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M. Kurian
University College London, United Kingdom

PLEN02_5 | What research and patients may teach us: Parkinson's disease—A history and perspective of learning

D. Berg
Department of Neurology, Universitätsklinikum Schleswig Holstein, Campus Kiel, Kiel, Germany

PLEN02_7 | Non-invasive neuromodulation in neurology

J. Rothwell
Department of Motor Neuroscience and Movement Disorders, University College London, London, United Kingdom

PLEN02_9 | Progress in gliomas: From histology to molecular biology and from surgery to precision therapies

R. Soffietti
Department of Neuro-Oncology, University and City of Health and Science Hospital (Torino), Torino, Italy

Tuesday, June 30 2026

Highlights and Breaking News

PLEN03_92 | Jes Olesen Award Lecture: Peripherin: A novel early diagnostic and prognostic plasmatic biomarker in Amyotrophic Lateral Sclerosis

A. Bombaci
Vita-Salute San Raffaele University, Milan, Italy

Background: Motor neuron diseases (MND) are heterogeneous and complex neurodegenerative disorders. Biomarkers could facilitate early diagnosis, prognosis determination, and patient stratification. Among the most studied biomarkers are neurofilaments, with peripherin (PRPH), a specific type predominantly expressed in peripheral nervous system, gaining attention. To date, no studies have evaluated PRPH in human plasma.

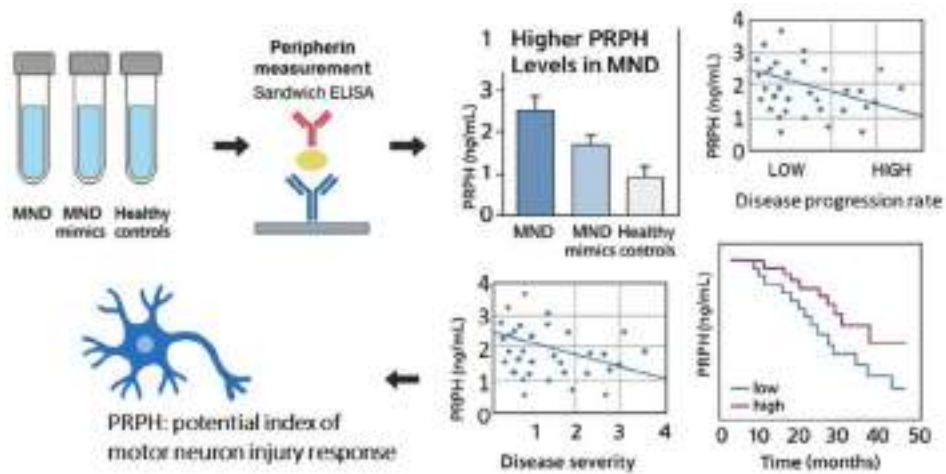
Methods: sandwich-ELISA was used to quantify plasma peripherin from 120 MND (100 ALS, 4 PMA, 15 PLS), 73 MND-mimics and 38 healthy-controls (HCs). Plasma was collected at diagnosis or some months earlier. 41 ALS were evaluated longitudinally. ALSFRS_r, MRC, spirometry, genetic tests, disease progression rate (PR), blood examinations, and neuropsychological tests were performed. Statistical analyses included Kruskal-Wallis, Mann-Whitney, Cox-regression, and Kaplan-Meier curves.

Results: Plasma PRPH levels differed significantly among groups ($p < 0.0001$), showing higher values in MND participants than MND mimics and HCs. Moreover, PRPH levels were elevated in PLS compared with HSP patients ($p = 0.0001$). Differences persisted after adjusting for age and sex. ROC curve demonstrated that PRPH discriminated MND from MND mimics (AUC = 0.85). Elevated PRPH correlated positively with ALSFRS_r and lower motor neuron index, while inversely with disease progression rate. Higher PRPH levels at the beginning of the disease were associated with longer survival.

Discussion: Plasma PRPH is raised in MND, particularly ALS, from the earliest stages, distinguishing MND from mimics and

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correlating with clinical parameters and survival. This suggests PRPH may reflect an endogenous response of lower motor neuron to injury. Further multicentre studies are required to refine the diagnostic and prognostic utility of PRPH in MND.

Disclosure: Nothing to disclose.

SUPPLEMENT ABSTRACTS

Symposia

Saturday, June 27 2026

EAN/MDS-ES: Tremor syndromes: From pathophysiology to emerging treatment approaches**SYMP01_1 | Pathophysiology of tremor syndromes**

R. Helmich
*Neurology Department, Radboud University Medical Centre,
Donders Institute, Nijmegen, the Netherlands*

Tremor syndromes are defined by clinical features, including tremor characteristics and accompanying symptoms. The pathophysiology of clinical tremor syndromes involves the mechanisms that give rise to the tremor. Knowledge about the pathophysiology of tremor is relevant, because it helps to understand clinical heterogeneity (e.g., in the response to treatment) and because it aids the development of new, mechanism-guided therapies. Here, I will discuss the pathophysiology of the most common tremor syndromes, including Parkinson's tremor, essential tremor, and dystonic tremor. To this end, I will discuss evidence from studies that have used neuroimaging and neurophysiology outcomes, with or without interventions (e.g., pharmacological, stereotactic surgery). I will discuss how rhythmic activity in the cerebello-thalamo-cortical circuit plays a key role in almost all tremor syndromes. These activities are governed by differing cerebral mechanisms, both within the cerebello-thalamo-cortical circuit (e.g., cerebellar atrophy), and outside the cerebello-thalamo-cortical circuit (e.g., triggering circuits like the basal ganglia and counter-acting circuits like recently discovered "anti-tremor networks").

Disclosure: Rick Helmich is consultant for Neurocrine Biosciences.

SYMP01_2 | Diagnostic criteria and clinical approach to tremor syndromes

M. Stamelou
Hygeia Hospital, Athens, Greece

SYMP01_3 | Pharmacological and other, non-surgical, treatments of tremor syndromes

R. Erro
University of Salerno, Salerno, Italy

SYMP01_4 | Surgical options for tremor syndromes: Clinical indications, efficacy and safety

Antonella Macerollo
*The Walton Centre NHS Foundation Trust, Neurology,
Liverpool, UK*

EAN/ESO: Future of medium size vessel occlusion stroke – Is there still a role for endovascular thrombectomy?**SYMP02_1 | The role of blood-based biomarkers in hyperacute risk stratification**

M. Katan
University Hospital of Zurich, Neurology, Zurich, Switzerland

SYMP02_2 | Use of imaging biomarkers for selection of patients who may still benefit from EVT?

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Jerusalem, Israel*

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**SYMP02_3 | IA lytics use in patients with MeVO:
The way forward**

J. Kaesmacher
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The treatment of Medium Vessel Occlusions (MeVO) represents on of the next frontiers in acute ischemic stroke intervention, moving beyond the established boundaries of large vessel mechanical thrombectomy (MT). While the 2015 “thrombectomy revolution” solidified MT as the gold standard for proximal occlusions, the management of more distal M2/M3, A2/A3, and P2/P3 segments remains a complex clinical challenge. This presentation evaluates the transition of intra-arterial (IA) thrombolysis from a historical “rescue” therapy to a potential primary tool for achieving micro-vascular patency in primary or secondary MeVos. From initial IA Lytics trial in the pre-EVT era the field is now reequipped with evidence for treatment of secondary MeVOs after LVO MT. Past and current evidence is reviewed and the role of patient selection, including intraoperative imaging is reviewed. The session concludes with a practical framework for clinical application, including dosing, timing and exact mode of administration, thereby providing a comprehensive roadmap for optimizing reperfusion in the distal cerebral vasculature.

Disclosure: TECNIO Trial funded by Boehringer Ingelheim. Research contracts with Siemens and Cercare for intra-operative Imaging.

**SYMP02_4 | New EVT devices and techniques for
patients with MeVO**

M. Psychogios
*University Hospital Basel, Diagnostic and Interventional
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**EAN/ILAE Europe: Seizures, cognition and
dementia: Hyper-exciting times ahead**

**SYMP03_1 | Size of the problem: Epidemiology of
seizures in dementia**

J. Zelano
*Department of Neuroscience and Physiology, University of
Gothenburg, Gothenburg, Sweden*

**SYMP03_2 | Time flies faster: Seizures and the aging
brain**

K. Kobow
*Department of Neuropathology, Universitätsklinikum Erlangen,
Erlangen, Germany*

**SYMP03_3 | Do antiseizure medications treat
dementia: Lowering hyper-excitability, improving
cognition**

M. Galovic
*Department of Neurology, University Hospital Zurich, Zurich,
Switzerland*

**SYMP03_4 | Putting it all together: Best practice
management of seizures in dementia**

A. Sen
Department of Neurology, John Radcliffe Hospital, Oxford, UK

**EAN/ESRS: Can we reduce the sleep disorders
burden with diagnostic and therapeutic
innovations?**

**SYMP04_1 | Epidemiology and economic burden of
sleep disorders in Europe**

C. Bassetti
*Department of Neurology, University of Bern, Inselspital, Bern,
Switzerland*

**SYMP04_2 | Sleep revolution: Digitalisation of sleep
diagnostics**

E. Arnardottir
*Department of Engineering, Department of Computer Science,
Reykjavik University, Reykjavik, Iceland*

**SYMP04_3 | New treatments for sleep disorders: From
insomnia to hypersomnia**

R. Fronczek
*Department of Neurology, Leiden, Leiden University Medical
Center, The Netherlands*

**SYMP04_4 | When the world doesn't sleep: How
modern environments shape our heart and brain health**

J. Reis
Hopital Universitaire de Haute-pierre CHU Strasbourg, France

The studies of human biorhythms have become an important and emerging discipline in the sixties along with an interest for the sleep quality and duration. The media assumption of its

decrease is debated. Sleep epidemiological studies are mostly based on self-reported sleep duration via questionnaires (self-reported wearable devices). They were first conducted in WEIRD (Western, Educated, Industrialized, Rich and Democratic) countries before considering nowadays non-WEIRD countries and even gather-hunter populations. Finally, the first question concerns sleep quality: which are the optimal sleep duration and pattern? The perceived sleep efficacy as well as duration/pattern are both individual and cultural. We will question genetic, age and sex variability, the effects of societal, cultural as well the geographical latitude on the sleep duration's characteristics. The description of the Insufficient Sleep Syndrome, characterized by a quantitative deficit of sleep seems triggered by specific lifestyles. Initially observed among New-York Yuppies, it seems nowadays related to some particular behaviors (e.g., work addiction). As well as imposed shift-work, these conditions lead considering some recent societal constraints, our "modern environments". These Zeitgebers include the effects of urbanization and technological revolution as well as lifestyle; they override the universal Zeitgeber which are light and the Day-Night alternance. Understanding the health consequences of poor sleep quality, needs a global view of biorhythms as sleep may be considered as the perfect clinical marker of bio-rhythmicity, or our Body Time. Amidst health risks, brain health, and cardio-vascular and metabolic problems are prominent.

Disclosure: Nothing to disclose.

EAN/WMS: Molecular therapies in neuromuscular disorders: From genes to clinical impact

SYMP05_1 | Spinal muscular atrophies: From molecular mechanisms to therapeutic breakthroughs

S. Corti

Foundation IRCCS Ca' Granda Ospedale Maggiore Policlinico, University of Milan, Milan, Italy

Spinal muscular atrophy (SMA) is a severe neurodegenerative disorder caused by mutations in the SMN1 gene, resulting in progressive motor neuron loss and muscle atrophy. Over the past decade, SMA has transitioned from being an untreatable condition to a model of successful molecular therapy, fundamentally transforming patient outcomes and quality of life.

This lecture will provide an integrated overview of the molecular pathogenesis of SMA and the revolutionary therapeutic strategies that have emerged. Gene replacement therapy using onasemnogene abeparvovec, an AAV9-mediated approach delivering functional SMN1 gene copies, has demonstrated remarkable efficacy, particularly when administered in pre-symptomatic or early symptomatic stages. The lecture will discuss clinical trial data, long-term outcomes, and practical considerations for implementation.

Splicing modulators, including nusinersen and risdiplam, represent groundbreaking RNA-targeted approaches that enhance SMN2 exon 7 inclusion to increase functional SMN protein production. Comparative analysis of antisense oligonucleotide and small-molecule strategies will be presented, highlighting their distinct pharmacological profiles and clinical applications across different SMA phenotypes.

The critical importance of therapy timing and personalization will be emphasized, drawing from newborn screening programs and natural history studies that underscore the window of therapeutic opportunity. Patient selection criteria, biomarkers for treatment response monitoring, and individualized treatment algorithms based on SMN2 copy number and disease severity will be discussed.

Finally, new and emerging therapies, including next-generation gene therapies, combination treatment strategies, and neuroprotective approaches targeting muscle and neuronal pathways beyond SMN restoration, will be explored as the future frontier of SMA management.

Disclosure: Participation in SAB of Novartis, Roche, Biogen.

SYMP05_2 | Advances in molecular therapies for muscular dystrophies: Targeting the underlying defects

G. Comi

Foundation IRCCS Ca' Granda Ospedale Maggiore Policlinico, University of Milan, Milan, Italy

Muscular dystrophies represent a heterogeneous group of inherited disorders characterized by progressive muscle weakness and degeneration. Recent advances in molecular medicine have revolutionized the therapeutic landscape, shifting from purely symptomatic management toward disease-modifying interventions that address the underlying genetic defects.

This lecture will provide a comprehensive overview of cutting-edge molecular therapies currently in development or clinical use. Gene and RNA-based therapies, including exon-skipping antisense oligonucleotides, stop-codon read-through agents, and adeno-associated virus (AAV)-mediated gene replacement strategies, have demonstrated promising results in conditions such as Duchenne muscular dystrophy and limb-girdle muscular dystrophies. The lecture will discuss the mechanistic principles, clinical trial outcomes, and real-world evidence supporting these approaches.

Targeted molecular approaches will be explored, focusing on precision medicine strategies tailored to specific genetic mutations and disease subtypes. Particular attention will be given to the development of personalized therapeutic protocols and the importance of genetic diagnosis in treatment selection.

Finally, the lecture will address current challenges and future directions, including immunological responses to viral vectors, limited tissue biodistribution, long-term safety concerns, and the need for biomarkers to monitor therapeutic efficacy. Emerging technologies, such as CRISPR-based gene editing and next-generation delivery systems, will be discussed as potential solutions to overcome existing limitations.

Disclosure: Participations to SAB of Roche, Novartis, Biogen, Sarepta.

SYMP05_3 | Emerging therapies in ALS: The use of Innovative Human In Vitro Systems

J. Pasterkamp

University Medical Center Utrecht, Utrecht, The Netherlands

SYMP05_4 | Human pluripotent stem cell-derived models for the evaluation of therapeutics in neuromuscular diseases

C. Martinat
INSERM UMR861 I-Stem, Corbeil-Essonnes, France

Monday, June 29 2026

Innovations in Neurology: From Brain Machine Interface to AI

SYMP06_1 | Adaptive deep brain stimulation for movement disorders

A. Kühn
Charité Universitätsmedizin Berlin, Neurology, Berlin, Germany

SYMP06_2 | Advances in non-invasive brain stimulation

F. Hummel
Swiss Federal Institute of Technology Lausanne (EPFL), Neuro-X Institute, Geneva, Switzerland

SYMP06_3 | Applying AI in clinical neurology

J. Teo
Kings College Hospital, London, UK

SYMP06_4 | Interacting with robots

A. Rosenthal-von der Pütten
RWTH Aachen University, Chair Individual and Technology, Aachen, Germany

EAN/MDS-ES: Dementia with Lewy bodies: Current and future perspectives

SYMP07_1 | Diagnosis of DLB within the framework of neuronal Lewy body diseases

E. Lemstra
Amsterdam University Medical Center, Neurology, Amsterdam, The Netherlands

SYMP07_2 | Role of imaging in the assessment of neurodegeneration, longitudinal monitoring and target engagement

D. Ferreira
Karolinska Institutet, Blickagången 16 (byggnad NEO), Huddinge, Sweden

SYMP07_3 | Neurophysiological biomarkers for disease stratification and longitudinal assessment

L. Bonanni
Università degli Studi "G. d'Annunzio" Chieti – Pescara, Chieti, Italy

SYMP07_4 | Treatment pathways, current and future perspectives

D. Arslan
King's College London, London, UK

Which are the best medicines to treat migraine patients: New international preventive and acute therapies guidelines

SYMP08_1 | Evidence based guidelines for preventive therapies

P. Pozo-Rosich
Vall d'Hebron University Hospital, Unidad de Cefalea – Hospital de la Dona (2ª planta)Ps., Barcelona, Spain

SYMP08_2 | Evidence based guidelines for acute therapies

V. Romanenko
International Association for the Study of Pain, Kyiv, Ukraine

SYMP08_3 | What to do in clinical practice: Expert consensus on acute and preventive therapies for migraine

S. Sacco
University of L'Aquila, Italy

SYMP08_4 | Central mechanism of CGRP target therapies: Will they modify migraine outcome?

M. de Tommaso
Policlinico bari, Neurology, Bari, Italy

EAN/ECTRIMS: Integrating the new revision of the McDonald criteria into clinical practice

SYMP09_1 | Overview of the 2024 revision of the McDonald criteria

M. Tintoré
Centre d'Esclerosi Múltiple de Catalunya (Cemcat), Barcelona, Spain

SYMP09_2 | Assessment of optic nerve involvement

C. Granziera
University of Basel, Basel, Switzerland

SYMP09_3 | MRI markers: Central vein sign and paramagnetic rim lesions

P. Preziosa
IRCCS San Raffaele Scientific Institute, Milan, Italy

SYMP09_4 | Application and challenges in the clinical scenario of revised McDonald criteria

B. Stankoff
Hopital Saint Antoine, Sorbonne University, Paris, France

EAN/EANO: The changing landscape of neuro-oncology: New frontiers of treatments

SYMP10_1 | Anatomical-physiological basis of the blood-brain barrier in brain tumours

A. Di Stefano
Spedali Riuniti di Livorno, Livorno, Italy

The blood-brain barrier (BBB) represents one of the most distinctive anatomical and physiological features of the central nervous system and plays a pivotal role in the pathogenesis, diagnosis, and treatment of brain tumours. While the BBB is essential for maintaining neural homeostasis by regulating molecular trafficking between the systemic circulation and the brain parenchyma, it also constitutes the principal obstacle to

the effective delivery of therapeutic agents to intracranial neoplasms. Indeed, many potentially active anticancer drugs fail to demonstrate clinical efficacy not because of inadequate target engagement, but because insufficient concentrations reach the tumour tissue. Understanding the anatomy and physiology of the BBB is therefore fundamental to modern neuro-oncology. This lecture will review the structural organization of the BBB, focusing on the neurovascular unit, a dynamic multicellular system composed of endothelial cells, pericytes, astrocytic end-feet, neurons, microglia, and extracellular matrix components. Particular attention will be given to the specialized properties of brain endothelial cells, including tight junction proteins such as claudin-5, occludin, and zonula occludens-1, the suppression of transcytosis pathways, and the expression of active efflux transporters including P-glycoprotein and breast cancer resistance protein. Together, these mechanisms tightly regulate molecular exchange, preserve ionic and metabolic homeostasis, and limit immune cell trafficking within the central nervous system.

The presentation will then examine how these physiological barriers are altered in brain tumours. In gliomas, particularly glioblastoma, the normal BBB undergoes profound but heterogeneous remodeling, giving rise to the so-called blood-tumour barrier (BTB). Contrary to the common perception of a uniformly disrupted barrier, permeability changes within gliomas are highly variable across different tumour regions. Tumour angiogenesis, driven in part by vascular endothelial growth factor (VEGF), results in abnormal endothelial differentiation, pericyte dysfunction, and increased vascular permeability. Nevertheless, infiltrative tumour cells frequently invade surrounding brain tissue where the BBB remains structurally and functionally intact. This preserved barrier at the tumour margins is a major contributor to treatment failure and disease recurrence.

The BBB in brain metastases exhibits distinct biological features, often showing greater permeability than primary gliomas, although substantial inter- and intra-tumour heterogeneity persists. These alterations have important clinical consequences, contributing to vasogenic edema, increased intracranial pressure, and the radiological appearance of contrast enhancement on magnetic resonance imaging (MRI).

The lecture will also discuss current approaches for assessing BBB integrity in clinical practice. Gadolinium-enhanced MRI remains the most widely used surrogate marker of BBB disruption; however, contrast enhancement does not necessarily reflect the full extent of tumour infiltration. Advanced imaging techniques, including dynamic contrast-enhanced and dynamic susceptibility contrast MRI, provide additional insights into vascular permeability and tumour physiology, although their ability to predict drug penetration remains limited.

Finally, the therapeutic implications of BBB and BTB biology will be explored. The differential permeability of the BBB explains why certain agents, such as temozolomide, achieve clinically meaningful concentrations within the brain, whereas many targeted therapies do not. Emerging strategies aimed at overcoming or bypassing the BBB include convection-enhanced delivery, focused ultrasound combined with microbubbles, nanoparticle-based drug delivery systems, and local cellular immunotherapies such as CAR-T approaches. Increasingly, the heterogeneity of the BTB is being recognized not only as an obstacle but also as a potential therapeutic opportunity.

A deeper understanding of BBB anatomy and physiology is essential for the development of next-generation neuro-oncological

therapies. Future advances may enable patient-specific characterization of BBB status, allowing treatment selection to be guided not only by tumour biology but also by the dynamic properties of the tumour microenvironment and its vascular barriers.
Disclosure: Nothing to disclose.

SYMP10_2 | Disrupting BBB to enhance treatment delivery

A. Idbaih
Sorbonne Université, AP-HP/La Pitié Salpêtrière, ICM

The blood–brain barrier (BBB) represents a major obstacle to drug delivery in treatment of central nervous system (CNS) disorders including brain cancer such as glioblastoma. The BBB is a highly selective biophysical and biochemical barrier that regulates molecular transport from the blood flow stream to the brain parenchyma.

In the setting of brain tumors, several strategies have been developed to overcome, bypass, and/or disrupt the BBB, to improve the bioavailability and the efficacy of therapeutic agents against tumor cells.

First, strategies aim at increasing drug delivery using systemic or local routes. High-dose intravenous chemotherapy has significantly improved outcomes in selected brain malignancies. Intra-arterial delivery approaches have also been explored. In addition, locally implanted biodegradable agents within the surgical cavity have demonstrated antitumor efficacy in recurrent glioblastoma.

Second, approaches focus on increasing drug penetration through drug or BBB modulation. Drug modulation strategies include several modalities, among which nanoparticle encapsulation is one of the most advanced, with encouraging signals of efficacy. Finally, direct BBB modulation represents a promising approach, including inhibition of efflux pumps and transient disruption of tight junctions. Ultrasound-mediated BBB opening is currently being evaluated in a phase III clinical trial in brain tumors.

Overall, the BBB remains a major limiting factor in the treatment of CNS diseases, including brain tumors. Strategies aimed at overcoming, bypassing, or disrupting the BBB have the potential to enhance local therapeutic efficacy while reducing systemic toxicity.

Disclosure: Travel funding: Carthera, Leo Pharma, Novocure; research grants: Transgene, Sanofi, Servier, Amgen and Nutritheragene; consulting: Novocure, Novartis, Polytone Laser, Leo Pharma, and Boehringer Ingelheim.

SYMP10_3 | Isocitrate dehydrogenase inhibitors for glioma: Efficacy and implications for service delivery

C. McBain
The Christie NHS FT, Department of Clinical Oncology, Manchester, UK

SYMP10_4 | Quality of life and neurocognitive functions in low grade glioma patients

F. Boele
Patient-Centred Outcomes Research Group, University of Leeds, Leeds, UK

SUPPLEMENT ABSTRACTS

Focused Workshop

Saturday, June 27 2026

AI in hospital neurology

FW01_1 | AI in acute stroke

S. Wegener
University Hospital Zurich and University of Zurich, Switzerland

FW01_2 | AI in disorders of consciousness

A. Estraneo
IRCCS, Don Gnocchi Foundation, Florence, Italy

FW01_3 | AI in identification & diagnosis of multiple sclerosis

C. Oreja-Guevara
Hospital Clinico San Carlos, Madrid, Spain

EAN/EPNS: How to deal with cerebrovascular diseases in children and adolescents

FW02_1 | Stroke across pediatric age

I. Kopyta
Department of Child Neurology, Medical University of Silesia, Katowice, Poland

Stroke is a rare condition in the pediatric population, but it can affect children of all ages, most commonly -newborns and young infants. Neonatal stroke is excluded from the definition of pediatric arterial ischemic stroke due to its specific risk factors

and prognosis. The diagnosis of PAIS (Pediatric arterial ischemic stroke) can be made in the case of sudden neurological symptoms, at age between 29 days and 18 years, with vascular pathology detected by neuroimaging, correspond in its location to clinical symptoms. The incidence of PAIS is estimated at 3–13/100,000 per year; the risk factors are arteriopathies (acquired and congenital, with a progressive, stationary or remitting course), heart diseases and thrombophilia. Confirmation of PAIS is based on the results of a neuroimaging and the method of choice is magnetic resonance imaging with MRA of the cerebral and neck vessels. If the probable delay in MRI performance exceeds 25 min, then CT imaging should be performed. A child diagnosed with PAIS is currently a potential candidate for thrombolytic therapy and thrombectomy. Antiplatelet therapy is an alternative in the case of exceeding the therapeutic window and is a method used in secondary prevention. Most children with PAIS will present the permanent neurological deficits; approximately 3% of children with PAIS die during the acute phase of the disease. A key issue in improving prognosis and increasing access to modern therapeutic methods for children with PAIS is improving awareness on PAIS.

Disclosure: Nothing to disclose.

FW02_2 | Arteriopathies in pediatric stroke

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Pediatric arterial ischemic stroke (PAIS) has been associated with a wide range of underlying conditions. In developed countries, the most common etiologies include cerebral arteriopathies, congenital or acquired heart diseases, and severe systemic infections. Cerebral arteriopathy is a major contributor to PAIS, accounting for nearly half of all cases and significantly increasing the risk of both initial and recurrent stroke. These arteriopathies may be congenital, reflecting abnormal vascular development, or acquired as a result of disruption of vascular homeostasis. The term encompasses a broad spectrum of vascular imaging abnormalities, including cervicocervical arterial dissection, moyamoya, vasculitis, post-varicella

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angiopathy, and idiopathic focal cerebral arteriopathy. Their classification has undergone several revisions over the past two decades. Among intracranial arteriopathies, focal cerebral arteriopathy (FCA) is the most frequently identified subtype, accounting for up to 25% of cases. FCA is a dynamic entity that may exhibit progression or relapse and is characterized by inflammatory arterial stenosis, often developing secondary to infection, particularly viral infections such as upper respiratory tract infections or varicella. The vascular involvement typically affects the carotid T, especially the proximal segment of the middle cerebral artery. Diagnosis relies on clinical presentation and neuroimaging. Brain MRI is the first-line imaging modality in pediatric stroke, enabling early differentiation from stroke mimics, while in cases of suspected FCA, vessel wall imaging is particularly useful. Therapeutic approaches remain heterogeneous. Antithrombotic therapy is most commonly used for secondary stroke prevention, while corticosteroids have been proposed to limit disease progression, with several studies currently ongoing.

Disclosure: Nothing to disclose.

FW02_3 | Hyperacute treatment for pediatric stroke (IVT, EVT)

P. Sporns

Stadtspital Zürich, Diagnostic and Interventional Neuroradiology, Switzerland

Timely evaluation of a first-time seizure

FW03_1 | Timely evaluation of a first seizure - insights from the SWISS FIRST Study

B. Jin

Sleep-Wake-Epilepsy-Center, Department of Neurology, Inselspital, Bern University Hospital, University of Bern, Bern, Switzerland

Early diagnosis of a first unprovoked epileptic seizure and estimation of seizure recurrence risk is challenging and important to decide about initiating antiseizure medication. In SWISS FIRST, a prospective, observational, multicenter study across seven hospitals in Switzerland, we assessed adult patients suspected for first unprovoked epileptic seizure and their development over two years. We determined diagnostic accuracy of baseline EEG and MRI based on their diagnosis two years after first event. We developed a binary logistic regression model for seizure recurrences on the baseline EEG and MRI and clinical characteristics of the patients.

We recruited 636 patients with suspected first unprovoked epileptic seizure. 238 were finally diagnosed with epilepsy. Sensitivity for epileptiform activity in EEG was 31.5%, specificity was 97.3%. Potential epileptogenic lesions were present in 40.3% amongst epilepsy patients, but also abundantly in patients with only a first unprovoked seizure (31%) and non-epileptic seizure

		Epileptic seizure group N = 389			Non-epileptic seizure group N = 148		Information incomplete N = 99	
Category		Epilepsy	First unprovoked seizure	Acute symptomatic seizures	Epileptic seizure mimics	Unknown	Lost to follow-up	Blind
Number of patients		238	113	38	122*	28	45	24
Age	Mean and IQR	34.5 [18-75]	39.2 [17-75]	32.2 [15-71]	33.8 [15-73]	38.1 [18-78]	37.3 [15-78]	72.7 [60-82]
Sex	Male	128 (52.1%)	53 (45.9%)	26 (67.4%)	67 (54.9%)	6 (22.2%)	19 (42.2%)	52 (59.3%)
	Female	110 (47.9%)	60 (53.1%)	12 (32.6%)	55 (45.1%)	22 (77.8%)	26 (57.8%)	22 (40.7%)
Comorbidities	Neurological	98 (41.6%)	38 (33.6%)	11 (28.9%)	41 (33.6%)	18 (64.3%)	13 (28.9%)	23 (48.7%)
	Cardiac	59 (24.8%)	17 (15.0%)	11 (28.9%)	21 (17.2%)	0 (0.0%)	15 (33.3%)	28 (53.7%)
	Psychiatric	23 (9.7%)	10 (8.8%)	7 (18.4%)	11 (9.0%)	1 (3.6%)	1 (2.2%)	0 (0.0%)
	Febrile convulsions	4 (1.7%)	1 (0.9%)	0 (0%)	3 (2.5%)	0 (0%)	0 (0%)	0 (0%)
	Intellectual disability	4 (1.7%)	2 (1.8%)	0 (0%)	5 (4.1%)	0 (0%)	0 (0%)	2 (3.7%)
Family history of epilepsy		24 (10.1%)	11 (9.7%)	2 (5.3%)	6 (4.9%)	1 (3.6%)	0 (0%)	2 (3.7%)
Event semiology	In wake state	185 (77.8%)	80 (70.8%)	38 (97.4%)	115 (94.3%)	28 (100%)	32 (71.1%)	40 (74.1%)
	In sleep state	52 (22.2%)	24 (21.2%)	0 (0%)	7 (5.7%)	0 (0%)	12 (26.7%)	6 (11.4%)
	Unclassified	7 (2.9%)	9 (8.0%)	7 (18.4%)	17 (13.9%)	0 (0%)	9 (20.0%)	10 (18.8%)
	Impaired awareness	143 (60.1%)	77 (67.3%)	30 (76.3%)	54 (44.3%)	18 (64.3%)	25 (55.6%)	38 (70.4%)
	Motor symptoms	150 (63.0%)	85 (75.2%)	21 (55.3%)	23 (18.8%)	7 (25.0%)	24 (53.3%)	17 (31.1%)
EEG	Showing generalized focal	18 (7.5%)	14 (12.4%)	7 (18.4%)	10 (8.2%)	1 (3.6%)	1 (2.2%)	10 (18.5%)
		126 (52.9%)	50 (44.2%)	0 (0%)	28 (23.0%)	8 (30.8%)	19 (42.2%)	36 (59.3%)
	Epileptiform activity	15 (6.3%)	0 (0%)	1 (2.6%)	0 (0%)	1 (3.6%)	0 (0%)	0 (0%)
		68 (28.6%)	18 (16.0%)	6 (15.8%)	5 (4.1%)	0 (0%)	7 (15.6%)	11 (20.4%)
Structural MRI	Non-lesional	28 (11.7%)	16 (14.2%)	3 (7.9%)	22 (18.0%)	6 (21.4%)	8 (17.8%)	0 (0%)
	PAGE	186 (78.3%)	87 (76.8%)	23 (59.5%)	61 (50.0%)	13 (46.4%)	24 (53.3%)	33 (61.4%)
	PEI	185 (77.8%)	15 (13.3%)	14 (36.8%)	37 (30.3%)	6 (21.4%)	11 (24.4%)	32 (59.3%)
Transient peritonal MRI abnormalities (PMI)	Diffusion restriction	44 (18.5%)	10 (8.8%)	3 (7.9%)	11 (9.0%)	0 (0%)	3 (6.7%)	28 (51.9%)
Baseline diagnosis	Epileptic seizure	228 (95.8%)	106 (93.8%)	34 (89.5%)	22 (18.0%)	7 (25.0%)	28 (62.2%)	40 (74.1%)
	Non-epileptic seizure	7 (2.9%)	6 (5.3%)	1 (2.6%)	7 (5.7%)	7 (25.0%)	12 (26.7%)	7 (13.0%)
	Unknown	3 (1.3%)	1 (0.9%)	3 (7.9%)	3 (2.5%)	14 (50.0%)	4 (8.9%)	4 (7.4%)
Treated with ASM after baseline assessment		184 (77.3%)	78 (68.9%)	28 (73.7%)	17 (13.9%)	3 (10.7%)	28 (62.2%)	30 (55.6%)

FIGURE 1 Clinical characteristics, baseline EEG and MRI findings.

	Epilepsy	First unprovoked seizure	Acute symptomatic seizure	Epileptic seizure relapse	Unknown	Lost to follow-up	Dead
	238	113	38	122	25	45	18
Structural lesions	59 (18.8%)	30 (17.7%)	4 (10.5%)	39 (18.8%)	8 (23.1%)	7 (13.6%)	15 (27.8%)
Brain tumours	23 (7.1%)	6 (5.3%)	0 (0%)	6 (6.3%)	0 (0%)	3 (6.4%)	11 (20.8%)
Vascular lesions	60 (18.2%)	20 (11.4%)	1 (2.6%)	3 (1.4%)	0 (0%)	1 (5.2%)	1 (1.9%)
Malformation of cortical development	2 (0.6%)	1 (0.9%)	1 (2.6%)	1 (0.8%)	0 (0%)	0 (0%)	0 (0%)
Medicological signal abnormalities	3 (2.3%)	1 (0.9%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Extracranial findings	1 (1.3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (1.9%)
Acute neurological disorders	3 (1.2%)	4 (3.5%)	3 (7.9%)	3 (4.2%)	0 (0%)	2 (4.4%)	4 (7.4%)
Structural lesions in genetic generalized epilepsy	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Inborn errors and metabolic disorders	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Brain pathologies	6 (2.3%)	0 (0%)	0 (0%)	1 (2.3%)	0 (0%)	1 (2.2%)	0 (0%)
Not otherwise specified	1 (2.3%)	1 (0.9%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)

FIGURE 2 Specification of the potentially structural epileptogenic lesions.

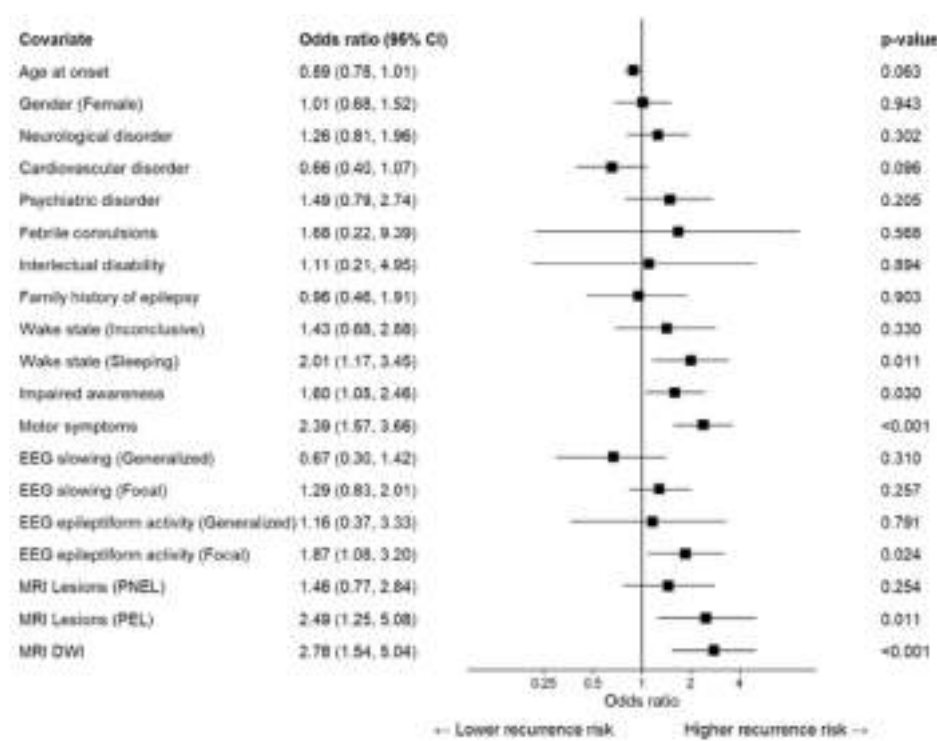


FIGURE 3 Multivariable logistic regression model for seizure recurrences.

patients (28.4%). Patients with sleep-related seizures (OR 2.01, 95% CI 1.17-3.45; $p=0.01$), motor seizures (OR 2.39, 95% CI 1.57-3.66; $p<0.001$), seizures with impaired consciousness (OR 1.6, 95% CI 1.05 to 2.46; $p=0.03$), focal epileptiform activity (OR 1.87, 95% CI 1.08-3.2 $p=0.02$), presence of potential epileptic lesion (OR 2.49, 95% CI 1.25-5.08; $p=0.01$), and diffusion restriction (OR 2.78, 95% CI 1.54-5.04; $p<0.001$) were associated with increased seizure recurrence risk.

These findings of the SWISS FIRST study are expected to influence future patient management and therapy guidelines after first unprovoked epileptic seizure.

Disclosure: Nothing to disclose.

FW03_2 | Timely evaluation of a first-time seizure – the need for seizure units

M. Seeck
Hopitaux Universitaires de Genève-HUG, Department of Neurology, Geneva, Switzerland

FW03_3 | Timely evaluation of a first-time seizure – how to fill the gaps?

T. Marson
The Walton Centre NHS Foundation Trust, Unit of Neuroscience Research, Liverpool, United Kingdom

EAN/IFCN-EMEAC: New developments in diagnostics and non-invasive therapy in cognitive disorders

FW04_1 | TMS-EMG for differential diagnosis of dementias

A. Benussi
University of Trieste, Italy

FW04_2 | Hyperexcitability in the pathogenesis of Alzheimer's disease and transcranial electrical stimulation for the treatment

A. Kamondi
Semmelweis University, Budapest, Hungary

FW04_3 | Repetitive TMS of the precuneus for the treatment in AD

A. Antal
Department of Neurology, UMG, Göttingen, Germany

Non-invasive Brain Stimulation (NiBS) techniques such as repetitive Transcranial Magnetic Stimulation (rTMS) and transcranial Electrical Stimulation (tES), including Direct Current and Alternating Current Stimulation (tDCS, tACS), allow to modulate neuronal excitability, oscillatory activity and to boost cortical functions, hereby possibly offering a therapeutic potential to slow down cognitive decline. Cognitive decline reaches medical attention for about 8–25% of individuals aged over 65, when suffering from Mild Cognitive Impairment (MCI). Around 15% of MCI patients develop dementia within two years, supporting a medical need for early diagnosis and treatment. Recent investigations propose rTMS of the precuneus as a potential therapeutic tool to improve network activity and cognitive functions in older individuals with cognitive impairments. However, several subject-specific factors, for example, anatomical and physiological differences, gender, brain state before and during stimulation, and also methodological factors, such as the type of the task applied in combination with the stimulation, can confound the effects of stimulation. This talk aims to provide an overview of the commonly utilized rTMS protocols, elucidating their individual merits and drawbacks.

Disclosure: Andrea Antal has nothing to disclose with regard to this talk. AA is a vice president of the European Society for Brain Stimulation, Member-at-Large at the EMEAC – IFCN Member of the safety or advisory boards: Boss-Stroke, Brady, Brainn, MemoSlap, GifteD, DisCoVer, PlatoScience Paid consultant at NeuroConn, Ilmenau, and she is a paid advisor at Electromedical Products International (Pulvinar), USA. Member of the advisory board at PlatoScience Paid Editor by Elsevier Supported by the DFG (AN 687/9-1), EU-Horizon 2020 (PAINLESS, No. 101057367), BMBF (NEURO VR-Plus 16SV9248), DAAD-PPP (57702891).

The neurological face of menopause: Rethinking migraine, stroke and Parkinson's in postmenopausal women

FW05_1 | Menopause and migraine

A. Szewczyk
Medical University of Lublin, Department of Neurology, Lublin, Poland

FW05_2 | Menopause and stroke

D. Aguiar de Sousa
Hospital de Santa Maria - Faculdade de Medicina de Lisboa, Lisbon, Portugal

FW05_3 | Menopause and Parkinson's disease

K. Rukavina
Berlin, Germany

The cerebellum beyond motor function: Uncovering cognitive-affective roles

FW06_1 | The 3 facets of the cerebellar syndrome: Emphasis on the cerebellar cognitive affective syndrome

M. Manto
Brussels, Belgium

FW06_2 | The role of the cerebellum in emotions

D. Timmann
Department of Neurology and Center for Translational Neuro- and Behavioral Sciences (C-TNBS), Essen University Hospital, University of Duisburg-Essen, Essen, Germany

The link between the cerebellum and emotional behavior will be discussed, beginning with an overview of known anatomical connections between the cerebellum and limbic structures, including the amygdala, PAG, hippocampus, and prefrontal cortex. Second, early animal studies demonstrating the involvement of the cerebellum in autonomic functions related to the expression of emotions will be summarized, including blood circulation and respiration. Third, functional brain imaging studies in healthy human participants examining emotional tasks will be reviewed, along with observations of altered emotional control in patients with cerebellar disorders, including degenerative ataxias and focal lesions. These findings will be complemented by structural and functional imaging evidence of disrupted cerebello–limbic interactions in patients with neuropsychiatric disorders, in particular anxiety disorders.

Finally, we will focus on fear, an important emotion for survival. The cerebellum contributes not only to innate affective and defensive behaviors but also to learned fear responses. Early rodent lesion studies showed that fear-conditioned bradycardia is markedly reduced after vermal lesions. To date, only a few studies have examined fear conditioning in patients with cerebellar disease, in which behavioral deficits appear to be milder. Most evidence for cerebellar involvement in human fear conditioning comes from neuroimaging studies in healthy participants. Threat-prediction-related activations extend beyond the vermis, with prominent involvement of the lateral cerebellum. There is initial evidence that the cerebellum contributes to prediction-error signals driving fear extinction via interactions with the VTA. These findings demonstrate that cerebellar control extends beyond autonomic functions and includes higher-order processes such as reward processing.

Disclosure: Dagmar Timmann received funding from the German Research Foundation (project number 316803389 – SFB1280), the European Union’s Horizon 2020 research and innovation program under the Marie Skłodowska-Curie grant agreement No 956414, and the Bernd Fink Foundation.

FW06_3 | A functional atlas of the cerebellum

C. Nettekoven

Department of Experimental Psychology, University of Oxford, Oxford, UK

Once considered primarily a motor structure, the cerebellum is now known to contribute to cognitive processes. Cerebellar damage can therefore produce cognitive disturbances in addition to motor deficits. However, limited knowledge of cerebellar functional organization continues to constrain the interpretation of cerebellar involvement in neurological disease.

This limitation largely reflects the absence of a comprehensive functional map of the human cerebellum. Although different cerebellar regions are engaged during motor, cognitive, and emotional tasks, these regions remain difficult to reliably delineate using existing atlases, particularly in the presence of substantial inter-individual variability.

In this talk, I will present the first comprehensive functional atlas of the human cerebellum, derived by integrating multiple large-scale functional MRI datasets spanning a wide range of motor, cognitive, and language tasks. Using a data-driven modelling approach, we identified distinct cerebellar regions with characteristic functional profiles and connectivity patterns. The atlas reveals a clear functional topography, with anterior cerebellar regions predominantly involved in motor control, posterior regions, particularly lobule VIIA (Crus I and II)—engaged in higher-order cognitive processes.

Importantly, the atlas enables precise mapping of cerebellar function at the individual level, addressing the limitations of group-based maps. I will discuss how this framework improves our ability to interpret cerebellar activation and lesion effects, and how it can inform the study of cognitive and affective symptoms in neurological disorders along with potentially improve surgical planning. By providing a unifying functional reference, this work supports a more integrated view of the cerebellum as a key contributor to both motor and non-motor brain function.

Disclosure: Nothing to disclosure.

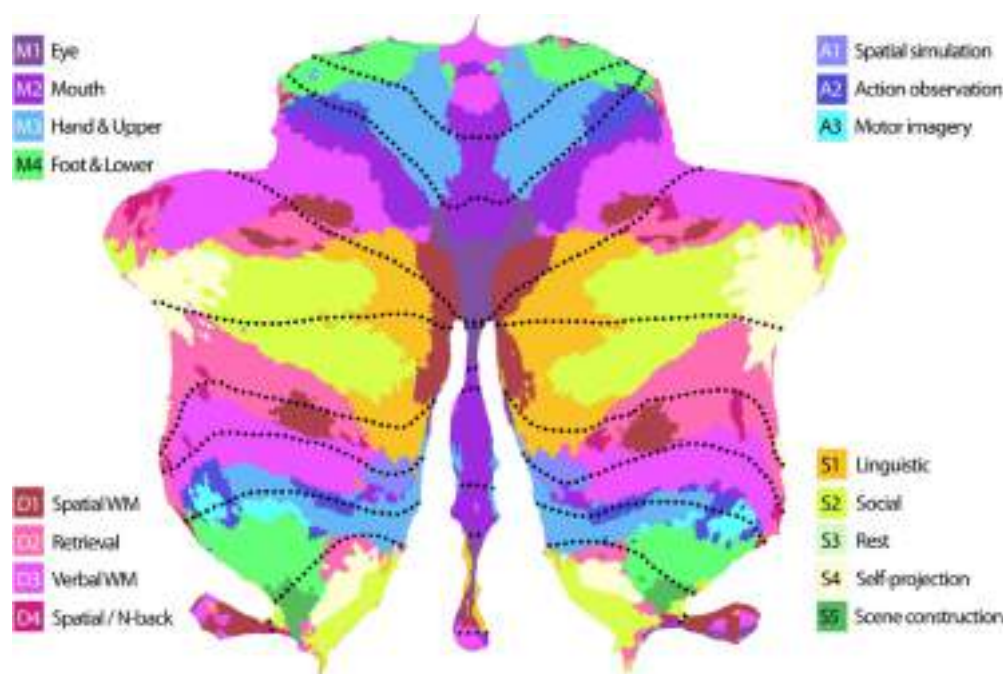


FIGURE 1 Cerebellar functional atlas. Symmetric atlas at medium level of granularity. The labels of each region consists of a capital for the domain (M, A, D, S), a number for region (1–4), and a lower- case letter for the subregion (a–d).

AI in outpatient neurology

FW07_1 | AI in epilepsy

S. Beniczky
*Epilepsy Hospital Filadelfia, Danish Epilepsy Centre -
Department of Clinical Neurophysiology, Dianalund, Denmark*

FW07_2 | AI for Parkinson's disease and rare parkinsonian conditions

C. Lambert
*National Hospital for Neurology and Neurosurgery/UCL,
Functional Imaging Laboratory, UCL Department of Imaging
Neuroscience, London, United Kingdom*

FW07_3 | AI in headache care

A. Stubberud
*NorHead Norwegian Centre for Headache Research, Department
of Neuromedicine and Movement Science, NTNU Norwegian
University of Science and Technology*

The use of artificial intelligence (AI) in headache care and research is rapidly expanding. AI has the potential to enhance clinical task such as diagnosis, treatment selection and prediction of disease trajectories. To date, several key areas have received the most attention in the headache literature. First, large language models have been used for automated extraction of patient-level data, as well as to support the training of healthcare providers and patient education. Second, a variety of AI models have been used to diagnose and classify headache disorders using both clinical and paraclinical data. There are also some indications that such models could improve diagnostic accuracy among non-neurologists. Third, AI models have shown promise in optimizing treatment selection and predicting treatment responses in primary headache disorders. Finally, predictive models that can forecast day-to-day headache occurrence, as well as longer-term disease trajectories, show encouraging results. While the current body of literature demonstrates promising developments, there remains a substantial need for external validation and clinical trials to enable the translation of these models into clinically applicable tools.

Disclosure: AS has received speaker honoraria from TEVA. He is co-founder and shareholder of Nordic Brain Tech AS and hold a patent related to the Cerebri biofeedback treatment for migraine. He is junior editor for Cephalalgia.

EAN/MDS-ES: Current role and future perspectives of neuromodulation in the treatment of Parkinson's disease and dementia with Lewy bodies

FW08_1 | Current role and future perspectives of deep brain stimulation in the treatment of PD

E. Moro
*CHU de Grenoble, Division of Neurology, Department of
Psychiatry and Neurological Rehabilitation, Grenoble, France*

FW08_2 | Focused ultrasound brain therapy is a new tool in the box

G. Deuschl
*Universitätsklinikum Schleswig Holstein Campus Kiel,
Department of Neurology, Kiel, Germany*

FW08_3 | Potential of non-invasive brain stimulation (NIBS) for the treatment of PD and DLB

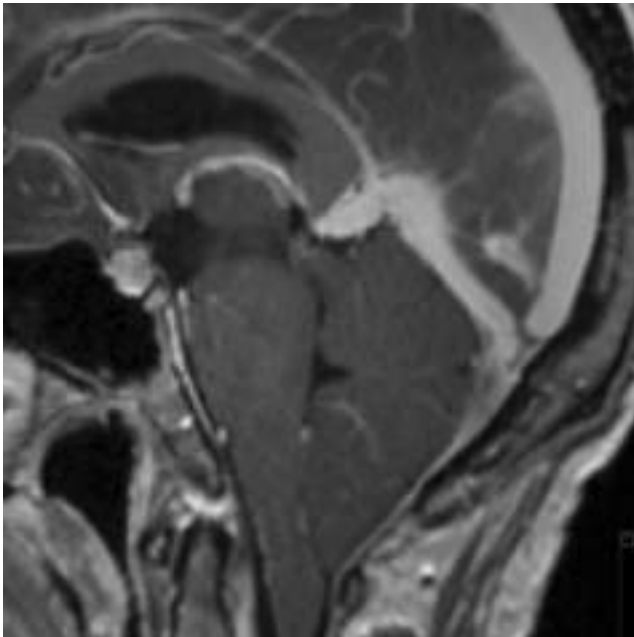
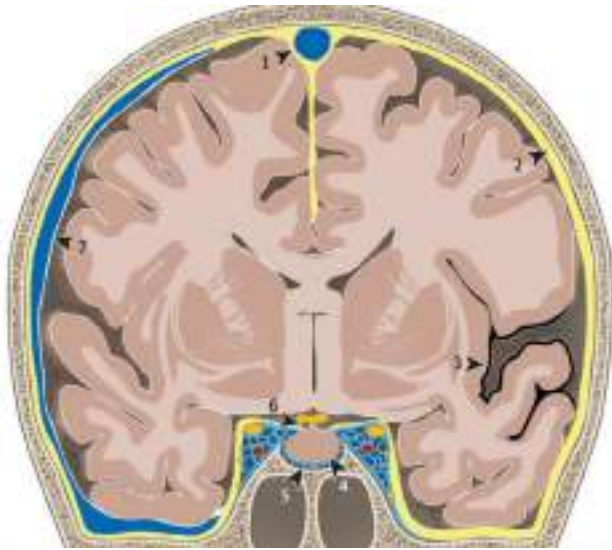
I. Rektorova
*Mararyk University, Brno Teaching Hospital Svata Anna, 1st
Department of Neurology, Brno, Czech Republic*

EAN/EANS: Spontaneous intracranial hypotension: The headache neglected by neurologist

FW09_1 | Clinical spectrum and imaging essentials in Spontaneous Intracranial Hypotension

T. Dobrocky
*Diagnostic and Interventional Neuroradiology, University of
Bern, Inselspital, Bern, Switzerland*

Spontaneous intracranial hypotension (SIH) is an important and frequently underrecognized cause of secondary headache with a broad and sometimes misleading clinical spectrum. Although orthostatic headache is the classic presentation, patients may develop atypical symptoms including non-orthostatic headache, cranial nerve palsies, cognitive and behavioral changes, movement disorders, or impaired consciousness, often leading to delayed diagnosis. Over the past years, substantial progress has been made in elucidating the underlying etiologies of SIH, particularly distinct types of spinal cerebrospinal fluid (CSF) leaks and CSF-venous fistulas. In parallel, standardized diagnostic algorithms integrating brain MRI and advanced spinal imaging have been established, transforming SIH into a well-defined and treatable condition. Neuroimaging is central to diagnosis and management, with characteristic brain MRI findings and targeted spinal techniques such as dynamic myelography enabling precise leak localization and guiding therapy. This session



reviews the clinical spectrum of SIH and highlights imaging essentials for accurate diagnosis and effective treatment.

Disclosure: No disclosures.

FW09_2 | Blood patching and percutaneous therapy for spontaneous intracranial hypotension

L. Carlton Jones
Guys and St Thomas Hospital, Department of Radiology,
Westminster Bridge Road, London, United Kingdom

FW09_3 | Surgical therapy, outcome and long-term complications in spontaneous intracranial hypotension

J. Beck
Universitätsklinikum Freiburg, Neurosurgery, Freiburg,
Germany

Technologies to identify and preserve residual brain functions in the ICU

FW10_1 | TMS-EEG to probe brain networks in unconsciousness

S. Casarotto
Dept. Biomedical and Clinical Sciences, University of Milan;
IRCCS Fondazione Don Carlo Gnocchi ONLUS, Milan

Introduction: Attributing consciousness to others always implies an inferential process, which is particularly challenging when evaluating patients who remain in prolonged unresponsive wakefulness syndrome (UWS) after severe brain injury. Behavioral evaluations alone may be misleading due to impaired sensory-motor functions. Neural responses to verbal commands can indicate intentional processing, but require intact language, executive functions, and sustained attention. Auditory paradigms with increasing complexity may reveal residual perceptual awareness without requiring active participation, though they still depend on attention and preserved sensory pathways. Measures of intrinsic brain activity, such as perturbational complexity, can inform about capacity for consciousness even when sensory-motor channels are compromised.

Methods: This study applied a multilayer neurophysiological assessment in 16 UWS patients hospitalized in an Intensive Rehabilitation Unit.

Results: Twelve patients recovered at least minimal behavioral signs of responsiveness. Before this occurred, intrinsic brain activity suggested a capacity for consciousness in all these patients, 25% of whom also showed neural responses to verbal commands and 8% perceptual activations. Clinical evoked potentials revealed significant sensory-motor impairments in most patients, likely contributing to negative findings of behavioral, task-based and perceptual evaluation.

Discussion: These results demonstrate feasibility and sensitivity of multilayer neurophysiological assessment in subacute rehabilitation. This approach is necessary to evaluate residual consciousness beyond behavioral responsiveness, progressively moving from task-based modulation of brain activity to perceptual awareness to intrinsic brain dynamics. The accurate detection of covert capacity for consciousness is crucial to guide treatment, since 75% of patients who remain in prolonged UWS may show late recovery.

Disclosure: I am currently advisor and share-holder of Intrinsic Powers, a spin-off of the University of Milan.

FW10_2 | Pupillometry and functional NIRS to identify covert consciousness in the ICU

D. Kondziella

Copenhagen University Hospital – Rigshospitalet, Department of Neurology, Copenhagen, Denmark

FW10_3 | Multimodal neuromonitoring to prevent secondary brain injury in the ICU

R. Helbok

Johannes Kepler University Linz, Department of Neurology, Linz, Austria

Monday, June 28 2026

EAN/MDS-ES: Repurposing GLP-1 receptor agonists: New frontiers for neuroprotection in neurological diseases

FW12_1 | Beyond glycemic control: Uncovering the neuroprotective role of GLP-1 receptor agonists

F. Ferrari

Department of Health Sciences, Università del Piemonte Orientale, Novara Italy

Introduction: Glucagon-like peptide-1 receptor agonists (GLP-1 RAs), developed for type 2 diabetes mellitus and obesity, are emerging as neuroprotective candidates in neurological disorders [1,2]. This lecture provides a mechanistic overview of their central nervous system (CNS) actions.

Methods: Literature review focused on GLP-1 RAs pharmacology, intracellular signaling and downstream pathways relevant to neuroprotection.

Results: GLP-1 receptors are broadly expressed in CNS and their activation engages cyclic AMP-dependent and phosphoinositide 3-kinase signaling, producing pleiotropic effects [3,4]: (i) attenuation of neuroinflammation via microglial polarization to anti-inflammatory phenotype, NFkB and NLRP3 inflammasome inhibition, suppression of TNF-alpha, IL-1-beta and IL-6, and prevention of neurotoxic reactive astrocyte conversion; (ii) anti-apoptotic activity through Bcl-2 upregulation and Bax/caspase-3 suppression; (iii) mitochondrial biogenesis via PGC-1-alpha and sirtuin-1, with reduction of oxidative stress; (iv) neurotrophic and synaptic support through brain-derived neurotrophic factor/TrkB signaling, AMPA receptor trafficking, postsynaptic density protein-95 upregulation, hippocampal neurogenesis and long-term potentiation; (v) proteostasis restoration via ULK1-dependent autophagy, reducing amyloid-beta, hyperphosphorylated tau and alfa-synuclein aggregates; (vi) preservation of blood-brain barrier and neurovascular unit. Additional body-to-brain signaling via vagal afferents contributes to indirect central neuroprotection.

Conclusion: GLP-1 RAs display multifaceted, converging neuroprotective mechanisms targeting the triad of neurodegeneration:

neuroinflammation, mitochondrial dysfunction and proteinopathy. Their established safety profile and pleiotropy make them strong candidates for repurposing in neurology.

References: [1] Ferrari et al. (2022). *Pharmacol Ther.* 239:108277; [2] Ferrari et al. (2020). *Pharmacol Res.* 160:105018; [3] Ros-Madrid et al. (2025). *Can J Physiol Pharmacol.* 103:369–377; [4] Roy et al. (2025). *Neurotherapeutics.* 22:e00712.

Disclosure: The author declares no conflicts of interest.

FW12_2 | GLP-1 receptor agonists in neurodegenerative diseases: What we have learned so far?

D. Athauda

University College London, Sobell Department of Motor Neuroscience and Movement Disorders, London, United Kingdom

FW12_3 | GLP-1 Receptor agonists for the treatment and prevention of stroke

E. Vercauteren

Karolinska Institute, Department of Clinical Science and Education, Stockholm, Sweden

EAN/EFIC: Revised guidelines for neuropathic pain: Diagnostic tests, pharmacotherapy and neuromodulation

FW13_1 | How to diagnose neuropathic pain. Evidence-based recommendations

A. Truini

University of Rome “La Sapienza”, Department of Human Neuroscience, Roma, Italy

FW13_2 | Revised Recommendations on the use of pharmacological therapies for neuropathic pain relief

X. Moisset

Université Clermont Auvergne, CHU Clermont-Ferrand, Inserm, Neuro-Dol, Clermont-Ferrand, France

Neuropathic Pain remains a major clinical challenge, with many patients experiencing insufficient pain relief and treatment-related adverse effects. The Neuropathic Pain Special Interest Group undertook a comprehensive update of its treatment recommendations, incorporating new randomized controlled trials and advances in evidence synthesis.

In this presentation, we will summarize the pharmacological findings of a large systematic review and meta-analysis including double-blind, randomized, placebo-controlled trials published up to February 2024. Across 284 pharmacological trials involving tens of thousands of participants, treatments were evaluated using standardized efficacy and safety outcomes, including responder rates, numbers needed to treat (NNT), and

numbers needed to harm (NNH), with evidence certainty assessed using the GRADE framework.

Participants will gain an updated overview of evidence-based pharmacological strategies for neuropathic pain. Strong recommendations support the use of tricyclic antidepressants, $\alpha 2\delta$ -ligands (gabapentinoids), and serotonin–noradrenaline reuptake inhibitors as first-line treatments. Second-line options include topical therapies such as high-concentration capsaicin patches, capsaicin cream, and lidocaine 5% plasters. Third-line strategies include botulinum toxin A and opioids in selected situations. The presentation will also highlight the magnitude of treatment effects, safety considerations, and how these therapies compare in terms of benefit–risk balance.

Beyond summarizing the evidence, the session will provide practical guidance on how to translate these updated recommendations into everyday clinical decision-making. Attendees will leave with a clear understanding of current pharmacological treatment hierarchies for neuropathic pain and the strengths and limitations of the available evidence.

Disclosure: Funding by NeuPSIG (IASP), ERANET-Neuron and Dr Jennie Gwynn Legacy Fund.

FW13_3 | New recommendations for the use of neuromodulation to relieve neuropathic pain

N. Attal
*Hôpital Ambroise Paré, Department of Pain Treatment,
Boulogne-Billancourt, France*

EAN/EFAS: Digital dysautonomia: The role of wearables and AI revolution

FW14_1 | Role of wearables and remote monitoring in assessing autonomic nervous system parameters

C. Falup Pecurariu
*Transilvania University Brasov, Department of Neurology,
Brasov, Romania*

FW14_2 | AI in the autonomic area

W. Struhal
*Universitätsklinik Tulln, Department of Neurology, Tulln,
Austria*

FW14_3 | Personalized assessment in autonomic dysfunction – role of digital biomarkers

A. Fanciulli
*Innsbruck Medical University, Department of Neurology,
Innsbruck, Austria*

Tuesday, June 30 2026

Understanding Alzheimer's disease: Disease models and diagnostic criteria

FW15_1 | The clinical context of treatment and prevention of Alzheimer's disease

K. Frederiksen
*Copenhagen University Hospital – Rigshospitalet, Memory
Disorders Research Unit, Copenhagen, Denmark*

FW15_2 | The case in favour of the Alzheimer Association criteria and Alzheimer's as a biological condition

C. Teunissen
Neurochemistry Lab, Amsterdam, The Netherlands

FW15_3 | International Working Group criteria and the clinical eco system they come with

G. Frisoni
Hôpitaux Universitaires de Genève, Geneva, Switzerland

Neurorehabilitation goes digital – management of cognitive and behavioral deficits

FW16_1 | Brain computer interface for post-coma patients

E. Molteni
*University College London, Institute of Health Informatics,
London, United Kingdom*

FW16_2 | Enhancing cognitive neurorehabilitation through virtual reality

A. Sokolov
*Centre Hospitalier Universitaire Vaudoise - CHUV BH07,
Department of Neurology, Lausanne, Switzerland*

FW16_3 | Digital solutions for diagnostics and treatment of psychiatric disorders in neurorehabilitation

K. Rauen

Institute of Emergency Medicine, University Hospital Zurich & Neuroscience Center Zurich, University of Zurich, Switzerland

Digital solutions are increasingly accepted as add-on tools for diagnostics and treatment in patients and their caregivers. Despite rapid developments and rising approvals only 39% of Digital Health Applications target psychiatric disorders and none of them addresses psychiatric burden in neurological patients during neurorehabilitation. Today, most digital solutions target depression (27%), anxiety (23%), insomnia (10%), cognition (7%), and various psychiatric conditions. It is well known that neurological patients after stroke or traumatic brain injury (TBI) particularly suffer from brain-injury induced depression, cognitive dysfunction and/or sleep disorders with raising evidence that brain-injury induced affective disorders differ in their pathophysiology and clinical presentation from Major Depressive Disorder (MDD). Consecutively, current digital solutions may not adequately address neurological patients' needs and often require caregivers' support. Therefore, it is time to close the gap on brain-injury specific Digital Health Applications for patients during and beyond neurorehabilitation aiming for better neuropsychiatric and quality of life outcomes. Here, we pinpoint (i) the communalities and differences of the brain-injury induced affective disorder and MDD, (ii) the specific personalized needs regarding neurobiological, functional and psychiatric characteristics to improve affective, cognitive and sleep burden during neurorehabilitation, and (iii) future digital solutions to enable effective, scalable, and patient-centered advances for better long-term outcomes as well as good quality of life for our neuropsychiatric patients during and beyond neurorehabilitation.

Disclosure: Nothing to disclose.

FW16_4 | Network-targeted brain stimulation for neurorehabilitation of cognition and behaviour

S. Filipović

Institute for Medical Research, University of Belgrade, Serbia

Non-invasive brain stimulation (NIBS) has been increasingly studied as a tool to promote neurorehabilitation for cognitive and behavioural impairments. A considerable body of evidence suggests that NIBS can have a beneficial effect, especially when coupled with other rehabilitation approaches. However, although encouraging, the results so far have not been unequivocal. One reason for the apparent lack of more substantial effects is the dominant single-target focus in most clinical studies. NIBS techniques (such as transcranial magnetic stimulation – TMS, or transcranial direct current stimulation – tDCS) were typically applied over either the injured area or the homologue (contralateral) healthy area, or, in the case of cognitive impairments, over the dorsolateral prefrontal cortex (DLPFC). However, it is increasingly recognised that this approach is not effective enough to reset physiological mechanisms into a sustainable and robust recovery mode. Injury- or disease-induced structural changes, beyond a loss of function directly associated with the lesion site(s), also disrupt large-scale distributed brain networks (such

as the default mode, salience, central executive, and sensorimotor). These disruptions can be a significant factor behind symptoms and impairments, but, even more importantly, they often prevent the recovery of functions, whether by restoration or by compensation. There is a growing understanding that individualised multi-locus targeting, guided by functional and structural connectivity determined by functional imaging techniques (e.g., fMRI, DTI, EEG), can provide a breakthrough towards meaningful and enduring clinical applications.

Disclosure: Nothing to disclose.

Vaccines technologies to protect brain health

FW17_1 | Vaccines to prevent neurological infectious disease

A. Chaudhuri

Queen's Hospital, Department of Neurology, Glasgow, United Kingdom

FW17_2 | Vaccine hesitancy and return of old neurological disease

E. Schmutzhard

Medizinische Universität Innsbruck, Universitätsklinik für Neurologie, Department of Neurology, Innsbruck, Austria

FW17_3 | Vaccines to prevent and treat non-infectious neurological disease

B. Willekens

Antwerp University Hospital and University of Antwerp, Department of Neurology, Edegem, Belgium

SUPPLEMENT ABSTRACTS

Special Sessions

Saturday, June 27 2026

EAN/Neurotorium: Precision medicine in brain diseases: Biomarkers to personalised care**SPS01_1 | From empirical to personalized prevention of Parkinson's disease**

E. Schäffer
Christian-Albrechts-Universität zu Kiel, Kiel, Germany

SPS01_2 | AI, biomarkers, and ethical leadership: Transforming neurological procedures towards cognitive health and care

I. Haraldsen
Oslo University Hospital, Oslo, Norway

SPS01_3 | Experimental medicine to predict individual differences in drug response: Lessons from dementia

J. Rowe
Addenbrooke's Hospital/University of Cambridge, Department of Neurology, University of Cambridge Herchel Smith Building Cambridge Biomedical Campus, Cambridge, United Kingdom

SPS01_4 | Precision medicine in epilepsy

D. Pal
King's College London, Basic & Clinical Neuroscience, London, United Kingdom

EAN/EPA: The biological and social determinants of neurological and mental health: Toward a unified brain health perspective**SPS02_1 | Integrating biological and environmental factors in mental health prevention**

C. Arango
Hospital Universitario La Paz, Chair, Department of Child and Adolescent Psychiatry, Madrid, Spain

SPS02_2 | Harmonising practices: Bridging biological aspects and social contexts across Europe

U. Volpe
Unit of Clinical Psychiatry, Department of Clinical Neurosciences/DIMSC, Università Politecnica delle Marche, Ancona, Italy

Achieving sustained improvements in brain and mental health requires approaches that integrate biological knowledge with social, cultural, and systemic determinants of health. Across Europe, advances in neuroscience, genetics, and clinical neurobiology coexist with persistent disparities in mental health outcomes, access to care, and implementation of evidence-based interventions. These gaps highlight the need for harmonised approaches that connect biological insights with social realities, healthcare systems, and public health strategies.

A unified brain health perspective can be applied across diverse European contexts by addressing interactions between biological vulnerability and social determinants across the lifespan. Biological risk factors—such as neurodevelopmental trajectories, genetic susceptibility, inflammation, and stress-related neurobiological changes—are shaped by environmental exposure, socioeconomic conditions, education, migration, discrimination, and healthcare access. Understanding these interactions

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is essential for effective prevention, early intervention, and service planning.

Also, cross-national differences will be considered in mental health policies, service organisation, and preventive strategies, and how these influence the translation of neuroscientific knowledge into routine practice. Particular attention is given to the challenge of aligning neurobiological advances with population-level and community-based interventions, and to the importance of interdisciplinary collaboration across neurology, psychiatry, public health, and social sciences.

Ultimately, advancing brain health in Europe requires preventive, equity-oriented strategies that integrate biological and social perspectives while remaining adaptable to local contexts.

Disclosure: Nothing to disclose.

SPS02_3 | A child neurology perspective on the origins of neurodevelopmental disorders and their impact in adolescence

L. Haataja

Department of Pediatric Neurology, Helsinki University Hospital, Helsinki, Finland

Children and adolescents with neurodevelopmental disabilities represent a growing population of individuals with varying functional everyday impairments. These health conditions cause impairments in motor, cognitive, language, behavioral and/or sensory functioning. In 2023, WHO and UNICEF reported that there are approximately 317 million children and adolescents affected by developmental disabilities globally. In the Global Burden of Disease study (2019) the prevalence of these conditions ranged from 7.5% among children under 5 years to 13.9% for those aged 15–19 years.

The origins of neurodevelopmental disorders are diverse and often multifactorial combinations of genetic, epigenetic and environmental factors affect critical periods of neurogenesis and connectivity. Furthermore, adverse perinatal incidents, like preterm birth and birth asphyxia, play a major role in adverse neurodevelopmental outcomes.

Neurodevelopmental disabilities are associated with poor academic outcomes, bullying, loneliness, discrimination, unemployment and somatic health problems like obesity, diabetes, cardiac and respiratory diseases, and mental health conditions. The vicious circle may be accelerated by the fact that the diagnoses of neurodevelopmental disorders are often based on various combinations of symptoms, blurring the line between so-called typical and atypical development, and, at present, the neuro-anatomic correlation is to a large degree not detected by the currently available imaging tools in clinics.

There is an immense global need for increasing evidence-based awareness of neurodevelopmental disorders and developing community-based strategies for providing opportunities to support individual needs and interventions and secure equal rights to individuals affected by these disorders as members of society.

Disclosure: Nothing to disclose.

MDS-ES European Basal Ganglia Club

SPS03_1 | C. David Marsden Award Lecture: What to do if even David Marsden would not have a clue?

B. Bloem

Radboud Medical Centre, Radboud UMC Center of Expertise for Parkinson & Movement Disorders, Nijmegen, The Netherlands

SPS03_2 | Dystonia Europe – David Marsden Award Lecture: Striato-pallidal oscillatory connectivity correlates with symptom severity in dystonia patients

R. Lofredi

Charité Universitätsmedizin Berlin, Klinik für Neurologie und Experimentelle Neurologie, Berlin, Germany

Sunday, June 28 2026

What's new on EAN guidelines?

SPS04_1 | EAN/ILAE/EpiCare/EPNS clinical practice guidelines in the field of rare and complex epilepsies: Lennox-Gastaut syndrome (LGS)

E. Trinka

Uniklinikum Salzburg, Christian-Doppler-Klinik, Department of Neurology, Salzburg, Austria

SPS04_2 | Management of SUNCT/SUNA, paroxysmal hemicrania, and hemicrania continua: Expert EAN consensus recommendations

D. Wei

King's College Hospital, Department of Basic and Clinical Neuroscience, London, United Kingdom

SPS04_3 | European guidelines for the symptomatic treatment of atypical Parkinsonian syndromes (MSA, PSP, CBD)

C. Palleis

LMU University Hospital, Department of Neurology, Munich, Germany

M. Majoie

Department of Neurology, Academic Centre for Epileptology, Epilepsy Centre Kempenhaeghe & Maastricht University Medical Centre, Netherlands

EAN CoCoCare Graduation 2025: Training the Next Generation of Cost-Conscious Neurology Guideline Developers

Marian HJM Majoie Affiliation Department of Neurology, Academic Centre for Epileptology, Epilepsy Centre Kempenhaeghe & Maastricht University Medical Centre, Netherlands

Dauren Ramankulov EAN Scientific Department Coordinator
Clinical guidelines are essential for ensuring high-quality, cost-efficient healthcare. They reflect scientific knowledge, guide clinical practices, and identify gaps in research, ultimately improving healthcare outcomes. However, the challenge lies in balancing quality, efficiency, accessibility, and sustainability. In response to this challenge, the Cost-Conscious Healthcare (CoCoCare) training programme was launched in 2018 at Maastricht University and later adopted by the European Academy of Neurology (EAN) in 2023. This year-long educational initiative targets neurology residents and early-career neurologists, equipping them with the skills to develop evidence-based, economically sustainable clinical guidelines. The programme combines e-learning, workshops, webinars, self-directed learning, and practical guideline development, culminating in project presentations at the EAN Congress. Over 140 participants from more than 25 European countries have completed the programme, which has received positive feedback for fostering cost-conscious decision-making and promoting active participation in guideline development. CoCoCare's fourth edition, launched at the 2026 EAN Congress, continues to nurture a new generation of experts poised to lead future guideline development within the EAN framework. Graduates gain mentorship, congress grants, and professional recognition, enhancing their careers and contributing to the long-term sustainability and quality of neurological care across Europe.

Disclosure: Nothing to disclose.

Breakthroughs in treatment neurology – Part 1

SPS05_2 | Chimeric antigen receptor (CAR) T-cell therapy in neurological disorders

H. Durmus

Istanbul Faculty of Medicine, Istanbul, Türkiye

SPS05_3 | Broad rim lesions as new biomarker for rapid disease progression in Multiple Sclerosis

V. Konstantinidis

Department of Neurology, Erasmus MC, Rotterdam, The Netherlands

The high diversity in individual clinical disease courses of multiple sclerosis (MS) is likely caused by heterogeneity in proportional contribution of underlying biology. Our understanding of mechanisms driving progressive disease in MS is limited, hampering the development of effective therapies. In our work, we aim to study differentiating underlying mechanisms by extensive biological characterization of people with opposite MS disease trajectories. In the current study, we investigated with an international team MS white matter lesion pathology of selected Netherlands Brain Bank (NBB) donors with slow versus rapid MS progression (26 vs. 18 individuals, respectively).

Donors with a rapid disease progression were characterized by high prevalence of a distinct histological MS lesion type marked by a hypocellular demyelinated core surrounded by extensive hypercellular myeloid cell rims. This broad rim lesion (BRL) type was characterized by cellular and transcriptional signatures of innate immune activation, inflammatory cytokine production and apoptosis. Within the entire NBB cohort, presence of BRL associated with rapid disease progression, homozygous carrier-ship of the rs10191329 severity-associated common genetic risk variant and higher levels of cerebrospinal fluid neurofilament light chain. Myeloid cell fractions in BRL rims stained positive for translocator protein 18-kDa (TSPO). An independent TSPO positron emission tomography study (114 individuals) revealed variation in white matter lesion myeloid cell rim size, and validated the association between lesions with a broad rim and disability progression in individuals with MS.

Our findings offer crucial insights into mechanisms behind MS progression, identifying BRL as a biomarker for rapid disease progression. Presence of BRL could potentially guide participant selection for future therapeutic trials targeting central nervous system intrinsic inflammation in progressive MS. Overall, our findings demonstrate the potential of translational studies comparing individual disease trajectories to reveal novel aspects of MS biology.

Disclosure: Nothing to disclose.

SPS05_4 | Innovative pharmacological therapies to promote the recovery of patients with disorders of consciousness

L. Sanz

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Disorders of consciousness is a group of neurological conditions resulting from heterogeneous etiologies yet with a common clinical phenotype. There are only very scarce therapeutic options at hand to improve clinical outcomes and promote the recovery of consciousness among these patients at the moment, despite the fact that even slim beneficial effects may result in tremendous implications both for the patients' quality of life and the financial burden on healthcare systems. Growing evidence suggests that early interventions may carry the most significant chances of efficacy by targeting brain plasticity and neurotransmitter imbalances shortly after injury, already in the ICU setting. Othman and colleagues have explored the use of pharmacological stimulants (methylphenidate and apomorphine) among patients with disorders of consciousness in the ICU and demonstrated preliminary evidence of increased arousal after the administration of these drugs, which may facilitate later recovery.

Pupillary responsiveness was identified as a potential marker of patients responding to these treatments, which may help identify candidates and guide therapy. On a similar note, Alnagger et al. are investigating the use of novel experimental pharmacological agents to enhance recovery of consciousness, using different mechanisms of action. They designed a virtual clinical trial using advanced computational modeling of brain activity to test the effect of psilocybin and lysergic acid diethylamide (LSD), two psychedelics, on brain dynamics of simulated patients, and demonstrated the potential future role of these innovative treatments in disorders of consciousness.

Disclosure: Nothing to disclose.

Clinical Grand Round

SPS06_1 | Not your average white matter story

R. Vernard-Valnet
*Centre Hospitalier Universitaire Vaudoise – CHUV BH07,
Department of Clinical, Neuroscience, Lausanne, Switzerland*

SPS06_2 | Beyond the tremor: A path to a hidden diagnosis

S. Gehri
*CHUV (Centre Hospitalier Universitaire Vaudois), Grandson,
Switzerland*

SPS06_3 | A seemingly simple head trauma with an unexpected diagnosis

P. Lalive
Geneva University Hospitals, Geneva, Switzerland

EAN/ESC: Brain & heart interaction for cognitive function and longevity

SPS07_1 | Bilateral brain-heart interaction – An introduction

M. Endres
*Charité Universitätsmedizin Berlin, Department of Neurology
and Experimental Neurology, Berlin, Germany*

SPS07_2 | The neuronal system and cardiac function

D. Carnevale
*Sapienza University of Rome and IRCCS Neuromed, Neuro and
Cardiovascular, Pathophysiology, Pozzilli, Italy*

SPS07_4 | Individualized cognitive risk in CV disease

R. Proietti
University of Liverpool, United Kingdom

SPS07_5 | Stroke-heart-syndrome – An update

M. Endres
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and Experimental Neurology, Berlin, Germany*

Omnipresent stress, its detrimental effects, and the beneficial effects of autogenic training

SPS08_1 | Physiology and detrimental sequelae of acute and chronic stress, and relaxation techniques reducing noxious stress-effects

M. Hilz
University Erlangen-Nuremberg, Erlangen, Germany

SPS08_2 | How autogenic training induces autonomic nervous system changes that cause relaxation and comfort

M. Hilz
University Erlangen-Nuremberg, Erlangen, Germany

SPS08_3 | Condensed introductory autogenic training session

M. Hilz
University Erlangen-Nuremberg, Erlangen, Germany

Monday, June 29 2026

EJON: Emerging technologies driving the next generation of neurological biomarkers

SPS09_1 | Between hope and uncertainty: Biomarker-led early diagnosis in neurodegenerative disease

P. Scheltens
*Amsterdam University Medical Centers, Alzheimer Center,
Amstelveen, The Netherlands*

SPS09_2 | In search of certainty: Biomarkers and the reproducibility challenge in polyneuropathies

A. Roos

University Medicine Essen, Department of Pediatric Neurology, Essen, Germany

SPS09_3 | Neurotechnology and biomarkers for precision diagnosis and prognosis in epilepsy

K. Vonck

Ghent University Hospital, Department of Neurology, Ghent, Belgium

SPS10_1 | Posterior fossa stroke: Update on thrombolysis and thrombectomy

P. Michel

Centre Hospitalier Universitaire Vaudoise – CHUV BH13, Neurology Service, Lausanne Switzerland

SPS10_2 | Antiepileptogenesis within reach: Preventing epilepsy after stroke

E. Trinka

Uniklinikum Salzburg, Christian-Doppler-Klinik, Department of Neurology, Salzburg, Austria

Breakthroughs in treatment neurology – Part 2

SPS10_3 | Disease-modifying therapies in frontotemporal dementia: Breakthrough clinical trials and emerging treatment paradigms

R. Vandenberghe

University Hospitals Leuven, Leuven, Belgium

The term “frontotemporal dementia” (FTD) covers a variety of rare diseases with distinct phenotypes, etiologies, and neuropathological features. All FTD diseases have in common that there is no symptomatic or disease-modifying therapy of proven efficacy and no in vivo biomarkers that accurately reflect the underlying neuropathological hallmark lesions. Current trials mostly target the forms with known genetic causes where the disease mechanism is clear. Progranulin (PGRN) mutations lead to a loss of function with a decrease in PGRN protein levels. At the time of writing, more than five industry-sponsored trials are ongoing in FTLT due to PGRN mutations. The main mechanisms of action under study are PGRN gene replacement via various delivery modes and PGRN protein substitution. We will review the therapeutic strategies, the opportunities and challenges. For the two other

main genetic causes (c9orf72 and MAPT) the therapeutic path forward is currently less clear for various reasons. In sporadic FTLT, an investigator-initiated study of a myeloperoxidase (MPO) inhibitor, verdiperstat, is ongoing in the semantic variant of primary progressive aphasia.

Progress in clinical development of novel therapies in FTLT also depends on readiness of clinical trial networks organized around hubs at a transnational, European level. Furthermore, due to the historical absence of treatment options, persons who are potentially interested to participate in trials may not have received a diagnosis or may not receive the information. The search for novel therapies for FTLT has intensified over the past five years offering hope to patients and families.

Disclosure: RV’s institution has a clinical trial agreement (RV as PI) with AviadoBio, BMS, Denali, Eli Lilly, J&J, Merck, and UCB. RV’s institution has a consultancy agreement (RV as chair or member of Data Safety Monitoring board or Data Monitoring Committee) with AC Immune and Novartis. RV’s institution has a consultancy agreement (RV as international Advisory Board Member) with Roche. RV’s research has been sponsored by the Mady Browaeys Fonds voor Onderzoek naar Frontotemporale Degeneratie.

The neurology of a changing planet

SPS11_1 | Effects of heat on human physiology – A perspective on climate change and human resilience

H. Pallubinsky

Department of Nutrition and Human Movement Sciences, NUTRIM Institute for Nutrition and Translational Research in Metabolism, Maastricht University, the Netherlands

According to the World Health Organization (WHO), heat is among the most dangerous of natural hazards for humans. Projections anticipate the death toll to rise sharply in the upcoming decades, unless effective prevention plans and long-term strategies are implemented on a wide scale. Understanding how heat affects the human body, who is most vulnerable, and how resilience can be strengthened is therefore critical.

The effects of heat exposure on the human body are dependent on environmental parameters (e.g., ambient temperature, solar radiation, humidity, air velocity), duration of exposure, and individual characteristics such as sex, age, fitness, and health status. Ageing and chronic disease, such as cardiovascular diseases or type 2 diabetes, increase vulnerability to heat strain. Moreover, people living with certain neurological conditions, such as epilepsy, may be negatively affected by climate change, as seizures may be triggered by heat stress.

However, importantly, heat should not be viewed solely as a threat. Repeated exposure induces a range of thermophysiological adaptations, including enhanced sweating, improved skin blood flow, reduced core temperature, and greater cardiovascular stability. Heat therapy, or heat training, has also been shown to induce health benefits, such as enhanced cardiovascular function and improved glucose homeostasis. This talk presents heat as a double-edged sword. It argues for a shift toward individualized and context-aware strategies that both protect

vulnerable populations and harness the adaptive potential of heat to strengthen human resilience and reduce health risks.

Disclosure: None.

SPS11_2 | Environmental neuroscience: Insights from experimental models and human tissue

E. Aronica

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Climate change and environmental degradation are critical determinants of brain health. Heat stress, air pollution, chemical toxicants, and other environmental exposures increase the risk and progression of neurological disorders. Environmental Neuroscience integrates epidemiology, experimental models, and human tissue studies to uncover how these exposures affect neural structure and function.

The neural exposome, reflecting cumulative exposures across the lifespan, provides a framework to understand how physical, chemical, biological, and social stressors shape neurological vulnerability. Exposures trigger oxidative stress, inflammation, blood–brain barrier disruption, metabolic imbalance, and epigenetic modulation, converging on networks that regulate neuronal excitability, glial activation, and neurovascular integrity. Experimental approaches, including in vitro systems, organ-on-chip platforms, and in vivo models, allow controlled investigation of heat, particulate matter, and chemical exposures. Multi-tier strategies, from computational screening to rodent models, assess dose dependency, interactions, and gene–environment effects. Human brain tissue studies confirm exposure accumulation and validate mechanisms.

A key convergent pathway involves hypothalamic thermoregulation. Warm-sensitive neurons and ion channels (TRPV1, TRPC4, SCN1A/Nav1.1) maintain thermal homeostasis; dysfunction increases neuronal excitability, seizure susceptibility, and promotes glial activation, extracellular matrix remodeling, and inflammatory signaling. These mechanisms occur across neurological conditions, including neurodegenerative disorders and epilepsy.

Understanding these pathways is essential for identifying vulnerable populations and guiding preventive and therapeutic strategies. Environmental Neuroscience provides a vital framework to mitigate climate-related neurological risk.

Disclosure: Nothing to disclose.

SPS11_3 | Adapting neurological care to environmental change

S. Sisodiya

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SPS11_4 | Transforming healthcare systems for a climate-resilient future

D. Campbell-Lendrum

World Health Organization, Geneva, Switzerland

Brief summary of stress and its sequelae and intensified autogenic training sessions

SPS12_1 | Brief repetition of the mechanisms during stress and the methods of autogenic training that mitigate the autonomic imbalance induced by acute and chronic stress

M. Hilz

University Erlangen-Nuremberg, Erlangen, Germany

SPS12_2 | Intensified autogenic training session 1

M. Hilz

University Erlangen-Nuremberg, Erlangen, Germany

SPS12_3 | Intensified autogenic training session 2 and summary

M. Hilz

University Erlangen-Nuremberg, Erlangen, Germany

Partnering for the brain in Europe and beyond

SPS13_1 | Global brain health: iGAP and beyond

SPS13_2 | Panel 1: Reducing the burden of neurological disorders in Europe: Focus on prevention

SPS13_3 | Panel 2: Global advocacy for neurology: How to join forces?

ABSTRACTS

Oral Presentation

Saturday, June 27 2026

Autonomic Nervous System Diseases 1

OPR-001 | Development of autonomic dysreflexia card

E. Hagen

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Background and Aims: Autonomic dysreflexia (AD) is a potentially life-threatening medical emergency, most commonly occurring in individuals with spinal cord injury (SCI) at or above the T6 neurological level. AD is characterized by a sudden increase in systolic blood pressure of >20 mmHg above a person's baseline, which is the result of an unopposed sympathetic response to noxious or non-noxious stimuli below the injury. To facilitate prompt recognition and targeted intervention of AD by clinicians, rapid access to essential information, including patient-specific information, is critical.

Methods: Through joint online surveys and meetings, the International Spinal Cord Society (ISCoS) and the European Society of Physical and Rehabilitation Medicine (ESPRM)

have systematically developed an International AD Medical Emergency Card highlighting causes, management, and prevention of AD.

Results: The International AD Medical Emergency Card has been uploaded on ESPRM and ISCoS platforms. It contains Personal information, General information concerning AD, Management of AD and QR codes linking to international standards, guidelines and a review. It is a pocket size-Instructions for completing and editing the Medical Emergency Card has been developed, as well as a translation process. The card is bilingual, in English and a national language.

Conclusion: The International AD Medical Emergency Card has been developed and will be updated regularly. It is presently in English and Greek and translations to other languages is underway.

Disclosure: Nothing to disclose.

OPR-002 | Diagnosing prodromal synucleinopathies: What is the role of cardiac metaiodobenzylguanidine scintigraphy?

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Background and Aims: Cardiac metaiodobenzylguanidine (MIBG) scintigraphy differentiates Lewy body disorders (LBD), such as Parkinson's disease and dementia with Lewy bodies, from multiple system atrophy (MSA). Reduced cardiac MIBG uptake reflects postganglionic sympathetic denervation typical of LBD, while uptake is usually preserved in MSA. Abnormal uptake has also been reported in pure autonomic failure (PAF). We aimed to characterize cardiac MIBG uptake in patients with an initial diagnosis of PAF (iPAF) and evaluate its role as a prognostic tool in stratifying risk of phenoconversion.

Methods: As part of our longitudinal natural history study, consecutive patients with iPAF underwent prospective clinical and autonomic evaluations, including cardiac MIBG scintigraphy.



FIGURE 1 Colour version of the card in full size for printing on a colour printer. There are 8 sections. Four in Greek and four in English.

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Patients were followed until conversion to LBD or MSA, or until last follow-up in 2025.

Results: Among 106 patients with iPAF, 63 (59%) remained clinically stable for ≥ 3 years; 62 (98%) had abnormal MIBG uptake. The single patient with normal uptake was under 50 years old with severe urogenital dysfunction. Thirty-four patients (32%) converted to LBD; 33 (97%) showed abnormal MIBG uptake up to 9 years before phenoconversion. Eight patients (8%) converted to MSA, all with normal uptake up to 1 year before phenoconversion. One patient with parkinsonism of uncertain diagnosis also had normal uptake.

Conclusion: Cardiac MIBG scintigraphy was highly sensitive for detecting cardiac sympathetic denervation in a large cohort of iPAF and accurately predicted disease trajectories up to 9 years before phenoconversion, supporting its value for risk stratification and counseling.

Disclosure: Nothing to disclose.

OPR-003 | Cardiovascular reflex tests as early indicators of disease-modifying treatments eligibility in asymptomatic hereditary transthyretin amyloidosis carriers: Introducing the “Two or more rule”

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Background and Aims: Hereditary transthyretin amyloidosis (ATTRv) is a progressive multisystem disorder in which autonomic dysfunction may precede both neuropathy and cardiomyopathy. Early identification of autonomic involvement is crucial, as disease-modifying treatments (DMTs) are most effective in the earliest disease stages.

Methods: In this monocentric, cross-sectional study, 47 asymptomatic ATTRv carriers and 47 healthy controls underwent cardiovascular reflex testing (CRTs), including head-up tilt test (HUTT), Valsalva manoeuvre (VM), deep breathing (DB), cold face (CF), and handgrip (HG). Forty carriers also underwent ^{99m}Tc-DPD scintigraphy.

Results: ATTRv carriers exhibited a significantly reduced overshoot during VM ($p < 0.001$) and attenuated blood pressure responses during HUTT, despite the absence of overt orthostatic hypotension. Overall, 21 of 47 carriers (44.7%) demonstrated at least one abnormal CRT and 7 of 21 were initiated on DMTs. All treated individuals presented with two or more abnormal CRTs, most frequently involving overshoot and Valsalva ratio (VR), or the combination of overshoot, VR, and DB abnormalities.

Conclusion: CRTs therefore appear to be sensitive markers of early autonomic dysfunction in ATTRv carriers. The presence of two or more abnormal CRTs (e.g., overshoot < 15 mmHg, VR < 1.24 , $\Delta I-E$ in DB < 10 bpm) identifies a subgroup at higher risk and may represent a practical threshold for advanced evaluation and early therapeutic intervention. Integrating this “two-or-more rule” into routine monitoring may enable timely treatment initiation and improve long-term outcomes in presymptomatic carriers of ATTRv.

Disclosure: Nothing to disclose.

OPR-004 | Autonomic involvement in small fiber neuropathy

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Background and Aims: The diagnosis of small fiber neuropathy (SFN) is based on sensory symptoms and signs, supported by sensory dysfunction assessment using quantitative sensory testing and/or evidence of intraepidermal nerve fiber (IENF) loss. However, autonomic impairment is frequently part of the clinical picture.

Methods: We assessed sensory and autonomic symptoms, as well as cardiovascular and sudomotor dysfunction, using the SFN-SIQ and COMPASS-31 questionnaires, cardiovascular reflex tests (Ewing score), sympathetic skin response (SSR), dynamic sweat test (DST), and IENF density at the distal leg. A total of 337 patients with SFN (age 49.0 ± 14.27 years; 237 women) from three centers were included.

Results: According to etiology, patients were classified into five groups: dysimmune ($n = 103$), metabolic ($n = 39$), toxic ($n = 5$), genetic ($n = 22$), and idiopathic ($n = 168$). No significant differences in functional or morphological assessments were observed among groups, except for higher COMPASS-31 scores in patients with dysimmune etiology. In the overall population, a COMPASS-31 total score > 30 was found in 59.3% of patients, while an Ewing score ≥ 2 was observed in 26.9%. SSR was absent in 7 patients and showed reduced amplitude in 10.6%. DST at the distal leg demonstrated hypohidrosis in 79.1% of patients compared with cut-off values derived from 210 healthy controls. IENF loss was detected in 81% of patients. Overall, sensory and

autonomic symptom severity (SFN-SIQ and COMPASS-31 total scores) was higher in women, who also exhibited greater sudomotor impairment.

Conclusion: These findings indicate that autonomic involvement is common in SFN and support the inclusion of postganglionic sudomotor function assessment in the diagnostic criteria for SFN.

Disclosure: Nothing to disclose.

OPR-005 | To tilt or to stand? Comparing the accuracy of current diagnostic tests for orthostatic hypotension in Parkinson's disease

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Background and Aims: Classic orthostatic hypotension (cOH) affects approximately 30% of persons with Parkinson's disease (pwPD), being associated with functional read-outs like orthostatic intolerance, syncope and falls. cOH is defined by a systolic and/or diastolic blood pressure drop $\geq 20/10$ mmHg within 3 min of an orthostatic challenge, be it an active standing test (AST) or a passive head-up tilt-test (HUT). However, the diagnostic accuracy of these tests in pwPD remains unclear.

Methods: In a retrospective cohort of 173 pwPD, who underwent both the AST and the HUT at the Medical University of Innsbruck between 2007 and 2020, we compared the diagnostic accuracy for cOH of HUT and AST.

Results: Both AST and HUT showed acceptable sensitivity for cOH in pwPD [63% (confidence interval, CI 48%–77%) vs. 73% (CI 59%–85%)] with moderate agreement between the two tests (Cohen's k 0.43, CI 0.26–0.60). Compared to AST, the HUT warranted a 10% sensitivity gain for cOH. We found no differences in the frequency of orthostatic intolerance, syncope and falls across persons with cOH at both the AST and the HUT and those with cOH at either the AST or the HUT only ($p=1$, $p=0.26$ and $p=0.69$ respectively).

Conclusion: Identifying and managing cOH improves the quality of life and prevents adverse outcomes in pwPD. The HUT shows higher sensitivity for cOH compared to AST but a proportion of pwPD shows cOH only at either the AST or the HUT. In clinical practice, AST and HUT represent therefore complementary tests, with similar sensitivity for cOH-related functional read-outs.

Disclosure: Academic study without external financial support. The authors have no conflict of interest to declare.

Cognitive Neurology/Neuropsychology

OPR-006 | Substrates of consolidated resilience and impact on cognitive outcomes in multiple sclerosis

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Background and Aims: Consolidated resilience integrates cognitive reserve and cognitive resilience. Its relevance in multiple sclerosis (MS) and the underlying functional mechanisms remain unexplored.

Methods: Structural and functional 3.0 T MRI scans were acquired from 522 MS patients and 304 healthy controls (HCs). Consolidated resilience scores were estimated in patients using a partial least-squares path model, combining proxies of cognitive reserve (education and intelligence quotient) and cognitive resilience (residuals from the regression of cognitive scores on T2-hyperintense white matter lesion volumes and normalized brain volumes standardized to HCs). After a median follow-up of 2.0 years (interquartile range = 1.1; 2.9 years), 179 patients underwent cognitive and MRI reassessment. Resting state (RS) functional connectivity (FC) was estimated using independent component analysis. Linear mixed models tested the interaction between consolidated resilience and RS FC changes in cognitive networks in predicting variations in cognitive scores.

Results: At follow-up, higher consolidated resilience was associated with better verbal and visuospatial memory, processing speed and verbal fluency (all $p < 0.001$). Significant three-way interactions (consolidated resilience $\times$ RS FC changes $\times$ time) were observed in verbal memory (executive control network [$\beta = -1.36$, $p = 0.029$], left frontoparietal network [$\beta = -0.79$, $p = 0.034$] and default mode network [$\beta = -0.71$, $p = 0.035$]) and processing speed changes (salience network [$\beta = -1.43$, $p < 0.001$], executive control network [$\beta = -0.92$, $p = 0.030$], left frontoparietal network [$\beta = -0.66$, $p = 0.034$] and default mode network [$\beta = -0.83$, $p = 0.003$]).

Conclusion: Consolidated resilience moderated the association between cognitive functioning and FC changes. In highly resilient patients, better cognitive performance was associated with stable or decreased FC, whereas in patients with lower resilience, better cognitive outcomes correlated with increased FC.

Disclosure: D. Mistri, P. Valsasina, A. Meani, N. Tedone, M. Rubin, G. Guido and F. Esposito report no competing interests. M. Margoni reports grants and personal fees from Sanofi Genzyme, Merck Serono, Roche, Biogen, Amgen and Novartis. Filippi is Editor-in-Chief of the Journal of Neurology, Associate



FIGURE 1 Consolidated resilience integrates cognitive reserve and cognitive resilience.

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OPR-007 | Glymphatic dysfunction by DTI-ALPS links to GFAP, brain atrophy, white matter hyperintensity progression, and processing speed decline in ONDRI cohort

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Background and Aims: Glymphatic clearance is implicated in neurocognitive disorders, but its role in human is unclear. The diffusion tensor image analysis along the perivascular space (DTI-ALPS) index provides a noninvasive MRI marker

of glymphatic function. We examined the association between baseline DTI-ALPS and 1-year changes in gray matter (GM) and white matter hyperintensity (WMH) volumes. Secondary aims were to assess whether plasma GFAP mediates DTI-ALPS effects on GM atrophy and the effect of DTI-ALPS on processing speed decline via GM volume.

Methods: We studied 304 participants from the Ontario Neurodegenerative Disease Research Initiative (ONDRI): AD/MCI ($n=120$), cerebrovascular disease (CVD, $n=139$), and frontotemporal dementia (FTD, $n=45$). MRI-derived GM, WMH, perivascular space, DTI-ALPS indices, total intracranial volumes were measured. Plasma GFAP was quantified. Processing speed was assessed with a composite z-score from the Digit Symbol Modalities Test and Trail Making Test A. Linear regression and mediation analyses were performed.

Results: Baseline DTI-ALPS was significantly associated with 1-year GM loss ($\beta=0.111$, $p=0.048$) and WMH increase ($\beta=-0.148$, $p=0.013$). Baseline plasma GFAP mediated the effect of DTI-ALPS on GM atrophy (indirect effect: 845.94; 95% CI [73.89, 3370.62]). Baseline left DTI-ALPS influenced 1-year processing speed decline via GM volume (indirect effect: 0.1385; 95% CI [0.042, 0.3016]).

Conclusion: Glymphatic dysfunction, measured by DTI-ALPS, is linked to structural brain changes and cognitive decline in mixed neurodegenerative and cerebrovascular disorders. Plasma GFAP mediates GM atrophy, highlighting astroglial involvement. DTI-ALPS, combined with plasma GFAP, may serve as a noninvasive biomarker to guide mechanistic studies and therapeutic strategies targeting glymphatic pathways.

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OPR-008 | Structural and functional brain correlates of fatigue in neuromyelitis optica spectrum disorder

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Background and Aims: Fatigue in NMOSD remains poorly understood. We investigated structural and functional MRI correlates of fatigue in NMOSD patients.

Methods: Twenty-five NMOSD patients and 60 healthy controls (HC) underwent 3T brain structural and functional MRI scans, and fatigue assessment using the Modified Fatigue Impact Scale (MFIS). In patients, associations were evaluated between MFIS and regional distribution of T2-hyperintense lesions, microstructural white matter (WM) damage, regional grey matter (GM) atrophy, and seed-based resting-state (RS) functional connectivity (FC) abnormalities within main brain networks.

Results: Higher MFIS scores were associated with lower GM volume in the right superior frontal gyrus, right posterior cingulate cortex, left inferior frontal gyrus, and right parahippocampal gyrus (r range = $-0.37/-0.40$, $p < 0.001$ uncorrected). Higher MFIS was also associated with increased RS FC within the two primary motor cortices (BA4), as well as between the right BA4 and the left postcentral gyrus, and between the left BA4 and the right temporal cortex (r range = $0.36/0.47$, $p < 0.001$ uncorrected). Finally, higher MFIS scores were associated with: (1) increased RS FC between the right insula and the right inferior frontal gyrus ($r = 0.46$, $p < 0.001$), (2) decreased RS FC within the left posterior cingulate cortex of the default-mode network, and (3) decreased RS FC between the left caudate and bilateral thalami (r range = $-0.45/-0.48$, $p < 0.001$). No associations were found between fatigue and distribution of T2-hyperintense lesions or WM microstructural abnormalities.

Conclusion: Fatigue in NMOSD is primarily related to large-scale brain network dysfunction rather than focal WM damage, supporting a network-based MRI biomarker framework for this symptom.

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OPR-009 | Blood biomarkers of Alzheimer's disease and progression of brain atrophy and cerebrovascular burden in cognitively healthy older adults

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Background and Aims: Blood biomarkers of Alzheimer's disease (AD) have been linked to structural brain alterations in clinical cohorts, but evidence in cognitively healthy populations remains limited. We investigated the associations between AD blood biomarkers and changes in brain volumes and cerebrovascular burden in cognitively healthy community-dwelling older adults.

Methods: In a cohort of 372 dementia-free older adults from Sweden, baseline blood levels of amyloid- $\beta_{42/40}$ (A $\beta_{42/40}$), phosphorylated tau (p-tau)181 and 217, total tau (t-tau), neurofilament light chain (NfL), and glial fibrillary acidic protein (GFAP) were measured and their z-scores derived. Brain MRI was performed at baseline and after 3 and/or 6 years. Associations between AD blood biomarkers and changes in

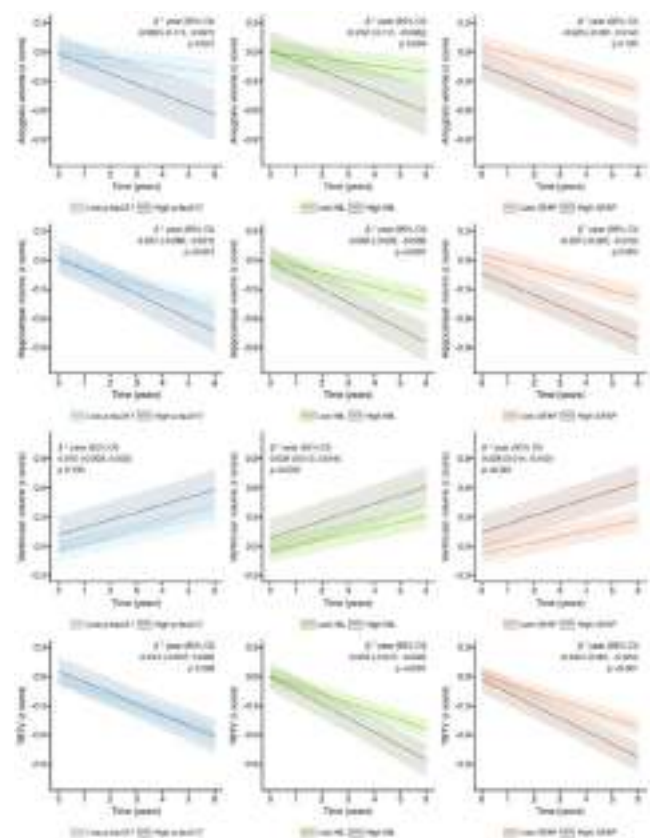


FIGURE 1 Annual rate of change in total brain tissue (TBTv), ventricular and hippocampus and amygdala volumes, by baseline levels of p-tau217, NfL and GFAP.

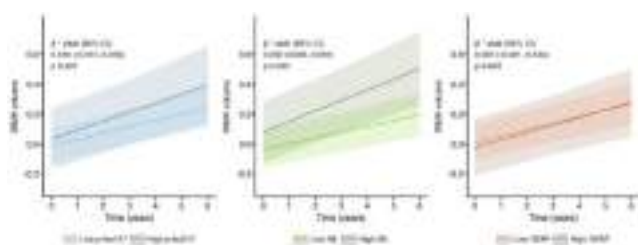


FIGURE 2 Annual rate of change in white matter hyperintensities (WMH) volume by baseline levels of p-tau217, NfL and GFAP.

volumes of total brain tissue (TBTv), ventricles, hippocampus, amygdala and white matter hyperintensities (WMH) were examined using linear mixed models.

Results: Higher p-tau181 and p-tau217 were associated with faster hippocampal volume loss (beta*year -0.036 , 95% CI -0.063 , -0.009 ; and beta*year -0.051 , 95% CI -0.080 , -0.021 , respectively), and p-tau217 was additionally associated with amygdala volume decline. Elevated GFAP was linked to faster TBTv decline, ventricular enlargement, and hippocampal shrinkage. Higher NfL levels were associated with steeper TBTv decline, ventricular enlargement, hippocampal and amygdala shrinkage, and faster WMH accumulation.

Conclusion: In cognitively healthy older adults, elevated blood levels of p-tau isoforms were linked to decline in AD-specific regions, whereas NfL and GFAP were associated with widespread atrophy and NfL also with WMH accumulation. These findings suggest that altered AD blood biomarkers may capture the earliest stages of dementia-related brain changes in the general population.

Disclosure: Nothing to disclose.

OPR-010 | Precuneus and posterior cingulate functional connectivity in multiple sclerosis: Age, onset timing and cognitive implications

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Background and Aims: Precuneus and posterior cingulate cortex (PCC) are cognitive hubs frequently involved in multiple

sclerosis (MS). We explored the effects of age and age at onset on static (sFC) and time-varying functional connectivity (TVFC) of precuneus/PCC and their association with cognition.

Methods: We enrolled 279 right-handed MS patients and 130 matched healthy controls (HC) from three Italian sites. All subjects underwent Rao's battery and 3T resting-state fMRI assessment. sFC was computed with seed-to-voxel correlation; TVFC with sliding-window correlation using bilateral precuneus and PCC as seeds.

Results: Compared with HC, MS patients showed limited sFC increase between the precuneus/PCC and parieto-frontal regions, and widespread sFC decrease within the default-mode network. Time-varying analysis revealed localized TVFC increase of the precuneus/PCC with lingual and cerebellar regions. In MS, a modulation of age on connectivity (significant at disease-by-age interaction analysis) was found: older age was associated with decreased sFC between the precuneus and cerebellum, and between PCC and anterior cingulate cortex. Similarly, older age was associated with decreased TVFC between the precuneus and left caudate (associated with better visual memory performance [$\rho = -0.19$, $p = 0.002$]) and between PCC and right superior frontal gyrus. Older age at disease onset was associated with decreased TVFC between the PCC and right thalamus, right angular gyrus, right middle frontal and left postcentral gyri, these latter two were associated with worse fluency performance ($\rho = 0.13-0.15$, $p = 0.02-0.03$).

Conclusion: In MS, age and age at disease onset significantly influenced sFC and TVFC of the precuneus and PCC, contributing to explain cognitive performances.

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Pharma, Neuraxpharm, Novartis, Roche, Sandoz, and Sanofi. She receives research support from the MS Society of Canada, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. She is Associate Editor for Multiple Sclerosis and Related Disorders; and Associate Co-Editor for Europe and Africa for Multiple Sclerosis Journal.

Neuro-Oncology

OPR-011 | Leptomeningeal metastases from primary brain tumors: Still a neglected complication

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Background and Aims: Leptomeningeal metastases (LM) from primary brain tumors are rare and aggressive with information about optimal management limited to small retrospective series. We report the characteristics and outcomes of a large cohort of patients with LM from primary brain tumors treated at our Institution.

Methods: Clinico-therapeutic and outcome data of adult patients with LM from primary brain tumors were retrospectively collected. LM were classified according to EANO–ESMO criteria. Time-to-LM was defined as the interval from first surgery to LM diagnosis, and overall survival from LM (OS-LM) as the interval from LM diagnosis to death or censoring. Statistical analyses included chi-square tests and Cox regression models.

Results: 111 patients diagnosed between 1994 and 2025 were included (median age 50 years). The most frequent histologies were glioblastoma IDH-wildtype (33.7%), astrocytoma IDH-mutant (16.8%), medulloblastoma (15.8%), and ependymoma (15.8%). LM were synchronous with the primary tumor in 15.8% of cases and occurred mainly at local tumor progression. Diagnosis was based on clinico-radiological findings in 72.6%. Median time-to-LM was 17.0 months and significantly differed by histology ($p < 0.001$), with the shortest interval in glioblastoma. Median OS-LM was 4.0 months overall and was strongly influenced by histology ($p < 0.001$). On multivariable analysis, Karnofsky Performance Status ≥ 70 and LM from ependymomas were independently associated with longer OS-LM.

Conclusion: LM represent a dismal complication of primary brain tumors. In this cohort, tumor histology and performance status were the main determinants of time-to-LM and survival, whereas EANO–ESMO class and treatment modalities did not significantly impact OS-LM. Molecular analysis in the different subgroups are ongoing.

Disclosure: Nothing to disclose.

OPR-012 | IDH-mutant gliomas treated with vorasidenib: Early seizure control correlates with [18F]-DOPA PET response and D-2-hydroxyglutarate plasma reduction

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Background and Aims: Given the role of IDH mutations and D-2-hydroxyglutarate (D2HG) in epileptogenesis, the impact of IDH inhibitors on seizure control is appealing. This prospective study assessed patterns and timing of seizure response in grade 2 IDH-mutant glioma patients receiving vorasidenib after surgery.

Methods: Seizures were recorded at the end of each 28-day cycle. MRI, [18F] fluorodopa (F-DOPA) PET, and D2HG plasma levels were performed at baseline and every 3 cycles.

Results: Of 56 patients, 12 (7 astrocytomas, 5 oligodendrogliomas) had persistent seizures prior to vorasidenib (mean frequency: 4/month); 9/12 had measurable F-DOPA PET uptake; median D2HG plasma concentration was 39.7 ng/mL. Over a median treatment duration of 11.6 months, 6/12 achieved seizure freedom within 1 cycle, whereas 5/12 improved gradually. One patient, after initial improvement, worsened by cycle 4. All patients showed stable disease (SD) per RANO criteria on MRI after 3 and 6 cycles. 8/9 patients with seizure reduction showed reduced PET uptake by cycle 3 (mean changes: SUVmax -6.3% ; TBRmax -7.5% ; MTV -4.7 cm^3) and 6 (SUVmax -20.9% ; TBRmax -10.9% ; MTV -35.9 cm^3), translating into 3 partial responses and 5 SD by PET RANO. All 11 patients with durable seizure response showed reduced D2HG at cycle 3 (mean change: -51%) and cycle 6 (-60%). In contrast, the only patient, who experienced seizure worsening over time, showed D2HG reduction at cycle 3 (-53%), but no further reduction, and PET progression disease, at cycle 6.

Conclusion: These findings suggest that F-DOPA PET and D2HG plasma levels may correlate with seizure improvement after vorasidenib.

Disclosure: F.B., A.P., R.R.: Servier (Honoraria for Preceptorships) All other Authors: nothing to disclose.

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Background and Aims: Oncolytic immunovirotherapy is an emerging therapeutic approach in neuro-oncology. DNX-2401, a tumour-selective oncolytic adenovirus with enhanced infectivity, has demonstrated clinical activity in adults with recurrent glioblastoma. DNX-2440 is a next-generation construct incorporating the OX40-ligand to enhance antitumour immune responses through T cell co-stimulation and modulation of the immunosuppressive tumour microenvironment. The primary objective was to evaluate the safety and feasibility of DNX-2440, with secondary exploration of preliminary clinical activity.

Methods: This open-label, single-centre, phase I dose-escalation study used a 3+3 design in patients with first or second glioblastoma recurrence. Participants underwent stereotactic biopsy followed by single intratumoral DNX-2440 administration. Two dose levels were evaluated: 4×10^9 and 4×10^{10} viral particles. Eligibility required a single measurable lesion of at least 10 mm in two perpendicular diameters and a maximum tumour volume of 25 cc.

Results: Sixteen patients were enrolled, including 6 women and 10 men, with a median age of 55 years (range 39–80) and a median KPS of 80 (range 70–90). No dose-limiting toxicities occurred during escalation, with 3 patients treated per dose level. Remaining patients received 4×10^{10} vp in the expansion cohort. DNX-2440 was well tolerated, with no treatment-related serious adverse events. Clinical benefit, including tumour reduction and prolonged survival, was observed in 3 patients. Correlative studies assessed systemic immune responses and characterised the tumour immune microenvironment.

Conclusion: Single intratumoral DNX-2440 administration is feasible, with a favourable safety profile and hints of antitumour activity, supporting further investigation of oncolytic virotherapy as an immunomodulatory strategy in glioblastoma.

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Background and Aims: This study aimed to describe clinical features and long-term outcomes of patients with glycine receptor (GlyR) antibody-mediated progressive encephalomyelitis with rigidity and myoclonus (PERM).

Methods: We retrospectively analyzed 41 patients with PERM and GlyR antibodies (22 diagnosed in our laboratory, 19 previously reported patients identified through a systematic literature review) with sufficient clinical data and ≥ 12 months of follow-up. Neurologic disability was assessed using the modified Rankin Scale (mRS). Relapses were defined as events occurring > 6 months after onset requiring immunotherapy.

Results: Median age was 58 years (IQR: 43–66 years), and 36 (88%) were male. Median time from symptom onset to admission was 2 weeks (IQR: 1–4 weeks). Predominant presentations included brainstem symptoms, mainly dysphagia and trismus, in 23 patients (56%); muscle stiffness and myoclonus in 9 (22%); dysesthesias or pruritus in 7 (17%); and cacosmia with dysgeusia in 2 (5%). Five patients (12%) never developed stiffness. All received immunotherapy. Eleven patients died, 8 (20%) from complications of PERM. Ten patients relapsed but responded well to immunotherapy. At last follow-up [median time 24 months (IQR: 18–72 months)] 23 (70%) of the 33 patients who did not die from PERM presented good functional status (mRs score < 3).

Age and admission to the intensive care unit independently predicted poor outcomes (mRS score ≥ 3).

Conclusion: GlyR antibody-mediated PERM is a rapidly progressive and severe disease that predominantly affects men and frequently presents with brainstem involvement. Its distinct demographic and clinical features support considering it separately from stiff-person spectrum disorders.

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OPR-015 | Reinforcing peritumoral GABAergic inhibition modulates glioma progression and enhances immunotherapy efficacy

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Background and Aims: Gliomas disrupt cortical excitatory-inhibitory balance, contributing to neurological dysfunction and tumor progression. Parvalbumin (PV)-expressing GABAergic interneurons are key regulators of network stability within the peritumoral microenvironment. This study aimed to determine whether chemogenetic modulation of peritumoral PV-interneurons and pharmacological enhancement of GABAergic signaling influence glioma growth, cortical function, and survival in murine models.

Methods: PV-Cre mice were implanted with GL261 glioma cells. To selectively modulate peritumoral PV-interneurons, AAV-hSyn-DIO-hM4D(Gi)-mCherry or AAV-hSyn-DIO-hM3D(Gq)-mCherry were bilaterally injected into the motor cortex adjacent to the tumor site. From day 12 to day 22 post-tumor induction, mice received clozapine-N-oxide (CNO) or vehicle twice daily. As readouts, we evaluated tumor volume by magnetic resonance imaging, longitudinal motor performance, and animal survival. To evaluate a pharmacological strategy, GL261- and CT-2A-bearing mice were treated with the GABAB receptor agonist baclofen, alone or combined with anti-PD-L1 immunotherapy. In vivo and in vitro assays were performed to assess direct drug effects.

Results: PV-interneuron inactivation exacerbated neurological symptoms and epileptiform activity without affecting tumor size or survival. Conversely, PV-interneuron activation reduced tumor proliferation, preserved motor and network function, and partially maintained visual acuity and contrast sensitivity. Baclofen modestly reduced tumor volume and motor decline in GL261 but not CT-2A gliomas, consistent with indirect microenvironment-mediated effects. Notably, baclofen

combined with anti-PD-L1 therapy synergistically reduced tumor burden, achieved complete tumor eradication in 66% of mice, and significantly extended survival.

Conclusion: Enhancing peritumoral GABAergic inhibition stabilizes cortical networks, limits glioma progression, and potentiates immunotherapy, representing a promising adjuvant strategy for glioma treatment.

Disclosure: Nothing to disclose.

OPR-016 | Vascular complications in IDH-wildtype glioblastoma: Incidence, timing, and clinical impact

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Background and Aims: Patients with high-grade glioma (HGG) are at elevated risk for venous thromboembolism (VTE) and cerebrovascular events; however, the frequency, timing, and underlying mechanisms remain insufficiently characterized. This study aimed to determine the incidence and potential risk factors for vascular complications in a well-defined glioblastoma cohort.

Methods: We retrospectively analyzed 337 consecutive patients with histopathologically confirmed IDH-wildtype glioblastoma (WHO 2021) treated at the neuro-oncological outpatient clinic of University Hospital Graz. Demographic data, treatment characteristics, and vascular events were systematically collected using the electronic hospital documentation system, covering all acute vascular events within the regional catchment area.

Results: Vascular complications occurred in 25.2% of patients ($n=85$), including VTE (18.4%), ischemic stroke (4.2%), intracerebral hemorrhage (3.6%), cerebral sinus venous thrombosis (0.6%), and myocardial infarction (0.6%). Most events occurred early: 31.8% within 1 month and 74.1% within 6 months of diagnosis; 22.4% occurred during first-line therapy. Vascular events were associated with a case fatality rate of 12.9%. Complications were treated according to current guidelines; VTE was mainly managed with low-molecular-weight heparins and, less frequently, with direct oral anticoagulants, without major intracerebral bleeding. Overall survival did not differ significantly between patients with and without vascular events, and no analyzed risk factor was significantly associated with outcome. TP53 mutations were less frequent in patients with vascular events.

Conclusion: Incidence rates were consistent with previous retrospective studies. The temporal clustering of events in the postoperative period and during radiochemotherapy confirms known high-risk phases. Vascular complications may affect treatment continuity and increase case fatality rates, warranting prospective risk stratification studies.

Disclosure: Nothing to disclose.

OPR-017 | Comparative effectiveness of IV pulses cyclophosphamide versus siponimod on disability accrual in secondary progressive multiple sclerosis

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Background and Aims: Effectiveness of available disease-modifying treatments (DMTs) on disability progression in multiple sclerosis (MS) is marginal, hence therapeutic options for secondary progressive (SP) MS are limited to Siponimod (SIP) in Europe. Cyclophosphamide (CY), a small highly immunosuppressive molecule with high blood-brain-barrier penetration, is a potential optimal treatment, although yet never compared to SIP.

Methods: SPMS patients recruited at the Tuscany region MS Referral Centre of the Careggi University-Hospital (Florence, Italy) treated with intravenous CY-pulses according to a standardized protocol were included as index-cases and compared to SIP-treated controls from the same MS Centre or extracted from the Italian Multiple Sclerosis Register. To correct for unbalances between the two groups, an overlap weighting approach based on propensity scores was used, censoring results at month 36. The primary endpoint was disability Worsening-Free Survival (WFS). Key secondary endpoints were Progression-Free Survival (PFS), No-Evidence of Disease Activity-2 (NEDA-2) and Time To First-Relapse (TTFR). Safety outcomes included mortality for any cause and incidence of neoplasms. Disability outcomes were assessed through Cox regression models.

Results: 123 CY-treated and 81 SIP-treated SPMS patients were included thus far. WFS was remarkably higher in the CY-treated compared to the SIP-treated cohort (Survival Probability Causal Effect, SPCE, close to significance, Figure 1); NEDA-2 (Figure 2) and disability improvement were significantly more frequently obtained and TTFR delayed (Figure 3) in the CY-cohort. No difference was observed in terms of safety.

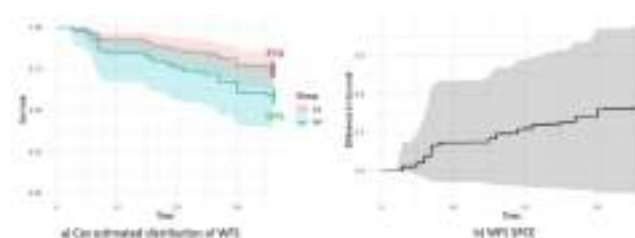


FIGURE 1 (a) Cox estimated distribution of WFS following treatment with Cyclophosphamide (CY; red) or Siponimod (SIP; green). WFS at Year 3 was 75% in CY and 60% in SIP group. (b) WFS SPCE was positive and close to significance.

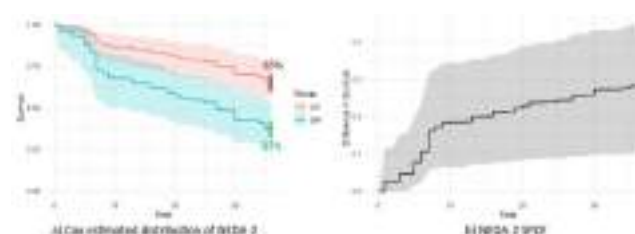


FIGURE 2 (a) Cox estimated distribution of NEDA-2 following treatment with Cyclophosphamide (CY; red) or Siponimod (SIP; green). NEDA-2 at Year 3 was 65% in CY and 37% in SIP group. (b) NEDA-2 SPCE was positive and statistically significant

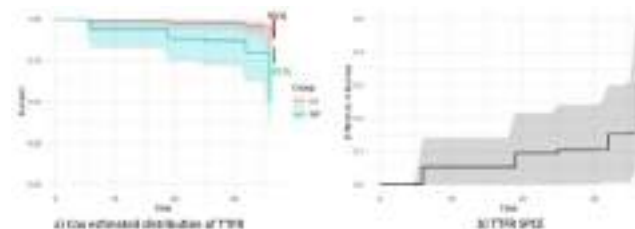


FIGURE 3 (a) Cox estimated distribution of TTFR following treatment with Cyclophosphamide (CY; red) or Siponimod (SIP; green). TTFR at Year 3 was 96% in CY and 81% in SIP group. (b) TTFR SPCE was positive and statistically significant.

Conclusion: The preliminary analysis of the first three years of this study in SPMS suggests a superior effectiveness of CY vs. SIP on disability progression and NEDA-2.

Disclosure: Nothing to disclose.

speaker/consulting honoraria and/or travel grants from Sanofi, Roche, Janssen, Merck, Novartis, Alexion and Amgen, and research grants from Roche, Sanofi, and Brugmann Foundation. S.E.S received grants and/or travel and/or consultancy fees from Biogen, Merck Serono, Sanofi, Roche, Novartis, Teva Pharma, and Bayer Schering. E.L. received travel and consultancy fees from Sanofi, Biogen, Merck Serono and Roche and is supported by National Fund for Scientific Research (FRS-FNRS), Belgium and received research funding from the Belgian Charcot Foundation, the King Baudouin Foundation as well as from the MS Belgian National League. V.V.P. received consulting honoraria and research grants from Biogen, Merck, Sanofi, BMS, Novartis, Janssen, Almirall, Roche, Alexion, Medtronic, and Teva Pharmaceuticals. P.M. research activity is supported by the Fondation Charcot Stichting Research Fund 2023, the Fund for Scientific Research (F.R.S, FNRS; grant #40,008,331), Cliniques universitaires Saint-Luc “Fonds de Recherche Clinique” and Biogen and received consulting honoraria from Sanofi, Biogen and Merck. F.G. and F.L. have nothing to disclose.

OPR-019 | Abnormal B-cell exosome pro- and anti-apoptotic cargo in multiple sclerosis: Implication in progressive disease biology

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Background and Aims: Compartmentalized central nervous system (CNS) inflammation involving B cells is implicated in gray matter injury and disease progression in multiple sclerosis (MS). Products secreted by B cells of MS patients can kill oligodendrocytes and neurons, a cytotoxicity conferred by their exosome-enriched extracellular vesicle (Ex-En) fraction.

Methods: To explore the potential molecular mediators of this cytotoxicity, we profiled proteomic and transcriptomic cargo of Ex-En isolated from B cells of treatment-naïve MS patients and matched healthy controls.

Results: MS B cell-derived Ex-En appeared enriched in cell-death associated proteins (including fibrinogen, complement C9, APP, and SPARC) and deficient in cell-survival associated proteins (such as galectin-3). Abnormal enrichment for cell-death proteins was supported by Gene Set Enrichment Analysis. Protein pathway analysis revealed densely connected pro-death modules in the MS B cell-derived EVs, contrasting with homeostatic signatures in controls. Transcriptomic analysis further revealed that Ex-En of MS B cells appeared to carry reduced levels of miRNAs (miR-182, miR-212, miR-1270) known to inhibit apoptosis.

Conclusion: Our findings indicate that B cell-derived exosomes of MS patients, previously shown to impair neuronal and glial survival, harbor an abnormal cytotoxic molecular profile that may contribute to CNS-compartmentalized injury and progressive MS biology.

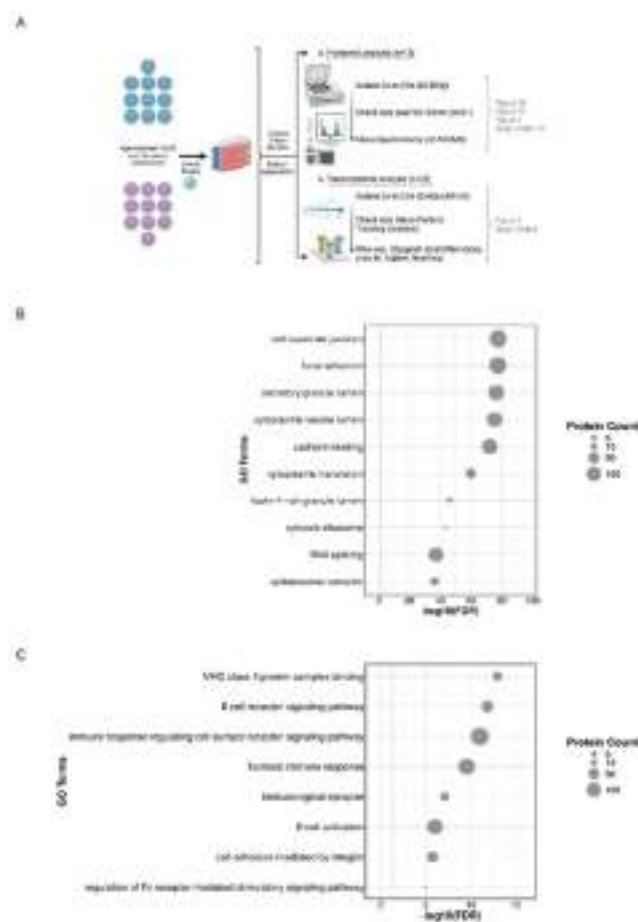


FIGURE 1 Proteomic Gene Ontology analysis of normal human B cell-derived exosomes identifies enrichment of vesicular elements, immune pathways and complexes implicated in inter-cellular communication.

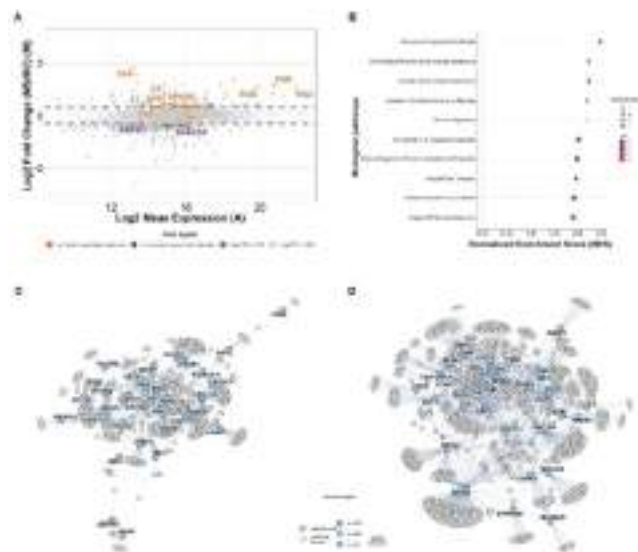


FIGURE 2 Abnormal enrichment of cell-death pathways in B cell-derived exosomes of MS patients.

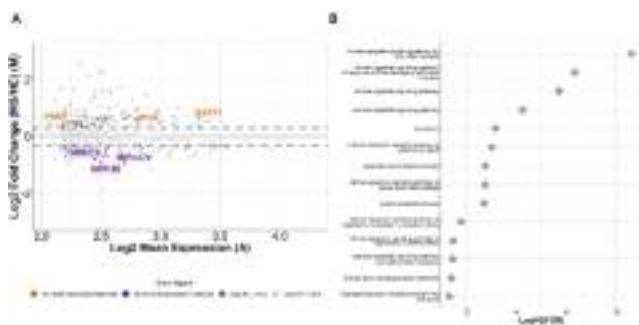


FIGURE 3 Deficiency of anti-apoptotic miR-182, miR-212, and miR-1270 in B cell-derived exosomes of MS patients.

Disclosure: Nothing to disclose.

OPR-020 | Disentangling age-related and disease-specific upper cervical cord atrophy in multiple sclerosis

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Background and Aims: This study aimed to model lifespan trajectories of upper cervical cord atrophy in healthy controls (HC) and identify multiple sclerosis (MS)-specific patterns of cord atrophy in a large multicenter cohort.

Methods: We analyzed 3D T1-weighted brain MRIs and clinical data from 480 HC and 1295 MS patients. Mean upper cervical cord cross-sectional area (MUCCA) was measured between C1 and C2/3 intervertebral disk using the active surface method and normalized for head size (nMUCCA). nMUCCA was modelled across the lifespan in HC using linear regression accounting for age, age², sex, scanner and interactions, and thereafter, applied to the MS cohort to derive nMUCCA Z-scores. Analyses were stratified by sex and age at onset (pediatric-onset [POMS], adult-onset [AOMS], late onset [LOMS]).

Results: In HC, nMUCCA showed a non-linear relationship with age, increasing until the late 30s ($p \leq 0.033$) and declining after age 50 ($p \leq 0.008$). In MS, nMUCCA Z-scores decreased up to late 40s ($p \leq 0.001$). Sex did not significantly influence atrophy patterns in either group. POMS patients exhibited significantly lower Z-scores compared to AOMS and LOMS ($p < 0.001$), although age-related MS-driven decline did not differ among onset groups. Lower nMUCCA Z-scores were

associated with longer disease duration and greater clinical disability (all $p < 0.001$).

Conclusion: In MS, upper cervical cord atrophy exceeded effects of normal ageing, especially during midlife, independent of the effect of sex and age at disease onset, and was associated with longer disease duration and disability. Upper cord atrophy holds promise as a sensitive biomarker for tracking MS progression beyond ageing effects.

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Background and Aims: Prognosticating disease progression is difficult in MS. We investigated how serum neurofilament light chain (NfL) and glial fibrillary acidic protein levels (GFAP) can support assessment of progression-risk and reflect protective effects of disease-modifying therapy (DMT) against long-term 'progression independent of relapse activity' (PIRA).

Methods: Patients from two large real-world MS cohorts, the Swiss MS Cohort (SMSC) and the EPIC study (University of California, San Francisco), were included. Clinical data and

biomarker Z scores were collected from 18629 visits at 6/12-month intervals.

Results: SMSC and EPIC consisted of 1709 and 620 patients, yielding 13375 and 5254 samples (Table 1). Elevated NfL (Z score >1.5; 93rd percentile) was associated with a 2-fold risk of relapse within the next year in SMSC (OR 2.00, 95% CI 1.63–2.45; $p < 0.0001$) and a 1.69-fold risk in EPIC (1.69, 1.24–2.32; $p = 0.00094$, Figure 1), while GFAP was not. Elevated GFAP (Z score >1.0; 84th percentile) prognosticated a 45% higher risk of PIRA at next visit in SMSC (1.45, 1.21–1.75; $p < 0.0001$). This was confirmed in EPIC (1.36, 1.07–1.71; $p = 0.011$, Figure 1). In SMSC, every yearly GFAP Z score unit reduction during the first 2 years of fingolimod or B-cell depleting therapy was associated with a 54% (HR 0.46, 95% CI 0.26–0.84; $p = 0.012$) and 67% (0.33, 0.18–0.61; $p = 0.00036$) lower risk of PIRA (Figure 2), respectively. No such associations were observed for NfL Z scores.

Conclusion: NfL and GFAP are powerful complementary tools for prognostication of relapse activity and PIRA in MS. DMT-associated GFAP-reduction predicted lower PIRA-risk. Together, these biomarkers may improve prognostic accuracy and personalized treatment.

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TABLE 1 Characteristics of included patients.

	SMSC (1709 patients)	EPIC (620 patients)
Baseline		
Sex, female	1126 (66.0%)	432 (69.7%)
Age, years	46.6 [32.1–50.1]	42.0 [35.0–50.0]
MS subtype		
RMS	1539 (90.1%)	536 (86.8%)
SPMS	170 (9.9%)	82 (13.2%)
Disease duration, years	6.3 [2.0–13.6]	6.3 [2.3–12.8]
EDSS	2.0 [1.5–3.0]	1.5 [1.0–3.0]
DMT category		
Untreated	456 (26.7%)	245 (39.5%)
Platform	174 (10.2%)	156 (25.2%)
Oral	524 (30.7%)	4 (0.6%)
High-efficacy	555 (32.5%)	16 (2.6%)
NfL Z score	0.6 [−0.3–1.8]	1.0 [−0.2–1.6]
GFAP Z score	0.2 [−0.7–1.0]	0.5 [−0.4–1.2]
Follow-up		
Follow-up, years	6.9 [2.5–10.7]	18.1 [9.8–14.0]
≥1 relapse during follow-up	412 (24.1%)	523 (84.3%)
≥1 PIRA event during follow-up	512 (30.0%)	267 (43.1%)
Next visit		
EDSS	2.5 [1.5–4.0]	2.5 [2.0–4.0]
DMT category		
Untreated	283 (16.6%)	161 (26.1%)
Platform	62 (3.7%)	150 (24.2%)
Oral	560 (32.8%)	85 (13.7%)
High-efficacy	803 (47.0%)	124 (20.0%)
Samples	13375	5254
NfL Z score	0.3 [−0.6–1.1]	0.4 [−0.4–1.2]
GFAP Z score	0.2 [−0.7–0.9]	0.4 [−0.2–1.3]
Days between sample timepoints	346.0 [190.0–375.0]	385.0 [355.0–518.0]

Data are n (%) or median [IQR].

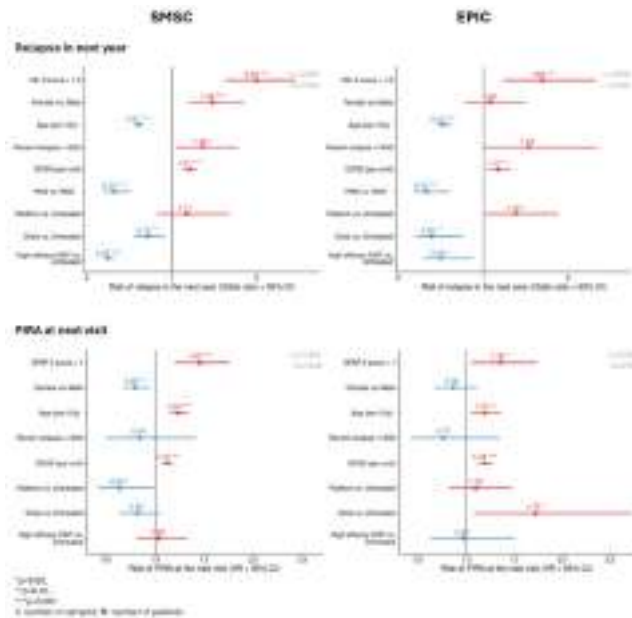


FIGURE 1 Serum NfL prognosticated subsequent relapses (logistic mixed models, 2.0- and 1.69-fold risk), while serum GFAP was independently associated with progression independent of relapse activity (PIRA; 1.45- and 1.36-fold risk) in both cohorts.

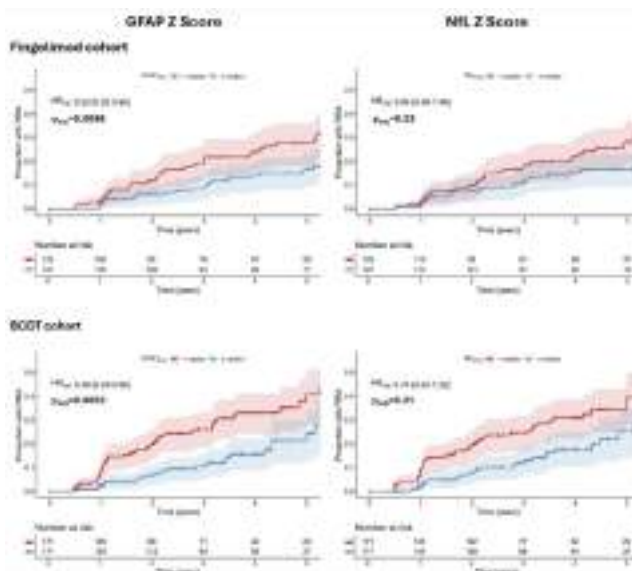


FIGURE 2 Patients with GFAP Z score reduction after DMT start had lower risk of future PIRA than those without. Kaplan-Meier curves for subsequent PIRA stratified by biomarker slope within 2 years of DMT start. HR/p values from multivariable Cox models.

OPR-022 | Genetic and early-life vulnerability in functional neurological disorder: A large-scale retrospective cohort study

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Background and Aims: Contemporary models of functional neurological disorder (FND) emphasise biological vulnerability affecting motor, sensory, and integrative processing as a predisposing factor. While early-life influences were traditionally viewed within psychological frameworks, little is known about early-life biological vulnerabilities that may predispose to FND or shape its clinical presentation.

Methods: Using TriNetX, a large electronic health records dataset, we assessed rates of genetic, congenital, perinatal, and neurodevelopmental diagnoses, selected to capture a broad range of early-life conditions, including those that may lead to sensory and motor system vulnerability. Comparisons were performed between 188,868 individuals with FND and demographically matched migraine and depression cohorts, and between matched cohorts of 42,015 individuals with motor FND and functional seizures.

Results: Compared with migraine and depression, FND was associated with increased odds of heritable connective tissue disorders, heritable neuromuscular disorders, and congenital malformations, including those of the musculoskeletal system and sensory organs (Table 1). Apart from attention-deficit/hyperactivity disorder, which was more prevalent in the depression

TABLE 1 Genetic, congenital, perinatal, and neurodevelopmental factors in functional neurological disorder (FND) compared to matched cohorts with migraine and depression.

	FND		Epilepsy		Depression		
	N (%)	R (%)	OR (95% CI)	p	N (%)	OR (95% CI)	p
GENETIC/CONGENITAL FACTORS							
Epilepsy	1,618 (0.8)	3.22 (2.15-5.03)	<0.001		465 (1.1)	1.58 (1.22-2.05)	<0.001
Hypertrophic cardiomyopathy	964 (0.5)	2.15 (1.41-3.38)	<0.001		342 (0.8)	2.52 (1.48-4.30)	<0.001
Hereditary motor and sensory neuropathy	312 (0.2)	2.84 (1.14-7.01)	<0.001		102 (0.2)	2.58 (1.18-5.25)	<0.001
Primary disorders of muscles	380 (0.2)	2.84 (1.41-5.68)	<0.001		127 (0.3)	2.44 (1.24-4.86)	<0.001
Congenital malformations and deformations of the musculoskeletal system	1,152 (0.6)	1.76 (1.11-2.80)	<0.001		354 (0.8)	1.75 (1.47-2.07)	<0.001
Congenital malformations of the nervous system	2,430 (1.3)	2.44 (1.37-4.35)	<0.001		665 (1.6)	1.54 (1.28-1.85)	<0.001
Congenital malformations (any)	16,414 (8.4)	1.51 (1.43-1.59)	<0.001		11,188 (2.7)	1.72 (1.67-1.78)	<0.001
Congenital ear malformations causing impairment of hearing	86 (0.0)	1.86 (1.18-2.93)	0.006		42 (0.1)	1.57 (1.07-2.31)	<0.001
Congenital myasthenia	11 (0.0)	2.88 (1.41-6.08)	<0.001		34 (0.1)	3.37 (1.81-6.68)	<0.001
Chromosomal abnormalities	670 (0.3)	2.85 (1.50-5.45)	<0.001		301 (0.7)	2.47 (1.38-4.45)	<0.001
PERINATAL FACTORS							
Maternal and perinatal factors affecting the newborn	287 (0.1)	1.34 (1.15-1.55)	0.011		304 (0.7)	1.39 (1.24-1.55)	0.010
Disorders of newborn related to slow fetal growth and fetal malnutrition	151 (0.0)	1.86 (1.03-3.35)	0.041		214 (0.5)	1.58 (1.22-2.05)	<0.001
Disorders of newborn related to short gestation and low birth weight	480 (0.2)	1.76 (1.48-2.07)	<0.001		305 (0.7)	1.66 (1.47-1.88)	<0.001
Disorders of newborn related to long gestation and high birth weight	71 (0.0)	1.86 (1.03-3.35)	0.041		165 (0.4)	1.57 (1.22-2.05)	<0.001
Respiratory distress of newborn	34 (0.0)	2.88 (1.41-6.08)	<0.001		34 (0.1)	3.37 (1.81-6.68)	<0.001
NEURODEVELOPMENTAL FACTORS							
Intellectual disabilities	1,124 (0.6)	5.41 (4.55-6.41)	<0.001		321 (0.8)	2.43 (2.18-2.69)	<0.001
Communication disorders	1,707 (0.9)	1.86 (1.74-2.00)	<0.001		1,506 (3.6)	1.58 (1.52-1.65)	<0.001
Autism spectrum disorder	2,208 (1.2)	1.55 (1.48-1.62)	<0.001		1,506 (3.6)	1.54 (1.48-1.60)	<0.001
Attention-deficit/hyperactivity disorder	12,886 (6.8)	1.86 (1.74-2.00)	<0.001		14,031 (3.4)	1.58 (1.52-1.65)	<0.001
Specific learning disorders	1,124 (0.6)	1.86 (1.74-2.00)	<0.001		1,506 (3.6)	1.58 (1.52-1.65)	<0.001
Motor disorders	481 (0.2)	2.88 (1.41-6.08)	<0.001		34 (0.1)	3.37 (1.81-6.68)	<0.001

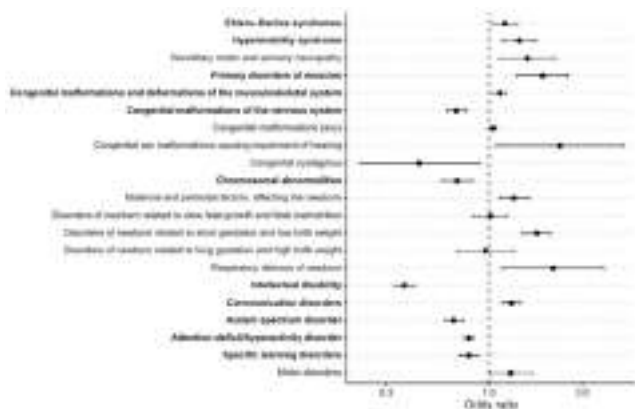


FIGURE 1 Congenital and developmental factors in functional motor disorder versus functional seizures. OR >1 indicate higher odds in the motor subtype, whereas OR <1 indicate higher odds in the seizure subtype.

cohort, neurodevelopmental disorders were consistently enriched in FND, alongside increased rates of perinatal adversity related to short gestation, low birth weight, and newborn respiratory distress. Motor FND showed stronger associations with connective tissue, neuromuscular, and musculoskeletal conditions, whereas functional seizures were more enriched for chromosomal abnormalities and neurodevelopmental disorders (Figure 1).

Conclusion: Genetic and early-life biological factors may predispose to functional symptoms. Distinct associations were observed for functional motor and seizure presentations. Our findings support a more biologically informed understanding of FND.

Disclosure: E.I. has no disclosures. R.B. is supported by the Slovenian Research and Innovation Agency as a member of the research programme Medical Physics (P1-0389). T.A.P. does medical expert reporting in personal injury and clinical negligence cases, including in cases of FND. He has received consultancy fees from Arialys Therapeutics. T.R.N. does medical expert reporting in personal injury and clinical negligence cases, including in cases of FND. He has received financial support for lectures from the FND Society (FNDS). He receives royalties from CRC Press for The Pocket Prescriber textbook series. He also has received grant funding, including for studies related to FND, from the UK National Institute for Health and Care Research (NIHR) and the Medical Research Council (MRC). He is co-chair of the FNDS patient liaison committee and on the medical advisory boards of the charities FND Hope UK and FND Action, and a trustee of FND Action. M.J.E. does medical expert reporting in personal injury and clinical negligence cases, has shares in Brain & Mind, which provides neuropsychiatric and neurological rehabilitation in the independent medical sector, has received financial support for lectures from the International Parkinson's and Movement Disorders Society and the FND Society (FNDS), receives royalties from Oxford University Press for his book, The Oxford Specialist Handbook of Parkinson's Disease and Other Movement Disorder, has received honoraria for medical advice to Teva Pharmaceuticals and educational events, receives grant funding from the National Institute for Health and Care Research (NIHR), is the deputy editor of the European Journal of Neurology, and is on the medical advisory boards of the charities FND Hope and the British Association of Performing Arts Medicine.

OPR-023 | Risk of epilepsy in people with adult-onset hydrocephalus: Insights from the UK Biobank

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Background and Aims: Epileptic seizures frequently occur in Alzheimer's disease (AD) and other neurodegenerative dementia; conversely, little is known about epilepsy risk in idiopathic adult-onset hydrocephalus.

Methods: We analyzed data from the UK Biobank cohort. Diagnoses of "hydrocephalus" and "epilepsy" were identified through ICD-10-coded health records, excluding congenital and secondary cases. The association between these two diseases was investigated using logistic regression (adjusted for age and sex); subsequently, we performed a nested case-control study to evaluate the hazard ratio (HR) for incident epilepsy in people with adult-onset hydrocephalus using Cox proportional hazards models with a dedicated sensitivity analysis including different sets of covariates such as demographic, lifestyle, vascular, and genetic factors.

Results: Our cohort included 483,790 controls, 5028 individuals with epilepsy, and 320 with adult-onset hydrocephalus. Hydrocephalus was strongly associated with epilepsy (OR: 9.6, 95% CI: 6.4–13.8, $p < 0.001$). Adjusted Cox models demonstrated a markedly increased risk of incident epilepsy in people with adult-onset hydrocephalus, with HR values ranging from 14.62 (95% CI: 7.91–27.00, $p < 0.001$) to 23.80 (95% CI: 12.87–44.03, $p < 0.001$) across models, after adjusting for multiple covariates. Results were also consistent after excluding people with comorbid neurodegenerative dementias such as Alzheimer's disease.

Conclusion: We demonstrated a markedly increased risk of epilepsy in adult-onset hydrocephalus, independent of vascular and neurodegenerative comorbidities and not attributable to shunt-related complications. These findings underscore the importance of increased diagnostic vigilance, as seizures may be under-recognized in this population, and support further research to clarify mechanisms and optimize management strategies.

Disclosure: Nothing to disclose.

OPR-024 | Mental health outcomes among refugees and internally displaced people in Africa: A systematic review

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Background and Aims: Refugees and internally displaced people in Africa are exposed to conflict, violence, and displacement-related stressors that substantially increase the risk of mental health disorders. However, a comprehensive continent-wide synthesis of mental health outcomes in these populations is lacking. This study aimed to systematically review the prevalence, risk factors, and interventions related to mental health among displaced populations in Africa.

Methods: This systematic review followed Preferred Reporting Items for Systematic Reviews and Meta-Analyses 2020 guidelines and was registered with the International Prospective Register of Systematic Reviews. Five databases and grey literature were searched up to January 20, 2025. Eligible studies assessed mental health outcomes among refugees, internally displaced persons, or asylum seekers in African countries. Risk of bias was evaluated using Joanna Briggs Institute tools and the revised Cochrane Risk of Bias tool.

Results: Seventy-eight studies from over 20 African countries were included. Depression, post-traumatic stress disorder, anxiety, and general psychological distress were the most frequently reported conditions, with prevalence estimates ranging from 6.8 percent to over 80 percent. Common risk factors included exposure to violence, gender-based violence, prolonged displacement, and socioeconomic hardship. Protective factors included social support, community cohesion, and access to basic services. Intervention studies were limited but suggested potential benefits of group therapy, task-shifting approaches, and culturally adapted psychosocial interventions.

Conclusion: Displaced populations in Africa experience a high burden of mental health disorders, while access to evidence-based interventions remains limited. Strengthening culturally appropriate mental health services and integrating them into humanitarian and national health systems is urgently needed.

Disclosure: Nothing to disclose.

OPR-025 | Changing incidence patterns for multiple sclerosis in a large population-based study in the province of Palermo, southern Italy

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Background and Aims: The global epidemiology of multiple sclerosis (MS) is changing over time and the understanding of its epidemiological features in low-prevalence areas is crucial for tailoring healthcare strategies and optimizing therapeutic interventions (1).

Methods: We collected demographic and clinical information of people with MS (pwMS) starting from 01st January 2000 to 31st December 2024 living in the Province of Palermo, southern Italy (pop. 1,268,000 inhabitants). The observational time was divided into five-year intervals. We calculated crude and sex and age-adjusted incidence rates in the analysed time frame. MS prevalence was estimated on the prevalence day, December 31st, 2024.

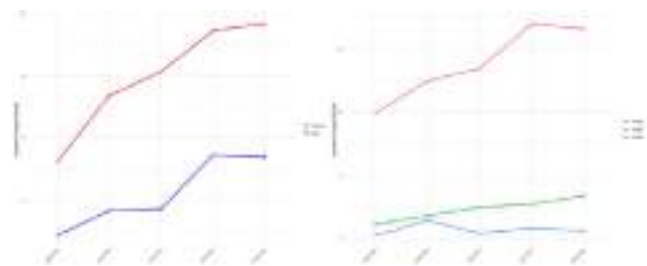


FIGURE 1 Crude and age and sex adjusted incidence rates for MS in the Province of Palermo

Results: A total of 2423 pwMS were diagnosed as of 31st December 2024, giving a prevalence of 201/100.000 person (95% CIs: 193–209). The annual incidence rate increased from 4.1/100.000 person/year to 7.6/100.000 person/year over time ($p=0.007$; Figure 1). Incidence rate was higher in women, reaching the peak of 9.7/100.000 person/year after 2020 (Figure 2A). While the incidence of early-onset MS was almost similar across eras ($p=0.9$), we observed an increasing trend of incidence rate for adult-onset ($p=0.011$) and late-onset MS ($p=0.001$; Figure 2B).

Conclusion: Incidence and prevalence of MS in Palermo Province are higher than previously reported, making this province a high-risk area. Our population-based study confirms increasing incidence rates of late-onset MS over time, confirming the impact that cases with late-onset MS are achieving in the clinical and pharmaco-economical management of this disease.

Disclosure: Nothing to disclose.

OPR-026 | Drivers of rising prevalence in major motor neurodegenerative diseases: Temporal trends in Sweden and France (2003–2021)

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Background and Aims: The prevalence of Parkinson's disease (PD), multiple sclerosis (MS), and motor neuron diseases (MND) is rising globally. However, it is unclear to what degree this reflects increased incidence versus improved survival after diagnosis.

Methods: We performed two nationwide, population-based cohort studies including all individuals in Sweden (2001–2016) and France (2009–2022). Pooled mixed-effects models determined temporal trends in prevalence, crude and age-sex-standardized incidence, and life expectancy at diagnosis.

Results: Prevalence of PD, MS, and MND increased significantly between 2003 and 2021. While crude incidence of PD and MS remained stable (PD: IRR=0.998; MS: IRR=0.992), standardized incidence decreased for PD (IRR=0.986) but not MS (IRR=0.995). For MND, both crude and standardized incidence increased (IRR=1.018 and 1.008, respectively). Life expectancy at diagnosis increased for PD between 2003 and 2013 (+0.95 months/year) then decreased 2013–2022 (−1.20 months/year), while increasing throughout for MS (+2.35 months/year) and MND (+0.34 months/year).

OPR-027 | Global SPG11 genotype-phenotype landscape: Integrated analysis of 560 patients

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Background and Aims: SPG11 is the most frequent cause of autosomal recessive hereditary spastic paraplegia (HSP). However, its clinical spectrum and genotype-phenotype correlations remain incompletely defined.

Methods: We assembled a multicenter international cohort of 75 genetically confirmed SPG11 patients with detailed epidemiological and clinical data and performed a literature review of published SPG11 cases. We analyzed clinical, radiological and genetic features to investigate genotype-phenotype associations.

Results: The merged dataset comprised 560 SPG11 patients (75 in-house; 485 literature) with >200 unique variants, without mutational hotspot. Mean age at onset was ~16 years. In addition to spastic paraparesis, the most frequent feature was cognitive impairment/intellectual disability (91%), with the vast majority presenting with complicated HSP (96%), while only 4% exhibited

TABLE 1 Epidemiological, clinical and radiological characteristics of the international SPG11 cohort compared with the combined dataset (75 in-house patients and 485 cases extracted from the literature). Values are presented as percentages.

	INTERNATIONAL COHORT (n=75)	COMBINED COHORT (n=560)
EPIDEMIOLOGY		
Sex (Male/Female)	~ 1:1 (40/35)	~ 1:1
Mean age of onset	16.8 years (Range 2-58)	15.9 years (Range 1-78)
Consanguinity	31%	41% (172/416)
Positive Family History	37%	29% (119/416)
CLINICAL FEATURES		
Spastic paraparesis/pyramidal signs	100%	97%
Cognitive impairment	80%	91%
Dysarthria	56%	43%
Cerebellar signs	47%	30%
Peripheral neuropathy	15%	17%
Distal amyotrophy (ALS mimics)	12%	18%
Parkinsonism	16%	10%
Dystonia	7%	4%
Tremor	9%	12%
Epilepsy	0%	5%
Optic atrophy	5%	5%
Psychiatric or behavioral symptoms	5%	6%
Pure HSP	10%	4%
BRAIN MRI		
Abnormal MRI	80% (37/44)	95% (401/422)
ICC	77% (34/44)	89% (378/422)
WMA	61% (27/44)	63% (264/422)
Cortical atrophy	23% (10/44)	31% (130/422)
Cerebellar Atrophy	7% (3/44)	9% (38/422)

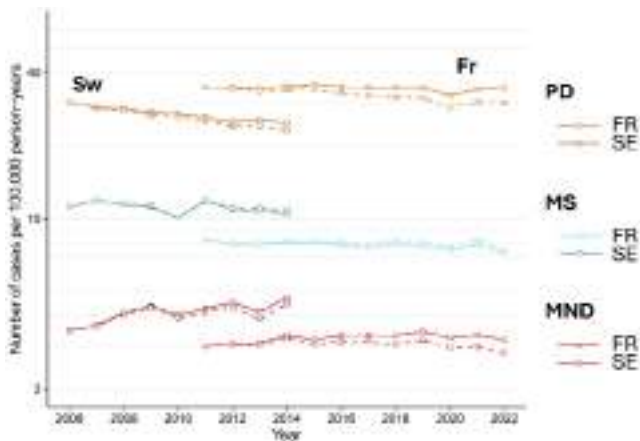


FIGURE 1 Country-specific of secular trends in incidence rates of Amyotrophic Lateral Sclerosis, Multiple Sclerosis and Parkinson's disease assuming a linear relationship with time in France and Sweden (2003–2022).

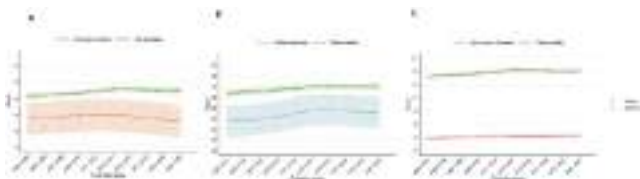


FIGURE 2 Secular trends in life expectancy at diagnosis of patients with (A) PD, (B) MS, and (C) MND compared with the general population.

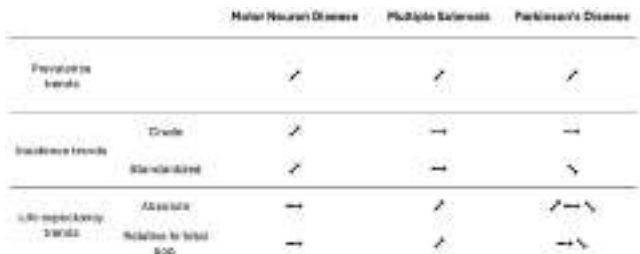


FIGURE 3 Varying dynamics explaining increases in prevalence across diseases.

Conclusion: These findings reveal fundamentally different prevalence drivers with critical implications for disease etiology and healthcare planning. MS prevalence is survival-driven through therapeutic advances, with no evidence of changing population-level risk factors. MND shows true incidence increases, suggesting emerging or intensifying environmental or lifestyle risk factors. PD demonstrates declining age-adjusted incidence, with prevalence growth historically driven by improved survival. These distinct mechanisms demand disease-specific strategies.

Disclosure: Nothing to disclose.

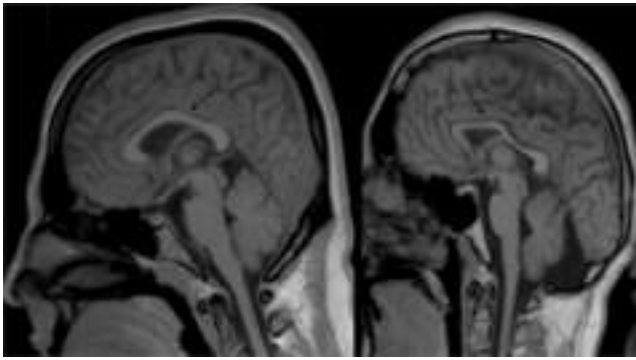


FIGURE 1 Sagittal T1-weighted brain MRI from two SPG11 patients showing marked thinning of the corpus callosum (TCC), as representative examples of the most common radiological finding in SPG11 (arrows).

a pure HSP phenotype. Distinct SPG11-associated phenotypic subtypes overlapping with other neurodegenerative diseases, included a predominately ataxic phenotype (30%), a parkinsonism/dystonia subtype (10%) and an amyotrophic lateral sclerosis (ALS)-like phenotype (18%). Additional manifestations included dysarthria (43%), neuropathy (17%), and epilepsy, optic atrophy and psychiatric symptoms (~5% each). Brain MRI was abnormal in 95%, most commonly showing thin corpus callosum (89%), white-matter hyperintensities (63%) and cortical atrophy (31%). Ten recurrent variants were identified; some with specific clinical patterns. The most frequent worldwide, c.733_734del ($n=50$), was associated with increased cerebellar/extrapyr- amidal manifestations; whereas, the recurrent missense variant c.5381T>C was linked to strikingly later-onset (mean 52 years) and milder phenotype.

Conclusion: This integrated analysis provides the largest SPG11 genotype-phenotype dataset to date, refining the SPG11 landscape. Despite extensive allelic diversity, recurrent variants are associated with distinct clinical features, potentially facilitating diagnosis and prognosis in SPG11-HSP.

Disclosure: Nothing to disclose.

OPR-028 | Genome sequencing as a diagnostic tool for common hereditary spinocerebellar ataxias with repeat expansions

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Background and Aims: Spinocerebellar ataxias (SCAs) is a group of autosomal dominant neurodegenerative disorders. Several common SCAs are caused by CAG repeat expansions, a type of variant that is a challenge for short read next-generation sequencing. We used genome sequencing (GS) with the application of ExpansionHunter (EH) for CAG repeat detection in SCA1, 2, 3, 6, 7, and 17.

Methods: The study cohort comprised 301 adult ataxia patients. PCR-free GS was performed on an Illumina platform. CAG repeat loci associated with SCA1, 2, 3, 6, 7, and 17 were sized using EH. Expanded CAG alleles of all loci were reviewed, and

orthogonal confirmation was performed using PCR-based fragment length analyses.

Results: EH detected expanded CAG alleles in all SCA loci (Table 1). The presence of all expanded alleles was confirmed orthogonally. 4/5 SCA1 calls had repeat size deviation of 2-18 repeats between methods and one expanded allele was reclassified to “normal expanded” due to repeat interruptions. 4/8 SCA2 and 3/4 SCA6 expanded calls resulted in the same repeat size between methods, while 4/8 SCA2 and 1/4 SCA6 calls deviated by 1–2 CAGs. SCA3, SCA7 and SCA17 calls had repeat size deviation of 7–15, 1–27 and 1–4 repeats between methods, respectively, with the PCR-based method showing higher repeat size.

TABLE 1 The GS results via EH for the different SCA loci and their orthogonal confirmation results. One of the SCA1 calls was reclassified to normal due to the presence of interruptions in the CAG repeats (*).

Disorder	Number of patients identified in the cohort	EH range of expanded alleles	Confirmation of expanded allele	PCR-based range of expanded alleles
SCA1	5	49-53	5/5*	48-71
SCA2	8	38-44	8/8	38-43
SCA3	5	13-20	5/5	67-77
SCA6	4	22-25	4/4	21-25
SCA7	1	45-70	3/3	75-108
SCA17	2	41-69	2/2	42-58

Conclusion: Our data show that EH application for SCA repeat expansion detection can increase the diagnostic utility of GS. Orthogonal confirmation remains relevant for positive EH calls: whilst shorter range CAG expansions show the same or ± 1 CAG size between GS and conventional fragment length analysis, larger expanded alleles require orthogonal method for accurate repeat sizing.

Disclosure: All authors are employees of CENTOGENE GmbH.

OPR-029 | Parkinsonian manifestations in primary mitochondrial diseases: Evidence from a large Italian multicenter cohort

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Background and Aims: Parkinsonism represents a common hypokinetic manifestation in individuals with primary mitochondrial diseases (PMDs) and has been reported in association with both mitochondrial and nuclear DNA variants. Despite this, its phenotypic features, associated comorbidities, and genetic landscape remain insufficiently defined. This study aimed to characterize the clinical and genetic profile of parkinsonism in a large, multicenter PMD cohort through a detailed phenotyping approach.

Methods: Patients were identified from the Italian Mitochondrial Disease Registry (Genomit). Available clinical records were reviewed to collect demographic, genetic, imaging, and comorbidity data.

Results: Sixty-six patients with genetically confirmed PMDs were included, of whom 45 carried nuclear DNA variants and 21 mitochondrial DNA variants. The most frequently implicated genes were POLG ($n=15$), TWNK ($n=10$), OPA1 ($n=9$). Parkinsonism onset occurred at a median age of 62.2 years, notably later than the median age at PMD onset (40.0 years), although in approximately 20% of cases both conditions manifested within 1 year. The most prevalent clinical syndrome was CPEO plus with several other mitochondrial syndromes observed less frequently. DAT-SCAN imaging mostly demonstrated bilateral dopaminergic deficits in over half of cases, along with additional abnormalities such as substantia nigra alterations and asymmetric dopamine uptake.

Conclusion: In PMDs, parkinsonism typically emerges later than the underlying mitochondrial disorder. These findings expand the clinical characterization of parkinsonism in PMDs and support the need for larger, longitudinal studies to better delineate genotype–phenotype relationships and disease evolution.

Disclosure: Nothing to disclose.

clinical and molecular spectrum remains unclear. This study characterizes phenotypic and genotypic variability in multinational patients with Twinkle-related disorders.

Methods: A retrospective cohort study included patients from specialized centers in Italy, France, Germany, Spain, Denmark, Hungary, and the United States, establishing the Twinkle-Related Disorders International Consortium for Trial Readiness (TReDIC). Data from medical records included clinical features, age at onset, disease progression, and genetic testing results. Phenotypes were classified as infantile-onset cerebellar ataxia, parkinsonism, primary mitochondrial myopathy, multisystem involvement, asymptomatic carriers, undetermined, or other. All diagnoses were genetically confirmed, with variants documented. Outcomes included phenotype prevalence, symptom chronology, and mutational patterns.

Results: The study analyzed 189 patients (116 females), mean onset 40.3 years; 70.4% were alive at analysis. Primary mitochondrial myopathy was predominant (85.2%). Common features included progressive external ophthalmoplegia (84.7%), skeletal myopathy (55.6%), hearing loss (17.5%), and psychiatric symptoms (15.3%). At onset, 76.8% had neuromuscular symptoms, 19.6% CNS involvement, and 3.6% multi-organ; after >8 years, proportions shifted to 54.4%, 23.3%, and 23.3%. Seventy-three TWNK variants (16 novel), mostly missense, clustered in critical functional regions.

Conclusion: This multinational cohort clarifies the broad phenotypic spectrum, progression patterns, and clinically relevant mutational hotspots in Twinkle-related disorders. International collaborations like TReDIC are essential to improve understanding and support future clinical trial design in this rare disease context.

Disclosure: Nothing to disclose.

OPR-030 | Clinical and genotypic spectrum of twinkle-related disorders: Insights from a multinational cohort study

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Background and Aims: Twinkle, encoded by the TWNK gene, is a mitochondrial DNA helicase that unwinds DNA during replication, crucial for mitochondrial function. Twinkle-related disorders encompass diverse genetic conditions with mitochondrial dysfunction. Despite several described phenotypes, the full

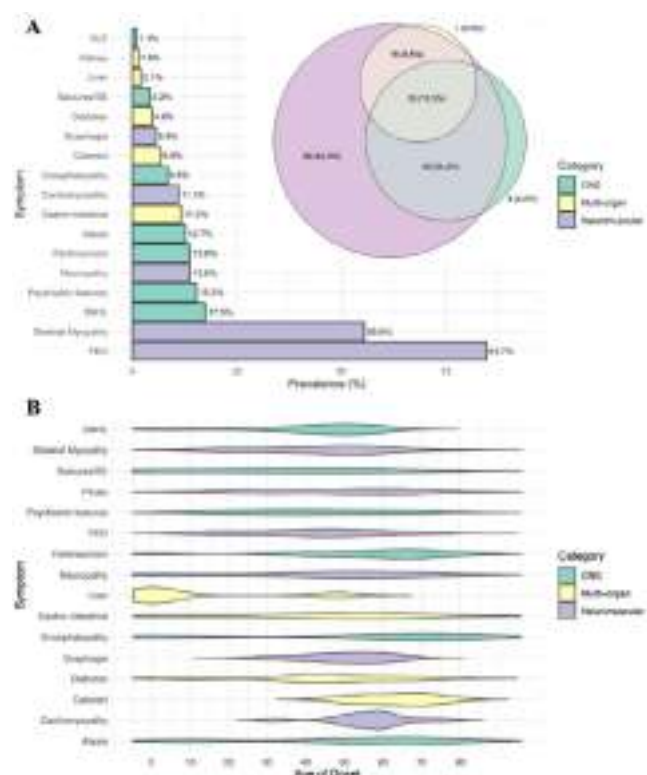


FIGURE 1 Clinical features and years into disease of each individual symptom in patients with Twinkle-related disorder.

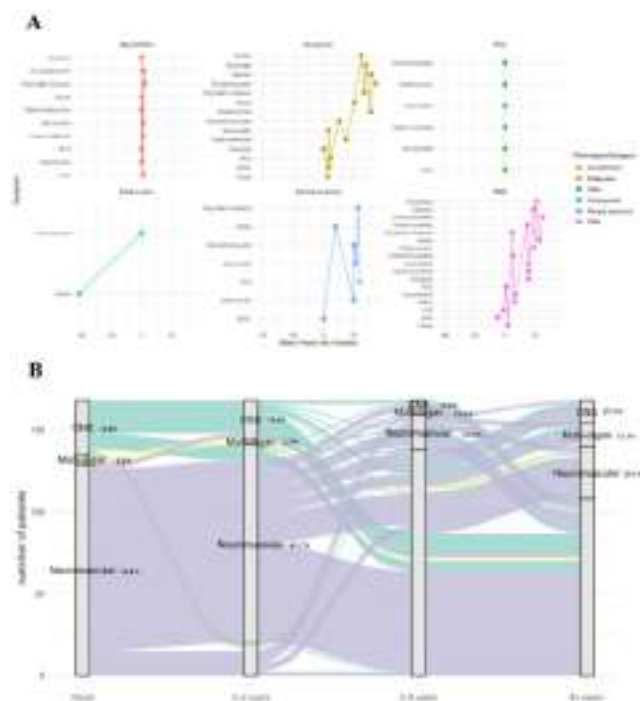


FIGURE 2 Disease progression in patients with Twinkle-related disorder.

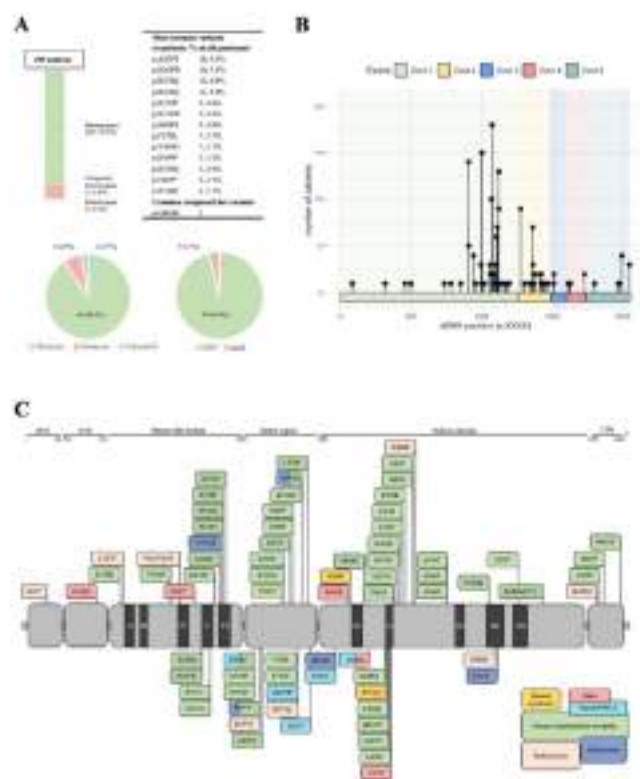


FIGURE 3 Variants across TWNK gene and protein domains.

OPR-031 | CANVAS-associated AAGGG repeat expansions cause cell-specific reduction in RFC1 expression and impaired DNA damage response

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Background and Aims: Cerebellar ataxia, neuropathy, vestibular areflexia syndrome (CANVAS), caused by biallelic AAGGG expansions in RFC1, is increasingly recognised as one of the most common causes of ataxia and sensory neuropathy. Despite previous studies failed to detect a decrease in RFC1 transcript and protein expression, the recessive mode of inheritance and the identification of patients carrying compound heterozygous null variants suggest a loss-of-function mechanism underlying this disease. **Methods:** We assessed the effects of AAGGG expansions on RFC1 expression and function, leveraging reporter assays, brain tissue, patient-derived cell lines, and a Drosophila model.

Moreover, we investigated the association between RFC1 expansions and chemotherapy-induced neuropathy in a cohort of cancer patients who underwent oxaliplatin treatment.

Results: AAGGG expansions cause a length-dependent impairment of transcription efficiency in reporter assays and decrease RFC1 expression in human cerebellum and iPSC-neurons. Patient-derived cell lines showed an increased apoptotic response after exposure to platinum compounds. CRISPR/Cas9-mediated excision of the AAGGG repeat restored RFC1 expression and the DNA damage response. Furthermore, neuronal-specific knockdown of RFC1 in flies led to reduced survival, motor impairment, and increased DNA damage. Finally, individuals carrying RFC1 expansions and exposed to oxaliplatin treatment presented a higher risk to develop a clinically significant neuropathy compared to non-carriers.

Conclusion: Our data suggest that AAGGG expansions cause a cell- and tissue-specific decrease in RFC1 expression with consequent impairment of DNA damage response. The rescue of RFC1 expression and DNA repair after repeat-excision lays the groundwork for future therapeutic avenues.

Disclosure: Nothing to disclose.

Neurorehabilitation

OPR-032 | Auditory cueing modulates motor performance and brain activity in people with Parkinson's disease: A kinematic and fMRI study

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Background and Aims: Cues are specific references that support motor performance by improving speed, timing, and amplitude while reducing variability. This study aimed to investigate the effects of external auditory cueing on bradykinesia-related kinematic deficits and their neural correlates during a hand-tapping task in people with Parkinson's disease (pwPD) and healthy controls (HC).

Methods: Twenty-six pwPD and 20 HC performed a right hand-tapping fMRI task either in a self-paced condition (NOCue) or following an auditory cue (Cue). Hand-tapping kinematics were objectively quantified using an optical-fiber glove.

Results: PwPD showed reduced hand-tapping amplitude and a greater sequence effect on amplitude than HC during NOCue condition. Cueing attenuated the sequence effect on amplitude in pwPD. During the NOCue condition, pwPD showed reduced activity of pallidum relative to HC. No group differences emerged during Cue condition. During Cue relative to NOCue condition, pwPD showed higher activity of pallidum, parietal, temporal, sensorimotor, insular, and cerebellar regions, alongside reduced activation in of paracentral gyrus and cerebellum. Higher activity of pallidum and lower activity of paracentral gyrus correlated with better motor performance and higher positive effects of cueing.

Conclusion: Our results demonstrated that auditory cueing reduces bradykinesia in pwPD by facilitating basal ganglia activity and reducing the need of movement programming.

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Background and Aims: Standard neuropsychological assessments of post-stroke attentional deficits often suffer from limited ecological validity and insufficient sensitivity to complex behavioral patterns. Immersive virtual reality (iVR) combined with eye tracking (ET) offers a high-dimensional characterization of attentional functioning; however, the structural substrates underlying these digital oculomotor biomarkers remain insufficiently understood. This study uses lesion-based connectomics to link ET-derived metrics with focal brain damage.

Methods: Fifty patients with acquired brain injury underwent iVR-based assessments of spatial attention. Random forest classifiers were trained to identify attentional deficits from ET metrics. Structural lesion impact was quantified using the Lesion Quantification Toolkit (LQT), estimating parcelwise gray matter lesion load (Schaefer–Yeo atlas) and white matter disconnection severity (HCP-1065 tractography atlas). Principal component analysis was applied to derive latent oculomotor dimensions. To explore prognostic relevance, longitudinal changes in oculomotor behavior were related to baseline structural disconnectivity.

Results: Classification models achieved high diagnostic performance ($F1=0.88$). Lesion quantification revealed that the principal oculomotor component characterizing spatial neglect—reflecting increased gaze bias and reduced visual exploration—was associated with cumulative damage within dorsal and ventral attention networks, notably involving the right frontal aslant tract and superior longitudinal fasciculi. Ongoing longitudinal analyses aim to determine whether these structural-functional associations predict recovery trajectories.

Conclusion: By integrating iVR-based eye tracking with lesion-informed connectomics, this study provides a novel framework for characterizing attentional deficits and their neural

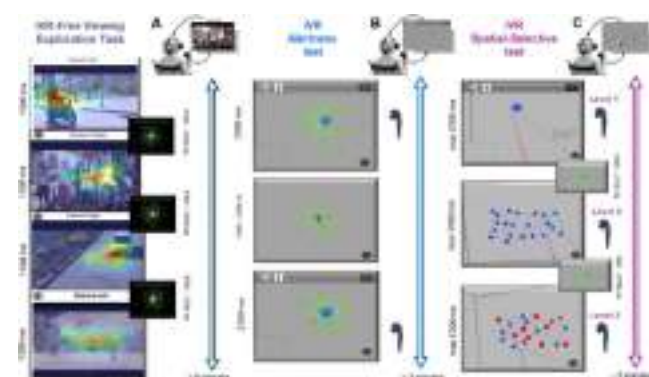


FIGURE 1 iVR tasks. (A) Free viewing: 4 conditions (lateral/bilateral x left/right) with fixation cross. (B) Alertness: target detection. (C) Spatial selective attention: target localization and selection.

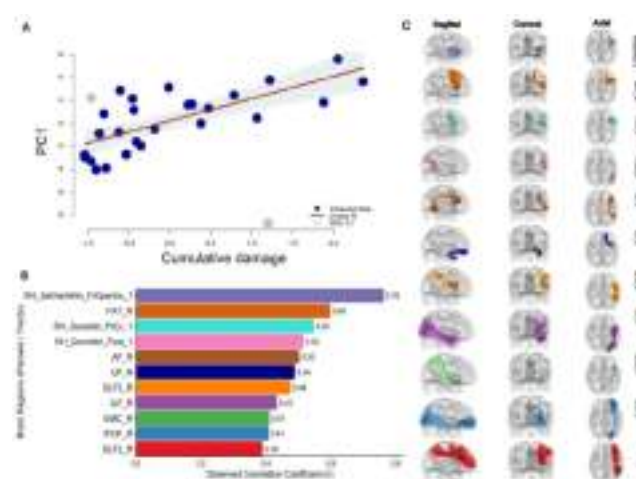


FIGURE 2 Structural correlates of the oculomotor latent factor (PC1). (A) Linear association between cumulative structural damage across the Sal-VAN/DAN and the PC1. (B) Region-wise permutation-based Spearman correlations. (C) Brain renderings

substrates. The findings suggest that network-level structural integrity plays a key role in shaping oculomotor manifestations of spatial attention deficits and may inform future digital diagnostic and prognostic tools.

Disclosure: Nothing to disclose.

OPR-034 | Toward personalized neurorehabilitation: A deep reinforcement learning framework for predicting attentional deficits via in-silico synthetic lesions

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Background and Aims: Attentional deficits, notably hemispatial neglect, are highly disabling consequences of acquired brain injury (ABI) that significantly impair recovery. Current rehabilitation lacks a granular understanding of the underlying behavioral dynamics. This study develops a predictive Deep Neural Network (DNN) framework to model attentional-motor behavior and assess deficits via synthetic lesions, aiming to provide a foundational tool for automated diagnosis and personalized neurorehabilitation (See Figure 1).

Methods: We integrated a Convolutional Neural Network (CNN) for foveated visual perception with a Deep Reinforcement Learning (DRL) agent using Proximal Policy Optimization (PPO) to resolve a visuospatial selective attention task (Figure 2). Lesion models were generated by selectively deactivating neurons across neural networks layers. A Multilayer Perceptron (MLP) was trained on 17 behavioral metrics (e.g., mean step size, velocity variance) to predict lesion location and magnitude. Behavioral data were validated against a cohort of 43 ABI patients and 19 healthy controls.

Results: The DRL agent successfully mimicked healthy human exploration patterns and trajectories. The MLP demonstrated

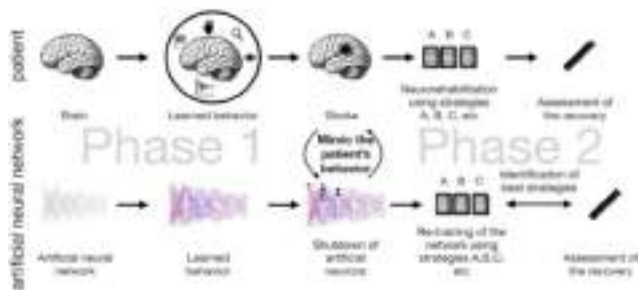


FIGURE 1 Schematic representation of the different phases of the project.

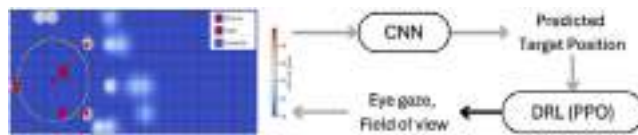


FIGURE 2 Overview of the deep neural network framework designed to mimic healthy human behavior during a visuospatial selective attention task.

discriminative performance in identifying in-silico lesion patterns. While group-level behavioral metrics showed convergence between in-silico and in-vivo data, direct transfer of the MLP’s predictive performance to patient data remains limited and need further improvements.

Conclusion: This project establishes a robust methodology for computational cognitive modeling. It demonstrates that synthetic lesions in DNNs can reflect specific behavioral impairments, offering a promising path toward data-driven diagnostic tools and the optimization of individualized rehabilitation strategies in clinical neurology.

Disclosure: Nothing to disclose.

OPR-035 | Investigating the relationship of trunk and postural control with pulmonary functions in subacute stroke patients

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Background and Aims: Stroke is a disease with high mortality and morbidity that not only causes weakness in the extremities, loss of balance and disturbances in trunk and postural control, but also affects respiratory function. The aim of this study was to investigate the relationship between trunk and postural control and pulmonary function in stroke patients.

Methods: 32 volunteer patients who were diagnosed with hemiplegia and who met the inclusion criteria participated in the study. Functional independence of the participants was evaluated using Modified Rankin Scale (mRS) and their cognitive function was assessed with the Standardized Mini Mental State Examination. Respiratory function was evaluated with spirometric measurements, inspiratory muscle strength was evaluated with intraoral pressure measurements, trunk control was evaluated using the Trunk Impairment Scale (TIS), postural

control was evaluated using the Postural Assessment Scale for Stroke Patients (PASS-T), computerized postural sway evaluation, and static posture analysis.

Results: A significant correlation was found between the TIS scores and inspiratory muscle strength ($p < 0.05$). A significant correlation was also found between the PASS-T scores and inspiratory muscle strength and pulmonary function ($p < 0.05$). All of the center of pressure parameters measured were significantly correlated with the PEF(L/s) and FEF25–75 (L/s) ($p < 0.05$).

TABLE 1 Demographic characteristics of the participants.

Sex	n	%
Female	20	62.5
Male	12	37.5
Age (years)	Mean	SD
Age (years)	58.56	12.31
Height (cm)	169	8.3
Weight (kg)	79.00	11.54
BMI (kg/m ²)	27.70	3.87
Time elapsed since stroke diagnosis (months)	6.03	3.89
Affected side (left/right) (n)	14/18	
Stroke Type (ischemic/hybrid/hemorrhagic) (n)	11/21	
Smoking status	n	%
Smoker	8	25
Non-smoker	32	68.8
Quit	2	6.3

TABLE 2 Relationship between functional independence and trunk-postural control and inspiratory muscle strength and respiratory functions.

[illegible]

Conclusion: In conclusion, this study showed that trunk and postural control are associated with inspiratory muscle strength and pulmonary function. It is recommended that evaluation of trunk and postural control and respiratory functions, as well as exercise training to improve these parameters, should be included in rehabilitation programs for individuals with stroke.

Disclosure: Nothing to disclose.

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Background and Aims: In multiple sclerosis (MS) patients, aerobic exercise may exert a neuroprotective role on the brain by stimulating neurogenesis in the dentate gyrus (DG) of the hippocampus and in the subventricular zone (SVZ), but further evidence is needed. We assessed the effects of aerobic exercise

on the volume of the hippocampus and its subfields and on diffusivity measures of the SVZ in progressive MS.

Methods: We retrospectively analyzed data from 84 patients with progressive MS enrolled at four sites in the CogEx MRI sub-study. Thirty-nine patients performed aerobic training, while 45 undertook a balance- and stretching-based sham intervention. Both groups trained twice weekly for twelve weeks. At baseline, post-intervention and 6-month follow-up, patients underwent MRI assessment on a 3.0 T scanner. FreeSurfer's longitudinal pipeline was used to analyze hippocampal subfields' volumes. Fractional anisotropy and mean diffusivity were extracted from the SVZ and the thalamus (as control region).

Results: No baseline differences were observed between the aerobic and the sham exercise groups ($p \geq 0.070$). The DG showed a significant volume increase post-intervention in the aerobic group (mean change = 0.63%, 95% confidence interval [95% CI] = 0.04%; 1.22%, $p = 0.035$), but not in the sham exercise group (mean change = -0.26%, 95% CI = -0.81%; 0.28%, $p = 0.337$). Between-group difference in DG volume change was significant ($p = 0.029$). No significant changes were found in the other hippocampal subfields or SVZ diffusivity metrics ($p \geq 0.057$). No changes in either group were found between post-intervention and 6-month follow-up ($p \geq 0.061$).

Conclusion: Aerobic exercise increases DG volume in progressive MS patients but does not modify microstructural integrity of the SVZ.

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Headache 1

OPR-037 | Migraine in older adults: A multicenter real-world analysis from the Italian RICE registry

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Background and Aims: Primary headaches, particularly migraine, are prevalent disorders insufficiently characterized in the growing elderly population. This multicentre study provides a evaluation of headache diagnoses, with a focus on migraine, across different age groups in tertiary headache centers participating in the Italian RICE registry.

Methods: We retrospectively analyzed 3472 patients evaluated at thirty Headache Centers between 2021 and 2025. Patients were stratified into four age groups: young adults (18–30 years),

adults (31–45 years), middle-aged (46–59 years), and elderly (≥ 60 years). Data collected included International Headache Society (IHS) headache diagnoses (ICHD3) and demographic and clinical features. Statistical analyses included chi-square tests and Kruskal–Wallis ANOVAs.

Results: Of the total sample, 643 patients (18.5%) were elderly, 1285 (37.0%) middle-aged, 988 (28.5%) adults, and 556 (16.0%) young adults. The distribution of diagnoses differed significantly across groups [$\chi^2(117)=254$, $p<0.001$]. Elderly patients were more likely to receive a diagnosis of chronic migraine ($z=3.41$). Migraine with aura was more prevalent in younger individuals ($z=5.32$). Among other primary headaches, tension-type headache was more frequent in the elderly ($z=2.47$). Medication overuse headache (MOH) occurred more often in middle-aged patients ($z=4.64$). Older patients reported a longer disease duration, but with less intense pain severity, and fewer associated symptoms (all $p<0.01$).

Conclusion: Headache diagnoses vary significantly with age. In elderly patients, chronic migraine predominates and it is characterized by longer attacks with reduced associated symptoms. MOH represents a relevant concern in middle-aged individuals. These findings highlight the need for age-specific diagnostic and therapeutic strategies, particularly in older adults.

Disclosure: Nothing to disclose.

OPR-038 | Onabotulinum toxin type-A for the management of medically refractory SUNHA: A preliminary retrospective single centre experience

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Background and Aims: Short-lasting unilateral neuralgiform headache attacks (SUNHA) are rare primary head and facial pain disorders that share clinical features and treatment challenges with trigeminal neuralgia (TN). Onabotulinum Toxin A (OnaBoNT) demonstrated promising efficacy in refractory TN. Based on this rationale, we began offering OnaBoNT to SUNHA patients refractory to medical management. Here we aim to evaluate the effectiveness of OnaBoNT in patients with refractory SUNHA.

Methods: Medically refractory SUNHA patients diagnosed between January 2024 and January 2026, were offered OnaBoNT using a “follow-the-pain protocol”, performed 3–4 months apart. Efficacy outcomes were retrospectively assessed using the Patient Global Impression of Change (PGIC) scale, percentage of symptom improvement, and duration of benefit. Side effects were collated.

Results: Of the 214 patients with SUNHA evaluated during the study period, outcomes were available for 18 individuals who received at least one OnaBoNT treatment (Tables 1 and 2). A median dose of 112.5U [IQR 80–145U] was administered per treatment cycle. At 3- and 6-month follow-ups, 9/18 (50%) and 6/12 (50%) patients respectively reported a PGIC of ≥ 6 and a $\geq 50\%$ pain reduction (median: 90%, IQR: 80%–100%). The mean duration of benefit was 9.5 weeks (3–16 weeks). The six responders to

the first two treatments displayed a sustained improvement up to their last treatment cycle (range: 5–13 cycles). Mild transient facial asymmetry was reported in 5/18 (28%) patients.

TABLE 1 Demographic characteristics.

Number of patients	18
Gender	
Females, N	10/18 (55%)
Males, N	8/18 (44%)
Diagnosis	
Chronic SUNHA, N	15/18 (83%)
Episodic SUNHA, N	3/18 (17%)
Other primary headaches	
Migraine, N	2/18 (11%)
Tension-type Headache, N	1/18 (6%)
Others, N	0/18 (0%)
Age at onset, Median [SD]	51 [11.7]
Years of disease, Median [IQR]	6 [5.3–14.8]

IQR: Interquartile Range; N: number; SD: Standard Deviation; SUNHA: Short-lasting unilateral neuralgiform headache attacks

TABLE 2 Clinical features, previous and current treatment.

Clinical features	
Side	
Left-sided, N	14/18 (78%)
Right-sided, N	4/18 (22%)
Trigeminal distribution of pain	
V1 only, N	0/18 (0%)
V2 only, N	1/18 (6%)
V3 only, N	0/18 (0%)
V1+V2, N	4/18 (22%)
V2+V3, N	0/18 (0%)
V1+V2+V3, N	7/18 (39%)
Duration of attack, Median [IQR]	90 seconds [12.5–180]
Frequency of attacks/day, Median [IQR]	17.5 [10–52.5]
Ipsilateral cranial autonomic symptoms, N	
1	5/18 (28%)
2	7/18 (39%)
3	6/18 (33%)
Previous and current treatment	
Previous oral prophylaxis failed N, Median [IQR]	4 [2–5.7]
Patient with failed trigeminal MVD, N	3/18 (17%)
Patients on oral prophylaxis at time of OnaBoNT, N	15/18 (83%)

IQR: Interquartile Range; MVD: microvascular decompression; N: number; OnaBoNT: Onabotulinum toxin type A; V1: cutaneous territory innervated by the first division of the trigeminal nerve; V2: cutaneous territory innervated by the second division of the trigeminal nerve; V3: cutaneous territory innervated by the third division of the trigeminal nerve

Conclusion: Our initial experience with OnaBoNT injected with a “follow-the-pain protocol” may indicate its safety and sustained efficacy overtime in a subpopulation of patients with refractory SUNHA.

Disclosure: Nothing to disclose.

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Background and Aims: Migraine is common in people with multiple sclerosis (PwMS) and substantially contributes to disability and impaired quality of life. Although calcitonin gene-related peptide (CGRP)-targeting therapies have reshaped migraine prevention, evidence on their use in PwMS remains scarce, particularly in patients receiving concomitant disease-modifying therapies (DMTs).

Methods: We retrospectively collected data from 17 Italian MS centers on adult PwMS with comorbid migraine treated with anti-CGRP monoclonal antibodies or atogepant in addition to stable DMTs. Monthly headache days (MHDs) and analgesic intake were compared between treatment initiation and last available follow-up. MS activity was assessed through relapses, Expanded Disability Status Scale (EDSS), and MRI findings. A $\geq 50\%$ reduction in MHDs defined treatment response. Multivariate regression models were used to explore predictors of clinical improvement.

Results: Fifty-four patients were included (46 women; mean age 42.3 years; 85% relapsing MS). Baseline MHDs averaged 19.9 ± 7.0 and declined to 11.4 ± 9.4 at follow-up ($p < 0.001$), with a parallel decrease in analgesic use ($p < 0.001$). A responder rate

TABLE 1 Patient demographics and clinical features at baseline.

Table 1	Overall population (n=54)
Demographics	
Age (years), mean $\pm$ SD	42.33 $\pm$ 9.06
Sex, n (%)	46 F (85%); 8 M (15%)
Psychiatric comorbidities, n (%)	9 (17%)
Other comorbidities, n (%)	36 (66%)
Migraine features	
Age at onset, mean $\pm$ SD ^a	39.69 $\pm$ 7.80
Disease duration (years), mean $\pm$ SD ^b	24.8 $\pm$ 18.03
Ataxia, n (%) ^c	7 (13%)
Episodic migraine, n (%) ^d	17 (27%)
Chronic migraine, n (%) ^e	24 (44%)
Medication overuse headache, n (%) ^f	16 (27%)
Monthly headache days, mean $\pm$ SD ^g	20.09 $\pm$ 8.38
Absolute number of analgesics, mean $\pm$ SD ^h	22.97 $\pm$ 15.35
Multiple Sclerosis features	
Age at onset, mean $\pm$ SD ⁱ	23.80 $\pm$ 10.06
Disease duration (years), mean $\pm$ SD ^j	30.88 $\pm$ 8.71
Months since the start of the current DMT, mean $\pm$ SD ^k	63.17 $\pm$ 44.24
Prior preventive DMTs, mean $\pm$ SD ^l	1.82 $\pm$ 4.23
Number of relapses one year prior anti-CGRP mAb, mean $\pm$ SD ^m	6.16 $\pm$ 9.47
EDSS one year prior anti-CGRP mAb, mean $\pm$ SD ⁿ	2.30 $\pm$ 1.63
New or T2 gadolinium+ MRI lesions one year prior anti-CGRP mAb, n (%) ^o	3 (5%)

^aCalculated on 47 patients; ^b43 patients; ^c93 patients; ^d52 patients; ^e34 patients; ^f43 patients; ^g29 patients; ^h37 patients; ⁱ49 patients; ^j45 patients.

TABLE 2 Patient demographics and clinical features at baseline: Responders vs. non responders.

Table 2	Responders ($\geq 50\%$ reduction MHDs)	Non-responders ($< 50\%$ reduction MHDs)	p-value
Demographics			
Age (years), mean $\pm$ SD	38.79 $\pm$ 8.48	46.6 $\pm$ 7.36	0.086
Sex, n (%)	21 F (38%); 4 M (7%)	25 F (40%); 4 M (7%)	0.771
Psychiatric comorbidities, n (%)	5 (9%)	3 (5%)	0.288
Other comorbidities, n (%)	7 (12%)	9 (16%)	0.883
Migraine features			
Age at onset, mean $\pm$ SD ^a	19.28 $\pm$ 6.43	21.47 $\pm$ 8.36	0.083
Disease duration (years), mean $\pm$ SD ^b	22.90 $\pm$ 10.97	24.88 $\pm$ 9.28	0.384
Ataxia, n (%) ^c	2 (3%)	3 (5%)	0.888
Episodic migraine, n (%) ^d	4 (7%)	11 (18%)	0.087
Chronic migraine, n (%) ^e	11 (20%)	13 (24%)	0.817
Medication overuse headache, n (%) ^f	10 (18%)	4 (7%)	0.022
Monthly headache days, mean $\pm$ SD ^g	21.84 $\pm$ 7.49	19.46 $\pm$ 6.19	0.422
Absolute number of analgesics, mean $\pm$ SD ^h	19.27 $\pm$ 15.89	18.75 $\pm$ 12.89	0.124
Multiple Sclerosis features			
Age at onset, mean $\pm$ SD ⁱ	28.12 $\pm$ 11.02	28.5 $\pm$ 9.68	0.595
Disease duration (years), mean $\pm$ SD ^j	8.8 $\pm$ 4.99	17.84 $\pm$ 10.83	< 0.001
Months since the start of the current DMT, mean $\pm$ SD ^k	62.22 $\pm$ 46.94	71.26 $\pm$ 42.89	0.547
Prior preventive DMTs, mean $\pm$ SD ^l	2.27 $\pm$ 6.37	1.16 $\pm$ 1.16	0.674
Number of relapses one year prior anti-CGRP mAb, mean $\pm$ SD ^m	0.16 $\pm$ 0.48	0.16 $\pm$ 0.48	1.00
EDSS one year prior anti-CGRP mAb, mean $\pm$ SD ⁿ	2.26 $\pm$ 1.62	2.62 $\pm$ 1.62	0.799
New or T2 gadolinium+ MRI lesions one year prior anti-CGRP mAb, n (%) ^o	2 (3%)	1 (1%)	0.525

of 53.7% was observed. MS disease activity remained stable, with no significant changes in relapse frequency, EDSS, or MRI activity. Higher baseline headache burden was associated with

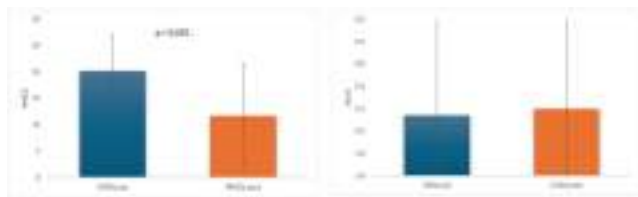


FIGURE 1 Number of MHD and EDSS score at baseline, and the last follow-up during treatment with CGRP-mAbs in combination with DMT for multiple sclerosis.

greater reduction in MHDs ($\beta=0.64$, $p<0.001$), whereas longer MS duration predicted poorer response (OR=1.70, 95% CI 1.11–2.63). Mild adverse events occurred in four patients, without treatment discontinuation.

Conclusion: In this real-world multicenter cohort, anti-CGRP therapies provided meaningful migraine improvement in PwMS while maintaining MS stability. Disease duration and baseline headache frequency may influence therapeutic response.

Disclosure: Nothing to disclose.

OPR-040 | Randomised controlled trial with translational validation: Migraine induced by vascular KATP channel activation is independent of HCN channel activity

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Background and Aims: Vascular ATP-sensitive potassium (KATP) channel activation can trigger migraine, but the mechanisms remain unclear. Proposed pathways involve either direct neuronal activation via hyperpolarisation-activated cyclic nucleotide-gated (HCN) channels or signalling at the vessel-to-neuron interface. We tested whether HCN channel blockade alters KATP channel-induced migraine.

Methods: We conducted a single-centre, randomised, double-blind, placebo-controlled, 2-way crossover study in individuals with migraine without aura and mechanistically aligned pre-clinical experiments in a validated mouse model. Participants received intravenous levocromakalim followed by oral ivabradine or placebo on separate days. The primary outcome was the 12-h incidence of levocromakalim-induced migraine; secondary outcomes included area under the curve (AUC) for headache intensity and haemodynamic responses. In mice, von Frey testing was used to assess whether ivabradine, given as pre- or rescue treatment, modified levocromakalim-induced hypersensitivity.

Results: Twenty-seven participants completed the human study and were included in the analysis. Ivabradine had no effect on the incidence of levocromakalim-induced migraine (22/27 after ivabradine and 22/27 after placebo; $p>0.99$) or on AUC for

headache intensity ($p=0.11$). Haemodynamic responses did not differ between study days. In mice, multiple doses of ivabradine neither prevented nor reversed tactile hypersensitivity caused by repeated levocromakalim administration.

Conclusion: HCN channel blockade does not affect migraine or nociceptive behaviour triggered by vascular KATP channel activation, suggesting that HCN channels are not required for translating vascular KATP channel activation into migraine pain. Instead, our findings support a model in which migraine is initiated via signalling at the vessel-to-neuron interface.

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Neuroimaging 1

OPR-041 | APOE ε4 gene dose is associated with more typical and homogeneous amyloid deposition across the Alzheimer's disease continuum

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Background and Aims: Although amyloid deposition is a core pathological feature of Alzheimer's disease (AD), its spatial expression varies widely across individuals. The probabilistic amyloid hypothesis predicts that APOE ε4 constrains amyloid accumulation toward more stereotyped spatial patterns. Whether APOE ε4 gene dose increases the predictability and homogeneity of amyloid topography across disease stages remains unknown.

Methods: We performed voxel-wise similarity and spatial homogeneity analyses of amyloid-PET data in amyloid-positive participants from ADNI (CU, MCI, dementia). Participants were stratified by APOE ε4 genotype. Individual amyloid-PET maps were correlated with two independent canonical AD amyloid reference maps. Within-group spatial homogeneity was quantified using inter-subject voxel-wise correlation matrices. Replication was performed in an independent cohort from the Geneva Memory Center.

Results: APOE ε4 genotype predicted amyloid-PET pattern similarity to canonical AD maps, independent of age, sex, and

global amyloid burden. Similarity increased in a dose-dependent manner from non-carriers to heterozygotes and homozygotes. APOE $\epsilon 4$ carriers also showed significantly greater within-group spatial homogeneity of amyloid deposition, with the highest convergence observed in homozygotes. These effects were most pronounced in CU and MCI stages but persisted across the AD continuum. All findings replicated across cohorts, PET tracers, and canonical reference maps.

Conclusion: The APOE $\epsilon 4$ gene dose is associated with increasingly predictable and homogeneous amyloid topographies, supporting a gradient of pathological determinism in AD. These results provide in vivo support for the probabilistic model of AD and suggest that APOE $\epsilon 4$ homozygosity drives the topographical predictability and homogeneity of amyloid deposition, with implications for targeted anti-amyloid interventions.

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OPR-042 | Functional connectivity reveals gene expression-driven network vulnerability across frontotemporal lobar degeneration variants

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Background and Aims: Frontotemporal lobar degeneration (FTLD) includes several syndromes with specific regional susceptibility. How molecular architecture shapes degeneration across functional networks remains unclear. Stepwise functional connectivity (SFC) captures how disease epicenters communicate across network distances. We investigated whether

transcriptomic profiles of key FTLD-associated genes align with SFC patterns.

Methods: Variant-specific atrophy epicenters were identified using voxel-based morphometry in patients with behavioral variant FTD (bvFTD, $n=11$), semantic variant PPA (svPPA, $n=18$), nonfluent variant PPA (nfvPPA, $n=14$), and semantic behavioral variant FTD (sbvFTD, $n=15$). Regional SFC topological distances from epicenters were derived using resting-state fMRI in 50 healthy controls. Spatial correlations were computed between SFC distance and gene expression maps from Allen Human Brain Atlas. Genes enriched near epicenters underwent pathway analysis.

Results: Epicenters were located in the left anterior insula (bvFTD), left anterior inferior temporal gyrus (svPPA), left supplementary motor area (nfvPPA), and right temporal pole (sbvFTD). GRN and MAPT were enriched near epicenters, whereas C9orf72 expression increased with SFC distance. Pathway analysis identified two molecular clusters: bvFTD/nfvPPA-associated genes were linked to mitochondrial metabolism, oxidative stress, and proteostasis; svPPA/sbvFTD-associated genes were associated with neuromodulation and synaptic plasticity.

Conclusion: Regional transcriptomic patterns reflect functional network architecture in FTLD, suggesting molecular vulnerability shapes network-specific degeneration. GRN and MAPT enrichment near epicenters indicates local susceptibility, while C9orf72 patterns suggest distinct propagation. Variant-associated clusters may reflect differential vulnerability to tau or TDP-43 pathology, potentially explaining clinical and anatomical differences.

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OPR-043 | Glymphatic impairment in GRN-mutated frontotemporal dementia: A distinct pathophysiological signature

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Background and Aims: Diffusion tensor imaging analysis along the perivascular space (DTI-ALPS) is a non-invasive MRI-based proxy of glymphatic dysfunction, a mechanism implicated in the accumulation of protein aggregates in several neurodegenerative disorders. This study aimed to evaluate glymphatic function using DTI-ALPS in GRN-mutated frontotemporal dementia (FTD), comparing them to sporadic cases and healthy controls (HC), and investigate its clinical relevance and relationship with white-matter (WM) damage.

Methods: Eighteen GRN-mutated patients with an FTD syndrome (10 behavioural variant FTD [bvFTD], 5 non-fluent primary progressive aphasia [nfvPPA], 3 other PPA variants), 43 sporadic FTD patients, and 32 age- sex-matched HC underwent 3T MRI. The DTI-ALPS index was calculated for each participant. Group comparisons were performed using age- sex- and fractional anisotropy (FA)-adjusted ANCOVA. Associations with cognitive performance, behavioural symptoms, and sleep disturbances were assessed. WM microstructural integrity was evaluated using tract-based spatial statistics with voxel-wise FA.

Results: GRN-patients showed significantly lower DTI-ALPS values than HC ($p < 0.001$) and sporadic FTD patients with bvFTD ($p = 0.027$) and semantic variant of PPA ($p = 0.003$). Within the GRN-group, lower DTI-ALPS was associated with poorer executive performance, higher impairment on selected Frontal Behavioural Inventory items, and the presence of insomnia ($p = 0.025$). DTI-ALPS values positively correlated with FA in the anterior and superior corona radiata, internal and external capsules, corpus callosum, and left superior longitudinal fasciculus ($p < 0.05$, Family-Wise Error corrected for multiple comparisons).

Conclusion: Glymphatic dysfunction is more pronounced in GRN-FTD than in sporadic FTD and is associated with WM damage, executive, behavioural alterations, and sleep disturbances, supporting a gliovascular contribution to GRN-mediated neurodegeneration.

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OPR-044 | Metabolically defined lesion clusters show differential associations with clinical disability in multiple sclerosis

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Background and Aims: Lesion burden on conventional magnetic resonance imaging correlates only weakly with clinical disability in multiple sclerosis (MS). We investigated whether metabolically defined lesion clusters identified by 7T MR

spectroscopic imaging show differential associations with clinical disability.

Methods: In this cross-sectional observational study, 50 people with MS underwent 7T MR spectroscopic imaging. After quality control, 304 white matter lesions from 45 participants were analysed using metabolite ratios reflecting lesion metabolic state. Data-driven clustering identified metabolically distinct lesion clusters. Lesion burden within each cluster was correlated with clinical disability measures, including the Expanded Disability Status Scale (EDSS), Symbol Digit Modalities Test (SDMT), Timed 25-Foot Walk (T25FW), and 9-Hole Peg Test (9-HPT), using age-adjusted models.

Results: Three reproducible metabolic lesion clusters were identified (Figure 1). Cluster 1, consistent with newly emerging, actively inflammatory and expanding lesions, showed the strongest clinical associations, with higher lesion count correlating with worse SDMT performance ($r = -0.34$, $p = 0.027$). Cluster 2, reflecting metabolically exhausted lesions with ongoing tissue damage, was associated with impaired motor function, including slower T25FW ($r = 0.31$, $p = 0.044$) and 9-HPT ($r = 0.29$, $p = 0.031$). Cluster 3, characterized by metabolically stable lesions with features consistent with partial repair, showed no significant associations with disability ($p > 0.1$). Associations between cluster-specific lesion burden and disability are illustrated in Figure 2.

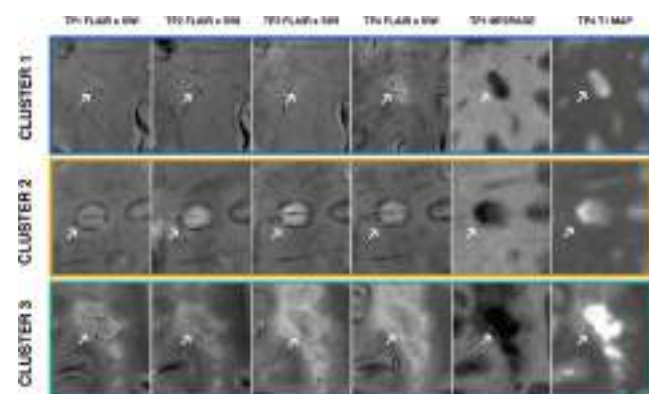


FIGURE 1 Longitudinal examples of metabolically clustered lesions (Clusters 1–3) across coregistered FLAIR + SWI, MP2RAGE, and quantitative T1 maps from baseline to 8-year follow-up.

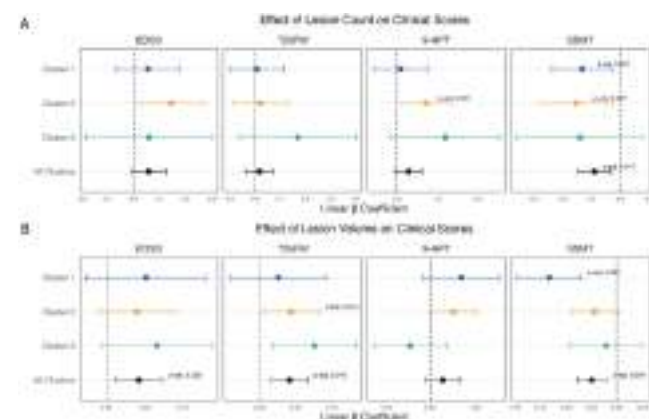


FIGURE 2 Linear regression models assessed the effect of lesion count (A) and log-transformed lesion volume (cm^3) on disability.

Conclusion: These findings indicate that lesion metabolic state, rather than lesion presence alone, determines clinical relevance, supporting metabolic imaging as a tool for improved stratification of disease impact.

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MS and Related Disorders 2

OPR-045 | Early cognitive trajectories over 5 years following the first demyelinating event and their baseline magnetic resonance imaging predictors

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Background and Aims: Cognitive decline in early MS remains understudied. We investigated longitudinal cognitive trajectories and baseline magnetic resonance imaging (MRI) predictors following the first demyelinating event.

Methods: Patients were enrolled within 3 months of symptom onset and followed for 5 years. Baseline MRI metrics included brain matter fractions and neurite orientation dispersion and density imaging (NODDI) parameters, specifically neurite density index (NDI), orientation dispersion index (ODI) and isotropic volume fraction (Viso). The Brief Visuospatial Memory Test-Revised (BVM-T-R), California Verbal Learning Test (CVLT) and Symbol Digit Modalities Test (SDMT) were collected at each visit. Quadratic and single-knot piecewise linear mixed models evaluated the impact of baseline MRI metrics on cognitive trajectories.

Results: 100 patients (mean age 32.3 ± 7.5 years, 33% male) were included, 35% of whom had MS at baseline. BVM-T-R and CVLT improved until year 3 by 0.15 (SE: 0.03; $p < 0.001$) and 0.21 (SE: 0.04; $p < 0.001$) z-scores/year, respectively, before declining. SDMT remained steady throughout the study period. A 1% decrease in deep grey matter fraction (DGMF) predicted 0.92 ($p = 0.009$, SE = 0.35) and 1.11 ($p = 0.022$, SE = 0.48) z-scores/year declines in BVM-T-R and CVLT, respectively, while a 0.1-unit increase in cortical grey matter isotropic volume fraction (CGMViso) predicted a 0.47 z-score/year decline in BVM-T-R ($p = 0.003$, SE = 0.15).

Conclusion: After the first demyelinating event, patients showed initial cognitive improvement followed by decline in multiple domains. Baseline DGMF and CGMViso predicted this decline, highlighting their value as prognostic markers for cognitive trajectories in early MS.

Disclosure: Nothing to disclose.

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Background and Aims: The choroid plexus (ChP) regulates cerebrospinal fluid production and central nervous system homeostasis. In multiple sclerosis (MS), ChP enlargement occurs early in the disease course, yet its normative lifespan trajectory and disease-specific associations remain unclear. We defined normative ChP volume trajectories across the healthy lifespan and assessed ChP enlargement in MS to evaluate associations with demographic, clinical, MRI, and genetic variables, including Human Leukocyte Antigen (HLA) and non-HLA polygenic risk scores (PRSs).

Methods: This monocentric retrospective study included 461 healthy controls (HC) and 727 MS patients (age 18–70 years) who underwent 3T brain MRI and neurological assessment. ChP volumes were quantified using ASCHOPLEX, normalized for head size, and modeled in HC to compute z-scores for MS patients. PRSs were calculated from established MS susceptibility loci, encompassing both HLA and non-HLA regions.

Results: In HC, normalized ChP volume (NChPV) was predicted by normalized brain volume, lateral ventricle volume, and its squared term ($R^2=0.54$), remaining stable until age 35, then increasing nonlinearly ($p\text{-FDR}<0.002$). MS patients had significantly higher NChPV z-scores than controls ($p\text{-FDR}<0.001$), independent of age or phenotype. NChPV z-scores rose during the first five years after onset ($p\text{-FDR}=0.012\text{--}0.015$) before plateauing. Higher z-scores were associated with greater T2-hyperintense white matter lesion volume ($p<0.001$) and HLA genetic burden ($p=0.027$) but not non-HLA PRSs ($p\geq 0.177$).

Conclusion: We provided normative ChP trajectories and showed early, sustained ChP enlargement in MS, linked to inflammatory lesion burden and HLA-mediated genetic risk. The ChP may represent a non-invasive biomarker of MS susceptibility and neuroinflammation.

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L. Storelli, E. Pagani, M. Rubin, F. Clarelli, E. Mascia, M. Sorosina, F. Esposito have nothing to disclose. M. Margoni reports grants and personal fees from Sanofi Genzyme, Merck Serono, Roche, Biogen, Amgen and Novartis. M.A. Rocca received consulting fees from Biogen, Bristol Myers Squibb, Roche; and speaker honoraria from Alexion, Biogen, Bristol Myers Squibb, Celgene, Horizon Therapeutics Italy, Merck Serono SpA, Mitsubishi-Tanabe Pharma, Neuraxpharm, Novartis, Roche, Sandoz, and Sanofi. She receives research support from the MS Society of Canada, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. She is Associate Editor for Multiple Sclerosis and Related Disorders; and Associate Co-Editor for Europe and Africa for Multiple Sclerosis Journal. M. Filippi is Editor-in-Chief of the Journal of Neurology, Associate Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmed Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla.

OPR-047 | The sphingosine-1-phosphate mimetic FTY720 inhibits mitochondrial activity in lymphocytes from healthy donors

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Background and Aims: A non-selective agonist of sphingosine-1-phosphate (S1P) receptors, FTY720, inhibits lymphocyte migration and mitigates subsequent inflammation. Mounting evidence supports the participation of S1P signaling in additional processes besides cell migration, but FTY720 mechanisms are not fully understood at present and possible effects on mitochondrial integrity are now explored. This study aims to investigate whether the S1P analog FTY720 modulates mitochondrial homeostasis in lymphocytes from healthy donors. Specifically, we evaluated its in vitro effects on peripheral blood mononuclear cells (PBMCs) isolated from controls.

Methods: Mitochondrial respiration and glycolytic activity were assessed by Seahorse XFp extracellular flux analysis. Mitochondrial mass, membrane potential, reactive oxygen species (ROS) production, and glycolytic mediators were evaluated by flow cytometry, and electron transport chain (ETC) complexes by Western Blot.

Results: FTY720 blocked early and late activation of PBMCs and inhibited basal and PHA-stimulated respiration (basal, maximal, and ATP-linked respiration) and glycolytic capacities (basal/maximal/glycolytic reserve). We observed significantly decreased mitochondrial depolarization and coupling in basal and stimulated conditions, with parallel increased ROS levels, together with lower fold-change of complex III of the ETC upon PHA stimulation. Both in basal and PHA-stimulated conditions, we found consistently reduced levels of fission/fusion (DRP1, FIS1, OPA1, MFN2) proteins and a reduced fold-change in mitochondrial mass of B cells. Upon PHA stimulation, lactate transporters (MCT1 and 4) remained at unstimulated levels, while PFKFB3 showed increased prestimulated levels already in the absence of PHA.

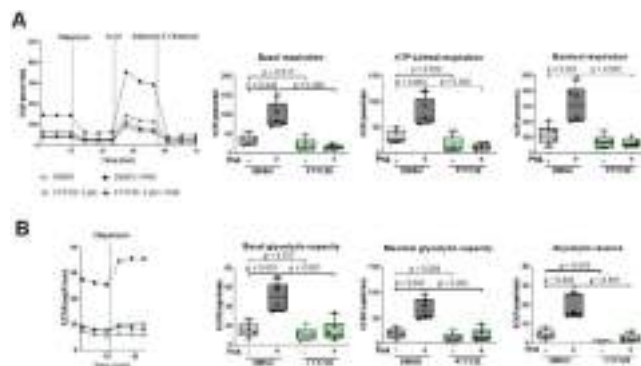


FIGURE 1 Respirometry of control lymphocytes incubated with FTY720. (A) Oxygen consumption rate and (B) extracellular acidification rate profiles and associated parameters in unstimulated and PHA-stimulated PBMCs in the presence of 5µM FTY720 or vehicle.

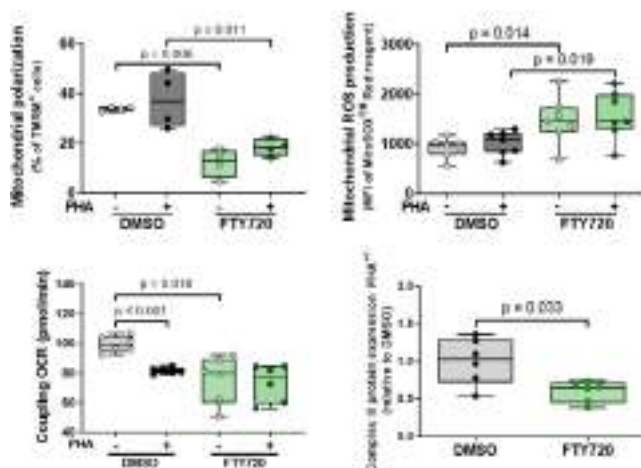


FIGURE 2 Changes in mitochondrial polarization, coupling, ROS and complex III levels upon in vitro PHA-stimulation of control lymphocytes incubated with FTY720.

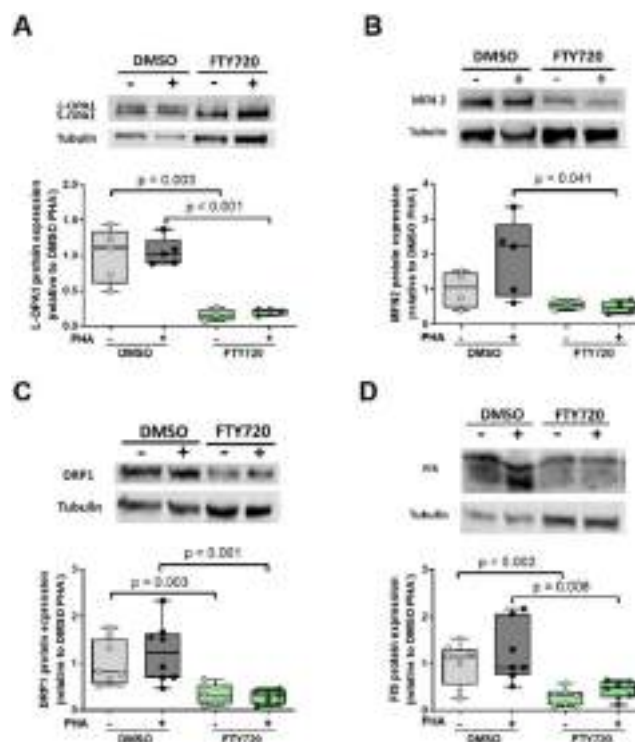


FIGURE 3 Changes in mitochondrial fusion and fission protein levels of lymphocytes incubated with FTY720. Representative Western blots and quantification of (A) OPA-L, (B) MFN2, (C) DRP1, and (D) FIS1 proteins from PBMCs.

Conclusion: FTY720 modulates mitochondrial activity of PBMCs isolated from healthy donors, indicative of additional pathways contributing to its therapeutic effects.

Disclosure: C.O.G. has received speaking and/or consultancy fees from Alexion, Biogen Idec, Bristol-Myers-Squibb, Janssen, Merck, Novartis, Roche, Sanofi-Genzyme, and Teva. Y.A. has received research grants, travel support, and lecturing and consulting fees from Bayer, Biogen, Roche, Merck, Novartis, Almirall, Sanofi-Genzyme, Janssen, and Bristol Myers Squibb. B.P. has received speaking, travel and/or training fees from Novartis, Almirall, Merck and Sanofi-Genzyme. The other authors have nothing to disclose.

OPR-048 | Mitochondrial DNA copy number correlates with brain atrophy in multiple sclerosis

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Background and Aims: Multiple sclerosis (MS) is a chronic inflammatory autoimmune disease of the central nervous system characterized by demyelination and neurodegeneration. Mitochondrial DNA copy number (mtDNA-CN) is a proposed biomarker of mitochondrial health, with reduced mtDNA-CN being related to disease duration and worsening in MS. However, its temporal dynamics and association with brain atrophy remain unclear. This study investigates longitudinal changes in mtDNA-CN in peripheral blood and its relationship with brain volume loss in people with MS (pwMS).

Methods: This study included 123 pwMS (mean ($\pm$ SD) age 33.2 ± 8.5 years, 59.3% female, median disease duration 3.1 years [IQR: 1.0–7.9]), median Expanded Disability Status Scale (EDSS) score 1.0 [IQR: 0.0–2.0] and 47 healthy controls (HC; mean ($\pm$ SD) age 34.1 ± 10.7 years, 72.3% female). In a subgroup of 62 pwMS, a second sample was collected after 2.6 ± 1.6 years. mtDNA-CN was quantified by real-time quantitative polymerase chain reaction in peripheral blood. Magnetic resonance imaging was performed on a 3-T Tim Trio system and longitudinal brain volume changes were assessed using SIENA.

Results: The mtDNA-CN was significantly lower in MS compared to HCs ($p < 0.05$). mtDNA-CN was unrelated to age, sex, disease duration and EDSS. No significant longitudinal change in mtDNA-CN was observed in the subgroup analysis. Of note, lower mtDNA-CN at baseline correlated with a higher percentage of annualized brain volume loss during the follow-up period ($\rho = 0.266$, $p < 0.05$).

Conclusion: These findings confirm lower mtDNA-CN in pwMS and suggest that mtDNA-CN might serve as a biomarker for the rate of brain atrophy in pwMS.

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Merck, Neuraxpharm, Novartis, Alexion, Amgen and Roche. He received research grants from Biogen, Novartis and Teva.

OPR-049 | Correlation of real-world digital biomarkers with clinical standards, findings from the MS-DETECT study

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Background and Aims: MSCopilot®, a Software as a Medical Device, remotely assesses key functions such as cognition, dexterity, vision and walking. French studies showed that MSCopilot® tests correlate with the Extended Disability Scale Score (EDSS) and the 4-item MS Functional composite (MSFC-4) when assessed in-clinic. The international MS-DETECT study (NCT05816122) evaluates MSCopilot®'s ability to detect disability worsening during 18–24 months. This analysis compares individual MSFC-4 scores with MSCopilot® digital biomarkers measured in-clinic or at home.

Methods: MSFC-4 scores were assessed in-clinic during the inclusion visit. MSCopilot® tests were performed with and without supervision in-clinic and at home, respectively. Association between the digital scores and their clinical counterparts was assessed using Pearson correlation coefficients.

Results: 244 patients (68.4% female, mean age 49.15 ± 8.74 , mean disease duration 17.57 ± 8.04 and mean EDSS: 3.8 ± 1.3) were included in this analysis. We collected 973 in-clinic and 870 remote MSCopilot® evaluations (243/218 cognition, 244/219 dexterity, 244/215 vision and 242/218 walking). Significant correlations were found between individual MSFC-4 items and digital tests: cognition ($r = 0.71/0.62$), dexterity ($0.45/0.47$), vision ($0.36/0.37$) and walking ($0.54/0.66$) tests. Composite MSFC-4 scores significantly correlated with MSCopilot®'s measured in-clinic (0.71) and remotely (0.74).

Conclusion: MSCopilot® digital tests, whether performed in-clinic or at home, correlated with their clinical counterparts. These findings emphasize their potential use in detecting disease worsening thus leading to improved clinical monitoring of MS.

Disclosure: P. Vermersch: AB Science, Ad Scientiam, Biogen, Imcyse, Janssen, Merck, Novartis, Roche, Sanofi, and Teva—consulting fees. Novartis, Roche, and Sanofi—research support. M. Filippi: The Journal of Neurology—Editor-in-Chief; Human Brain Mapping, Neurological Sciences, and Radiology—Associate editor; Alexion, AstraZeneca Rare Disease, Almirall, Biogen, Merck, Novartis, Roche, Sanofi—consulting fees.

Bayer, Biogen, Celgene, Chiesi Italia SpA, Eli Lilly, Genzyme, Janssen, Merck-Serono, Neopharmed Gentili, Novartis, Novo Nordisk, Roche, Sanofi, Takeda, and TEVA—speaking fees; Alexion, AstraZeneca Rare Disease, Biogen, Bristol-Myers Squibb, Merck, Novartis, Roche, Sanofi, Sanofi-Aventis, Sanofi-Genzyme, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi—advisory board member; Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla—research support. C. Oreja-Guevara: Alexion, AstraZeneca rare Disease, Amgen, Biogen Idec, BMS, Horizon, Janssen, Merck, Novartis, Roche, Sanofi-Genzyme, Sandoz, Viartis, Neuraxpharm and Teva—speaking and consulting fees; advisory board member. J. Oh: Amgen, Biogen, Eli Lilly and Company, EMD Serono, Novartis, Roche, Sanofi—consulting and/or speaking fees; Biogen, Roche—research support. T. Sejbæk: Tobias Sejbæk received travel grants from Biogen, Merck, Novartis, Roche and Sanofi—travel grants; Biogen, Merck and Sanofi—research grants; Biogen, Merck, Neuroxpharm, Novartis, Roche and Sanofi—advisory board member. JS. Graves: Sanofi, Genentech, Ad Scientium, Octave and EMD Serono—research grants; Octave, TR1X and Google—consulting fees. L. Kilaely, S. Bieuvelet, L. Carment, P. Drouin, S. Zinaï: Ad Scientiam employees; may hold shares or stock options. P. Rufi, B. Padrazzi: Sanofi employees; may hold shares or stock options. T. Ziemssen: Biogen, Roche, Neuraxpharm, Novartis, Viartis and Merck—scientific advisory board and/or consulting fees; Neuraxpharm, Roche, Novartis, Merck, Sanofi, BMS, and Biogen—speaking fees; Neuraxpharm, Roche, Novartis, Merck, and Sanofi—research support.

Ageing and Dementia 1

OPR-050 | Cortical thickness signature of early-onset Alzheimer's disease across preclinical and prodromal stages in a real-world memory clinic

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Background and Aims: The cortical thinning signature of early-onset Alzheimer's disease (EOAD) is well characterized in dementia, but its expression at earlier stages remains unclear. We aimed to determine when EOAD-related cortical atrophy becomes detectable and assess ATN biomarker contribution.

Methods: We studied individuals <65 years who underwent 3T structural MRI and cerebrospinal fluid-based ATN profiling. Cortical thickness was derived from T1-weighted images using FreeSurfer. EOAD signature regions were defined through vertex-wise comparisons between AD dementia and ATN-negative subjective cognitive decline (SCD). Regional cortical thickness and hippocampal volume were evaluated across SCD

and mild cognitive impairment (MCI) groups stratified by ATN status. Brain–cognition associations were assessed.

Results: The cohort included 507 individuals (mean age 57.7 ± 5.3 years), of whom 185 within the AD continuum. EOAD dementia showed marked thinning in parietal and lateral temporal cortices. In MCI with AD biomarkers, cortical thickness was significantly reduced across EOAD signature regions, including the inferior and superior parietal lobules, precuneus, and lateral temporal cortex, together with hippocampal volume (all $p < 0.01$; Cohen's $d \leq -0.50$). In SCD, cortical thinning was observed only

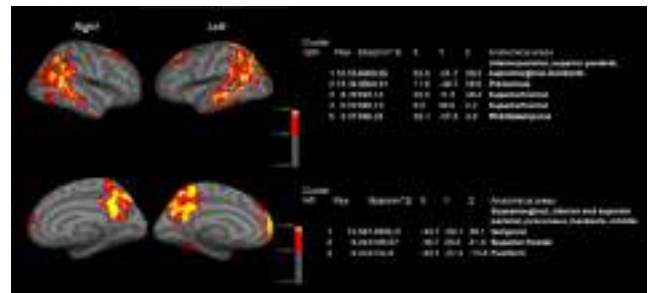


FIGURE 1 Cortical thinning pattern in EOAD dementia compared to the reference group of ATN-negative SCD.

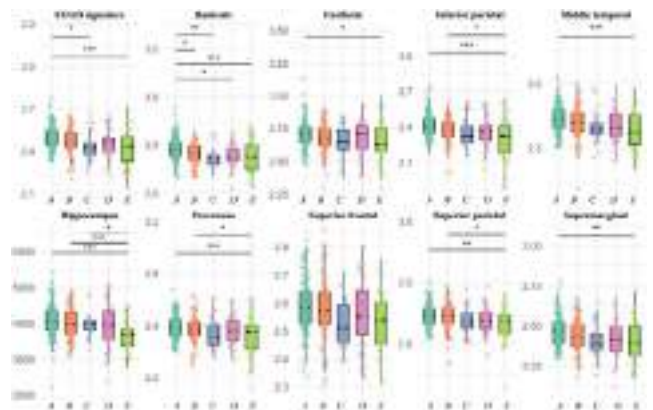


FIGURE 2 Regional cortical thickness and hippocampal volume in MCI subgroups. (A) SCD ATN negative, (B) MCI ATN negative, (C) MCI non AD-change, (D) MCI A+T-, (E) MCI A+T+.

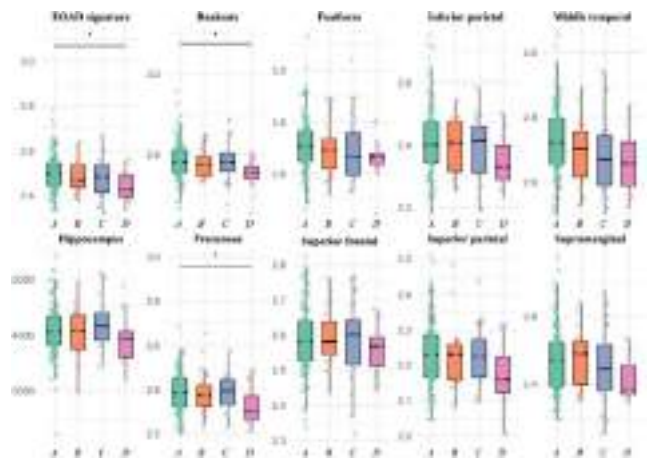


FIGURE 3 Regional cortical thickness and hippocampal volume in SCD groups. (A) SCD ATN negative, (B) SCD non AD change, (C) SCD A+T-, (D) SCD A+T+.

in A+T+ individuals, involving the precuneus and the banks of the superior temporal sulcus (all $p < 0.05$; $d \leq -0.60$), with no differences in A+T− SCD. In MCI-AD, reduced cortical thickness correlated with poorer global cognition and episodic and visuospatial memory (all $p < 0.05$), while no associations emerged in SCD.

Conclusion: EOAD-related cortical thinning at preclinical and prodromal stages depends on combined amyloid and tau pathology.

Disclosure: Nothing to disclose.

OPR-051 | Automatic primary progressive aphasia variant classification from transcripts and diffusion magnetic resonance imaging

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Background and Aims: Primary Progressive Aphasia (PPA) comprises three main clinical variants, nonfluent/agrammatic (nfvPPA), semantic (svPPA), and logopenic (lvPPA), each with distinct linguistic profiles and neurodegeneration patterns. Differential diagnosis relies heavily on subjective clinical judgment, limiting standardization. This study developed a multimodal pipeline combining speech-derived linguistic features with diffusion MRI markers for automated PPA classification.

Methods: This study included data from 91 healthy controls and 94 PPA patients (38 nfvPPA, 36 svPPA and 20 lvPPA). Connected speech was recorded during Picnic scene description task and automatically transcribed using Microsoft Azure after preprocessing optimization. Linguistic features were extracted via Python pipelines; diffusion features derived from atlas-based fractional anisotropy measures. A multinomial logistic regression classifier was trained with nested cross-validation. Transcriptions were compared against manual gold-standard using word error rate (WER). A web-based application integrating transcription with clinician-supervised revision and automatic feature extraction was implemented.

Results: Automatic transcription achieved mean WER of $12 \pm 7\%$ (controls) and $19 \pm 10\%$ (PPA). Linguistic features

captured variant-specific patterns (reduced output and slowed speech in nfvPPA; increased pauses and word-finding difficulty in lvPPA; fluent but semantically less informative speech in svPPA). The multimodal classifier achieved balanced accuracy of $87 \pm 6.9\%$, outperforming speech-only and imaging-only models. Per-variant accuracy was 93% (nfvPPA), 95% (svPPA), with lower performance for lvPPA.

Conclusion: This multimodal framework integrates speech and diffusion MRI to support standardized PPA variant differentiation with strong diagnostic performance. The interactive web platform improves transcription accuracy through clinician oversight, supporting harmonized diagnosis and longitudinal monitoring.

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OPR-052 | Independent effects of amyloid- β and tau pathology on synaptic damage across the Alzheimer's continuum

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Background and Aims: Synaptic dysfunction is the biological substrate of cognitive decline in Alzheimer's disease (AD). While amyloid- β and tau are known to interact in driving synaptic damage, their independent contributions remain unclear.

Methods: 109 cognitively unimpaired individuals and patients with mild cognitive impairment or dementia from the Geneva Memory Center with available CSF synaptic biomarkers (neurogranin, SNAP25, GAP43) were included and classified according to amyloid (A) and tau (T) status. Group comparisons and correlations between synaptic and AD biomarkers were performed. Multiple linear regression models assessed the independent associations of amyloid and tau with synaptic biomarkers. Model I included CSF A β 42 and CSF p-tau181, whereas Model II included amyloid PET centiloid and CSF p-tau181. Mediation analyses explored the relationships between amyloid, tau and synaptic biomarkers. All analyses were replicated in the Alzheimer's Disease Neuroimaging Initiative (ADNI) cohort ($n=787$).

Results: Synaptic biomarkers concentrations were significantly higher in A+/T+ participants ($n=27$) compared with A-/T- ($n=52$) and A+/T- ($n=30$) groups ($p<0.001$). CSF p-tau181 showed the strongest correlations with synaptic biomarkers (rs up to 0.83, $p<0.001$). In Model I, GAP43 was positively associated with CSF A β 42 ($\beta=0.31$, 95% CI 0.23–0.40, $p<0.001$; partial $R^2=0.321$) and p-tau181 ($\beta=0.94$, 95% CI 0.84–1.04, $p<0.001$; partial $R^2=0.809$). In Model II, lower centiloid ($\beta=-0.31$, 95% CI -0.46 to -0.16, $p<0.001$; partial $R^2=0.225$) and higher p-tau181 ($\beta=0.94$, 95% CI 0.84–1.04, $p<0.001$; partial $R^2=0.758$) predicted higher GAP43. Mediation analyses indicated tau as the mediator of the direct association between amyloid burden and GAP43.

Conclusion: These results indicate tau pathology as the main driver of synaptic damage across the Alzheimer's disease continuum.

Disclosure: Nothing to disclose.

OPR-053 | Residual cerebrospinal fluid NPTX2 predicts dementia progression beyond neurogranin and core pathology in prodromal Alzheimer's disease

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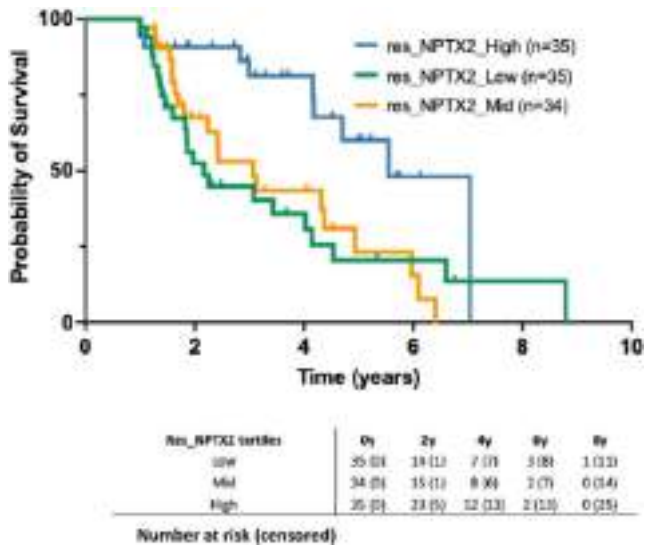


FIGURE 1 Kaplan–Meier curves for time-to-progression to AD dementia across tertiles of Res_NPTX2.

Background and Aims: In Alzheimer's disease (AD) core markers have limited ability in predicting progression from mild cognitive impairment (MCI) to dementia. Synaptic cerebrospinal fluid (CSF) biomarkers may add prognostic information, spanning processes from injury to adaptive network capacity. We tested whether a residual component of CSF neuronal pentraxin-2 (NPTX2), a synaptic protein linked to network homeostasis, beyond neurogranin (Ng) reflecting synaptic injury and core AD biomarker burden predicts dementia progression.

Methods: We retrospectively studied 104 patients with typical MCI-AD supported by core CSF biomarkers. CSF NPTX2 and Ng were quantified by commercial immunoassays. Early vs. late MCI sub-stages were defined by proximity to progression (LMCI ≤ 2 years, EMCI > 2 years or stable). A residual NPTX2 component (Res_NPTX2) was defined after regressing standardized (z-transformed) NPTX2 on Ng, a core AD biomarker burden composite (phosphorylated tau181 divided by amyloid- β 42/amyloid- β 40), and age. Associations with Mini-Mental State Examination (MMSE) and time to progression were tested using linear regression, Kaplan–Meier and Cox analyses, including ≤ 2 -year administrative censoring.

Results: Ng correlated with NPTX2 ($\rho=0.67$, $p<0.001$). Res_NPTX2 was higher in early than late MCI ($p=0.0006$) and associated with better MMSE at baseline ($p=0.008$) and less decline over time (Δ MMSE, $p=0.002$). During follow-up, 55/104 (52.9%) progressed to dementia. Higher Res_NPTX2 predicted lower hazard of progression (HR=0.63; $p=0.007$). Res_NPTX2 tertiles separated Kaplan–Meier curves (log-rank $p=0.006$), with stronger discrimination within 2 years (High vs. Low HR=0.18; $p=0.008$).

Conclusion: A residual CSF NPTX2 component beyond Ng and core biomarker burden identifies clinically meaningful heterogeneity and improves near-term prognostic stratification for dementia progression in MCI due to AD.

Disclosure: Nothing to disclose.

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Background and Aims: TREM2 (Triggering Receptor Expressed on Myeloid Cells 2) modulates microglial responses to neurodegeneration and plasma soluble TREM2 has been linked to white matter hyperintensity burden. However, whether plasma TREM2 relates to tract-specific white matter disconnection in Alzheimer's disease (AD) is unknown. To determine whether plasma TREM2 is associated with white matter tract disconnection in AD and whether such associations show anatomical specificity.

Methods: Fifty-seven AD patients (age 72 ± 6 years, 42% male) underwent 3T MRI and plasma collection. TREM2 was measured using the NULISA CNS Disease Panel. White matter hyperintensities were segmented on FLAIR and MPRAGE images and tract-specific disconnection probability was estimated for white matter tracts using volBrain DeepLesion, an automated deep learning-based pipeline. Tracts were grouped into limbic, association, callosal and projection categories. Partial

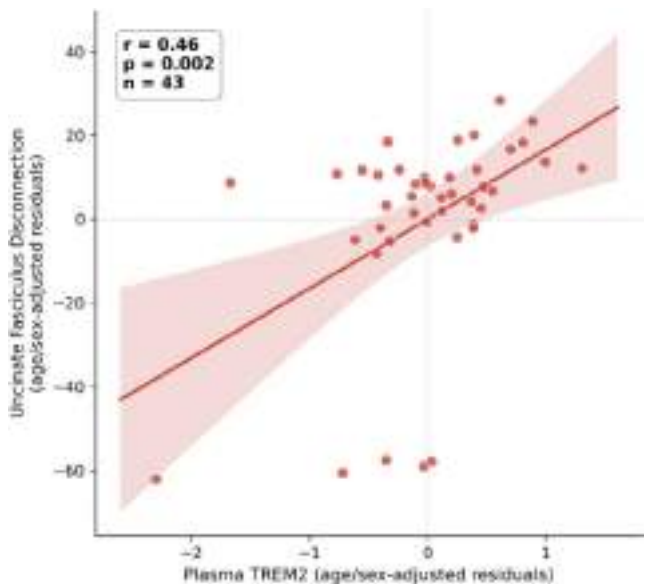


FIGURE 1 Plasma TREM2 and uncinete fasciculus disconnection in Alzheimer's disease. Scatter plot shows age- and sex-adjusted residuals.

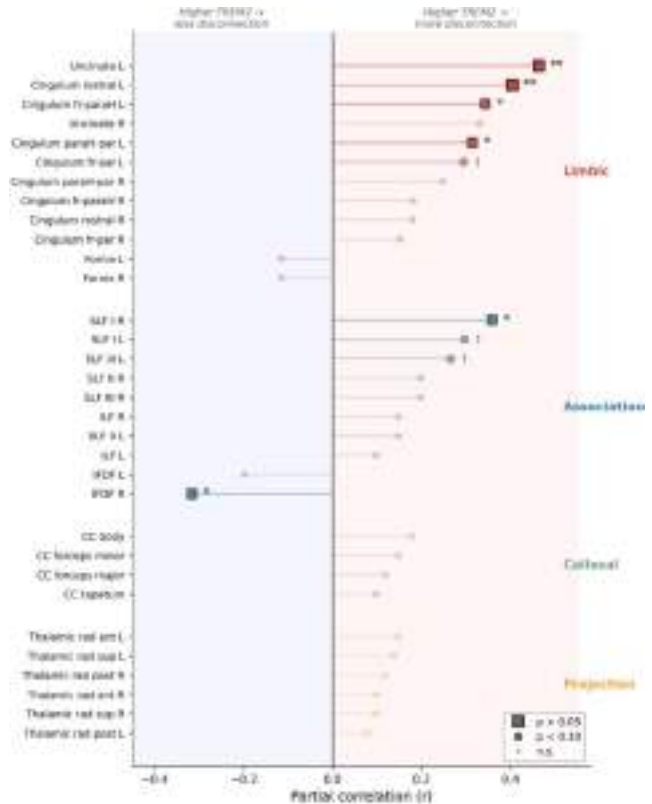


FIGURE 2 Forest plot of TREM2 associations with white matter tract disconnection.

correlations adjusted for age and sex tested associations between plasma TREM2 and each outcome.

Results: Plasma TREM2 showed a trend association with total WMH volume ($r=0.28$, $p=0.07$). Strikingly, TREM2 was selectively associated with limbic tract disconnection: uncinete fasciculus ($r=0.46$, $p=0.002$), rostral cingulum ($r=0.40$, $p=0.007$), frontal-parahippocampal cingulum ($r=0.34$, $p=0.024$), and parahippocampal-parietal cingulum ($r=0.32$, $p=0.040$). Overall, 4/12 limbic tracts reached significance (mean $r=+0.38$), compared to 0/4 callosal and 0/6 projection tracts (Figure 2). Higher TREM2 consistently predicted greater disconnection in memory-critical pathways (Figure 1).

Conclusion: Plasma TREM2 is specifically associated with limbic white matter disconnection in AD. This anatomical selectivity mirrors the regional vulnerability of AD and implicates microglial activation in disrupting memory-relevant circuits. Plasma TREM2 may serve as an accessible biomarker of limbic network integrity in AD.

Disclosure: Nothing to disclose.

OPR-055 | Serum inflammatory proteomic signatures stratify disease activity in chronic inflammatory demyelinating polyneuropathy

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Background and Aims: Chronic inflammatory demyelinating polyneuropathy (CIDP) is an immune-mediated neuropathy characterized by marked clinical heterogeneity and variable treatment response. Objective blood-based biomarkers reflecting disease activity and therapeutic responsiveness remain an unmet need, particularly in the context of immunoglobulin therapy, which may confound immune profiling. We aimed to identify serum inflammatory proteomic signatures associated with clinical phenotype, disease activity, and treatment exposure in CIDP.

Methods: In this case-control study, a multiplex proximity extension assay (PEA) and ELISA were used to quantify inflammatory proteins in serum from patients with CIDP and age- and sex-matched healthy controls, as well as from neurological disease controls including multiple sclerosis and IVIG-treated myasthenia gravis. Multivariable regression with correction for multiple testing, principal component analysis, and correlation analyses were performed to define disease-specific protein signatures and their associations with clinical characteristics, disease stability, and longitudinal outcomes.

Results: Distinct inflammatory proteomic patterns differentiated 51 patients with CIDP from 192 healthy controls and 105 neurological disease controls, indicating immune signatures beyond treatment-related effects. Specific protein clusters were associated with clinical phenotype, unstable disease, and subsequent clinical improvement, whereas others reflected immunoglobulin-modifiable pathways. Integrative analyses further suggested links between systemic inflammatory activity and markers of axonal injury.

Conclusion: This study identifies clinically relevant serum inflammatory signatures in CIDP and supports their potential as translational biomarkers for patient stratification, disease monitoring and therapy optimization.

Disclosure: F.S., A.R.P., S.H., and A.M. report speaker fees, consultancy honoraria, advisory board participation, or institutional research support from companies active in neuromuscular therapeutics, including Alexion, argenx, UCB, and others. These relationships are unrelated to the present study. All other authors declare no competing interests.

OPR-056 | Patient-reported carpal tunnel syndrome outcomes with and without surgery during COVID-19: A real-world follow-up study

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Background and Aims: During the COVID-19 period, some patients postponed or declined planned surgery for carpal tunnel syndrome (CTS). We assessed whether surgery was associated with better longitudinal outcomes on symptom severity and functional status.

Methods: We analyzed follow-up data from 73 patients (17.8% men, mean age 59.5 ± 12.0) first assessed pre-COVID and re-contacted later. Surgery status was self-reported (operated $n=29$; non-operated $n=44$). Overall, 40 (54.8%) used ≥ 1 additional option (rehabilitation, nocturnal splinting, reduced manual work). Baseline Padua severity was similar: median 3 (IQR 3–3) in both groups. Primary outcomes were Boston Carpal Tunnel Questionnaire subscales: Symptom Severity Scale (SSS) and Functional Status Scale (FSS). Between-group differences in improvement were tested, and multivariable linear models evaluated follow-up SSS/FSS adjusted for baseline score, follow-up interval, age, and Padua score.

Results: Both groups improved significantly over time. SSS improved from 2.77 ± 0.78 to 1.64 ± 0.80 in non-operated and from 2.94 ± 0.83 to 1.58 ± 0.65 in operated patients (both $p < 0.001$). FSS improved from 2.47 ± 0.95 to 1.70 ± 0.81 in non-operated

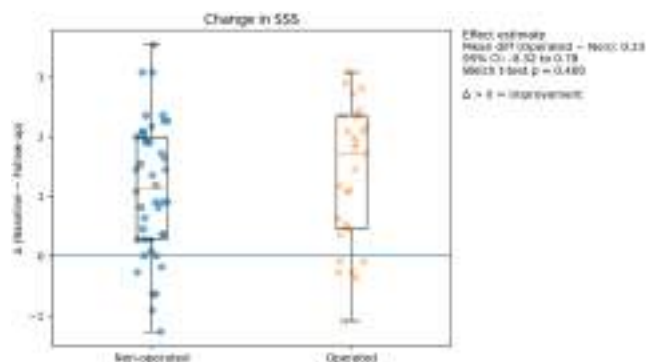


FIGURE 1 Change in symptom severity scale from baseline to follow-up by surgery status.

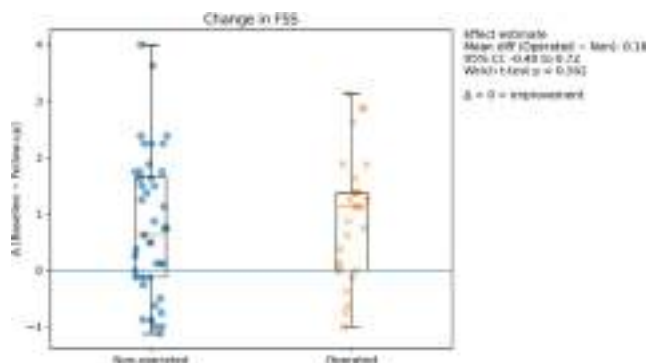


FIGURE 2 Change in functional status scale from baseline to follow-up by surgery status.

and from 2.58 ± 0.95 to 1.65 ± 0.75 in operated patients (both $p < 0.001$). Mean SSS improvement was 1.13 ± 1.12 vs. 1.36 ± 1.17 ($p = 0.40$; Figure 1). Mean FSS improvement was 0.77 ± 1.26 vs. 0.93 ± 1.11 ($p = 0.562$; Figure 2). In adjusted models, surgery was not independently associated with follow-up SSS ($\beta = -0.10$, 95% CI -0.49 to 0.28 ; $p = 0.60$) or FSS ($\beta = -0.04$, 95% CI -0.45 to 0.36 ; $p = 0.83$).

Conclusion: In this cohort, symptom and function scores improved over time regardless of surgery status. Surgery was not associated with greater longitudinal improvement in SSS or FSS after adjustment, suggesting that non-surgical factors and follow-up timing may substantially influence patient-reported outcomes.

Disclosure: Nothing to disclose.

OPR-057 | Diaphragmatic dysfunction after lung transplantation: Longitudinal assessments and intraoperative neurophysiological monitoring of the phrenic nerve

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Background and Aims: The diaphragm is the main respiratory muscle, controlled by the phrenic nerve, essential for breathing. Diaphragmatic dysfunction affects up to 43% of lung transplant (LTx) patients due to phrenic nerve injury from surgical factors, causing respiratory complications. This thesis combines two investigations: a prospective cohort study of longitudinal diaphragmatic function pre- and post-LTx, and an intraoperative neurophysiological monitoring sub-study of phrenic nerve damage dynamics.

Methods: Thirty patients were assessed for respiratory function, diaphragm ultrasound, and phrenic conduction preoperatively, at discharge, 6 and 12 months. Three patients underwent intraoperative testing at multiple surgical points, focusing on ice slush effects.

Results: Despite good clinical recovery (spirometry, 6MWT), phrenic cMAP amplitude reduced at discharge and 6 months but normalized by 12 months, underscoring multimodal monitoring needs. Intraoperative tests showed immediate cMAP drop with ice slush ($p < 0.05$) suggesting cryogenic axonal

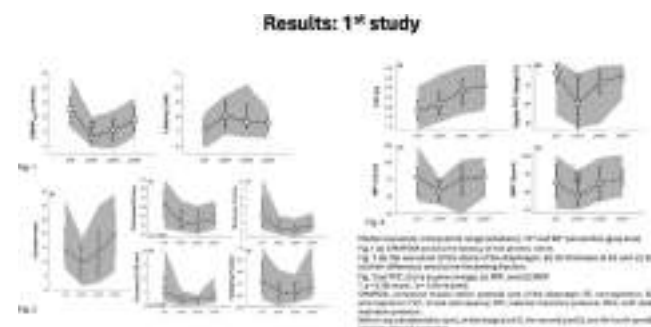


FIGURE 1 Longitudinal changes in phrenic nerve cMAP amplitude and respiratory parameters in 30 lung transplant patients.

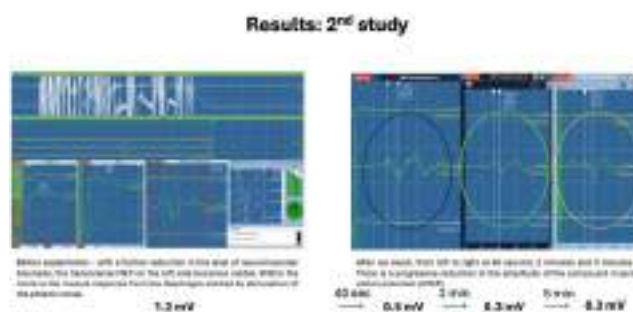


FIGURE 2 Intraoperative neurophysiological monitoring of phrenic nerve during lung transplantation. Immediate cMAP amplitude reduction after ice slush application, indicating cryogenic axonal damage.

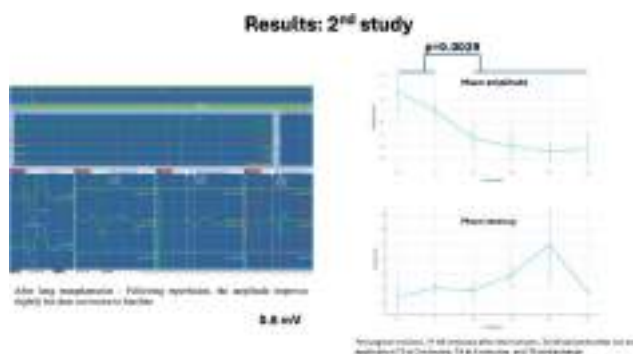


FIGURE 3 Statistical representation of phrenic cMAP reduction post-ice slush ($p = 0.0029$) and latency prolongation, indicating cryogenic nerve damage despite partial subsequent recovery.

damage, emphasizing the utility of intraoperative neurophysiological monitoring in detecting and potentially mitigating nerve damage.

Conclusion: This study highlights the importance of routine diaphragm assessment during and after surgery to identify phrenic nerve damage mechanisms, guide strategies to reduce respiratory complications, and ultimately improve management protocols and optimize transplant outcomes.

Disclosure: Nothing to disclose.

OPR-058 | Anti-PLP1 antibody as a biomarker in acute and chronic polyradiculoneuropathies

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Background and Aims: Up to 24% of patients with combined central and peripheral demyelination (CCPD) harbor IgG antibodies against proteolipid protein 1 (PLP1), a key component of CNS and PNS myelin. Given peripheral demyelination in these cases, we assessed anti-PLP1 prevalence in Guillain-Barré syndrome (GBS) and chronic inflammatory demyelinating polyradiculoneuropathy (CIDP).

Methods: Sera from GBS ($n=66$), CIDP ($n=46$), and CCPD ($n=16$) were tested for IgG/IgM against PLP1 using live cell-based assays on HEK293 cells as well as oligodendrocyte and myelinating Schwann cell/dorsal root ganglion cocultures. Clinical features of seropositive and seronegative patients were compared. Controls included autoimmune nodopathies ($n=25$), Charcot-Marie-Tooth disease ($n=20$), and healthy donors ($n=23$).

Results: Anti-PLP1 antibodies were found in 17% of GBS patients (IgG: $n=3$; IgM: $n=8$), associated with greater disability (ONLS 8 vs. 5; $p=0.019$), sensory-motor subtype (91% vs. 47%; $p=0.008$), AIDP (91% vs. 51%; $p=0.014$), and higher CSF protein (0.88 vs. 0.6 g/L; $p=0.011$). In CIDP, 24% were seropositive (IgG: $n=1$; IgM: $n=10$), linked to older age (66 vs. 55 years; $p=0.003$), greater disability (ONLS 6 vs. 4; $p=0.041$), and reduced IVIg response (36% vs. 70%; $p=0.05$). Four CCPD patients (25%) were positive. All controls were negative. Antibody binding to oligodendrocytes confirmed reactivity.

Conclusion: Anti-PLP1 antibodies, mainly IgM, correlate with severe demyelinating GBS and typical CIDP with greater disability and poorer IVIg response. These antibodies likely target conformational epitopes on myelin. Further studies are needed to confirm these findings and to determine the pathogenic actions of these antibodies.

Disclosure: Nothing to disclose.

OPR-059 | Evaluating peripherin as a biomarker of axonal damage in chemotherapy-induced peripheral neuropathy

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Background and Aims: Chemotherapy-induced peripheral neuropathy (CIPN) is a common, often dose-limiting adverse effect of several chemotherapeutic agents, preferentially affecting sensory neurons. Sensitive biomarkers of early axonal injury are currently lacking. Neurofilament light chain (NfL) is not specific to sensory neurons. As an initial, proof-of-concept approach, we investigated whether peripherin, an intermediate filament enriched in peripheral sensory axons, could serve as a biomarker of CIPN using human induced pluripotent stem cell (iPSC)-derived sensory neuron cultures, aiming to inform subsequent evaluation in patient samples.

Methods: Human iPSC-derived sensory neurons were exposed to a range of concentrations of vincristine or paclitaxel. Culture media were collected at 24, 48, and 72 h and analysed for peripherin using Simoa and NfL using an electrochemiluminescence assay. Axonal integrity was quantified by an axonal degeneration index using immunostaining and confocal microscopy. To explore mechanistic linkage, parallel cultures were treated with a small-molecule SARM1 inhibitor to block the Wallerian degeneration pathway.

Results: Peripherin release into culture media increased in a dose- and time-dependent manner with both vincristine and paclitaxel, correlating with the progression of the axonal

degeneration index and levels of NfL. Levels of both biomarkers were lower in cultures treated with SARM1 inhibitor, indicating reduced axonal degeneration.

Conclusion: In vitro, peripherin shows promise as a biomarker of chemotherapy-related axonal damage. Work is underway to measure peripherin and NfL in large, deeply phenotyped patient cohorts of CIPN. We will determine whether axonal fluid biomarkers correlate with clinical features and existing outcome measures, and evaluate their individual and combined contributions to clinical evaluation.

Disclosure: Nothing to disclose.

Coma and Chronic Disorders of Consciousness

OPR-060 | Reconstructing treatment preferences for patients with disorders of consciousness: A longitudinal qualitative study

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Background and Aims: For patients with neurological conditions that impair their capacity to participate in shared decision-making, guidelines recommend that physicians help surrogates to reconstruct these patients' treatment preferences. This study's objective was to explore the way physicians reconstruct preferences for patients with disorders of consciousness (DoC).

Methods: This 2-year longitudinal qualitative case study took place in the specialized Dutch chain of care patients with DoC. Eligible patients (>18 years of age) had a Coma Recovery Scale-revised based diagnosis of unresponsive wakefulness syndrome or minimally conscious state. Interactions between physicians and surrogates were audio-recorded. Additionally, we interviewed healthcare professionals.

Results: Characteristics of included patients are reported in Table 1. Across 16 observed healthcare trajectories, 46 physician-surrogate interactions were audio-recorded and 32 healthcare professionals were interviewed. We identified five recurring activities physicians' support of surrogates in reconstructing treatment preferences (Table 2): (1) clarifying responsibilities, (2) exploring clues that point to patient's preferences, e.g., the patient's described personality or identity and verbal advance directives, (3) discussing the preferences revealed by the patient's behavior, (4) establishing the minimal level of functioning thought to result in an acceptable quality of life, and (5) discussing sources of surrogate bias. More interactions involved an exploration of patients' personality or identity (51.2%) than prior expressed wishes (25.6%). Inferring preferences from the patient's behavior was important for continuing or withdrawing life-sustaining treatment.

Case	gender	Age at inclusion	Religion ^a	Race of inclusion ^b	Injury ^c	Time at inclusion ^d	Interaction (total minutes) ^e	WCP interview ^f
001	Male	<19	Rome	ERI	TB	UW1	7 (130)	9
002	Male	30-39	Rome	CC	TB	RC3 (+)	3 (113) ^g	3
003	Female	40-49	Rome	ERI	PE	RC3 (+)	3 (12)	2
004	Female	40-49	Yes	ERI	TB	UW1	8 (104)	2
005	Male	40-49	Yes	CC	TB	RC3 (+)	1 (30)	2
006	Female	40-49	Yes	ERI	CVA	UW1	1 (40)	—
007	Male	40-49	Rome	ERI	CVA	RC2 (-)	7 (105)	9
008	Female	40-49	Rome	Rome	PE	RC3 (+)	3	3
009	Female	20-29	Rome	Rome	PE	RC3 (-)	0	1
010	Female	40-49	Yes	FR	CVA	RC3 (+)	2 (54) ^h	1
011	Female	40-49	Yes	FR	CVA	RC3 (+)	3 (30)	1
012	Female	20-29	Rome	ERI	CVA	RC3 (+)	1 (23)	—
013	Female	40-49	Yes	ERI	CVA	RC3 (-)	3 (124)	2
014	Female	65-69	Rome	FR	CVA	RC3 (+)	0	1
015	Female	60-69	Yes	Rome	PE	RC3 (+)	1 (30)	—
016	Male	20-29	Yes	FR	TB	RC3 (+)	2 (11)	1

* LWB = undergraduate wilderness leadership; MCI = medically conscious state. *Respected as "healthcare professional." Participants requested withdrawal from the study. Therapies did not consent to audio-recording of the interaction.

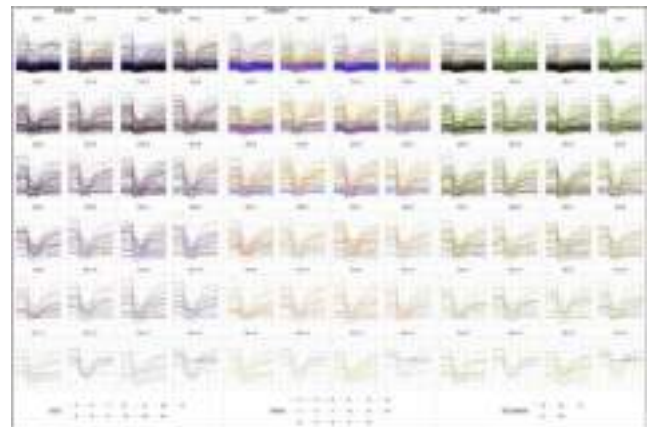
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Disclosure: Nothing to disclose.

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Methods: In a prospective longitudinal cohort ($n=250$) with impaired consciousness after traumatic or non traumatic brain

Results: Late LOR latency was dissociated from same day behavioral responsiveness yet independently predicted improvement



A PLR and late LOR: Mixed-effects modeling of current consciousness ('today')
 PLR is associated with same-day PFCM score, but not LOR

B Sedation-dependent effect of late LOR on consciousness recovery ('next week')
 Late LOR predicts PFCM 7 days later in non-sedated patients

C Etiology-dependent effect of late LOR on consciousness recovery ('next week')
 Late LOR and 7-day recovery in the fully sedated model

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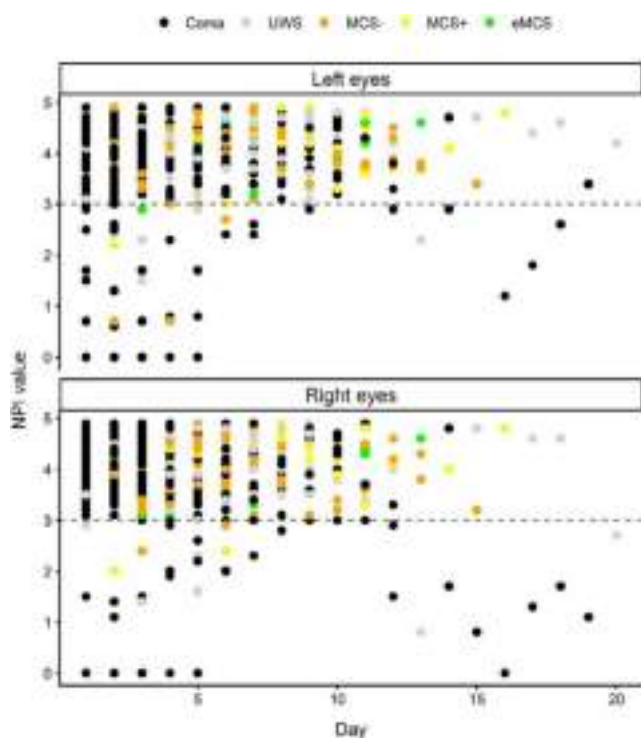


FIGURE 3 Longitudinal NPi values across ICU days and levels of consciousness. Daily NPi recordings are shown for 250 DoC patients.

in FOUR scores seven days later, beyond baseline FOUR and time since injury. The prognostic relationship was modified by sedation status and etiology, with effects most evident in non-sedated patients and predominantly among those with anoxic-ischemic injury (etiology interactions exploratory). In contrast, PLR based metrics, including Neurological Pupil Index and PLR latency, did not predict later gains in consciousness despite being within normal ranges early after injury.

Conclusion: Conclusion: Late LOR latency captures recovery related pupillary dynamics that anticipates clinically meaningful improvement in consciousness.

Disclosure: This project was funded by the Lundbeck Foundation (R507-2025-286) and the Independent Research Fund Denmark. Foundation (5333-00034B). Additional funding was supplied by Offerfonden. Material execution, content and results are the sole responsibility of the authors. The assessments and views expressed in the material are the authors' own and are not necessarily shared by the Offerfonden.

OPR-062 | Baseline pupillometry reveals dissociation between the pupillary light reflex and late light off response in acute brain injury

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Background and Aims: We investigated whether distinct components of the automated pupillary reflex – including the

pupillary light-off response (LOR) – measured in the intensive care unit (ICU) reflect level of consciousness after acute brain injury, beyond summary metrics such as the Neurological Pupil Index (NPI).

Methods: In a prospective cohort of 250 ICU patients with acute brain injury and impaired consciousness, alongside 30 age- and sex-matched healthy controls, we performed standardized automated pupillometry during the early ICU period. Within a single measurement, we quantified the pupillary light reflex (PLR), decomposed LOR into early and late components, and computed late LOR latency (T50: time to 50% dilation ≥ 3 s after light offset), normalized to controls. Associations with concurrent level of consciousness, measured by the Full Outline of UnResponsive-ness (FOUR) score, were assessed using mixed effects models adjusted for ICU Day, baseline severity, sedation, and injury etiology.

Results: In acute coma, PLR and early LOR components were largely preserved. In contrast, late LOR latency was markedly attenuated despite normal NPi-values and showed substantial interindividual variability. Unsupervised hierarchical clustering of raw pupillary waveforms demonstrated a selective loss of delayed pupillary dilation early after injury. PLR latency showed a strong association with same day FOUR scores, whereas late LOR latency was not correlated with concurrent behavioral responsiveness.

Conclusion: Pupillometry reveals dissociable features of the pupillary reflex arc in coma which are not captured by conventional metrics, indicating that PLR and late LOR index distinct aspects of brainstem–arousal network function early after brain injury.

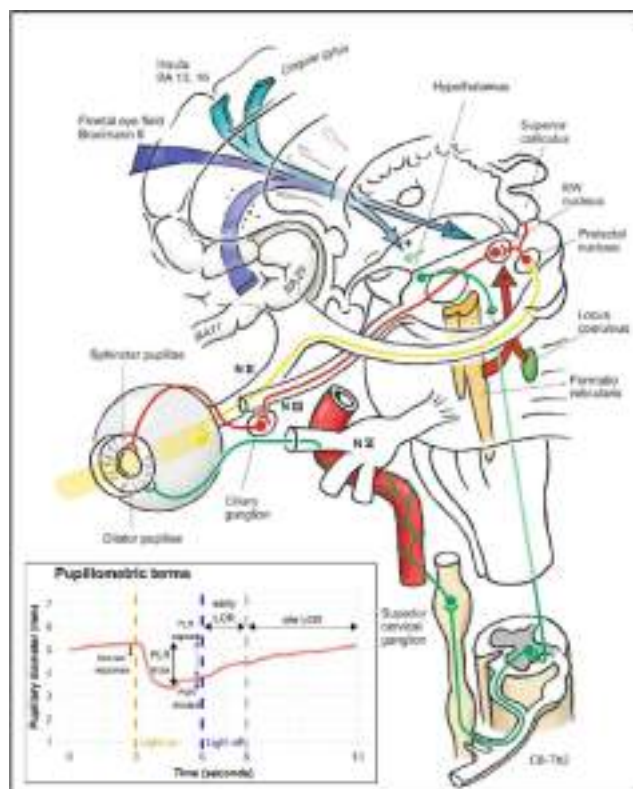


FIGURE 1 Neural pathways underlying pupillary reflexes and terminology used in this study.

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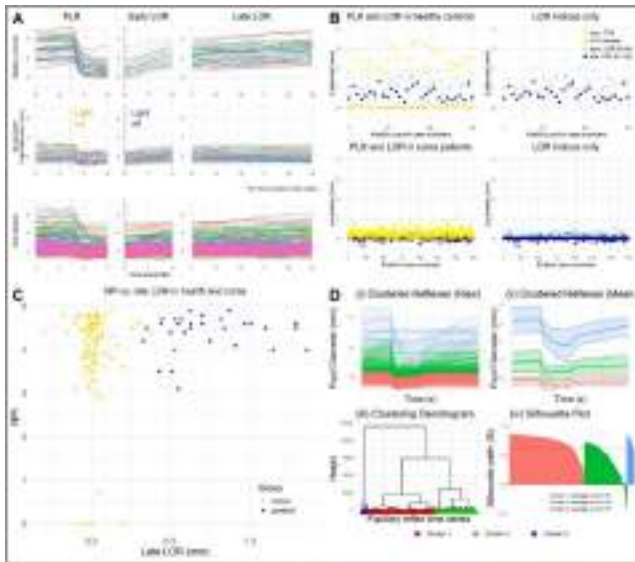


FIGURE 2 Pupillary indices in healthy controls and acute coma (ICU day 1).

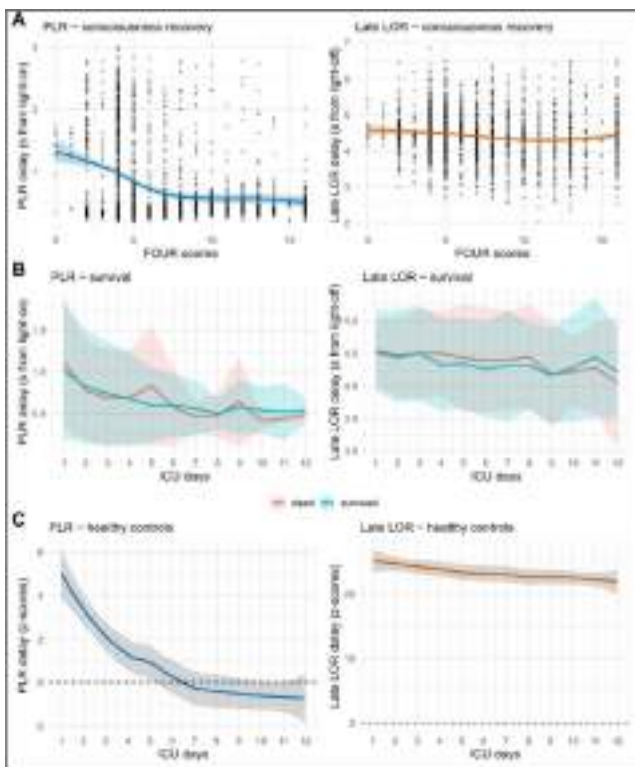


FIGURE 3 Dissociation between pupillary constriction, late dilation, clinical outcomes, and normalization to healthy controls.

Disclosure: This work was funded by the Lundbeck Foundation (R507-2025-286) and the Independent Research Fund Denmark. Foundation (5333-00034B). Additional funding was supplied by Offerfonden. Material execution, content and results are the sole responsibility of the authors. The assessments and views expressed in the material are the authors' own and are not necessarily shared by the Offerfonden.

Background and Aims: Prolonged disorders of consciousness (PDOC) include the unresponsive wakefulness syndrome and the minimally conscious state. Long-term outcomes following specialized intensive neurorehabilitation are largely unknown, complicating personalized decision-making. This study aimed to determine the 2-year outcomes of PDOC patients in the Netherlands regarding consciousness, mortality, disability, functioning, and independence.

Methods: Prospective cohort study including PDOC patients ≥ 16 years admitted to the nationwide Dutch PDOC chain of care (2019–2023). Baseline measurements at admission to Early Intensive Neurorehabilitation (EIN): Coma Recovery Scale-Revised (CRS-R), Disability Rating Scale (DRS), Functional Independence Measure (FIM). Follow-up at 14, 28, 40, 52, and 104 weeks post-admission. Mortality, recovery of consciousness, functional outcomes, and independence (FIM ≥ 78) were analysed using descriptive statistics and generalized estimating equations.

Results: Of 129 enrolled patients (mean age 38 years; 52% male), 43% regained consciousness during EIN, with an additional 19% within 2 years. Overall, 62% recovered consciousness. Functional independence increased from 4% at 14 weeks to 41% at 2 years among conscious survivors. The most substantial improvements in DRS and FIM occurred between 14–28 weeks post-admission, with continued, though smaller, gains up to 2 years. Mortality was 26%, predominantly following a decision to withhold or withdraw life-sustaining treatment.

Conclusion: PDOC patients demonstrate significant recovery of consciousness and functional independence during the first 2 years of specialized rehabilitation, predominantly within the first 9 months. These findings emphasize the importance of early, intensive, and prolonged neurorehabilitation and support informed decision-making in severe acquired brain injury.

Disclosure: Nothing to disclose.

OPR-064 | Alterations of cerebellar volumes and functional connectivity and gait changes in Parkinson's disease subjects with freezing of gait

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Background and Aims: Cerebellum plays an increasingly recognized role in gait modulation and freezing of gait (FoG) in Parkinson's disease (PD). This study investigated gait kinematics, cerebellar structural and functional connectivity (FC) changes in PD patients with and without FoG (PD-FoG, PD-NO-FoG).

Methods: Thirty PD-FoG, 30 PD-NO-FoG (with postural instability and gait disorders), and 30 healthy controls (HC) underwent clinical, gait analysis and MRI evaluations. Seed-based FC used cerebellar vermis as region of interest, based on volumetric findings.

Results: PD-FoG showed lower balance confidence and greater impact of symptoms on daily activities than PD-NO-FoG, while cognitive performance was similar. PD groups exhibited reduced stride length, foot-strike angle, and gait velocity. PD-FoG displayed higher asymmetry, slower Timed-Up-and-Go (TUG) relative to HC, and slower turning velocity, greater stride variability, and shorter turning stride length than PD-NO-FoG; while PD-NO-FoG showed reduced trunk mobility and gait rhythm relative to HC. MRI analyses revealed reduced cerebellar lobule-VI volume in PD vs. HC. PD-FoG showed increased volumes of lobule-VIIIb, vermis-X, and vermis-Crus-II then PD-NO-FoG. Resting-state FC analyses demonstrated increased vermis connectivity with frontal and cerebellar areas (VI, IX, vermis-VII, and Crus-I) in PD-FoG relative to HC, while PD-NO-FoG showed enhanced FC between vermis and postcentral, middle temporal, and occipital cortices. In PD-FoG, increased cerebellar FC correlated with increased cerebellar volume,

which also correlated with worse gait parameters; reduced frontal areas FC correlated with worse depression.

Conclusion: PD-FoG patients displayed specific alterations. Increased FC of cerebellum suggests an attempt to hyper-control movement that may result in increased volume related to overuse.

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OPR-065 | NIO752, a tau-targeting antisense oligonucleotide, in progressive supranuclear palsy: Phase I results and phase III design

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Background and Aims: Progressive supranuclear palsy (PSP) is a rare, debilitating primary tauopathy with no effective therapies. NIO752, an antisense oligonucleotide (ASO), inhibits MAPT mRNA translation, reducing production of all tau protein isoforms and pathogenic aggregation into neurofibrillary tangles, and thereby potentially attenuating tau-mediated neurodegeneration. NIO752 is being investigated in PSP for its potential to slow clinical progression. Here we present results from

the first-in-human Phase I study (NCT04539041) of NIO752 in PSP and Phase III development plans.

Methods: In the Phase I, randomized, double-blind, placebo-controlled, multiple dose-escalation study, eligible patients had a PSP diagnosis for <5 years classified as probable PSP Richardson syndrome (PSP-RS). Patients received intrathecal NIO752 or placebo. The Phase III randomized, double-blind, placebo-controlled, parallel group study will enroll patients with possible/probable PSP-RS for <5 years to receive NIO752 or placebo. The goal is to evaluate efficacy and safety of NIO752.

Results: In the Phase I study, 59 patients were enrolled to receive NIO752 ($n=45$) or placebo ($n=14$). Median time since diagnosis was 1.6 years (range: 0.3–4.8 years). NIO752 had an acceptable safety profile; most adverse events were mild to moderate and consistent with the nature of PSP. Serious adverse events were reported in treatment and placebo cohorts at similar rates. Pharmacokinetic profile and exploratory biomarker data will also be presented.

Conclusion: NIO752 is the first MAPT ASO investigated as a potential PSP treatment. Phase I results support proceeding to Phase III to establish the potential of NIO752 as a disease-modifying therapy in PSP-RS.

Disclosure: Adam Boxer: Paid consultant to Novartis Mirek Brys: Novartis employee and stockholder Ana Graf: Novartis employee and stockholder Handan Gunduz-Bruce: Novartis employee and stockholder Gunter Hoglinger: Received consultancy fee from Novartis Lidija Kovacic: Novartis employee and stockholder Abhijit Pethe: Novartis employee and stockholder Nicole Pezous: Novartis employee and stockholder Carmen Serban: Novartis employee and stockholder.

OPR-066 | Efficacy and safety of dimethyl fumarate in Friedreich ataxia: A phase 2, randomized, double-blind, placebo-controlled DMF-FA-201 trial

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Background and Aims: FA is an autosomal recessive neurodegenerative disease. Compared to controls, FXN gene expression is 20% in patients and 50% in healthy carriers. Treatment of multiple sclerosis patients with DMF is known to increase FXN expression by 70%. Objective of the study was to evaluate the efficacy and safety of Dimethyl fumarate (DMF) in patients with Friedreich Ataxia (FA).

Methods: We conducted a phase II trial with a randomized, placebo-controlled, 12 weeks phase, and an identical extension open-label phase (Figure 1). Primary endpoint was the efficacy of DMF on FXN gene transcription. Secondary objectives were: frataxin protein, stimulation of the nrf2 pathway and mitochondrial biogenesis, safety and tolerability, and clinical measures of disease (Figure 2). EudraCT number: 2021-006274-23.

Results: 140 patients (female, 57.5%; mean [SD] age, 36.1 [12.6] years; disease duration 16.8 [6.9] years), were randomized (DMF $n=20$, placebo $n=20$). At the end of the core phase, 33 patients continued to the open-label extension. DMF increased FXN gene expression after 12 weeks of treatment (difference compared to

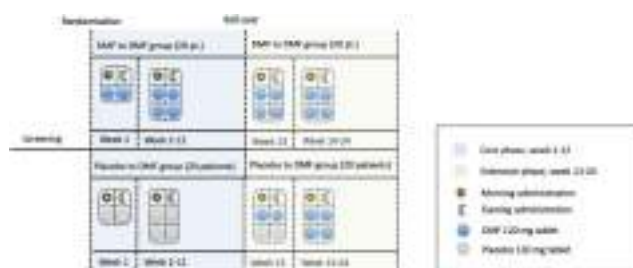


FIGURE 1



FIGURE 2

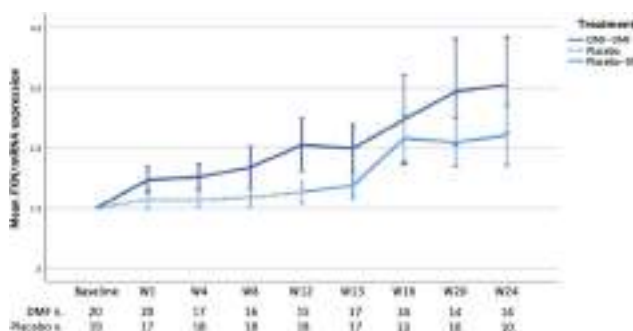


FIGURE 3

placebo +119.0%, 95% CI +55.3, +182.7, $p=0.018$; Figure 3). In the DMF-DMF group FXN expression increased to +205% after 24 weeks compared to baseline ($p<0.001$). Secondary endpoints showed a non-significant trend towards improvement after treatment. Frequent adverse events were gastrointestinal, flushing, eosinophilia, lymphopenia, and elevated liver enzymes.

Conclusion: DMF significantly increases transcription of the FXN gene, with long-term stability, to a degree that restores expression levels to those of healthy carriers. The drug appeared safe, with adverse events consistent with DMF pharmacovigilance.

Disclosure: Francesco Saccà received public speaking honoraria from Alexion, argenx, Biogen, Genpharm, Johnson&Johnson, Medpharma, Medison Pharma, Neopharm Israel, Novartis, UCB, Zai Lab; he also received compensation for Advisory boards or consultation fees from Alexion, Amgen, argenx, Astrazeneca, Avexis, Biogen, Dianthus, Johnson&Johnson, Lexeo, Novartis GmbH, Reata, Sandoz, UCB, Zai Lab; he is PI in clinical trials for Alexion, argenx, Carthesian, Dianthus, Immunovant, Lediand, Lexeo, Novartis, Prilenia, Remgen, Sanofi. The other authors report no disclosures. Funding: Agenzia Italiana del Farmaco (AIFA), Associazione Italiana per la lotta alle Sindromi Atassiche

OPR-067 | Safety and efficacy of dual-target MRgFUS thalamotomy for dystonic tremor: A prospective single-centre cohort

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Background and Aims: MR-guided focused ultrasound (MRgFUS) is an established treatment for essential tremor when targeting the ventral intermediate nucleus (VIM), whereas its efficacy in dystonic tremor (DT) remains uncertain. Emerging evidence suggests that a more anterior targeting may improve clinical outcomes. Based on the involvement of distinct circuits, we explored a dual-target approach combining VIM and Ventralis oralis (Vo) targeting.

Methods: Initial treatment targeted the VIM using standard stereotactic coordinates. In cases of partial tremor control, additional targeting of the most anterior (i.e., pallidal) portion of the Vo was performed. Efficacy was assessed by changes in CRST A + B scores of the treated hand at 1 and 6 months, while functional outcomes were evaluated using CRST Part C. Safety was assessed through systematic collection and grading of adverse events (AEs).

Results: Fifteen patients (6 female; mean age 68.3 ± 5.8 years) with DT underwent dual-target thalamotomy. Significant improvements in tremor severity were observed at both follow-up timepoints, reflected by reductions in CRST A + B scores of the treated hand (-56% and -60% at 1 and 6 months, respectively). Functional improvement was observed on CRST Part C (-60% and -61% at 1 and 6 months). A total of 22 AEs were recorded

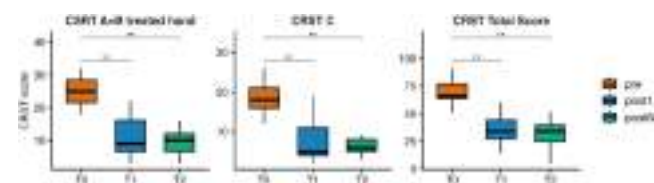


FIGURE 1 Boxplots of the Clinical Rating Scale for Tremor (CRST) scores at T0, T1, and T2, showing a significant post-treatment reduction in CRST A + B for the treated hand, CRST C, and total CRST scores.

across all timepoints, all of which were mild. At the last available follow-up, only two AEs persisted (worsening of pre-existing

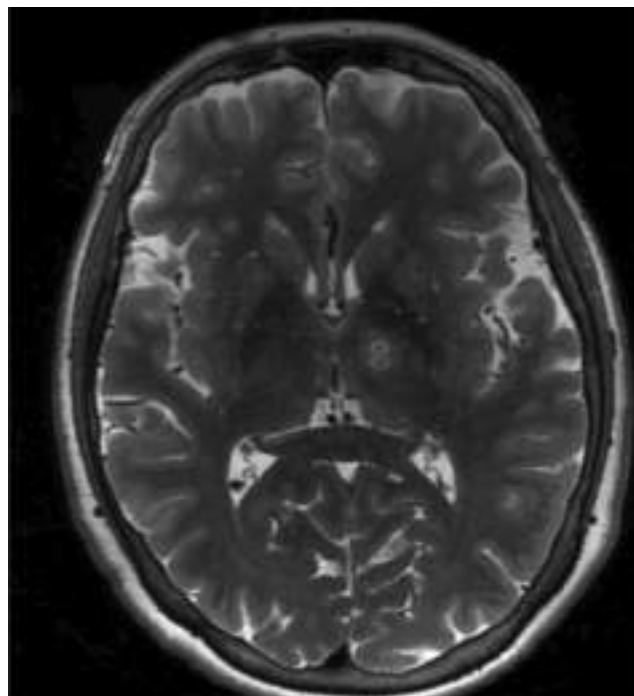


FIGURE 2 Axial T2 MRI sequence showing dual lesion at 1-month follow-up.

gait instability and finger hypoesthesia), both occurring in the same patient.

Conclusion: These preliminary findings suggest that a dual-target MRgFUS thalamotomy approach is feasible in patients with DT and is associated with an acceptable safety profile.

Disclosure: Nothing to disclose.

OPR-068 | Motor outcomes and network effects of adaptive deep brain stimulation programming in Parkinson's disease

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Background and Aims: In Parkinson's disease (PD), adaptive deep brain stimulation (aDBS) of the subthalamic nucleus (STN) may enhance clinical benefits and reduce side effects compared with conventional DBS (cDBS). In single-drive aDBS (SD-aDBS), bilateral stimulation is modulated using local field potentials (LFPs) recorded from one hemisphere, making interhemispheric coupling a critical determinant of appropriate

stimulation. Here, we investigated the effects of chronic cDBS and SD-aDBS on subthalamic interhemispheric coupling in PD. **Methods:** Twelve PD patients were treated in a randomized, double-blind crossover design with cDBS and SD-aDBS (AlphaDBS, Newronika) for two consecutive weeks per condition, with dopaminergic medication unchanged. In SD-aDBS, stimulation amplitude was adjusted every minute using a linear proportional algorithm based on patient-specific beta-range STN-LFP amplitude. Motor symptoms were assessed using the MDS-UPDRS-III at baseline (stim-OFF/meds-OFF) and after each treatment period (stim-ON/meds-ON). ON- and OFF-time percentages were derived from Hauser diaries. Continuous STN-LFP recordings were decomposed into periodic and aperiodic spectral components. Interhemispheric coupling was quantified using Pearson's r^2 for aperiodic offset and slope, and low-beta (12–20 Hz) and high-beta (21–30 Hz) oscillatory amplitudes.

Results: cDBS and SD-aDBS yielded comparable motor improvement (mean MDS-UPDRS-III change: 59% vs. 58%), with greater ON-time during SD-aDBS. Ten of twelve patients blindly preferred SD-aDBS. Interhemispheric coupling was significant under both conditions. Low-beta interhemispheric coupling was higher during cDBS than SD-aDBS and, during SD-aDBS, positively correlated with OFF-time.

Conclusion: Chronic cDBS and SD-aDBS differentially modulate subthalamic interhemispheric coupling, with a greater reduction during SD-aDBS. This mechanism may contribute to its additional clinical value.

Disclosure: M.A. is an employee of Newronika S.p.A. L.R., S.M., and A.P. are founders and shareholders of Newronika S.p.A. I.U.I. is a Newronika S.p.A. consultant and shareholder and received funding for research activities from Newronika S.p.A. I.U.I. received lecture honoraria and research funding from Medtronic Inc. I.U.I. is an Adjunct Professor at the Department of Neurology, NYU Grossman School of Medicine.

OPR-069 | α -Synuclein-associated mitochondria-Nrf2 dysfunction in Parkinson's disease olfactory mucosa

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Background and Aims: In this study we outline the dynamics of the mitochondrial network and cytoprotective response in patients with Parkinson's disease (PD)-derived olfactory mucosa neurons (ONs) across disease stages.

TABLE 1 Demographic, clinical, and biochemical features of the study population.

Variable	PD (n=41)	CTRL (n=28)	P	PDdn (n=24)	PDtr (n=17)	P
Age, yr	61.2 (±9.2)	59.8 (±18.1)	n.s.	61.3 (±9.4)	58.8 (±9.0)	n.s.
Sex (M/F)	26/15	12/16	n.s.	13/7	9/8	n.s.
Disease duration, yr	3.7 (±3.1)	—	—	3.92 (±1.2)	6.2 (±4.1)	<0.001
Medication, yr	—	—	—	—	—	—
MDS-UPDRS III	21.4 (±8.9)	—	—	28.96 (±9.2)	31.4 (±8.1)	n.s.
HoN	2.2 (±0.38)	—	—	2.08 (±0.6)	2.31 (±0.46)	n.s.
HoA	17.3 (±21.7)	—	—	31.2 (±29.3)	41.1 (±28.2)	n.s.
SCOPA-AUT	11.3 (±7.7)	—	—	6.5 (±5.9)	17.3 (±14.5)	n.s.
MMSE	26.1 (±2.2)	—	—	25.1 (±2.3)	26.2 (±1.9)	n.s.
HoCA	24.8 (±3.8)	—	—	34.3 (±1.4)	35.2 (±4.1)	n.s.
HRV	23.7 (±2.4)	—	—	23.3 (±1.8)	21.5 (±4.2)	n.s.
LEDD	225.1 (±366)	—	—	9	113 (±248.6)	<0.001

Values are reported as mean (±SD) where applicable; n.s. = not significant.

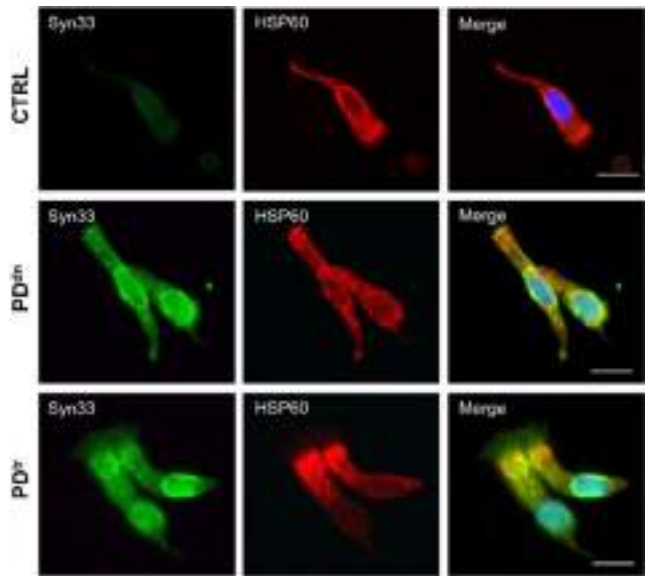


FIGURE 1 Immunolocalization of oligomeric α -syn and mitochondria. (A) Representative immunofluorescence of oligomeric α -syn (Syn33, green) and HSP60 (red) in ONs from healthy controls and PD. Nuclei: DAPI (blue). Scale bar: 10 μ m.

Methods: ONs obtained by nasal brush from 41 well-phenotyped patients with PD ($n=24$ PD de novo [PDdn] and $n=17$ under treatment [PDtr]) and 29 healthy controls were examined through real-time polymerase chain reaction (RT-PCR), immunofluorescence, and Western blot. An integrative set of experiments using SH-SY5Y neuronal cells was also performed. **Results:** PD ONs accumulated α -synuclein oligomers in association with an aberrant subcellular distribution of mitochondrial markers COX IV and HSP60. DJ-1 ONs expression was permanently reduced in PD, revealing the mitochondrial dysfunction and justifying the defective Nrf2-mediated cytoprotective response. The Nrf2/SOD-1 pathway was indeed downregulated in PD ONs, although with stage-specific differences. In PDdn ONs, Nrf2 mostly presented in the inactive cytosolic localization with a major reduction of SOD-1 expression, whereas in PDtr, the Nrf2 active nuclear fraction increased, and the SOD-1

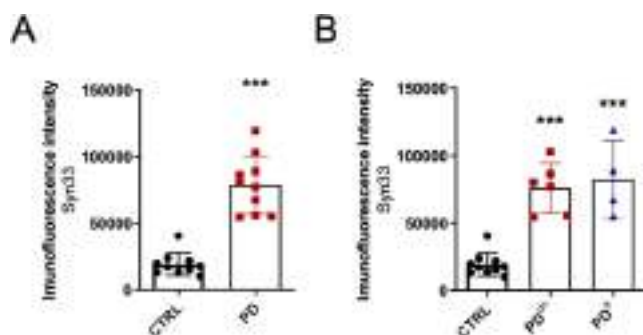


FIGURE 2 Graphs showing immunofluorescence intensity of oligomeric α -syn in each group, quantified with ImageJ. Data are mean $\pm$ SEM. Asterisks indicate significant differences by unpaired t test or 1-way ANOVA with Tukey post hoc. *** $p < 0.001$.

expression raised. In SH-SY5Y cells, we demonstrated that dopamine administration increases the Nrf2 nuclear fraction, acting as a possible pathway's inducer.

Conclusion: Human-derived ONs may recapitulate PD pathogenic milestones, exhibiting stage-specific interactions among α -synuclein oligomers, mitochondrial metabolism, and cytoprotective response. These findings highlighted mitochondrial dysfunction as primary target for therapeutic interventions and a potential axis for the biological stratification of patients. Moreover, they supported the translational value of ONs, as a source of biomarkers or models, which is critical in the current changing paradigm of PD toward a biological-based approach.

Disclosure: JB: Italian National Recovery and Resilience Plan (PNRR) "Next Generation EU", through the Italian Ministry of Health (MCNT1-2023-12378408), project titled "Exploring the upper airways through an integrated multidisciplinary approach to tackle pathogenic trajectories and disclose novel targets in Parkinson disease" (CUP MASTER E83C24000720001). DM: the author declares that there are no additional disclosures to report. MGC: the author declares that there are no additional disclosures to report. RM: the author declares that there are no additional disclosures to report. FV: the author declares that there are no additional disclosures to report. MR: the author declares that there are no additional disclosures to report. DM: the author declares that there are no additional disclosures to report. RB: the author declares that there are no additional disclosures to report. CS: the author declares that there are no additional disclosures to report. MM: the author declares that there are no additional disclosures to report. VB: the author declares that there are no additional disclosures to report. SV: the author declares that there are no additional disclosures to report. AG: the author declares that there are no additional disclosures to report. MGM: the author declares that there are no additional disclosures to report. AMR: the author declares that there are no additional disclosures to report. FG: the author declares that there are no additional disclosures to report. LS: the author declares that there are no additional disclosures to report. AS: the author declares that there are no additional disclosures to report. FMP: the author declares that there are no additional disclosures to report. SDG: the author declares that there are no additional disclosures to report. MP: the author declares that there are no additional disclosures to report. CV: the author declares that there are no additional disclosures to report. AF: the author declares that there are no additional disclosures to report. DC: the author declares that there are no additional disclosures to report. RL: the author declares that there are no additional disclosures

to report. CS: the author declares that there are no additional disclosures to report. TS: Italian National Recovery and Resilience Plan (PNRR) "Next Generation EU", through the Italian Ministry of Health (MCNT1-2023-12378408), project titled "Exploring the upper airways through an integrated multidisciplinary approach to tackle pathogenic trajectories and disclose novel targets in Parkinson disease" (CUP MASTER E83C24000720001).

Autonomic Nervous System Diseases 2

OPR-070 | Haemodynamics of supine graded exercise and orthostatic blood pressure in autonomic failure

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Background and Aims: The prevalence of exercise-induced hypotension and exacerbation of post-exercise orthostatic hypotension across phenotypes of autonomic failure is currently not well documented. In this work, we aimed to fill this gap as both symptoms can significantly impair mobility, quality of life and rehabilitation efforts.

Methods: We performed a retrospective analysis of blood pressure (BP) and heart rate (HR) recorded with Dinamap during supine rest, supine graded cycling exercise (25W, 50W, 75W) and an active standing test before and after exercise. Additionally, the orthostatic intolerance ratio (OIR) was calculated. Participants ($n=140$) were diagnosed with Parkinson's disease/Lewy body dementia (PD/LBD, $n=32$), multiple system atrophy (MSA, $n=16$), pure autonomic failure (PAF, $n=41$), autoimmune autonomic ganglionopathy (AAG, $n=13$), peripheral autonomic neuropathy ($n=27$), or rare disorders featuring autonomic failure ($n=11$).

Results: Systolic BP (SBP) did not increase during exercise in 69 (52%) people (Figure 1, Table 1). Different patterns were observed in post-exercise supine recovery of SBP (Figure 2). The magnitude of the SBP drop upon standing did not increase post-exercise compared to pre-exercise, but the absolute SBP

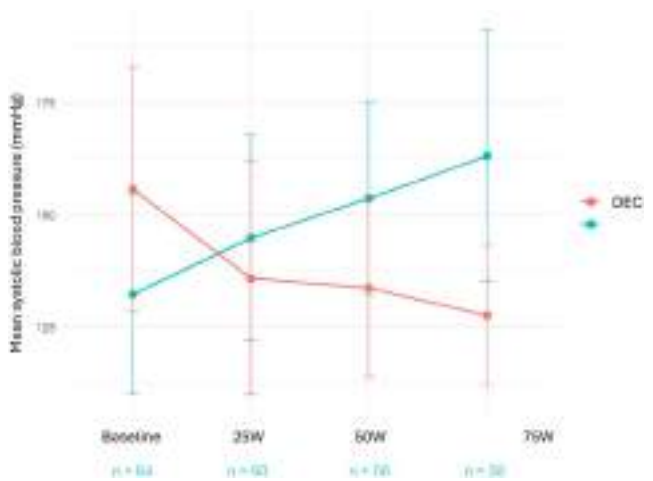


FIGURE 1 Systolic blood pressure during supine graded exercise, split into those who had an increase in systolic blood pressure during exercise (INC) and those who had a decrease during exercise (DEC) (-19.6 mmHg per exercise increment, $p < 0.001$).

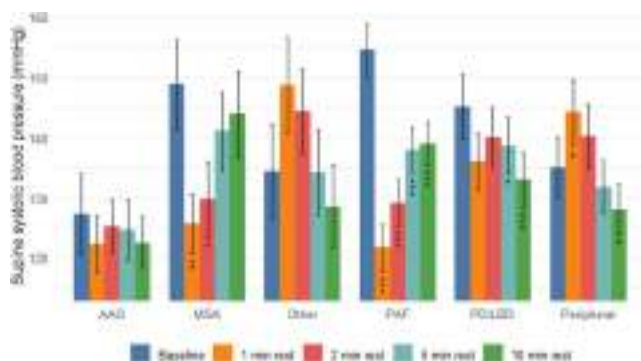


FIGURE 2 Recovery of supine systolic blood pressure during a 10-min post-exercise rest. Measurements were taken at 1, 2, 5 and 10 min after cessation of exercise, and compared to the pre-exercise supine baseline. * $p < 0.05$; ** $p < 0.001$.

TABLE 1 Blood pressure response during and after supine graded exercise. Includes supine and standing SBP, change in SBP and the Orthostatic Intolerance Ratio (OIR). * $p < 0.05$; ** $p < 0.001$.

	SBP decrease during exercise	Supine SBP (10 min post-exercise)	Standing SBP (post vs pre-exercise)	Delta SBP	OIR
PD/LBD	11 (38%)	+	+	+	+
MSA	10 (67%)	+	+	+	+
PAF	20 (73%)	+	+	+	+
Peripheral	0 (33%)	+	+	+	+
AAG	7 (54%)	+	+	+	+
Other	3 (20%)	+	+	+	+

was lower in those with alpha-synucleinopathies (Table 1). The OIR only worsened in those with MSA (Table 1). HR increased during exercise and did not fully recover during 10 min of post-exercise supine rest.

Conclusion: An abnormal BP response to exercise is common in autonomic failure, and the baroreflex impairment in alpha-synucleinopathies exacerbates orthostatic hypotension post-exercise. Both should be considered when advising on physical activity and rehabilitation in clinic.

Disclosure: Nothing to disclose.

OPR-071 | Autonomic involvement across the spectrum of genetic prion diseases

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Background and Aims: Genetic prion diseases (gPrDs) are fatal neurodegenerative disorders caused by pathogenic PRNP mutations. Among them, fatal familial insomnia (FFI) and PrP systemic amyloidosis (PrP-SA) show prominent autonomic nervous system involvement. FFI is marked by selective thalamic degeneration, leading to sleep disruption and cardiovascular sympathetic dysregulation. PrP-SA, in contrast, involves

systemic amyloid deposition affecting peripheral organs and nerves, resulting in early gastrointestinal dysfunction, sensorimotor neuropathy, and widespread autonomic failure.

Methods: This study aimed to assess cardiovascular autonomic involvement across gPrDs, focusing on FFI and PrP-SA. We retrospectively evaluated 5 patients with FFI, 6 with PrP-SA, and 3 asymptomatic PRNP variant carriers. Cardiovascular autonomic function was assessed using standardized autonomic testing (AFT) with Finapres® NOVA plus and 24-h blood pressure monitoring.

Results: At the time of assessment (median age 44 years; IQR: 33–52), all symptomatic patients ($n = 11$) reported autonomic symptoms, predominantly affecting cardiovascular, gastrointestinal, and urogenital systems. FFI patients exhibited sympathetic overactivity – supine hypertension, nocturnal hypertension, and resting tachycardia – alongside AFT evidence of cardiovagal and adrenergic impairment. In contrast, PrP-SA showed combined sympathetic and parasympathetic failure, characterized by neurogenic orthostatic hypotension, reduced heart rate variability, and poorer postural tolerance than FFI patients. Asymptomatic carriers displayed subtle AFT abnormalities despite the absence of clinical symptoms.

Conclusion: FFI and PrP systemic amyloidosis exhibit distinct autonomic phenotypes – central dysregulation in FFI and peripheral autonomic failure in PrP-SA. Routine autonomic assessment may facilitate early detection and targeted symptom management across the spectrum of gPrDs.

Disclosure: Nothing to disclose.

OPR-072 | Linking autonomic dysfunction, immune adrenergic signaling, and neurofilament light chain in multiple sclerosis

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Background and Aims: To investigate the association between autonomic dysfunction and immune cell neurotransmitter receptor expression in people with multiple sclerosis (pwMS), and to explore relationships with serum neurofilament light chain levels (sNfL) as a marker of disease activity.

Methods: Autonomic dysfunction was assessed in 68 pwMS using the Composite Autonomic Scoring Scale (CASS). Expression of Adrenoceptor Beta 2 (ADRB2), Nicotinic Acetylcholine Receptor $\alpha 7$, Dopamine Receptors D3 and D5, Interleukin-1 receptors (IL1R1, IL1R2, IL1R8), and Interleukin-23 receptor was measured on T- and B-lymphocyte subsets, natural killer (NK) cells, and monocytes. Serum neurofilament light chain levels were quantified using the Roche Elecsys NfL assay.

Results: PwMS with objective autonomic dysfunction showed significantly lower ADRB2 expression on total T cells ($p = 0.037$), cytotoxic T cells ($p = 0.016$), including memory and naïve subsets, B lymphocytes ($p = 0.019$), and NK cells ($p = 0.026$), compared with pwMS with normal autonomic function. No differences

were observed for other neurotransmitter or cytokine receptors. sNfL levels did not differ between groups; however, sNfL negatively correlated with ADRB2 expression on cytotoxic T cells ($r = -0.294$, $p = 0.015$), including effector/memory and naïve non-regulatory subsets.

Conclusion: Autonomic dysfunction in pwMS is associated with reduced ADRB2 expression across multiple immune cell populations, suggesting altered adrenergic neuroimmune regulation. The inverse relationship between ADRB2 expression on cytotoxic T cells and sNfL levels points to a potential link between impaired adrenergic signaling and inflammatory disease activity in multiple sclerosis.

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OPR-073 | Pattern and progression of sudomotor dysfunction in alpha synucleinopathies

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Background and Aims: Pure autonomic failure (PAF) is an alpha-synucleinopathy with a 30%–40% risk of phenoconversion to Parkinson's disease (PD), dementia with lewy bodies (DLB), or multiple system atrophy (MSA). While several biomarkers have been proposed to predict phenoconversion, sudomotor dysfunction remains underexplored. This study examined the distribution and longitudinal progression of sweat loss in patients with PAF.

Methods: As part of an ongoing natural history study, consecutive patients diagnosed with PAF were systematically phenotyped and followed longitudinally until phenoconversion or death. Sudomotor function was assessed using thermoregulatory sweat testing (TST) and dynamic sweat testing (DST). Combined results were used to localize sudomotor dysfunction as preganglionic, postganglionic, or mixed.

Results: 69 PAF patients underwent sudomotor testing: 40/69 (58%) completed TST and 45/69 (65%) completed DST. On TST, 29/40 (73%) demonstrated global anhidrosis, 8/40 (20%) length-dependent sweat loss, 2/40 (5%) regional anhidrosis, and 1/40 (2%) unilateral anhidrosis. Among those with global anhidrosis, 22/29 (76%) remained PAF, 3/29 (10%) developed DLB, and 4/29 (14%) developed MSA. Patients without global anhidrosis either remained PAF or phenoconverted to PD/DLB. DST showed reduced sweating in 43/45 patients (95.5%) and a predominantly length dependent distribution in patients with stable PAF compared with MSA, with no difference in the rate of sweat output decline over time.

Conclusion: Global anhidrosis is the most common sudomotor pattern in MSA and is associated with a non-length-dependent impairment of postganglionic sudomotor fibres. In contrast, a

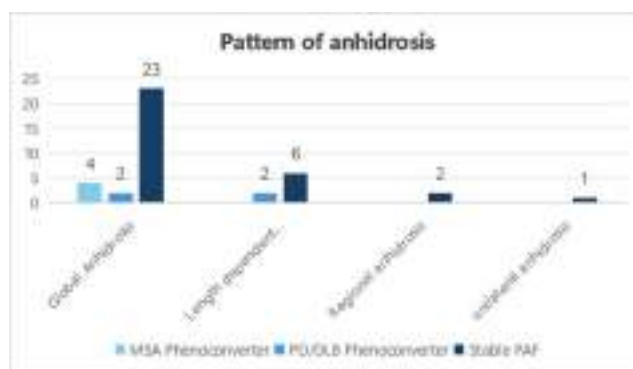


FIGURE 1 Pattern of anhidrosis.

length-dependent sudomotor deficit is more suggestive of a restricted postganglionic alpha-synucleinopathy, such as PAF.

Disclosure: Nothing to disclose.

Ageing and Dementia 2

OPR-074 | Plasma ApoE proteotyping on Lumipulse for ApoE epsilon4 determination: A clinically practical tool in the era of anti-amyloid therapies

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Background and Aims: To determine whether plasma-based quantification of apolipoprotein E4 (ApoE4) and total apolipoprotein E (PanApoE) using a Lumipulse immunoassay can reliably identify APOE epsilon4 carrier status as a practical alternative to genetic testing in patients along the Alzheimer's disease (AD) continuum.

Methods: We retrospectively included 110 patients with cognitive impairment evaluated at a tertiary memory clinic. APOE genotyping classified individuals as epsilon4 non-carriers, heterozygous, or homozygous carriers. Plasma ApoE4 and PanApoE were measured using the Lumipulse 600 G platform, and the ApoE4 to PanApoE ratio was calculated. Additional plasma biomarkers were assessed, including AB40, AB42, pTau181, pTau217, NfL, GFAP, and sTREM2. Diagnostic accuracy for epsilon4 carrier identification was assessed using receiver operating characteristic analyses, deriving area under the curve values and optimal Youden thresholds.

Results: The ApoE4 to PanApoE ratio increased stepwise with epsilon4 allele dose, with complete separation between carrier status. For identifying epsilon4 carriers, the ratio achieved perfect discrimination, with an area under the curve of 1.00 and no misclassified individuals at the optimal cut-off. ApoE4 concentration alone showed substantially lower accuracy. No significant differences were observed in other plasma AD biomarkers between epsilon4 carriers and non-carriers.

Conclusion: The ApoE ratio measured on the Lumipulse platform accurately reflects APOE epsilon4 status and allele dose. This prototype-based approach may represent a rapid and accessible alternative to genotyping to support risk stratification and decision-making in the anti-amyloid treatment era.

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OPR-075 | Automated diagnosis using deep neural networks: MRI-based classification of Alzheimer's disease and behavioral variant frontotemporal dementia

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Background and Aims: Frontotemporal lobar degeneration (FTLD) includes heterogeneous neuropathological and clinical profiles often overlapping with Alzheimer's disease (AD). Neuroimaging is essential for differential diagnosis alongside biomarkers and clinical assessment. We validated a deep learning approach for classifying behavioral variant frontotemporal dementia (bvFTD), AD, and healthy controls (HC) using single high-resolution T1-weighted MRI scans.

Methods: 3T T1-weighted MRI scans from 120 bvFTD, 147 AD, and 202 HC from five institutions were analyzed, plus 651 scans (293 AD, 358 HC) from ADNI. 3D convolutional neural networks (CNNs) were implemented in PyTorch using raw MRI volumes. Data were stratified by center and split into training (64%), validation (16%), and test (20%) sets, with random augmentation. Two architectures were evaluated: fully convolutional and convolutional-pooling networks. Performance was measured by balanced accuracy and F1-score. Grad-CAM maps were generated for the best model to identify regions driving class-specific predictions.

Results: The best model was the fully convolutional network, achieving 72% balanced accuracy and 77.7% F1-score with class-specific F1-scores of 67% (bvFTD), 76% (AD), and 82% (HC). Grad-CAM highlighted the right frontal lobe for bvFTD, bilateral parietal cortices for AD, and periventricular regions for HC, demonstrating anatomically plausible patterns.

Conclusion: Our findings support structural MRI as a potential biomarker for early disease screening using end-to-end CNN models. While performance was lower for bvFTD, likely

reflecting heterogeneity and sample imbalance, anatomically plausible patterns were identified. CNN-based MRI analysis may serve as a complementary tool for early differential diagnosis of neurodegenerative disorders.

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OPR-076 | 1-year real-world use of lecanemab and donanemab in an Italian memory clinic

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Background and Aims: To report feasibility, safety, and early biological outcomes of lecanemab and donanemab therapy for early Alzheimer's disease (AD) in a real-world tertiary setting.

Methods: This observational study included consecutive patients treated with anti-amyloid antibodies at the San Raffaele Memory Center (Milan, Italy) between September 2024 and October 2025. All patients fulfilled clinical and biomarker criteria for mild cognitive impairment or mild dementia due to AD. Lecanemab (10 mg/kg biweekly) and donanemab (350–1400 mg monthly titration) were administered under EMA-aligned protocols with standardized MRI monitoring. Safety outcomes included amyloid-related imaging abnormalities (ARIA) and infusion-related reactions (IRR). Biological effects were assessed using amyloid-PET Centiloid quantification, plasma biomarkers, and cognitive measures at 6 months.

Results: Twenty-nine treatment courses were delivered (9 lecanemab, 20 donanemab). ARIA-E occurred in 2 (10%) and ARIA-H in 3 (15%) donanemab-treated patients, all resolving with corticosteroids. One mild asymptomatic ARIA-H (11%) was observed with lecanemab. IRRs occurred in 6 patients (21%), all mild-to-moderate. At 6 months, cortical amyloid burden decreased by -61 ± 30 Centiloids ($p < 0.001$); 75% of donanemab-treated patients achieved PET negativity (< 11 CL). Plasma GFAP (-20 ± 26 pg/mL) and pTau181 (-0.8 ± 0.9 pg/mL) declined, while NfL showed a mild increase ($+4.4 \pm 4.7$ pg/mL). Cognitive scores (MMSE, CDR-SB) remained stable. No discontinuations occurred for safety or logistical reasons.

Conclusion: Anti-amyloid antibodies can be safely implemented in real-world tertiary centers. Amyloid clearance and plasma biomarker dynamics mirror pivotal trials, supporting the feasibility of disease-modifying therapy for AD in structured clinical practice.

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OPR-077 | Frailty is associated with structural and metabolic brain changes in cognitively unimpaired adults

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Background and Aims: Frailty is a multidimensional syndrome that entails an increased risk of dementia. Characterizing early frailty-related brain changes in cognitively unimpaired (CU) individuals may help to clarify its role in subsequent cognitive decline, particularly beyond Alzheimer's pathology.

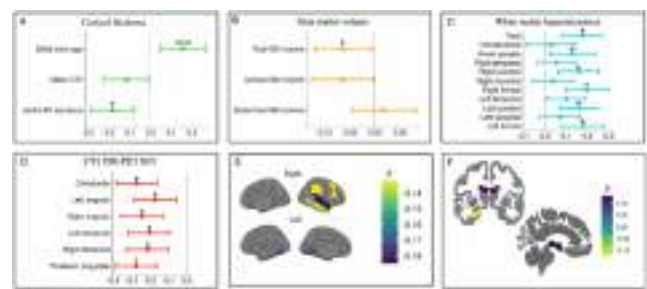


FIGURE 1 Forest plots of linear regression models, corrected by age, sex, CSF Aβ42/40, and total intracranial volume if necessary, exploring the impact of frailty on cortical thickness.

Methods: We investigated the relationship between frailty and 3T-MRI, and [18F] FDG PET biomarkers in CU middle-aged adults from the ALFA+ cohort with available cerebrospinal fluid (CSF) Alzheimer's disease (AD) biomarkers. Frailty was assessed using a 35-item Frailty Index (FI), and its relationship with predicted brain age, global and regional cortical thickness (CTh), brain volume, white matter hyperintensities (WMH), and cerebral hypometabolism was investigated using linear regression models.

Results: Out of the 418 included participants, 57 (13.64%) were classified as frail. Frailty was associated with older predicted brain age, decreased mean, fronto-temporal and Jack's signature CTh, and hippocampal volume, as well as mean, parieto-temporal and posterior cingulate [18F] FDG uptake. Frailty was also associated with augmented white matter hyperintensities (WMH), especially in fronto-parietal and basal ganglia regions. These associations remained significant also after adjusting for CSF Aβ42/40 levels.

Conclusion: Independent of amyloid pathology, frailty was associated with atrophy, vascular damage, and hypometabolism in AD and aging regions in CU adults. These findings highlight frailty as an early contributor to brain vulnerability, potentially preceding dementia.

Disclosure: Nothing to disclose.

OPR-078 | MRI-based atrophy subtypes in a young memory clinic cohort: Associations with clinical and biomarker profiles

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Background and Aims: MRI-based brain atrophy subtypes are increasingly recognized in Alzheimer's disease (AD) dementia, but their relevance across the real-world memory clinic spectrum remains unclear. We aimed to identify atrophy subtypes in a young memory clinic cohort and examine their associations with demographics, cerebrospinal fluid (CSF) biomarkers, and cerebrovascular burden.

Methods: We included consecutive patients (subjective cognitive impairment [SCI] to dementia) evaluated at the Karolinska

Memory Clinic (2018–2023) with available 3T MRI. Subtypes were defined using FreeSurfer volumetric measures and a validated algorithm combining categorical classification (typical, limbic predominant, cortical predominant, minimal atrophy) with continuous indices of typicality and severity. Cognitive, APOE $\epsilon 4$, CSF biomarkers (A β 42, A β 42/40, p-tau181, total tau, neurofilament light [NFL]), and cerebrovascular burden were compared across subtypes. Analyses were replicated in A β -positive and anti-A β -therapy-eligible patients.

Results: Among 809 patients (median age 60years; 56.1% female), 38.2% had SCI, 44.4% MCI, and 17.4% dementia. Limbic predominant and typical subtypes showed higher male prevalence, APOE $\epsilon 4$ frequency, cerebrovascular burden, poorer memory, and greater A β positivity (all $p \leq 0.02$). The cortical predominant subtype was more frequent in females, whereas minimal atrophy was associated with milder cognitive impairment and higher depressive symptoms. In A β -positive patients ($n = 231$), typical and limbic subtypes showed higher p-tau181 and NFL levels and lower A β 42/40 ratios (all $p < 0.05$). Results were consistent across continuous atrophy measures and in patients eligible for anti-A β therapy ($n = 89$).

Conclusion: MRI-based atrophy subtypes display distinct clinical and biomarker profiles across the memory clinic spectrum, supporting their diagnostic value and relevance for biologically targeted AD trials.

Disclosure: Nothing to disclose.

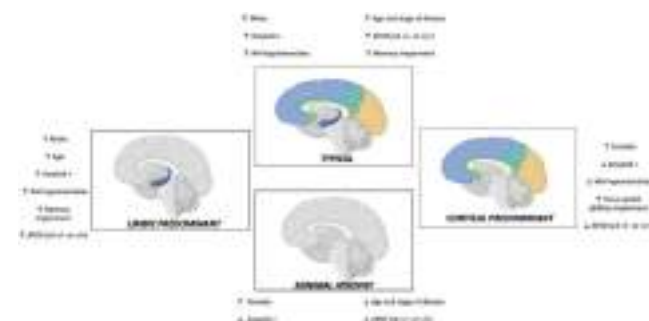


FIGURE 1 Summary of demographic, cognitive assessment, CSF, and vascular profiles among the MRI-based atrophy subtypes.

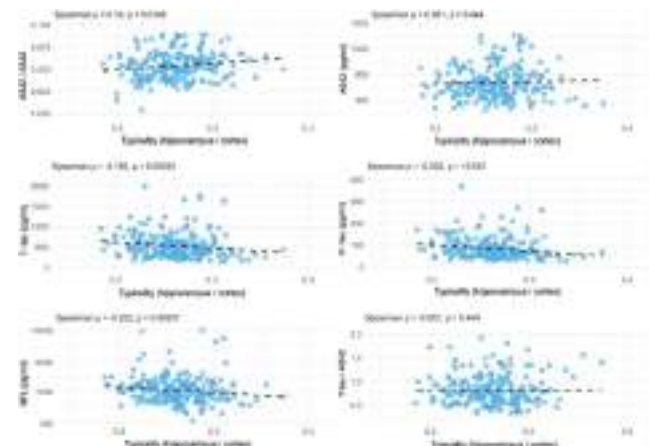


FIGURE 2 Scatterplots of correlation between typicality (hippocampus/cortex ratio) and CSF biomarkers in the A β -positive cohort.

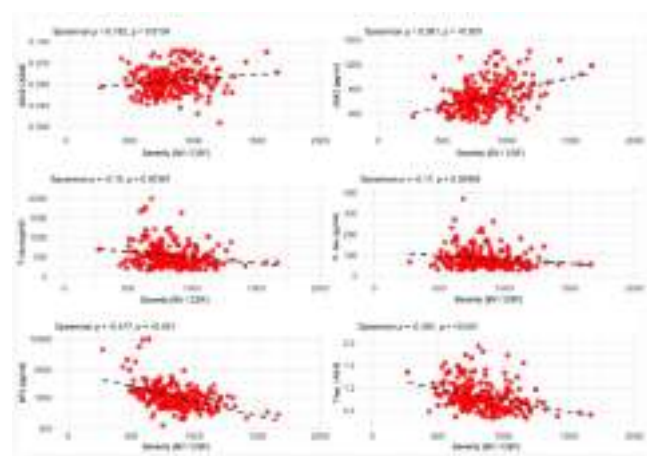


FIGURE 3 Scatterplots of correlation between severity (brain volume/CSF ratio) and CSF biomarkers in the A β -positive cohort.

Neuroimmunology 1

OPR-079 | Clinical and prognostic features of de novo central nervous system non-MS demyelinating disorders following immune checkpoint inhibitor therapy

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Background and Aims: Immune-related adverse events (irAEs) can occur after the administration of immune-checkpoint inhibitors (ICI). De novo demyelinating irAEs (DEM-irAEs) are rare, poorly characterized and often challenging. We aimed to define the demographic, clinical, therapeutic and prognostic features of DEM-irAEs.

Methods: We conducted a multicentric retrospective study including patients with DEM-irAEs evaluated between 2011 and 2024 in nine European neurology units. Additional cases were identified through a systematic literature review according to PRISMA guidelines. Statistical analyses were performed using R software version 4.0.3.

Results: We identified 80 patients with DEM-irAEs. Median age was 59 years (range: 9–84), with male predominance (52/80, 65%). DEM-irAEs developed after a median of 18 weeks (range: 2–178) following ICI initiation (anti-PD1 $n=49$, 61%; anti-PD-L1 $n=4$, 5%; anti-CTLA4 $n=5$, 6%; anti-PD1 plus anti-CTLA4 $n=22$, 28%). Clinical manifestations included optic neuritis ($n=17$, 8 bilateral), myelitis ($n=29$, including 24 LETM), acute disseminated encephalomyelitis ($n=6$), atypical DEM-irAEs ($n=7$), and overlapping syndromes ($n=21$). CSF-restricted oligoclonal bands (12/50, 24%) and glial autoantibodies were uncommon (11/80, 14%). Immunotherapy was administered in 74/80 (93%) of patients, mainly steroids ($n=61$), IVIg ($n=16$), plasmapheresis ($n=23$), rituximab ($n=8$), and cyclophosphamide ($n=8$). Outcomes were complete response in 15 patients (24%), partial response in 25 (40%), and no response in 22 (35%). Compared with AQP4-NMOSD ($n=42$) and MOGAD ($n=22$), DEM-irAEs showed greater attack severity and worse outcomes ($mRS>2$; $p<0.01$).

Conclusion: DEM-irAEs represent a rare and heterogeneous complication of ICI treatment. Long-term immunosuppression should be considered in selected cases, as approximately one-quarter of patients relapse, and outcomes appear worse than in AQP4-NMOSD and MOGAD.

Disclosure: Nothing to disclose.

OPR-080 | Circumventricular organ permeability to NMO IgG allows AQP4 targeting in the mouse brain

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Background and Aims: The penetration of circulating aquaporin-4 (AQP4) IgG into the CNS is a major event in neuromyelitis optica (NMO)-induced astrocytopathy. After intravenous injection in rodents, recombinant AQP4 IgG predominantly accumulates in circumventricular organs (CVOs), such as the median eminence. Physiologically, protein passage through the fenestrated blood vessels of CVOs importantly contributes to brain-periphery communication. The aim of the present study

was to determine whether the median eminence is an entry site for AQP4 IgG in the mouse CNS, leading to AQP4 targeting.

Methods: AQP4 expression was examined in the brain of C57BL/6Jrj female mice by immunofluorescence on coronal sections. The biodistribution of recombinant human anti-AQP4 antibody (rAb-53) was observed 24 h after intravenous injection (7.5 mg/kg body weight). Vascular endothelial cells and tanycytes were labeled using podocalyxin and vimentin, respectively.

Results: AQP4 was present in perivascular astrocytic endfeet and in ependymal cells lining the 3rd ventricle, but not in tanycyte cell bodies which occupy a more ventral position. After intravenous injection, a high level of rAb-53 was detected within the parenchyma and around podocalyxin-positive blood vessels of the median eminence. Ependymal cells, but not tanycyte cell bodies, were also positive for rAb-53. High-resolution microscopy revealed perivascular colocalization of AQP4 and rAb-53, thus suggesting antigen-antibody interaction.

Conclusion: Our data demonstrate that AQP4 IgG reaches AQP4 in astrocytes and ependymal cells through the leaky vasculature of the median eminence in the mouse brain, suggesting that CVOs are potential entry sites for pathogenic antibodies in NMO.

Disclosure: This project has been supported by the Swiss National Science Foundation (SNSF project grant #10003453 to VP and AC). Andrew Chan has served on advisory boards for, and received funding for travel or speaker honoraria from, Actelion-Janssen, Alexion, Almirall, Biogen, BMS, Celgene, Dresden International Uni, Horizon, Merck, Mylan (Viatris), Novartis, Roche, Sanofi-Genzyme, Teva, UCB, Uni Leipzig, Wiley, all for hospital research funds; and research support from Biogen, CSL Behring, Genzyme, Roche, UCB and European Union and Swiss National Research Foundation. A. Chan is associate editor of the European Journal of Neurology and serves on the editorial board for Clinical and Translational Neuroscience and as topic editor for the Journal of International Medical Research.

OPR-081 | GAD65 epitope predictive for Stiff-person syndrome

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Background and Aims: Stiff-person-syndrome (SPS) is a rare but treatable neurological autoimmune disease associated with GAD65-autoantibodies (aAb). However, GAD65aAb may also be associated with other diseases, like epilepsy or ataxia. We therefore aimed at determining SPS-specific GAD65 epitopes.

Methods: Epitope binding was screened in sera ($n=46$) and cerebrospinal fluid ($n=4$) from a SPS patient cohort ($n=30$) via microarray binding assay including full positional scans. Controls included healthy controls ($n=7$), a GAD65-aAb-positive diabetes cohort ($n=10$) and a GlyR α 1-aAb SPSD cohort ($n=24$).

Results: 80% (24/30 patients) showed aAb binding to at least one out of five epitopes (E1–E5) and a single dominant epitope

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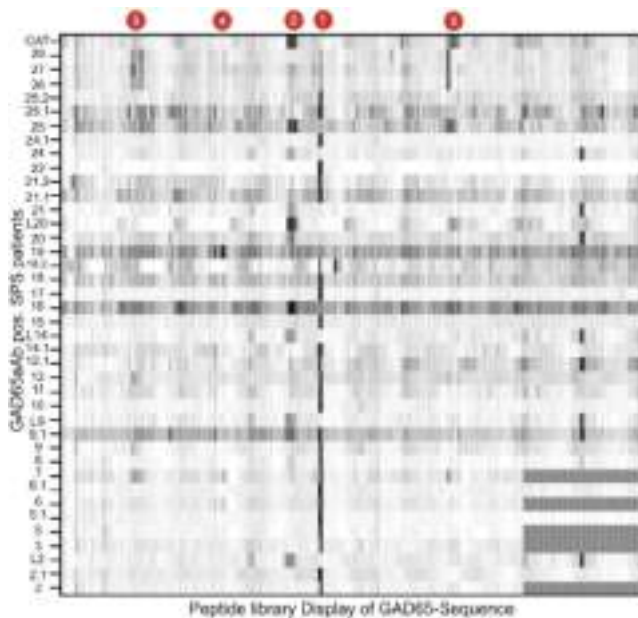


FIGURE 1 Epitope mapping in Stiff person syndrome cohort reveals five distinct epitopes. Heatmap representing overview of initial screening results for GAD65 epitopes with microarray binding assay including baseline, follow up and CSF samples.

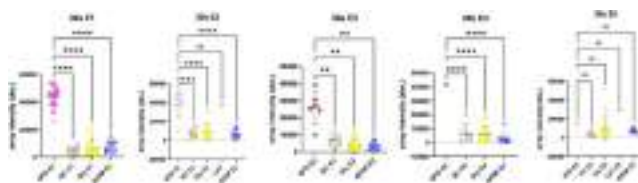


FIGURE 2 Correlation among disease and GAD65 epitope E1, E2, E3, E4 and E5. Horizontal line refers to median, error bars indicate interquartile range. Prior to conducting the one-way ANOVA, the assumption of homogeneity of variances was tested.

(E1) was recognized in 56% of all SPS samples. For 20% (6/30 patients) no aAb binding epitopes could be determined. In 14 patients epitopes were analysed before and after plasmapheresis (PE). Here we found a baseline polyclonal antibody response that disappeared after PE, only the response to E1 remained. No correlation between epitope profile and clinical phenotype was visible within the SPS spectrum. Statistical analysis validated E1 as the SPS-specific GAD65 epitope, not recognized by any of the tested controls ($p < 0.0001$).

Conclusion: Epitope screening, mapping and characterization defined five distinct GAD65 epitopes shared within SPS patients. Amongst all, E1 stands out as the strongest predictor for SPS due to its prevalence and consistency. Epitopes in SPS GAD65-aAbs and diabetes GAD65-aAbs were clearly distinct. This knowledge has the potential for more precise SPS diagnostics and broad implications for SPS pathophysiological research.

Disclosure: Nothing to disclose.

Background and Aims: Studies on the long-term cognitive abilities and psychosocial outcomes of individuals with anti-LGI1 encephalitis are scarce. Recently, a disease-specific Patient-Reported Outcomes Score for (autoimmune) Encephalitis (PROSE; score 0–100) was developed. We assessed cognitive and psychosocial recovery in a large national cohort, including the first patient-data using PROSE.

Methods: All adults diagnosed with anti-LGI1 encephalitis in the Netherlands were invited to participate, provided they could self-report and participate in cognitive testing. Patients were evaluated cross-sectionally, and those within 1.5 years of diagnosis were also followed prospectively. Functional (mRS, CASE), cognitive (comprehensive test-battery covering major cognitive domains) and self-reported outcomes (PROSE, SF-36, EQ-5D-5L, WHO-5, WHO-DAS-II, FSS, BDI, HADS) were assessed at various disease stages.

Results: We included 106 patients with anti-LGI1 encephalitis (mean age 68 years, 33% female). Two years after diagnosis, 50% of patients ($n = 37/74$) had fully recovered and returned to their regular life, while 40% had significant cognitive impairments (< -1.5 SD below norm) in at least one domain, most commonly verbal memory (average z-score > 2 years -1.12 , $SD = 0.90$). Cognitive recovery plateaued around 24 months post-treatment, whereas psychosocial recovery often continued for at least 3 years after treatment (PROSE mean score < 1 year: 30, > 3 years: 26).

Conclusion: Anti-LGI1 is a serious neurological condition. Around 50% fully recovers but many experience lasting cognitive and psychosocial difficulties, and would benefit from long-term monitoring and targeted rehabilitation. We outline the recovery trajectory of anti-LGI1 encephalitis and present the first patient data on the disease-specific PROSE.

Disclosure: J. Brenner and M.J. Titulaer, on behalf of the Erasmus University Medical Centre, hold intellectual property rights for the PROSE.

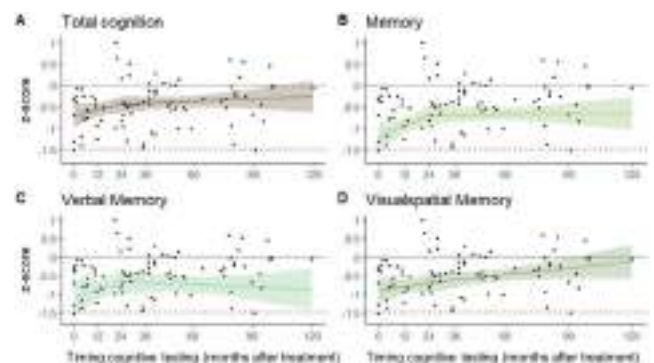


FIGURE 1 Cognitive outcomes in relation to time after diagnosis and treatment.

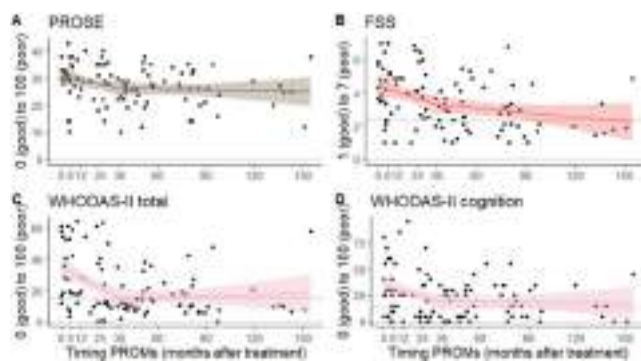


FIGURE 2 Self-reported psychosocial outcomes in relation to time after diagnosis and treatment, including the first patient-data on the PROSE.

OPR-083 | Prolonged vegetative state in anti-NMDA receptor encephalitis: A retrospective international cohort study of complications, outcomes, and implications

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of Neurology, Tokyo Dental College Ichikawa General Hospital, Ichikawa, Japan; ¹⁸Department of Neurology, Showa General Hospital, Tokyo, Japan; ¹⁹Division of Neurology, Department of Internal Medicine, Sakura Medical Center, Toho University, Sakura, Japan; ²⁰Neurology Department, Kagoshima City Medical Association Hospital, Kagoshima, Japan; ²¹Department of Neurology, Keio University School of Medicine, Tokyo, Japan; ²²Department of Clinical Neuroscience and Therapeutics, Hiroshima University Graduate School of Biomedical and Health Sciences, Hiroshima, Japan; ²³Medical Statistics, Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Barcelona, Spain; ²⁴Neuroimmunology Division, Children's Hospital of Pittsburgh/University of Pittsburgh Medical Center, USA; ²⁵Department of Neurology, Kyoto University Hospital, Kyoto, Japan; ²⁶Department of Neurology, Hannover Medical School, Germany; ²⁷Division of Neuropathology and Neurochemistry, Department of Neurology and Comprehensive Center for Clinical Neurosciences and Mental Health, Medical University of Vienna, Austria; ²⁸Department of Neurology, Kitasato University School of Medicine, Sagami-hara, Japan

Background and Aims: In anti-NMDA receptor encephalitis (NMDARe) delayed and slow improvement complicates assessment of treatment refractoriness, creating a larger knowledge gap in prolonged impaired consciousness. We aimed to assess these features and outcomes in patients with prolonged vegetative state. **Methods:** Retrospective cohort study of NMDARe patients who remained in vegetative state for ≥ 9 months. Outcomes were: death; achieving command-following, mRS=2, or recovery (mRS=0 with return to premorbid activities).

Results: Forty-five patients were identified (38 female; median age 22 [IQR 19–31]). All had impaired consciousness 9 (5–16) days after symptom onset. All received first-line immunotherapy; 41 (91%) second-line, and 18 (40%) third-line. Twenty-one (47%) had ovarian teratomas. Median durations were: vegetative state 399 days (307–698), ICU stay 275 days (189–354), and hospitalization 474 days (349–715). Thirteen (28%) patients were resuscitated from dysautonomic cardiac arrest. After a median of 5 years (2.5–6.8), 15 (33%) recovered, 13 (29%) substantially improved (mRS ≤ 2), 11 (24%) had mRS=3–5, and 6 (13%) died. Five began command-following 11 months (1.5–21) after last immunotherapy. Estimated cumulative incidences at 5 and 10 years were 66% and 76% for achieving mRS=2, and 32% and 54%, respectively, for recovery. Teratomas were associated with lower probability of achieving mRS=2, whereas older age and higher NEOS2 score were associated with increased mortality.

Conclusion: In NMDARe, recovery from prolonged impaired consciousness is more common than expected. Early assessment of treatment response or refractoriness may underestimate delayed improvement, prolonged therapy needs, or spontaneous recovery. Futility decisions should be individualized with multidisciplinary input after extended follow-up.

Disclosure: Dr. Dalmau holds patents licensed to Euroimmun for the use of NMDA, GABAB receptor, GABAA receptor, DPPX and IgLON5 as autoantibody tests, for which he receives royalties. Dr. Graus holds a patent licensed to Euroimmun for the use of IgLON5 in an autoantibody test, for which he receives royalties. He receives honoraria from MedLink Neurology for his role as associate editor. Dr. Brenner and Dr. Titulaer hold a copyright, on behalf of Erasmus MC, for the patient-reported outcome measure PROSE. Dr. Brenner is supported by the AEA

Seed Grant 2024. The Medical University of Vienna, Austria (employer of Dr. Höftberger) receives payment for antibody assays and for antibody validation experiments organized by Euroimmun (Lübeck, Germany). The rest of the authors (MG, MMS, GMS, KW, KW, PHL, GOC, JY, KPW, SM, GDL, EL, TC, MJMG, JPFC, MS, EF, TM, HN, TT, SS, YI, SN, RB, EA, LM, JP, AH, MT, AI, DW, MS, YB, RDP, KWS, FL, CF, TA, TI) declare no conflicts of interest related to this work.

Muscle and Neuromuscular Junction Disorder

OPR-084 | Investigating intravenous efgartigimod in juvenile generalized myasthenia gravis: Results from the ADAPT JR study

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Background and Aims: Efgartigimod is a human immunoglobulin G1 (IgG1) antibody Fc fragment that blocks the neonatal Fc receptor. Phase 3 trials (ADAPT/ADAPT+) demonstrated that intravenous (IV) efgartigimod is efficacious and well tolerated in adults with generalized myasthenia gravis (gMG). However, there remains an unmet need for treatments in patients with juvenile gMG. We present interim results from a Phase 2/3 trial assessing efgartigimod IV in juvenile participants with anti-acetylcholine receptor antibody-positive (AChR-Ab+) gMG (ADAPT JR; NCT04833894).

Methods: ADAPT JR will ultimately recruit ≥12 participants (aged 2–17 years), using a staggered design starting with an adolescent cohort (aged 12–17 years). The study comprises a single-dose confirmatory Part A (≥8 weeks) and a multiple-dose treatment-response confirmatory Part B (≥18 weeks).

Results: Eleven adolescent participants were enrolled as of February 2025. Efgartigimod treatment decreased both total IgG and AChR-Ab levels. In Part B, the mean (SE) total Myasthenia Gravis Activities of Daily Living (MG-ADL) score change between cycle baseline to Week 4 was −3.5 (0.94) and −4.0 (1.34) during Cycles 1 and 2, respectively. Quantitative Myasthenia Gravis (QMG) scores exhibited a similar trend. Minimal symptom expression (MSE; MG-ADL, 0–1) was achieved by 72.7% (Cycle 1) and 80.0% (Cycle 2) of participants. Efgartigimod was well tolerated.

Conclusion: Efgartigimod treatment was well tolerated in adolescents and resulted in decreased levels of both total IgG and AChR-Abs, similar to those observed in adult studies. Clinically meaningful improvements in MG-ADL and QMG occurred, with many participants achieving MSE. Efgartigimod may address an unmet need in juvenile gMG therapy.

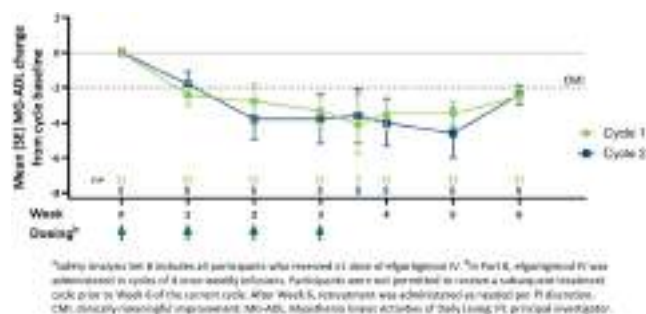


FIGURE 1

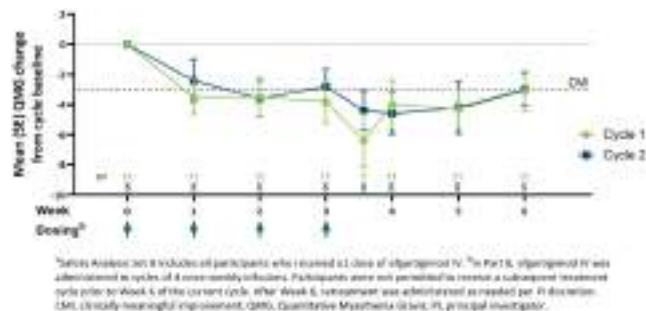


FIGURE 2

Disclosure: This study was sponsored by argenx; AK-P, ANS, NLK, SR, and EHN reported financial/nonfinancial relationships with argenx at the time of submission. AB, JG, FM, and LL are employees of argenx, and JN and TvB are consultants for argenx.

OPR-085 | Patient acceptable symptom state in MG patients: A cross-sectional study

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Background and Aims: To assess patient acceptable symptom state (PASS) in tertiary center cohort.

Methods: Cross-sectional study of 101 MG patients, age 60.7 ± 18.5 years, 67.3% F, 42.6% with EOMG, conducted between Feb2024 to Jun2025.

Results: 91% of patients received AChEI, 72.3% glucocorticoids (CS), 20.8% AZA, 19.8% MFM, 2% MTX, 44.6% were treated with IVIG (23.8% previous year), 12.9% with PLEX (5.9% previous year). 13.9% had history of myasthenic crisis, 15% had ocular symptoms only. 40.6% of patients were PASS-positive, with MG-ADL 4.4 ± 3.8 vs. 8.3 ± 4.1 in PASS-negative, MGQoL15 was 16.3 ± 10.5 vs. 32.2 ± 12.3, respectively ($p < 0.05$). Patients treated with CS had lower MGQoL15 (27.22 ± 13.7 vs. 16.9 ± 11.6, $p < 0.05$) as compared with untreated, with no significant differences in MG-ADL, age or sex between groups. Patients treated with IVIG or PLEX within last 12 months had worse MGQoL15 (29.9 ± 15.2 vs. 23.0 ± 13.7, $p < 0.05$) and MG-ADL (8.1 ± 5.1 vs. 5.5 ± 4.0, $p < 0.05$). Females, as compared with males were more often PASS-negative; (51.5% vs. 27.3%), had higher MG-ADL

(6.8+4.5 vs. 4.4+3.6) and MGQoL (26.8+14.0 vs. 19.7+14.2) $p<0.05$. There were no differences between PASS-positive and negative in age, history of myasthenic crisis. More patients with only ocular or no symptoms were PASS-positive (67%) compared with gMG (35%, $p<0.01$), with better MGQoL15 (9.7+10.3 vs. 28.2+12.8, $p<0.001$). Threshold for PASS-positive answer was ≤ 4.0 points in MG-ADL (sensitivity 58%, specificity 75%).

Conclusion: Nearly 60% of patients do not accept their MG state. Treatment with CS had negative impact on PASS-status despite no differences in MG-ADL, sex or age, compared with those who not CS-treated.

Disclosure: Nothing to disclose.

OPR-086 | From evidence to practice: Clinical audit of uptake of heart failure therapy in adult Duchenne muscular dystrophy at a tertiary centre

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Background and Aims: Duchenne muscular dystrophy (DMD) is a progressive X-linked neuromuscular disorder that leads to dilated cardiomyopathy and heart failure with reduced ejection fraction (HFrEF) in most patients by adulthood. Despite consensus recommendations, the extent of guideline-directed medical therapy (GDMT) utilization remains varied across institutions. The objective of the study is to evaluate the proportion of patients who are receiving GDMT under specialist care at an accredited Duchenne centre.

Methods: A retrospective audit was conducted of patients aged ≥ 18 years with confirmed DMD and HFrEF under the care of an accredited Duchenne centre in January 2025. Prescription rates for key GDMT classes which include renin-angiotensin-aldosterone-system inhibitors (RAASi), beta-blockers, mineralocorticoid receptor antagonists (MRA), and sodium-glucose-co-transporter-2 inhibitors were analysed.

Results: A total of 49 patients met inclusion criteria (mean age 23.5 ± 3.9 years; mean BMI 25.8 ± 8.4 kg/m²; mean LVEF $29.7 \pm 9.1\%$). Genetic variants in the dystrophin gene included deletions ($n=32$), duplications ($n=11$), nonsense ($n=5$), and point mutations ($n=1$). 29 (59%) were on long-term corticosteroids, and 28 (57%) used chronic non-invasive ventilation. Medication

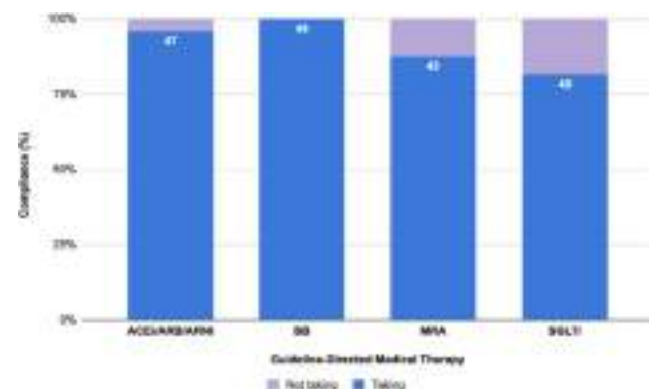


FIGURE 1 GDMT compliance.

utilisation was high: RAASi 96%, beta-blocker 100%, MRA 88%, and SGLT2 inhibitor 82% patients. Others included ivabradine in 24% and loop diuretics in 10%.

Conclusion: This audit demonstrated excellent adherence to GDMT among adults with DMD-related HFrEF under an accredited Duchenne centre, with near-universal prescription of beta-blockers and renin-angiotensin system inhibitors. SGLT2i and MRA use were also notably high which shows proactive adoption of evidence-based HF management.

Disclosure: Nothing to disclose.

OPR-087 | Rituximab in acetylcholine receptor antibody-positive generalised myasthenia gravis: A single-centre experience

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Background and Aims: This study evaluated the effectiveness of rituximab in acetylcholine receptor antibody (AChR-Ab)-positive generalised myasthenia gravis (MG) patients with explosive onset, steroid intolerance, or treatment resistance at Wessex Neurological Centre (2018–2025).

Methods: Retrospective analysis of 40 AChR-Ab-positive generalised MG patients (mean age 60 ± 17 years; 58% male; mean baseline Myasthenia Gravis Activities of Daily Living [MG-ADL] 10 ± 6 , Myasthenia Gravis Composite [MGC] 15 ± 9) receiving rituximab (median initial dose 2000 mg). Outcomes included MG-ADL/MGC at baseline and 3–24 months, CD19 B-cell counts, IgG, prednisolone dose. Repeated-measures ANOVA assessed changes; McNemar tests compared pre/post RTX hospitalisations/plasmapheresis; $p<0.05$ significant (JASP software).

Results: MG-ADL ($F(6,54)=4.67$, $p=0.001$) and MGC ($F(6,42)=3.68$, $p=0.005$) improved significantly over time, though post-hoc pairwise differences were non-significant after Bonferroni correction. CD19 counts depleted markedly ($F(6,12)=10.71$, $p=0.001$); Rituximab led to clinically meaningful improvement, with 71% of patients achieving MCID (≥ 2 -point MG-ADL and ≥ 3 -point MGC reduction) at one or more visits, alongside sustained B-cell depletion and reduced rescue

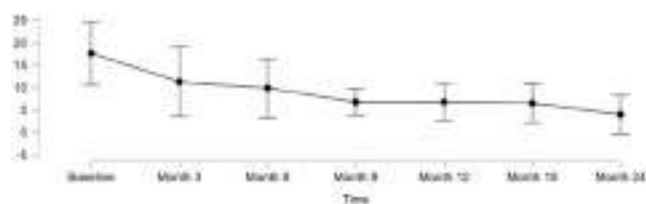


FIGURE 1 MG ADL reduction.

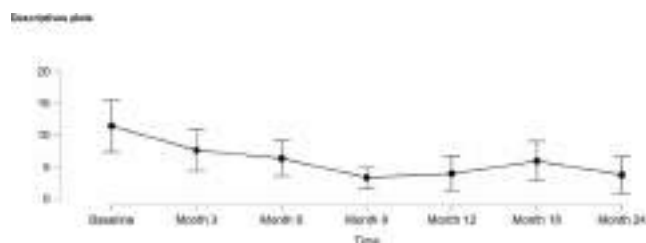


FIGURE 2 MGC reduction.

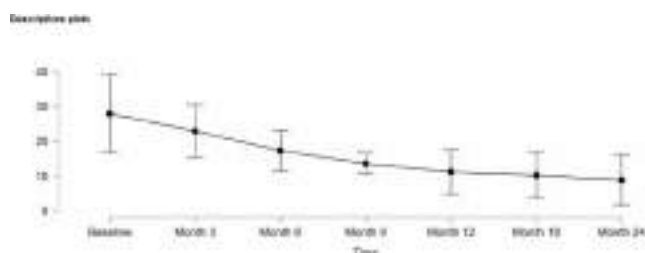


FIGURE 3 Prednisolone dose reduction.

therapies. IgG remained stable ($p=0.93$). Prednisolone dose reduced ($F(6,42)=5.49$, $p=0.001$). Hospital admissions dropped from 47.5% to 20% pre/post RTX ($p=0.013$); plasmapheresis from 50% to 25% ($p=0.021$). No serious adverse events with RTX.

Conclusion: Rituximab achieved sustained B-cell depletion, clinical improvement, and reduced rescue therapies/steroids in refractory AChR-MG without impacting IgG, supporting its role in difficult-to-treat cases.

Disclosure: Nothing to disclose.

OPR-088 | Natural progression of oxidative phosphorylation pathology in mitochondrial myopathy

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Background and Aims: Mitochondrial myopathies are progressive and disabling disorders. Although their hallmark muscle oxidative phosphorylation (OXPHOS) pathology is widely measured in diagnosis and research, its longitudinal behaviour remains poorly characterised, limiting mechanistic insight and the interpretability of OXPHOS markers. Building on our previous characterisation of cross-sectional intra-individual variability in muscle OXPHOS pathology (PMID:41419999), we now aim to assess its natural history using comparable methods.

Methods: Imaging mass cytometry was used to quantify myofibre deficiency across OXPHOS complex (C) I, III, IV and V. Repeat muscle biopsies from a genetically confirmed mitochondrial myopathy cohort were analysed using our previous methods (PMID:41419999), with rates of change estimated from per-patient bootstrapped differences.

Results: Myofibre deficiency in CI or CIV, and a combined CI-IV-V-deficient profile, were the most prevalent abnormalities. Longitudinal differences were heterogeneous, with CI, CIV and CV often changing in tandem and preserving baseline relationships, while CIII deficiency was rare and changed inconsistently. Some patients exhibited stable or declining pathology over time. Both the magnitude and direction of change were patient-specific and showed no association with major genotype-phenotype groups, demographics or biopsy-related factors.

Conclusion: These data show a slow, heterogeneous progression of muscle OXPHOS pathology in mitochondrial myopathy and indicate that extended follow-up or large effect sizes are likely to be required for its use as a reliable indicator of disease modification. Apparent declines in some patients further indicate that endogenous or exogenous biological factors and/or sampling effects must be considered in future longitudinal research.

Disclosure: Nothing to disclose. This work was funded by the Wellcome Trust, UK.

OPR-089 | Incidence of lipid storage myopathy in Southwestern Sweden

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Background and Aims: Lipid storage myopathies (LSM) are a group of genetic and acquired rare disorders affecting the lipid metabolism. Symptoms typically derive from organs with a high energy demand and onset can occur from birth to late adulthood. This study aimed to describe the incidence and primary causes of adult-onset LSM in a tertiary muscle biopsy centre serving >1.4 million adult inhabitants during 12 years.

Methods: A registry search identified patients with a histopathological diagnosis of adult-onset LSM (Figure 1) 2014–2025 at Sahlgrenska University Hospital, Gothenburg, Sweden. We retrospectively reviewed histopathology, genetic data and medical records.

Results: A total of 31 patients were included (17 women, 14 men). Median age at biopsy was 55 years (range 23–79 years). The primary cause of LSM was identified in 24 patients. Sixteen patients had sertraline-associated MADD (Figure 2A). Three patients had a genetically confirmed disease with variants in ETFDH, PNPLA2 and CPTII respectively. One patient was a symptomatic carrier of an ETFDH variant. Other causes included one patient with a pituitary tumour, and three patients with inflammatory myopathy. Seven patients lacked sufficient data to establish the cause of LSM. The mean incidence of LSM in adults in Region Västra Götaland, Sweden, was 1.45 patients per million inhabitants per year (Figure 2B). The mean incidence of sertraline-associated MADD was 1.76 patients per 100 000 inhabitants treated with sertraline per year.

Conclusion: Sertraline-associated MADD was the most common primary cause of LSM in this study and is important to consider in patients diagnosed with lipid storage myopathy without genetic cause.

Disclosure: Nothing to disclose.

OPR-090 | Brain and CSF volume changes in AI-assisted MRI volumetry in spontaneous intracranial hypotension

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Background and Aims: Surgical closure of spinal CSF leaks in SIH patients is associated with quantifiable MRI changes, such as increased intraventricular CSF volume and region-specific expansion and contraction of cerebral lobes, which could help monitoring these patients on MRI follow-up. The pathophysiological significance and prognostic value of brain and CSF volume changes remain to be elucidated. AI-assisted image quantification could be a useful, time-efficient tool for analyzing these changes on a larger scale.

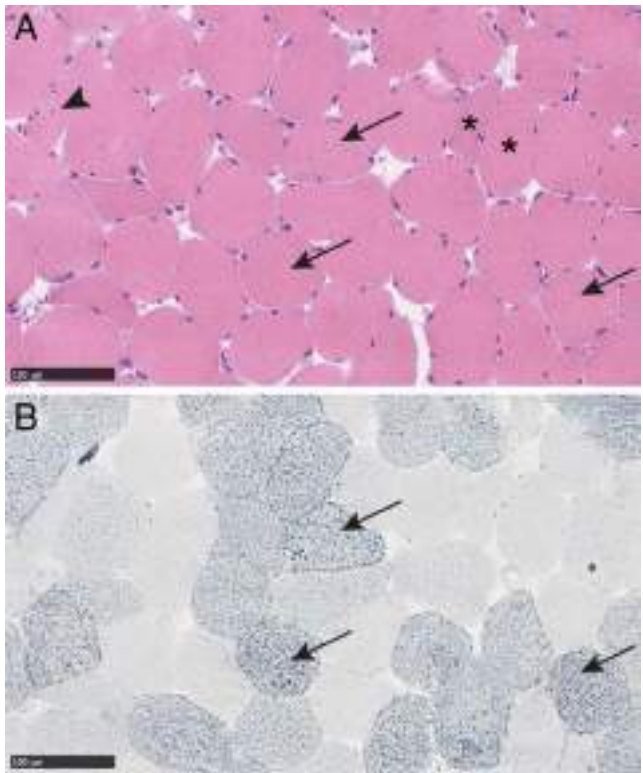


FIGURE 1 Muscle biopsy showing lipid storage myopathy. (A) Vacuoles (arrows), fibre size variation (asterix) and internal nuclei (arrowheads) are seen (Haematoxylin/eosin). (B) Multiple lipid droplets are stained with Sudan black (arrows).

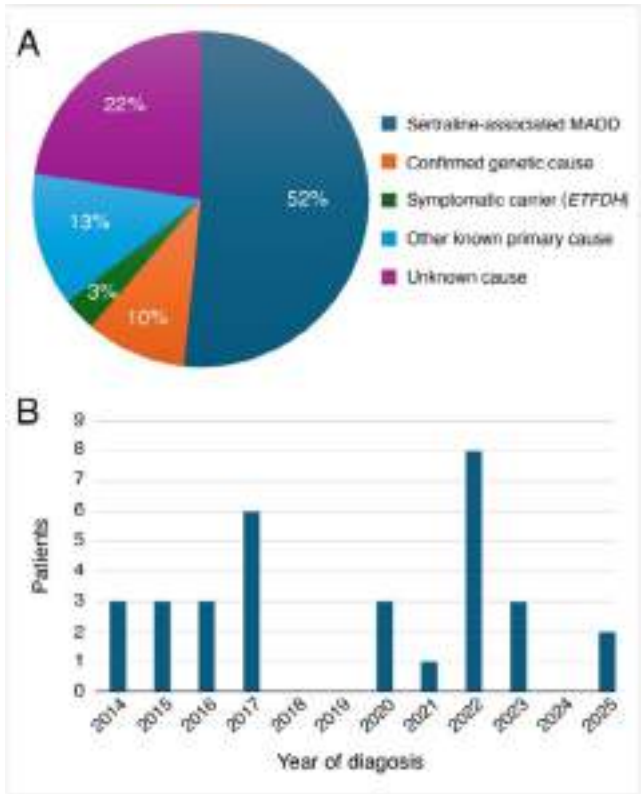


FIGURE 2 (A) Primary causes and (B) Incidence of adult-onset lipid storage myopathy in 31 adult patients diagnosed at Sahlgrenska University Hospital, Gothenburg, Sweden 2014–2025.

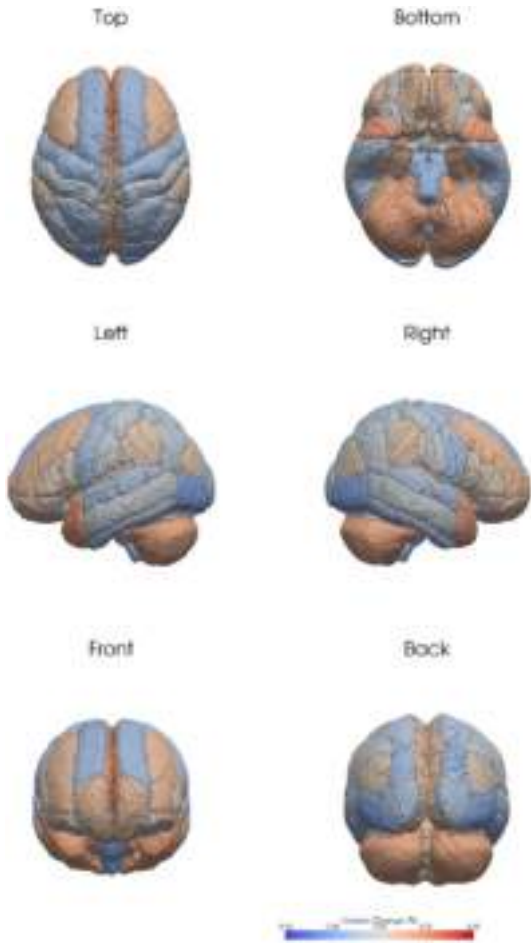


FIGURE 1 Illustration of volume changes by area. Areas in orange are expanding, areas in blue are contracting.

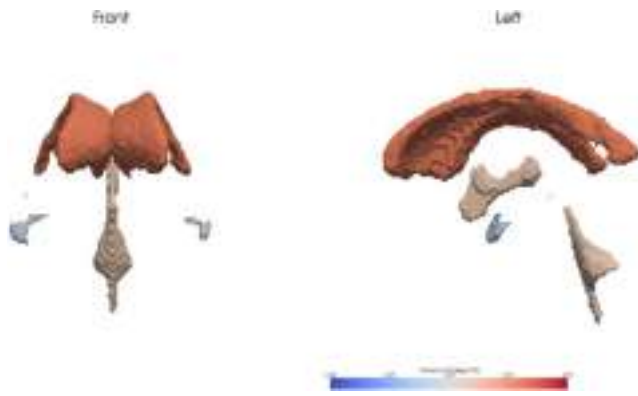


FIGURE 2 Illustration of volume changes within the ventricular system. Areas in red are expanding, areas in blue are contracting.

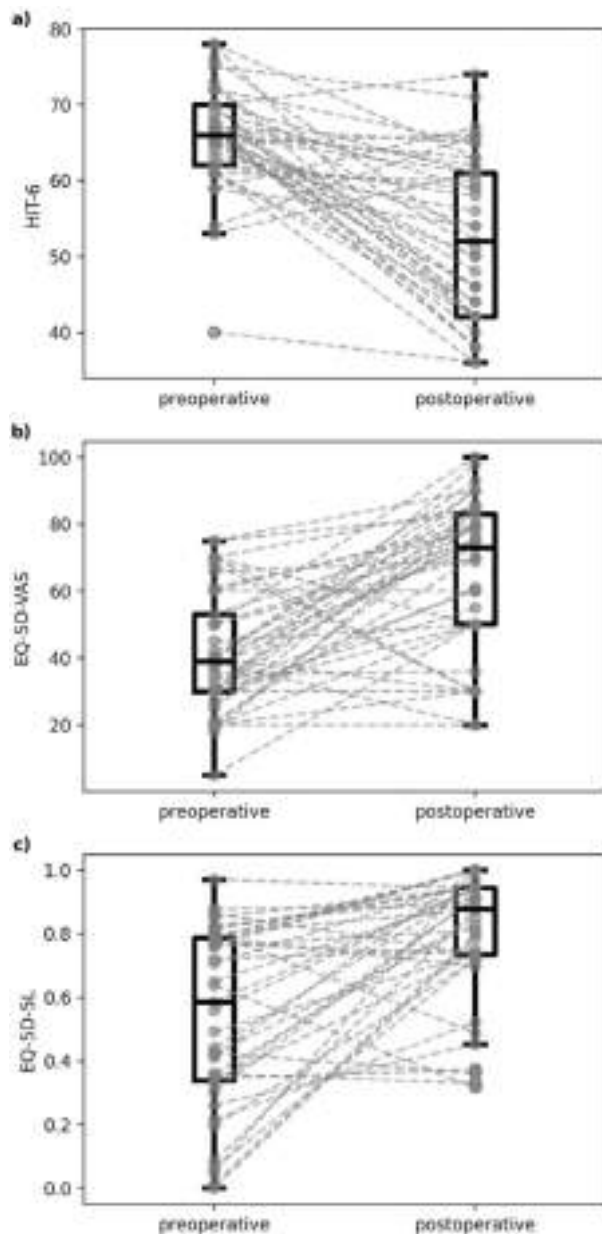


FIGURE 3 Boxplots compare preoperative and postoperative HIT-6 scores at (a), EQ-VAS scores at (b), and EQ-5D-SL scores at (c). The dotted lines represent the evolution of each patient's scores.

Methods: We analyzed 107 patients with SIH who underwent surgical closure of a spinal CSF leak. We used AI-assisted volumetry of pre- and postoperative MRIs using the AssemblyNet segmentation model to quantify CSF and brain region volumes, which were correlated with patient-reported clinical outcome measures.

Results: Patients demonstrated a significant increase in lateral ventricle volume ($+2.83 \text{ cm}^3$, $+17.8\%$) as well as third ($+0.22 \text{ cm}^3$, $+30.1\%$) and fourth ventricle volume ($+0.20 \text{ cm}^3$, $+14.1\%$), after SIH surgery ($p < 0.001$ for all). There was an increased frontal lobe volume ($+6.23 \text{ cm}^3$, $+3.1\%$, $p < 0.050$), and there were small but significant relative volume decreases in temporal horn volume, brainstem, and occipital lobe volume. Clinical scores improved significantly postoperatively but showed low correlation with volumetric changes.

Conclusion: Surgical closure of spinal CSF leaks in SIH patients is associated with quantifiable MRI changes: increased intraventricular CSF volume and region-specific expansion and contraction of cerebral lobes, which could help monitor these patients on MRI follow-up. AI-assisted image quantification could be a useful, time-efficient tool for analyzing these changes at scale.

Disclosure: Nothing to disclose.

OPR-091 | Artificial intelligence analysis of patient videos for neurological diagnosis: A systematic review

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Background and Aims: Neurological diagnosis frequently depends on visual assessment during examination and video review for paroxysmal and movement disorders. However, clinician interpretation remains subjective, variable, and unsuited to growing tele-neurology data volumes. Computer vision and deep learning advances enable quantitative analysis, potentially converting qualitative neurological signs into reproducible digital biomarkers.

Methods: Following a registered PROSPERO protocol (CRD420251070499), we searched databases from inception to June 2025 for studies using AI on video to detect, classify, or quantify neurological disorders. From included papers, we extracted clinical aim, video modality, model type, validation approach, diagnostic metrics. Risk of Bias assessment was conducted following the Newcastle–Ottawa Scale tool. Heterogeneity precluded meta-analytic synthesis.

Results: Thirty studies including 22,532 participants met inclusion criteria. Performance varied by condition: acute stroke/TIA identification reached 85.8% accuracy; post-stroke motor deficit quantification reported 78%–83% accuracy, while facial weakness detection reached 94.3% accuracy. Neonatal seizure detection achieved $>90\%$ specificity. Movement-disorder applications (bradykinesia, tremor, gait, tics) reported 73%–97% accuracies. Pediatric and fetal tasks reported 0.80–0.90 AUCs for early cerebral palsy and congenital malformations. Most studies were moderate quality with limited external validation.

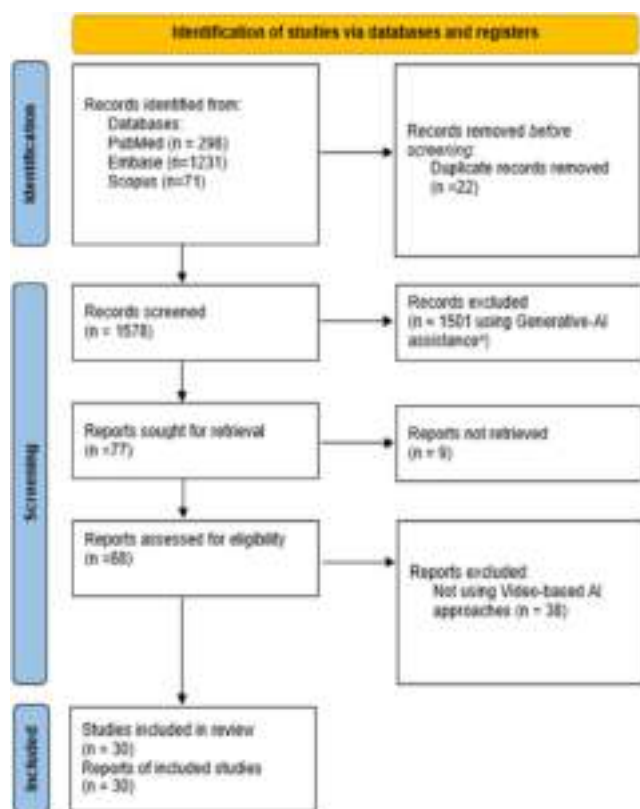


FIGURE 1 PRISMA flow diagram illustrating the screening process.

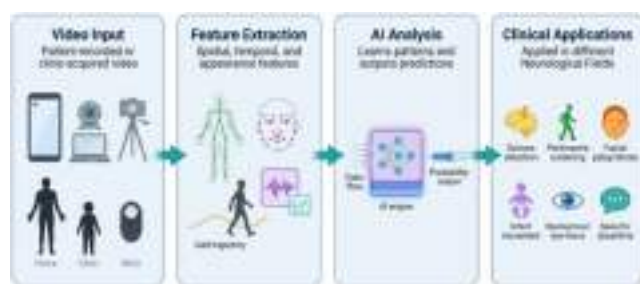


FIGURE 2 Illustration of the typical video-based AI workflow.



FIGURE 3 Graphical synthesis of included studies.

Conclusion: AI based video analysis shows potential to support neurological evaluation across acute, chronic, and developmental disorders, but translation to clinical practice is limited by small datasets, heterogeneous protocols, and scarce real-world evaluation. Prospective multicenter evidence with standardized

methodology and robust external validation are required to support the integration of AI-video analysis in neurological care.

Disclosure: Nothing to disclose.

OPR-092 | Speech-based biomarkers for scalable detection of Alzheimer's disease, mild cognitive impairment, and Parkinson's disease

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Background and Aims: Early detection of neurodegenerative disorders is limited by the cost, accessibility, and burden of standard clinical assessments. Speech analysis offers a low-burden, non-invasive, and scalable alternative. This study evaluates a unified speech-based framework for detecting Alzheimer's disease (AD), Mild Cognitive Impairment (MCI), and Parkinson's disease (PD) from spontaneous speech, with an emphasis on robustness across clinical sites, recording conditions, and speech varieties.

Methods: Spontaneous speech recordings were analyzed from multiple clinical and research cohorts, including six datasets for cognitive impairment and five for PD, each treated as an independent domain. Audio was standardized and processed using a shared self-supervised speech encoder. To reduce site-, language-, and recording-related bias, domain-adversarial learning was applied. For cognitive impairment, binary (AD vs. healthy control), three-class (healthy/MCI/AD), and ordinal ($\geq$ MCI, $\geq$ AD) classifiers were trained using a two-phase strategy combining pretraining and multitask learning. PD detection used binary classification, including a model adapted to Swiss-dialect speech. Performance was evaluated using five-fold cross-validation.

Results: For cognitive impairment, AD versus healthy classification achieved a sensitivity of 0.794 and specificity of 0.911, while MCI versus healthy reached a sensitivity of 0.851 and specificity of 0.782; three-class AD/MCI/healthy classification showed a macro-sensitivity of 0.723 and macro-specificity of 0.860. For Parkinson's disease, baseline detection achieved a sensitivity of 0.659 and specificity of 0.937, while dialect-adapted models improved sensitivity to 0.845 and specificity to 0.831.



FIGURE 1 Schematic representation of the neural, physiological, and acoustic levels involved in speech production, from cortical and brain-stem control to vocal tract articulation and the resulting acoustic signal.

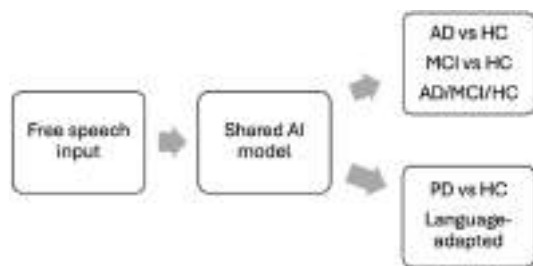


FIGURE 2 Overview of the unified speech analysis framework, in which a shared model processes spontaneous speech and supports multiple classification tasks for cognitive impairment and Parkinson's disease.

TABLE 1 Sensitivity and specificity of speech-based models for Alzheimer's disease, mild cognitive impairment, and Parkinson's disease.

Task	Sensitivity	Specificity
AD vs Healthy	0.794	0.911
MCI vs Healthy	0.851	0.782
AD / MCI / Healthy (macro)	0.723	0.86
Parkinson's Disease (baseline)	0.659	0.937
Parkinson's Disease (dialect-adapted)	0.845	0.831

Conclusion: Speech-based biomarkers enable scalable, low-burden screening and monitoring of neurodegenerative diseases and show robust performance across disorders, centers, and speech varieties, supporting their potential integration into routine neurological practice.

Disclosure: Lara Gervaise holds equity in Virtuosis.

OPR-093 | Machine learning for multiple sclerosis: Classification, phenotype differentiation and disability prediction using demographic, clinical and MRI data

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Background and Aims: Multiple sclerosis (MS) shows marked heterogeneity in clinical presentation and progression patterns. Here, we used machine learning (ML) to investigate the combined ability of demographic, clinical and MRI variables in: (i) identifying MS patients from healthy controls (HC); (ii) classify MS phenotypes; and (iii) predict disability, assessed using the Expanded Disability Status Scale (EDSS) score.

Methods: From the Italian Neuroimaging Network Initiative, we selected 1554 MS patients (1143 relapsing/411 progressive MS) and 520 age-matched HC. Participants underwent neurological evaluation and T2-/3D T1-weighted MRI, from which lesion volume (LV), normalized whole-brain and tissue compartment volumes were calculated. Different ML models (including support vector machines and multi-layer perceptron) were implemented for the above-mentioned classification tasks. SHAP analysis ranked the most important variables.

Results: The described ML tools were able to classify MS patients vs. HC with an accuracy ranging from 94% to 96%. Measures consistently showing top contributions included T2 LV and normalized cerebellar/brainstem volumes. ML models were able to classify relapsing from progressive MS patients with 92% accuracy. Top discriminating variables were EDSS score, age, normalized cortical grey matter volume, and normalized hippocampal/thalamic volumes. Finally, ML models were able to predict EDSS score with 0.74–0.76 intra-class correlation. The top variables contributing to EDSS prediction were T2 LV, sex, normalized cortical grey matter and thalamic volumes.

Conclusion: ML models were able to differentiate MS patients from HC, classify MS subtypes and predict disability with high accuracy, supporting the potential of ML in advancing personalized MS care.

Disclosure: Funded by the European Union, Next Generation EU, NRRP M6C2, Investment 2.1 Enhancement and strengthening of biomedical research in the NHS, PNRR-MAD-2022-12376530, CUP master C43C22001290007 MA Rocca received consulting fees from Biogen, Bristol Myers Squibb, Roche; and speaker honoraria from Alexion, Biogen, Bristol Myers Squibb, Celgene, Horizon Therapeutics Italy, Merck Serono SpA, Mitsubishi-Tanabe Pharma, Neuraxpharm, Novartis, Roche, Sandoz, and Sanofi. She receives research support from the MS Society of Canada, the Italian Ministry of Health, the Italian Ministry of University and Research, and FISM. P. Valsasina, L. Storelli, N. Tedone, S. Tommasin, A. Bisecco have nothing to disclose. P. Preziosa received speaker honoraria from Roche, Biogen, Novartis, Merck, Bristol Myers Squibb, Genzyme, Horizon and Sanofi. P. Pantano has received funding for travel from Novartis, Genzyme, and Bracco, and a speaking honorarium from Biogen. She receives research support from the Italian Ministry of Health, the Italian Ministry of University and Research, and FISM. A. Gallo received speaker and consulting fees from Biogen, Bristol Myers Squibb, Genzyme, Janssen, Juvisè, Merck Serono, Mylan, Novartis, Roche, Sandoz, and Teva, and receives research support from the Italian Ministry of Health, the Italian Ministry of University and Research, and FISM. A. Bianchi received a research grant from

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Movement Disorders 2

OPR-094 | Primary brain calcification: Survey on the efficacy of medical therapy on movement and psychiatric disorders in an Italian cohort

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Background and Aims: Primary Brain Calcification (PBC) is a neurodegenerative disorder characterized by brain calcium deposition, causing movement disorders, psychiatric/cognitive disturbances. Evidence on symptomatic treatment efficacy is limited. We here assess response to medical therapy in a cohort of PBC patients.

Methods: Patients diagnosed with PBC through CT-scan and blood tests at Padova University underwent clinical evaluation and pharmacological anamnesis; treatment response was rated by neurologists and subjective patients' perception as absent, partial, or satisfying.

Results: 35 PBC subjects with movement disorders underwent pharmacological (32) and/or rehabilitation treatment (24), with a multidrug approach in 65.5%. Parkinsonism was the most common phenotype (75%); all patients received Levodopa, dopamine agonists in 13 (54%), MAO/COMT-inhibitors in 6 (25%). Dystonia was treated with anticholinergics or botulinum toxins in 7 cases (22%), benzodiazepine/antiepileptics were added in resistant cases without further benefit; anticholinergic and carbamazepine were effective in 2 PKD subjects. Essential/dystonic tremor was treated with beta-blockers or antiepileptics (4 subjects, 12.5%). Levodopa trial was adopted in tremor or dystonia as second line treatment, and in 13 subjects with

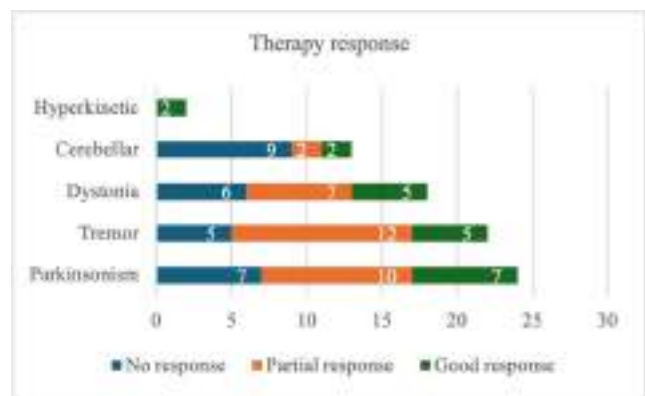


FIGURE 1 Therapy response according to patients and clinician's survey in different movement disorders.

cerebellar symptoms. Overall, 28% of patients had a satisfying response and 37% a partial response. Cerebellar symptoms, dysarthria, and atypical parkinsonism responded significantly worse ($p=0.03$) (Figure 1). Psychiatric symptoms required treatment in most patients (33), achieving satisfactory control in 80%; main drugs were SSRIs, atypical antipsychotics, anxiolytics and benzodiazepines.

Conclusion: Symptomatic medical treatment provides significant benefit in over half of PBC patients, except for cerebellar features, supporting the need for systematic therapeutic evaluation. Systematic assessment will help in defining therapy guidelines in the future.

Disclosure: Data partially submitted for 2026 Italian movement disorders congress. Nothing to disclose.

OPR-095 | Continuous subcutaneous foslevodopa/foscarbidopa infusion in advanced Parkinson's disease: results from a prospective real-world study

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Background and Aims: In advanced Parkinson's disease (APD), motor fluctuations and dyskinesias often persist despite optimized oral therapy, supporting the transition toward continuous dopaminergic delivery. Recently, continuous subcutaneous foslevodopa/foscarbidopa (LDp/CDp) became available, but real-world data remain limited. This study aimed to evaluate the effectiveness and tolerability of continuous subcutaneous LDp/CDp infusion in patients with APD in a real-world setting.

Methods: This prospective observational study included APD patients referred to the outpatient clinics for device-aided therapies at Fondazione IRCCS Istituto Neurologico "Carlo Besta", consecutively screened from March 2025 over the following 18 months and followed-up for 6 months after treatment initiation. Motor complications were assessed using the Movement Disorder Society–Unified Parkinson's Disease Rating Scale part

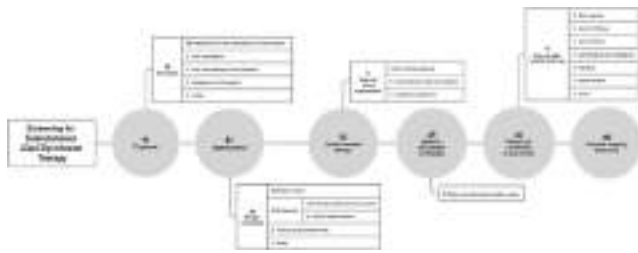


FIGURE 1 Flowchart of patients screened and initiated on continuous subcutaneous Foslevodopa/Foscarbidopa infusion therapy.

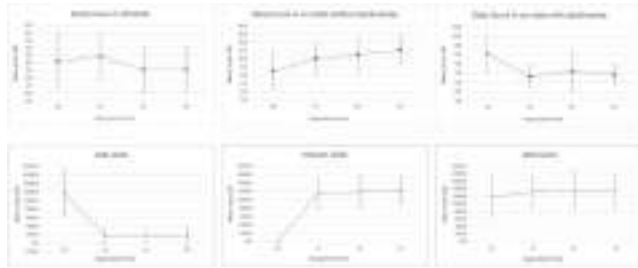


FIGURE 2 Longitudinal changes in motor-diary outcomes and LEDD.

IV (MDS-UPDRS IV), the MDS–Unified Dyskinesia Rating Scale (MDS-UDysRS) and 3-day Hauser diaries. Non-motor symptoms were assessed using validated scales. Levodopa equivalent daily dose (LEDD), adverse events and treatment discontinuations were recorded.

Results: Out of ninety-nine patients screened, 45 patients were initiated on LDp/CDp and followed up in outpatient clinics. Treatment discontinuation occurred in 9 (20.0%) patients, mainly due to local infusion-site reactions. Among patients who continued therapy, MDS-UPDRS IV, MDS-UDysRS scores and daily on-time with dyskinesias significantly decreased, while on-time without dyskinesias increased. Oral LEDD was reduced, while total LEDD increased, reflecting continuous 24-h dopaminergic delivery. Non-motor symptoms showed a tendency toward improvement.

Conclusion: Continuous subcutaneous LDp/CDp infusion reduced motor complications and improved on-time quality with acceptable tolerability in a real-world cohort of APD, supporting its use in clinical routine with appropriate patient selection and follow-up.

Disclosure: Nothing to disclose.

OPR-096 | Unraveling multiple system atrophy through skin biopsy: Biomarkers of autonomic dysfunction and α -synuclein aggregation

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Background and Aims: Multiple System Atrophy (MSA) is a progressive neurodegenerative disorder characterized by marked clinical heterogeneity. Identifying accessible biomarkers that mirror underlying neuropathology and differentiate MSA subtypes remains an unmet need. This study explores the skin as an accessible window, aiming to identify peripheral signatures that distinguish MSA phenotypes and capture clinically meaningful differences.

Methods: We conducted a cross-sectional study in 49 patients with clinically established MSA (22 MSA-P, 27 MSA-C). Participants underwent comprehensive clinical and instrumental evaluations, including brain magnetic resonance imaging, DaTSCAN, cardiovascular autonomic testing, and sudomotor assessment. Peripheral biomarkers included α -synuclein seed amplification assay (SAA) performed on olfactory mucosa, and skin biopsy analyses assessing phosphorylated α -synuclein (p-syn) deposits and synaptic terminal density. High-resolution confocal images were analyzed using an automated image software to quantify synaptic markers.

Results: MSA-C was associated with more severe orthostatic hypotension (OH) compared with MSA-P. A postganglionic cholinergic sudomotor impairment was observed across nearly all patients. Synaptic terminals co-expressing vasoactive intestinal peptide and tyrosine hydroxylase were significantly denser in MSA-P than in MSA-C, suggesting phenotype-specific compensatory or regenerative mechanisms; p-syn deposits exhibited a distal-to-proximal gradient in both somatic and autonomic fibers and correlated significantly with OH. Integrating p-syn pathology with SAA results increased sensitivity to 90% in differentiating MSA subtypes, with synaptic loss accounting for most remaining negative cases.

Conclusion: By using a multimodal approach that integrates clinical, molecular, and morphological data, this study provides novel insights into MSA heterogeneity and support the skin as a meaningful source of disease-relevant biomarkers to refine MSA characterization.

Disclosure: Nothing to disclose.

OPR-097 | Local field aperiodic spectral power modulated by deep brain stimulation in Parkinson's disease

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Background and Aims: Aperiodic spectral broadband power has been described recently as reflecting Parkinson's disease (PD) severity. It has therefore become an increasing focus of research interest in the context of the new adaptive deep brain stimulation (aDBS) approach. We aimed to study the influence of DBS on the main parameters of the aperiodic spectral component of local field potentials (LFPs).

Methods: LFPs were recorded from the subthalamic nucleus (STN) in patients with PD ($n = 22$) during a simple experimental paradigm that included 5 min of resting state and a short gait

task during DBS “off” and “on” conditions (“off” medication). Classical spectral analysis was performed using Fast Fourier Transform (FFT); the analysis of the aperiodic component was performed by fitting oscillations and a one-over-F (FOOOF) approach. In a subset of the patients ($n=14$), it was possible to evaluate the symptom progression over time after 1 year by analyzing the control measurement.

Results: The severity of hypokinetic/rigid symptomatology, measured by MDS-UPDRS, correlated significantly with both of the aperiodic components characteristics of the STN LFPs: the slope and the offset. These parameters were significantly modified by DBS, as evaluated with a Wilcoxon signed-rank test during the resting state and during the gait task. Aperiodic power remained stable over time during the control measurements.

Conclusion: The aperiodic component in LFPs may reflect pathological activity of the neuronal network and might serve as a new potential clinical marker in PD. The variable relation to conventional beta oscillopathy indicates the involvement of multiple neural mechanisms.

Disclosure: Nothing to disclose.

OPR-098 | Clinical progression and genetic pathways in body-first and brain-first Parkinson's disease

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Background and Aims: Recently, two main Parkinson's disease (PD) phenotypes have been proposed: “body-first,” where α -synuclein pathology begins in the peripheral nervous system and spreads symmetrically from bottom-up, and “brain-first,” where pathology starts in the brain and spreads asymmetrically downwards. However, no studies have assessed these phenotypes across both prodromal and clinical PD stages, tracked their pathological progression in vivo or identified potential underlying biological mechanisms.

Methods: We analyzed 910 prodromal and 1120 clinical PD cases with comprehensive longitudinal clinical, imaging, and genetic data from the Parkinson Progression Marker Initiative over a 12-year period.

Results: Both prodromal and clinical groups with body-first symptoms exhibited worse longitudinal motor progression and attention decline compared to brain-first cases. The body-first and brain-first phenotypes were stable over time, identifiable using unsupervised deep learning, and predicted conversion to clinical PD in prodromal cases. Additionally, body-first cases displayed more pronounced changes in the caudal locus coeruleus (LC), symmetrical alterations in the striatum and glymphatic system, consistent with the traditional Braak's bottom-up

progression of α -synuclein pathology and the more symmetric distribution proposed for body-first PD. In contrast, brain-first cases exhibited changes in the rostral LC and asymmetric alterations in the striatum and glymphatic system, suggesting a top-down progression. Genetic analysis identified new specific single-nucleotide polymorphisms associated with PD phenotypes (e.g., TRIM40, IP6K2) linked to worse outcomes in prodromal cases.

Conclusion: Recognising body-first and brain-first PD as distinct entities with unique clinical, imaging, and genetic profiles paves the way for targeted and personalised therapeutic strategies that address the specific pathophysiological mechanisms of PD.

Disclosure: Nothing to disclose.

OPR-099 | Activity of brainstem nuclei of PD patients during noninvasive vagal nerve stimulation: An fMRI study

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Background and Aims: Transcutaneous auricular vagus nerve electrostimulation (taVNS) is under investigation as add-on therapy for Parkinson's disease (PD). fMRI studies in healthy cohorts show taVNS activates nucleus tractus solitarius (NTS) and locus coeruleus (LC). Whether this applies to PD is unknown. We aimed to quantify taVNS effects on LC and NTS activation in PD, using fMRI.

Methods: We enrolled 40 PD patients in our double-blind, sham-controlled, within-subject, fMRI study (NCT05967598). 32 were analyzed. During a single ON-medication visit, each participant received taVNS at 100 Hz (taVNS100), taVNS at 25 Hz (taVNS25), and sham, in randomized order during which fMRI was obtained. Activity of LC and NTS was analysed using LME models accounting for stimulation type, degeneration-onset side (Deg_onset_side), and UPDRS-III-tertiles. After

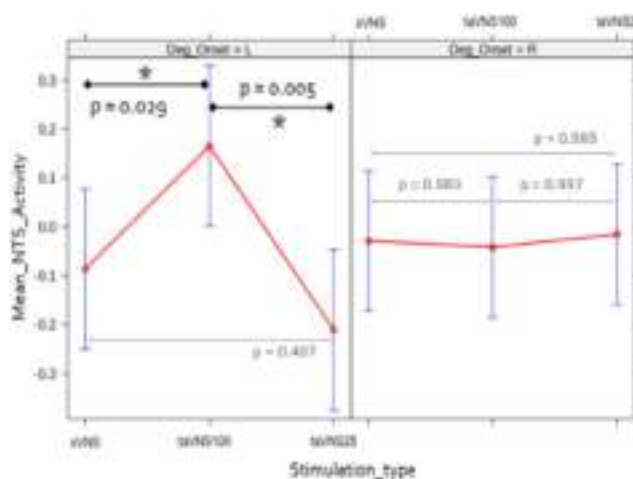


FIGURE 1 In left-onset PD, taVNS100 exceeded both taVNS25 ($p=0.005$) and sham ($p=0.029$). No NTS differences were detected in right-onset PD.

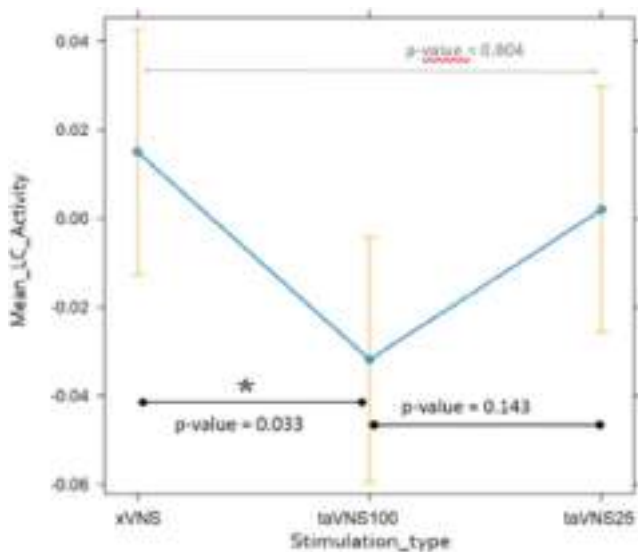


FIGURE 2 LC activity was decreased during taVNS100 regardless of the side of the participants degeneration onset.

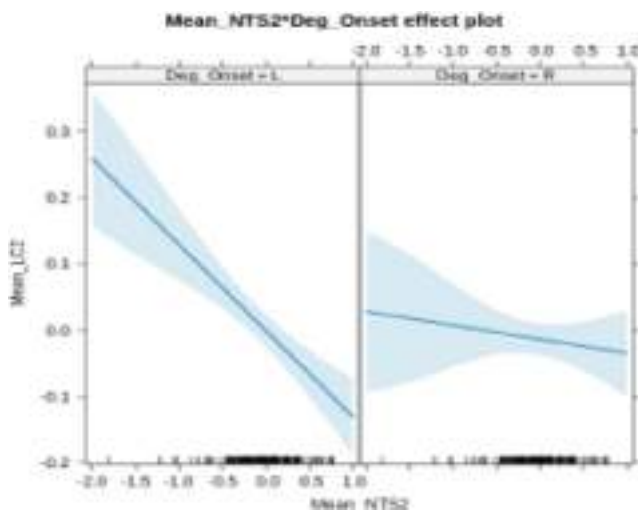


FIGURE 3 Correlation between the NTS and LC ROI signal.

imaging, we assessed gait using motion sensors across stimulation conditions.

Results: NTS activation showed a main effect of taVNS type ($p=0.021$) and an interaction with Deg_onset_side ($p=0.011$), with taVNS100 increasing NTS activity versus taVNS25 ($p=0.018$). In left-onset PD, taVNS100 exceeded taVNS25 ($p=0.005$) and sham ($p=0.029$), while no NTS differences appeared in right-onset PD. LC activity showed a stimulation-type effect ($p=0.034$), driven by reduced LC activity during taVNS100 versus sham ($p=0.033$). UPDRS-III-tertiles did not affect nuclei responses. LC activity was predicted by NTS activity with a Deg_onset_side interaction ($p=0.007$), showing a negative NTS–LC correlation in left-onset PD. Stimulation had no effect on gait.

Conclusion: taVNS100 reduced LC activity and elevated NTS activity in patients with left Deg_onset_side. This suggests taVNS has PD specific effects on brainstem-nuclei activation, which are driven by disease asymmetry.

Disclosure: Nothing to disclose.

OPR-100 | Genetic determinants of apixaban plasma exposure and clinical outcomes in ischemic stroke patients

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Background and Aims: Apixaban is a direct factor Xa inhibitor used for stroke prevention in patients with atrial fibrillation. Its absorption and elimination are influenced by drug transporters and metabolic enzymes encoded by the ABCG2, ABCB1, CYP3A5, and SULT1A1 genes. Genetic polymorphisms in these pathways may contribute to variability in apixaban plasma exposure, but data in ischemic stroke patients remain limited. This study evaluated the impact of selected genetic polymorphisms on minimal apixaban plasma concentrations (Cmin) and clinical outcomes.

Methods: This retrospective monocentric study included consecutive ischemic stroke patients treated with apixaban. Genotyping for ABCG2 rs2231142, ABCB1 rs4148738, CYP3A5 rs776746, and SULT1A1 rs9282861 was performed, and Cmin was measured 12h after administration. Clinical follow-up assessed recurrent ischemic stroke, clinically relevant bleeding, thromboembolic events, and mortality.

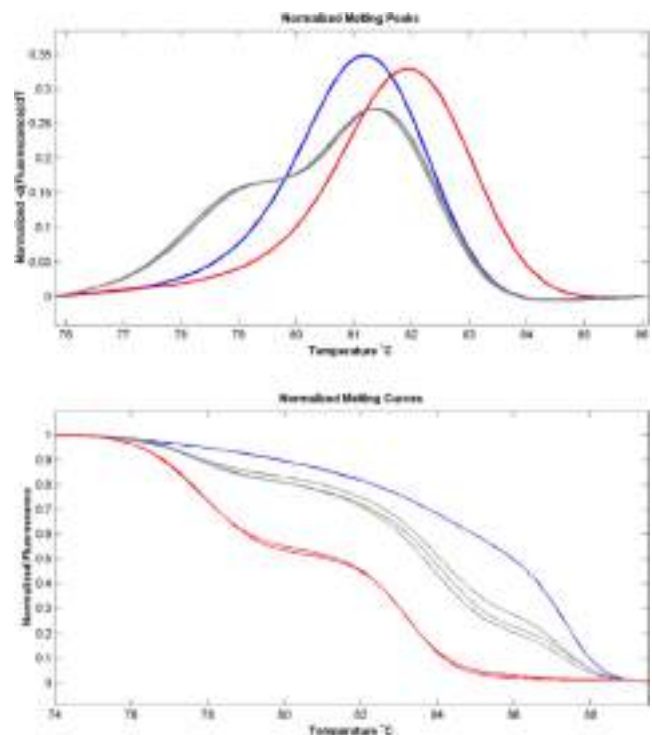


FIGURE 1 ABCB1 genotyping – graphical visualization of melting curves using HRM analysis with the LightScanner Master Mix system, where red curves represent patients without the ABCB1 rs4148738 allele (wild type), grey curves indicate heterozygotes, and blue curve.

Results: Eighty-three patients were included (53% male, mean age 70.4 years), with a mean follow-up of 47.2 months (326 patient-years); 37.3% received a reduced dose. Carriers of ABCG2 rs2231142 more frequently had apixaban plasma levels below the therapeutic range ($p=0.016$). No significant associations were observed for ABCB1, CYP3A5, or SULT1A1, and genetic variants were not independently associated with clinical outcomes.

TABLE 1 Distribution of genotypes and apixaban plasma levels in patients treated with apixaban.

Genotype	Patients, n (%)	Median apixaban plasma level (ICP, ng/ml)
CYP3A5 rs17746 - WT	71 (82.8)	111.34 (70.79–193.00)
CYP3A5 rs17746 - Heterozygous	6 (7.2)	93.77 (70.88–159.89)
CYP3A5 rs17746 - Homozygous	0	NA
CYP3A5 rs17746 - Variant allele carrier	6 (7.2)	93.77 (70.88–159.89)
ABCG2 rs2231142 - WT	60 (72.5)	130.48 (77.81–193.00)
ABCG2 rs2231142 - Heterozygous	22 (28.5)	187.28 (93.87–302.40)
ABCG2 rs2231142 - Homozygous	1 (1.2)	138
ABCG2 rs2231142 - Variant allele carrier	23 (27.7)	187.28 (93.87–302.40)
SULT1A1 rs9282861 - WT	29 (34.9)	111.34 (70.79–193.00)
SULT1A1 rs9282861 - Heterozygous	54 (65.1)	108.87 (75.80–155.80)
SULT1A1 rs9282861 - Homozygous	0	NA
SULT1A1 rs9282861 - Variant allele carrier	54 (65.1)	108.87 (75.80–155.80)
ABCB1 rs4148736 - WT	29 (34.9)	98.09 (74.58–129.99)
ABCB1 rs4148736 - Heterozygous	29 (34.9)	120.76 (89.87–174.80)
ABCB1 rs4148736 - Homozygous	70 (85.1)	90.00 (61.30–136.82)
ABCB1 rs4148736 - Variant allele carrier	58 (69.1)	114.27 (82.14–165.28)

Apixaban plasma levels are presented as median (interquartile range, ICP). * Plasma level is a constant value due to a single patient.

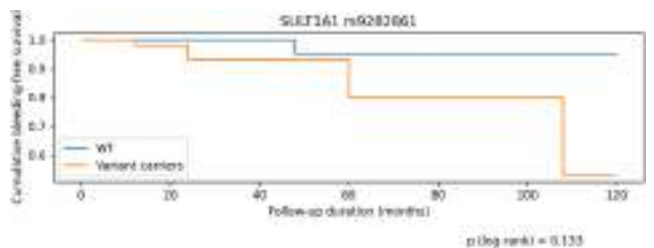


FIGURE 2 Kaplan–Meier curve for the occurrence of bleeding complications in carriers of SULT1A1 rs9282861.

Conclusion: The ABCG2 rs2231142 polymorphism is associated with subtherapeutic apixaban plasma levels in ischemic stroke patients, suggesting a potential role for pharmacogenetic guidance of apixaban dosing.

Disclosure: Nothing to disclose.

OPR-101 | Streamline-based lesion-symptom mapping reveals network substrates of post-stroke disability

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Background and Aims: Although many factors are associated with post-stroke disability (including initial severity, age, education, co-morbidity, and social support), the underlying anatomical determinants remain unclear.

Methods: We applied a novel method, whole-brain streamline-based lesion-symptom mapping (SLSM) (Figure 1) in 493 patients with stroke and a single ischaemic lesion on acute MRI (<1 week). This approach was used to delineate fibre bundles predicting the modified Rankin Scale (mRS) at the chronic stage (>3 months).

Results: SLSM identified afferent (spinothalamic) and efferent (corticospinal tract) projection fibres, interhemispheric transcallosal fibres, and three association fibre bundles as predictors of chronic mRS (Figure 2). The association bundles corresponded to dorsal and ventral pathways, as well as streamlines linking the temporal, parietal, and occipital lobes, consistent with the middle longitudinal fasciculus (“caudal pathway”).

Conclusion: SLSM is a new technique that constructs whole-brain connectomes at the single-streamline level without predefined tract templates. Disability after stroke is not only explained by damage to afferent and efferent projections. Dorsal and ventral pathways converge in frontal and posterior hubs to integrate sequential processing with deeper structural analysis (“syntax”). The caudal pathway supports multimodal integration in the angular gyrus and abstraction in the anterior temporal lobe (“semantics”). This framework enables abstraction and generalisation, crucial abilities when adapting to novel situations during recovery.

Disclosure: CW, FH, EvdH, MR are supported by EBRAINS2.0.

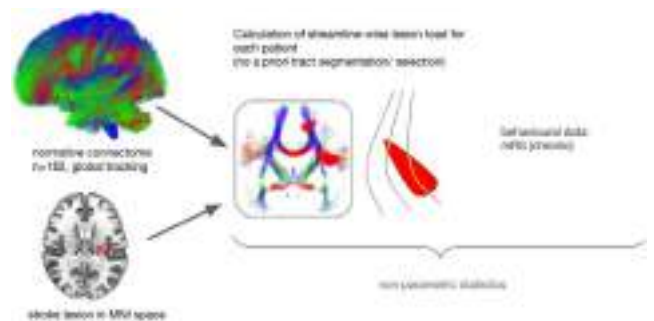


FIGURE 1 Streamline-based lesion symptom mapping (SLSM).

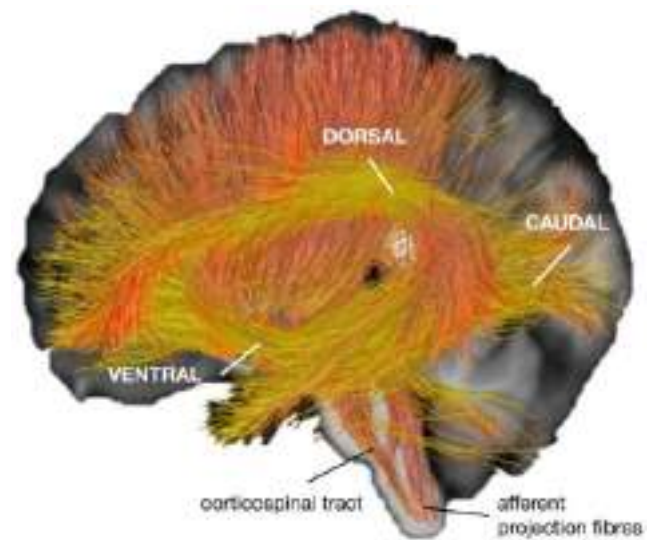


FIGURE 2 Affected streamlines correlating with mRS (chronic).

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Background and Aims: Oral anticoagulants (OACs), including vitamin K antagonists (VKAs) and direct oral anticoagulants (DOACs), reduce ischemic stroke risk in atrial fibrillation (AF), yet 1%–2% of patients still experience breakthrough strokes annually, not fully explained only by poor adherence. Clarifying underlying mechanisms is essential to improve prevention strategies.

Methods: ASPERA (NCT06823466) is a multicenter ambispective study; this analysis includes the retrospective arm of AF patients with neuroimaging-confirmed ischemic stroke despite ongoing therapeutic OAC between 2020 and 2025, with collection of baseline characteristics, comorbidities, treatments, and 90-day outcomes.

Results: Among 1649 patients (median age 80.4 years, 52.2% females) most were treated with DOACs (77.3%). Causal factors for breakthrough ischemic stroke included drug-to-drug interactions potentially affecting OAC efficacy (26.4%), competing etiologies (24.3%), and active cancer (4.4%). Overall, 180 (10.9%) had more than one potential cause and 724 (43.9%) of patients had at least one of those factors. Patients with almost one identifiable factor had a higher prevalence ($p < 0.01$) of arterial hypertension (87.3% vs. 76.3%), diabetes mellitus (30.1% vs. 24.3%), and dyslipidemia (58.1% vs. 45.8%) and a higher rate of minor stroke (NIHSS ≤ 5) presentation (33.8% vs. 26.7%, $p = 0.002$) compared with those without. These patients also showed higher 90-day mortality (22.7% vs. 18.4%, $p = 0.032$) and stroke recurrence rates (4.8% vs. 2.4%, $p = 0.007$).

Conclusion: Approximately half of patients experiencing ischemic stroke despite ongoing oral anticoagulation have an identifiable mechanism that may have contributed to the event. Improved recognition and characterization of these factors may help reduce the risk of breakthrough stroke and warrant investigation in prospective studies.

Disclosure: Nothing to disclose.

OPR-103 | Maraviroc for stroke recovery (MASTER): Protocol and status of a phase 2 double-blind placebo-controlled randomized clinical trial

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Background and Aims: Despite advances in acute stroke care, stroke remains the leading cause of acquired disability, and no pharmacological therapy is currently available to enhance recovery beyond the acute phase. Preclinical studies suggest that inhibition of the C-C chemokine receptor 5 (CCR5) may promote functional recovery by enhancing peri-infarct cortical plasticity. However, its role in human stroke recovery remains unknown.

Methods: MASTER (MARaviroc for STroke REcovery) is a phase II, single-centre, randomized, double-blind, placebo-controlled trial. Eighty patients with moderate, incomplete upper limb motor impairment will be enrolled within 7 days of acute ischemic stroke and randomized (1:1) to receive oral maraviroc (300 mg twice daily) or placebo for 90 days, in addition to standard rehabilitation. The primary outcome is upper limb motor recovery at day 90, assessed using the Fugl-Meyer Assessment for the Upper Extremity (FMA-UE). Secondary outcomes include measures of motor skill learning and imaging markers of peri-infarct plasticity at day 90, evaluated using MRI

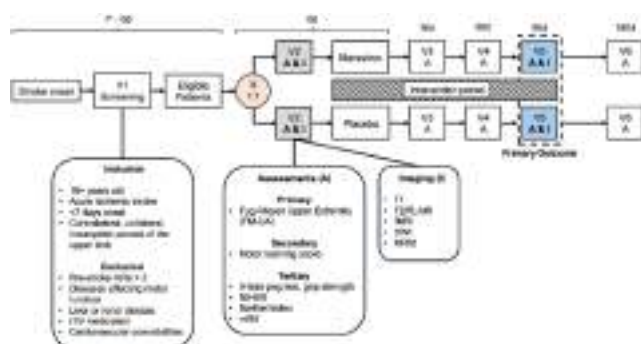


FIGURE 1 Study schematic for the MASTER trial. Patients are included following verification of inclusion and exclusion criteria. Randomization (R) is 1:1 to placebo (Mannitol) or Maraviroc group.

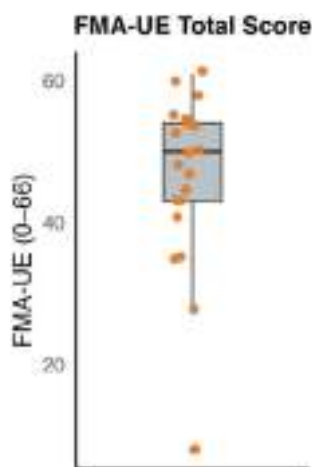


FIGURE 2 Box-plot of Fugl-Meyer scores for the upper extremity (FMA-UE) at baseline.

connectivity and spectroscopy. Additional clinical outcomes will be assessed longitudinally up to 6 months post-stroke.

Results: To date, 21 of 80 patients have been enrolled. Treatment compliance ranges from 69% to 100%. The cohort has a median age of 77 years, 43% are women, and median baseline NIHSS and FMA-UE scores are 5 and 50, respectively. Eight participants have completed the primary outcome assessment.

Conclusion: MASTER explores a novel pharmacological approach to enhance post-stroke motor recovery and to investigate its underlying mechanisms with the help of advanced MRI.

Disclosure: EC is supported by the Swiss National Science Foundation (SNSF; Grant no 215285).

OPR-104 | Sex-based outcomes after breakthrough ischemic stroke on oral anticoagulants for atrial fibrillation: ASPERA-R inverse probability weighted analysis

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Background and Aims: Sex-specific outcomes after breakthrough ischemic stroke on oral anticoagulation (OAC) are unexplored. We compared 90-day outcomes by sex and explored modifiers.

Methods: ASPERA-R (NCT06823466) was an international, multicenter, retrospective study enrolling adults (>18 years) with breakthrough ischemic stroke on OAC for AF. Primary outcome was 90-day return to baseline neurological (mRS 0–1 maintained if pre-stroke 0–1; or same/lower mRS if pre-stroke ≥ 2). Secondary outcome were 90-day mRS shift, recurrent ischemic stroke/TIA, myocardial infarction, all-cause and vascular death. Safety outcomes included 90-day moderate-to-severe bleeding, intracranial hemorrhage (ICH), 24-h hemorrhagic transformation, and 24-h symptomatic ICH. We applied inverse probability weighting and regression models to compare outcomes. Prespecified subgroup analysis tested sex-specific interactions.

Results: We included 1649 patients (women 52.2%; mean age 78.0 ± 10.7). Women were older (80.2 ± 9.6 vs. 76.3 ± 10.8 ; SMDunw = 0.376), had higher baseline NIHSS (13 [IQR 9–19] vs. 9 [IQR 4–17]; SMDunw = 0.227), and worse pre-stroke mRS (SMDunw = 0.237). After weighting, women were less likely to return to baseline neurological function (35.2% versus 42.7%; adjusted risk ratio 0.82, 95% CI 0.71–0.96; $p = 0.015$), had worse mRS distribution (adjusted OR 1.17, 95% CI 1.01–1.37; $p = 0.043$) and higher recurrent ischemic stroke/TIA (4.8% vs. 2.8%; adjusted HR 1.70, 95% CI 1.01–2.86; $p = 0.045$). Women showed a trend toward more moderate-to-severe bleeding (4.6% vs. 2.8%; adjusted HR 1.63, 95% CI 0.96–2.72; $p = 0.070$). Subgroup analyses revealed significant sex interactions for OAC type, competing etiology, endovascular treatment, and OAC restart.

Conclusion: Women had worse 90-day outcomes than men after breakthrough ischemic stroke on OAC for AF, highlighting the need for sex-aware management.

Disclosure: Prof Budincevic declares speaker's fees from Bayer, Boehringer Ingelheim, Viatrix, Novartis, Pliva-Teva, Abbott, Medis, Eli Lilly, Berlin Chemie, Pfizer, and Roche. Prof Pantoni

declares consultancy fees from Medtronic, PIAM and Amicus. Prof Sacco reports compensation from Novartis for other services; compensation from Novo Nordisk for consultant services; compensation from Boehringer Ingelheim for consultant services; compensation from Teva Pharmaceutical Industries for consultant services; compensation from Allergan for consultant services; employment by Università degli Studi dell'Aquila; compensation from Novartis for consultant services; compensation from Allergan for consultant services; compensation from PFIZER CANADA INC for consultant services; compensation from Abbott Canada for consultant services; compensation from H. Lundbeck A S for consultant services; compensation from AstraZeneca for consultant services; and compensation from Eli Lilly and Company for consultant services. Dr Zini declares consulting and speaker fees from Bayer, Boehringer-Ingelheim, Alexion, Daiichi Sankyo, Pfizer, PIAM, Amgen, fees for Advisory Board from Boehringer-Ingelheim, Daiichi Sankyo, Bayer and Astra Zeneca, not related to this study. The other authors report no conflicts.

OPR-105 | Impact of cancer on outcomes following breakthrough ischemic stroke on oral anticoagulants for atrial fibrillation: Insights from the ASPERA-R study

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Background and Aims: Breakthrough ischemic stroke during oral anticoagulation (OAC) for atrial fibrillation (AF) remains a major therapeutic challenge, particularly in patients with cancer, who have competing thrombotic and bleeding risks. We investigated the impact of cancer on 90-day outcomes after ischemic stroke during OAC.

Methods: We analyzed patients with AF experiencing ischemic stroke on continuous OAC enrolled in the international retrospective ASPERA-R study (35 stroke centers, 9 countries). Inverse probability weighting adjusted baseline imbalances. Weighted Cox, ordinal logistic, and generalized linear models estimated adjusted 90-day risks for primary (ischemic stroke or TIA), secondary (mRS shift, vascular/all-cause death), and safety outcomes (moderate-to-severe bleeding, intracranial hemorrhage, 24-h hemorrhagic transformation).

Results: Among 1649 patients (mean age 78.0 ± 10.7 years; 45.8% male), 247 (15.0%) had cancer (35.2% active, 64.8% remission). After weighting, cancer was associated with a higher 90-day risk of new ischemic stroke or TIA (8.2% vs. 2.8%; aHR 2.56, 95% CI 1.59–4.13; $p < 0.001$) and worse mRS distribution (aOR 1.29, 95% CI 1.08–1.54; $p = 0.005$). Active cancer conferred a >4-fold higher ischemic risk (HR 4.48, 95% CI 2.46–8.13; $p < 0.001$) and higher moderate-to-severe bleeding risk (HR 2.77, 95% CI 1.30–5.88; $p = 0.008$). Cancer in remission increased ischemic (HR 2.60, 95% CI 1.59–5.25; $p = 0.001$) but not bleeding risk. Hematological malignancies showed higher risks of ischemic events (HR 3.06, 95% CI 1.69–5.54; $p = 0.001$) and moderate-to-severe-bleeding (HR 3.47, 95% CI 1.57–7.70; $p = 0.006$) than solid tumors.

Conclusion: Cancer, particularly active and hematological malignancies, substantially worsens 90-day prognosis after breakthrough stroke on OAC, underscoring the need for refined risk stratification and tailored secondary prevention.

Disclosure: Nothing to disclose.

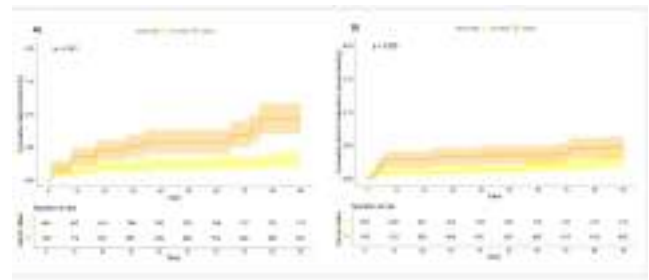


FIGURE 1

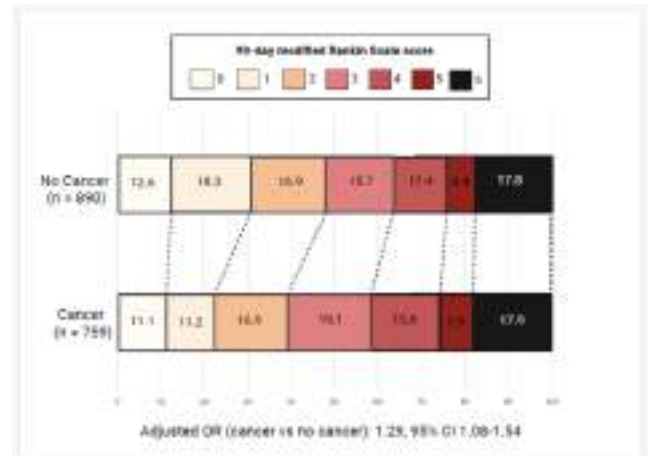


FIGURE 2 Adjusted OR (cancer vs. no cancer): 1.29, 95% CI 1.08–1.54.

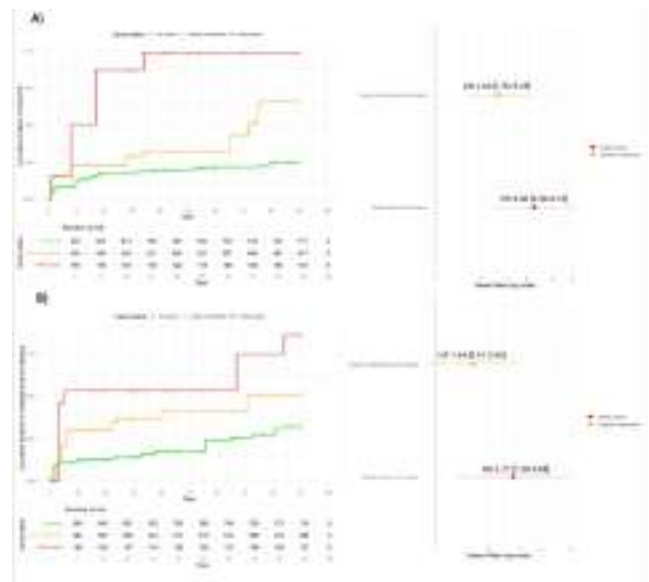


FIGURE 3

OPR-106 | Neuronal antibody discovery in unexplained cerebellar ataxia: Preliminary results

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Background and Aims: Over 40 autoantibodies associated to autoimmune cerebellar ataxia (ACA) have been discovered and this number is still growing. Antibody identification allows for definitive diagnosis and access to potentially beneficial immunotherapy. This is especially relevant for patients in whom an immune-mediated cause is not initially suspected, for example because of a chronic rather than subacute disease course.

Methods: We retrospectively identified patients with suspected ACA tested at the national referral center for antibody diagnostics and autoimmune encephalitis (cohort A) and patients with unexplained chronic cerebellar ataxia seen at the largest tertiary ataxia referral center in the Netherlands (cohort B). Patients positive for known ACA-associated antibodies were excluded. Serum or cerebrospinal fluid (CSF) samples were screened for antibodies by immunohistochemistry (IHC). Positive samples were tested on human induced pluripotent stem cell-derived live neurons (LN). Samples positive with both techniques were grouped according to clinical features and IHC/LN staining patterns and evaluated by immunoprecipitation/mass spectrometry (IP/MS) to identify novel antibody targets.

Results: After exclusion of 235 patients who tested positive for known antibodies (all cohort A), we enrolled 351 patients in cohort A (229 serum and CSF, 99 serum, 23 CSF) and 143 in cohort B (all serum). IHC and LN were both positive in 11/351 (3.1%) and 3/143 (2.1%) patients, respectively. Four patients of the IHC/LN double positive subgroup in cohort A had similar staining patterns and were tested by IP/MS (analysis pending).

Conclusion: The search for novel ACA-associated antibodies is not yet exhausted and should also include patients with a chronic disease course.

Disclosure: This study was funded by ZonMw (ACT-MD, 09120232310026). JK was supported by a Research Mobility Fellowship from the European Joint Programme on Rare Diseases (EJP RD) and is currently funded by the Erasmus Trustfonds. PSS holds a patent for the detection of anti-DNER and received research support from Euroimmun. MT has filed a patent, on behalf of the Erasmus MC, for methods for typing neurological disorders and cancer, and devices for use therein, and has received research funds for serving on a scientific advisory board of Horizon Therapeutics, for consultation at ArgenX, and for consultation at UCB. MT has received an unrestricted research grant from Euroimmun AG, and from CSL Behring.

BvdW receives research support from ZonMw, Hersenstichting, Dutch Scientific Organization, Christina Foundation, and FARA; has served on scientific advisory board or did paid consultancy for Biogen, Biohaven, Vico Therapeutics; and receives royalties from BSL – Springer. The other authors have nothing to disclose.

OPR-107 | Antibody functionalities during herpes-simplex-virus-1 infection are associated with disease severity and predict post-herpes autoimmune encephalitis

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Background and Aims: Herpes-simplex-virus-1 (HSV1) can cause recurrent cold sore (periphHSV) or encephalitis (HSE), which may be complicated by autoimmune encephalitis (postHSE-AE). The immunological mechanisms underlying HSE and postHSE-AE remain unclear.

Methods: We deeply characterized HSV1-antibody responses in paired serum/CSF of 45 HSE patients (12 who developed postHSE-AE, 33 who did not; at onset and 1 month), and in serum of 12 periphHSV. Profiles included Ig classes/subclasses, Fc receptor binding, and Antibody-Dependent Cellular/Neutrophil Phagocytosis (ADCP/ADNP), Complement Deposition (ADCD) and NK-cells activation (ADNKA). Antibody features were compared across groups and compartments, and correlated with disease severity and development of postHSE-AE. Neuronal death was quantified in HSV1-infected cultures treated with IgG from HSE, periphHSV or healthy controls.

Results: In HSE, HSV1-specific responses in CSF were low at onset, but significantly increased at 1 month, while serum responses remained elevated. LASSO/PLSDA analyses identified ADCD in serum and ADCP in CSF as features that differed most across compartments. Higher CSF-ADCP correlated with HSE severity. Compared to HSE, periphHSV patients showed higher serum ADNKA and FcγR3A binding despite similarly high IgG titers. Patients who developed PostHSE-AE exhibited higher titers, ADCD, ADNP and FcγR binding. IgG from HSE patients caused increased neuronal death in-vitro compared to IgG from periphHSV or healthy controls.

Conclusion: Our results indicate that, during HSE, HSV1-responses are compartmentalized and characterized by phagocytosis-activating antibodies in CSF that correlate with HSE severity and predict PostHSE-AE. NK-activating antibodies in periphHSV may protect the brain from HSE. Antibodies from HSE cause increased neuronal death, which likely promotes the development of autoimmune encephalitis.

Disclosure: Nothing to disclose.

OPR-108 | Frequent early cognitive impairment in both pediatric multiple sclerosis and myelin oligodendrocyte glycoprotein antibody-associated disease

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Background and Aims: The characterization of early cognitive impairment (CI) in pediatric multiple sclerosis (MS) remains limited due to methodological heterogeneity across previous studies. Moreover, standardized data for pediatric Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD) are scarce. Therefore, we aim to evaluate early cognitive dysfunction in both diseases and comprehensively characterize their neurocognitive profiles.

Methods: A cross-sectional study of children with MS and MOGAD (excluding acute demyelinating encephalomyelitis) assessed neurocognition within 2 years after disease onset using a standardized, comprehensive test battery covering all cognitive domains. Test results were converted to Z-scores. CI was defined as having Z-scores below -1.5 in ≥ 2 domains. Children were compared according to CI status and domain specific impairment was evaluated using composite domain Z-scores.

Results: Fifty-one participants were analyzed (MS $n=31$, MOGAD $n=20$). Full-scale intelligence was normal in both groups. Poor performance on the Bourdon-Vos Test indicated reduced sustained attention and processing speed. MS patients additionally showed lower scores on tasks of visuospatial processing and visuospatial memory. Overall, 48% of MS children and 45% of MOGAD children met criteria for CI. Domain-specific impairments were most prominent in complex attention, with significantly reduced composite Z-scores (MS: -1.26 ± 2.45 , $p < 0.001$; MOGAD: -1.08 ± 2.13 , $p = 0.006$). Only fatigue was significantly associated with the presence of CI (PedsQL fatigue Z-score: -2.55 vs. -0.75 , $p = 0.02$).

Conclusion: In both pediatric MS and MOGAD, early CI is prevalent with prominent difficulties in complex attention. Visuospatial functioning appears to be an additional vulnerable area for children with MS.

Disclosure: Michiel Buijze, Sandy Molenaar, Rosa Verkerk, Maud Quist and Willemijn van der Vlugt report no disclosures. Rinze Neuteboom participated in clinical trials with Roche, Novartis, Sanofi, Teva, Amgen and Livanova.

OPR-109 | Pathophysiology of pain in stiff person syndrome: An MRI and autoantibody study

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Background and Aims: Stiff-person syndrome (SPS) is a rare autoimmune neurological disorder characterized by stiffness,

spasms, anxiety, and pain. Pain is frequent but insufficiently understood and treated. Autoantibodies interfering with GABAergic inhibition may contribute to neuromuscular hyperexcitability and nociceptive processing. This study aimed to comprehensively characterize pain in SPS and to investigate its association with autoantibodies, peripheral nociceptor involvement, and central GABAergic dysfunction.

Methods: Thirty patients with SPS underwent detailed neurological examination and standardized pain assessment (NRS, GCPS, mRS). Structural brain MRI and proton MR spectroscopy of the insular cortex were performed. Serum autoantibodies were analyzed, peripheral blood mononuclear cells were stimulated to antibody-producing B cells, and serum binding to rat spinal cord and brain sections was assessed.

Results: Patients reported high levels of acute and chronic pain. Greater serum binding to GABAergic interneurons in the spinal cord was associated with higher pain intensity. Burning and electrifying pain, predominantly affecting the back and legs, were frequently reported. SPS patients showed reduced cortical thickness in the bilateral superior frontal gyrus, which correlated negatively with pain severity. Insular GABA levels were reduced compared with healthy controls and showed an association with autoantibody titers.

Conclusion: Pain is a prominent and clinically relevant feature of SPS and appears to be linked to autoantibody-associated peripheral nociceptor involvement and central GABAergic dysfunction. Improved understanding of pain mechanisms may support more targeted therapeutic strategies to reduce pain and disability in SPS.

Disclosure: Nothing to disclose.

OPR-110 | Long-term outcomes in anti-LGI1 encephalitis: A nationwide Dutch cohort study

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Background and Aims: Anti-leucine-rich glioma-inactivated 1 (LGI1) encephalitis is one of the most prevalent autoimmune encephalitides, yet data regarding long-term outcomes and treatment efficacy remain scarce. This study aims to assess long-term clinical outcomes, characterize relapses and compare the efficacy of different therapeutic approaches.

Methods: In this nationwide cohort study, patients diagnosed with anti-LGI1 encephalitis between August 2010 and January 2026 were included. Retrospective and prospective clinical data were collected. Primary outcomes included the modified Rankin Scale (mRS) and the Clinical Assessment Scale for Autoimmune Encephalitis (CASE).

Results: 215/240 patients were eligible for analysis. An interim analysis ($N=100$) demonstrated a median follow-up of 60 months (IQR 42–84; range 8–211). At least one relapse occurred in 24%,

with significantly lower incidence among those treated with rituximab. Median time to first relapse following immunotherapy was 6 months (IQR 4–37; range 3–83). Relapses typically mirrored the symptoms of the initial episode, albeit with reduced severity: median maximal mRS during the initial disease episode was 3 (IQR 2–3; range 1–5), compared to 2 (IQR 2–3; range 2–3) at the time of first relapse. At 2 years post-diagnosis, the median mRS score was 1 (IQR 1–2; range 0–3) and symptomatic epilepsy was present in 6% of patients.

Conclusion: This study will provide novel insights into long-term prognosis. Ongoing analyses aim to identify predictors for relapse and functional outcome. Furthermore, we seek to provide guidance on optimal treatment strategies regarding immunotherapy and duration of anti-seizure medication to improve patient care.

Disclosure: Peter A.E. Sillevius Smitt holds a patent for the detection of anti-DNER and received research support from Euroimmun. Maarten J. Titulaer has filed a patent, on behalf of the Erasmus MC, for methods for typing neurological disorders and cancer, and devices for use therein, and has received research funds for EpilepsieNL (NEF 14-19 & 19-08), Dioraphte (20010403), ZonMW (Memorabel fellowship, VIMP scheme and Open Competition [ACT-MD]), ItsME, Erasmus MC Foundation and Erasmus Trustfonds. MJT has served on a scientific advisory board of Horizon Therapeutics/AmGen, ArgenX and Arianys; for consultation at Guidepoint Global LLC, and at UCB. MT is co-PI of the EXTINGUISH trial. MT has received an unrestricted research grant from Euroimmun AG, and from CSL Behring. MJT and Juliette Brenner, on behalf of ErasmusMC, have filed a copyright for the PROSE (Patient-Reported Outcome Scale in Encephalitis). Jeroen Kerstens received a Research Mobility Fellowship from the European Joint Program on Rare Diseases (EJP-RD) and funding from the Erasmus Trust Foundation. Robin W. Van Steenhoven is funded by ItsME foundation and Erasmus MC foundation. Juna M. de Vries is local investigator for the CIELO Satralizumab in anti-NMDAR and anti-LGI1 encephalitis (Roche). JV received the AEA Community seed grant in 2021 for the PROMISE study. The other authors report no relevant disclosures.

OPR-111 | Duchenne muscular dystrophy drives T cell remodeling

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Background and Aims: Duchenne muscular dystrophy (DMD) is characterized by progressive muscle degeneration, yet systemic immunological consequences remain poorly understood. Recent fatalities following AAV-mediated gene therapy highlight critical gaps in baseline immune dysfunction knowledge. This study aimed to comprehensively characterize systemic immune profiles in glucocorticoid-free DMD patients using multi-omic approaches.

Methods: We performed single-cell transcriptomic and epigenomic profiling coupled with systemic cytokine analysis in glucocorticoid-free DMD patients. Immune cell populations

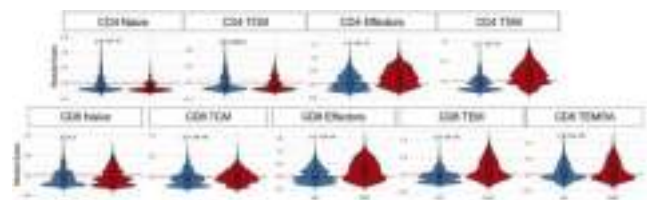


FIGURE 1 DMD muscles showed marked increases in CD4+ and CD8+ T cells and myeloid dendritic cells, while B cells were notably absent, suggesting selective immune cell recruitment driven by the inflammatory muscle.

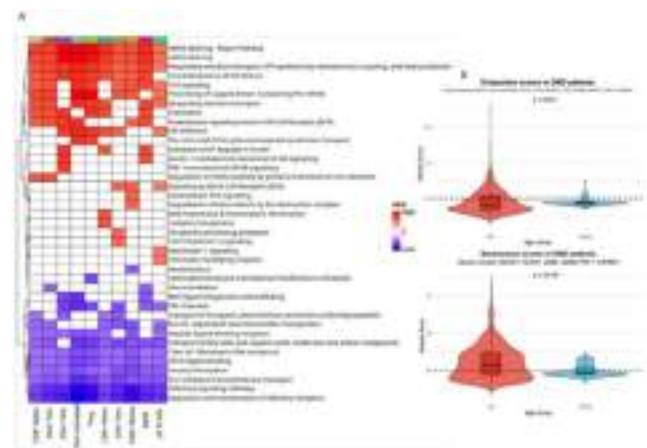


FIGURE 2 DMD patients exhibited significantly higher T cell exhaustion scores.

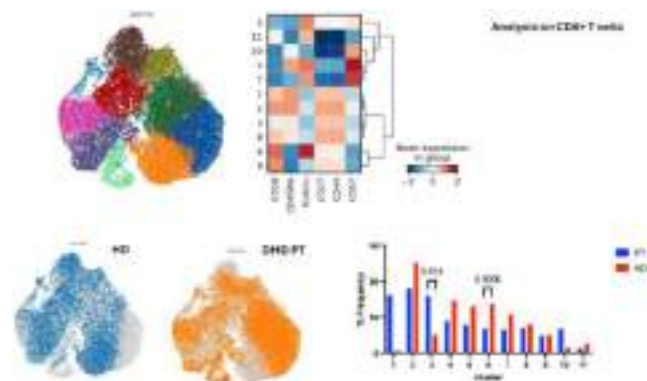


FIGURE 3 Circulating CD8+ T cells from DMD patients and healthy controls, revealing specific T cell subsets expressing multiple markers of both exhaustion and senescence.

were characterized across bone marrow, dystrophic muscle, and peripheral blood compartments. Cell-cell communication networks were analyzed, and findings were validated through meta-analysis across independent cohorts.

Results: DMD exhibited distinct immune microenvironments: bone marrow harbored transcriptionally primed yet phenotypically naïve lymphocytes with stress-responsive signatures (ATF3, FOSB) and metabolic reprogramming. Dystrophic muscle showed chronic T cell exhaustion with epigenetic imprinting. Paradoxically, circulating CD8+ memory T cells maintained TCF1+TIGIT– stem-like features despite tissue exhaustion. Systemic profiling revealed elevated

pro-inflammatory mediators (IL-6, TNF receptors, interferons) with impaired chemokine networks. Bone marrow networks centered on monocyte-mediated T cell priming, while muscle networks involved FAP-myofiber-macrophage triads driving remodeling.

Conclusion: Pre-existing immune priming in DMD—characterized by stress-responsive transcription, enhanced OXPHOS metabolism, and preserved T cell stemness—may predispose patients to exaggerated therapeutic responses, providing essential groundwork for predicting complications and designing immunomodulatory strategies.

Disclosure: Nothing to disclose.

Epilepsy 1

OPR-112 | AutoStim mode versus traditional vagus nerve stimulation in drug-resistant epilepsy: Long-term real-world outcomes from a large vagus nerve stimulation cohort

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Background and Aims: Vagus nerve stimulation (VNS) is an adjunctive therapy for drug-resistant epilepsy. Newer devices incorporating AutoStim deliver additional stimulation in response to ictal tachycardia (1–3). However, comparative data between AutoStim-enabled and conventional VNS remain limited. This study compared long-term clinical outcomes of VNS with AutoStim ON versus AutoStim OFF.

Methods: We conducted a retrospective observational cohort study of adults implanted with VNS between 2010 and 2024 at Ghent University Hospital (minimum follow-up of 12 months) (Table 1). Patients were classified according to exposure to AutoStim during follow-up. The AutoStim ON group included patients in whom AutoStim was activated at any time, while the AutoStim OFF group comprised patients treated exclusively with conventional VNS (ON: *n* = 142; OFF: *n* = 94). Outcomes included seizure frequency reduction, responder and best-responder rates (≥50% and ≥90% reduction), seizure freedom, total daily antiseizure medication load (TDL), and mortality. Mixed-effects negative binomial regression and analysis of covariance were applied.

Results: Responder rates were higher in the AutoStim ON group (51% vs. 41%, *p* = 0.011), as were best-responder rates (22% vs. 13%, *p* = 0.002) (Figure 1). Seizure freedom occurred in 17% versus 12% of patients, respectively (*p* > 0.05). Stimulation parameters did not differ significantly between groups (Table 2). Among responders, antiseizure TDL remained stable or decreased in the AutoStim ON group. Mortality was lower in the AutoStim ON group (6% vs. 15%, *p* = 0.03).

Conclusion: In this wide real-world cohort, VNS with AutoStim ON was associated with better seizure outcomes, stable

TABLE 1 Demographic description of the cohort. *Chi-square and Mann-Whitney tests applied. °8/21 (38.1%) patients had SE recurrence after VNS implantation on Open Loop Group, 11/40 (27.5%) in the Closed loop.

	AutoStim ON (n=142)	AutoStim OFF (n=94)	<i>p</i> -value*	Total (n=236)
Gender female, n (%)	81 (57)	47 (50)	0.35	128 (54)
Right-handed, n (%)	118 (83)	81 (86)	0.67	199 (84)
Depressive symptoms, n (%)	25 (18)	11 (12)	0.44	36 (15)
Impaired cognitive status, n (%)	66 (46)	45 (48)	0.64	111 (47)
Epilepsy surgery, n (%)	11 (8)	7 (7)	1	18 (8)
History of SE, n (%)°	40 (28)	21 (22)	<0.0001	61 (26)
Duration of follow-up, median (range) [y]	40.89±22	39.82±46	<0.0001	40±45
Death rate, n (%)	9 (6)	14 (15)	0.03	23 (10)
Timing access to VNS				
Age at epilepsy diagnosis, y (median - range)	8.5 (0 - 38)	8.6 (0 - 14)	0.27	8 (0 - 48)
Age at VNS implantation, y (median - range)	25.1 (1 - 85)	22.12 - 69	0.01	23 (0 - 89)
Duration of epilepsy, y (median - range)	15 (0 - 60)	23 (1 - 80)	0.008	17 (0 - 60)
ASMs failed prior to VNS, n (median - range)	9 (4 - 17)	30 (4 - 19)	0.008	9 (4 - 17)

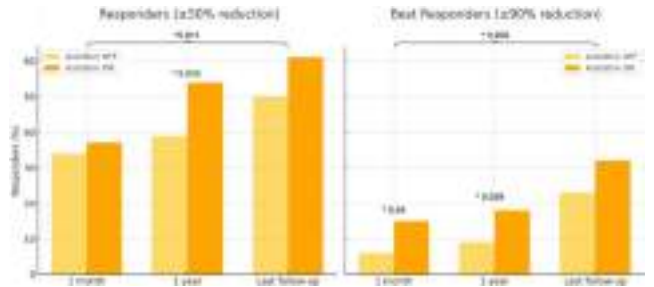


FIGURE 1 Comparison of the percentage of responders (≥50% seizure reduction) and best responders (≥90% reduction) at 1 month, 1 year, and last follow-up, with AutoStim ON versus OFF. Across all timepoints, AutoStim ON is associated with higher responder.

TABLE 2 Comparison of VNS stimulation parameters between AutoStim ON and OFF groups.

Parameter	AutoStim ON Mean±SD	AutoStim OFF Mean±SD	<i>p</i> -value
Output current (mA)	1.85±0.47	1.88±0.61	0.0722
Frequency (Hz)	24.37±5.25	24.73±5.65	0.5763
Pulse width (ms)	280.86±130.64	281.78±127.58	0.6524
Duty cycle (%)	28.87±12.18	19.73±13.89	0.3023
Sensory threshold (%)	11.02±17.22		NA

medication burden, and lower mortality compared with conventional VNS, supporting its use in drug-resistant epilepsy (4,5).

Disclosure: Nothing to disclose.

OPR-113 | Medial pulvinar stereo-electroencephalography biomarkers associated with deep brain stimulation response in focal drug-resistant epilepsy

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Background and Aims: Thalamic deep brain stimulation (DBS) represents an emerging therapeutic option for patients with focal drug-resistant epilepsy who are ineligible for or have failed resective surgery. To optimize outcomes and guide DBS lead placement, thalamic stereo-electroencephalography (SEEG) has been proposed.

Methods: This monocentric retrospective study aimed to identify interictal and ictal SEEG biomarkers of the medial pulvinar (PuM) associated with favourable PuM-DBS response. Six patients (four female, two male) underwent SEEG including PuM sampling, were deemed unsuitable for resective surgery, and subsequently received bilateral PuM-DBS. In total, eight PuM were sampled in SEEG: four bilaterally (two patients) and four unilaterally (four patients). The SEEG exploration covered both the PuM and the ipsilateral epileptogenic-zone network (EZN) in four patients, whereas in the other two the EZN was bilateral but PuM sampling was unilateral. All SEEG signal analyses were performed on the PuM sampling contacts available in each patient. Interictal SEEG analysis included spike-rates and non-linear functional connectivity (h2), while ictal analyses combined visual inspection with quantitative biomarkers: Epileptogenicity Index (EI), Connectivity Epileptogenicity Index (cEI), Permutation Entropy Index (PEI) and Delta Entropy (ΔE).

Results: Two patients were responders ($\geq 50\%$ seizure reduction at 1 year). PuM spike-rates, connectivity, EI and cEI values did not differentiate responders from non-responders. In contrast, entropy-based measures were significantly higher in responders: PEI (FDR- $p=0.024$) and ΔE (FDR- $p=0.034$).

Conclusion: Ictal PuM complexity disruption, quantified through entropy-based SEEG metrics (PEI and ΔE), may represent a candidate biomarker of response to medial pulvinar DBS and warrants validation in larger cohorts.

Disclosure: Nothing to disclose.

OPR-114 | Acute brain injury in cryptogenic new onset refractory status epilepticus

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Background and Aims: New onset refractory status epilepticus (NORSE) is an extremely severe form of status epilepticus. We aim to assess how extensive is seizure-induced acute brain damage in patients with cryptogenic NORSE (cNORSE) compared to etiology-defined status epilepticus (eSE), chronic epilepsy, and healthy controls.

Methods: This international cross-sectional cohort study includes 78 patients with cNORSE, 211 with eSE, 100 subjects with chronic epilepsy and 36 healthy participants. We measured neurofilament light chain (NfL) and S100-beta (S100B), respectively for neuronal and astroglial damage, in serum and cerebrospinal fluid (CSF).

Results: In cNORSE, NfL concentrations were tenfold higher in CSF and fourfold higher in serum compared with eSE ($p<0.0001$). Serum levels of NfL in cNORSE were twentyfold higher than in epilepsy and healthy controls' groups. Serum and CSF NfL values were strongly correlated (Spearman's $\rho=0.75$, $p<0.001$) and NfL levels rose sharply from week 1 to week 3 after cNORSE onset ($p<0.001$). On the contrary, S100B concentrations in serum and CSF did not differ between cNORSE and eSE. NfL levels discriminated cNORSE from other cohorts with an area under the receiver operating characteristic (ROC) curve between 0.79 (95% CI 0.68–0.90) versus eSE and 0.99 (95% CI 0.78–1.00) versus non-SE groups.

Conclusion: Acute neuroaxonal injury in cNORSE is extraordinarily severe, disproportionate to glial damage, and far greater than in other forms of status epilepticus. The rapid early rise in NfL highlights a narrow therapeutic window, underscoring the need for early, aggressive, and neuroprotective interventions.

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Background and Aims: Brain tumor-related epilepsy (BTRE) is a disabling complication of brain tumors and often remains poorly controlled despite multiple antiseizure medications (ASMs). Cenobamate (CNB) showed high efficacy in drug-resistant focal epilepsy, but evidence in BTRE is limited. This study evaluated the effectiveness and safety of CNB as add-on therapy in a real-world multicenter cohort of patients with BTRE.

Methods: We conducted a multicenter retrospective observational study across tertiary epilepsy centers in Italy. Adult patients with drug-resistant BTRE treated with adjunctive CNB were included. Monthly seizure frequency was assessed at baseline and follow-up. Outcomes were seizure frequency changes, responder rates, and safety. Effectiveness was analyzed in the intention-to-treat (ITT) population.

Results: Twenty patients (10 females; median age 48 years) were included. Tumor histologies were meningioma (35%), astrocytoma (20%), glioblastoma (15%), ganglioglioma (15%), oligodendroglioma (10%), and DNET (5%). Median follow-up duration was 12 months (IQR: 9.8–12). In the ITT cohort ($n = 20$), monthly seizure frequency significantly decreased from a median of 6 seizures (IQR 18.75) at baseline to 1 seizure (IQR 2.55) at last follow-up ($p = 0.006$). Median seizure reduction was 49.7%, with 50% of patients achieving a $\geq 50\%$ reduction, 30% achieving a $\geq 75\%$ reduction, and 20% becoming seizure-free. One patient discontinued treatment before the 3-month follow-up, and one patient with glioblastoma died before the 6-month follow-up. Adverse events were predominantly mild to moderate in severity.

Conclusion: In this cohort of patients with drug-resistant BTRE, CNB add-on therapy was associated with a substantial reduction in seizure frequency and acceptable tolerability, supporting CNB as a promising option in this challenging population.

Disclosure: Nothing to disclose.

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Background and Aims: Epilepsy is markedly more prevalent in individuals with intellectual disability (ID) than in the general population and is often associated with drug resistance and increased adverse effects [1]. This study aimed to evaluate the efficacy and safety of perampanel (PER) as the sole add-on therapy in patients with epilepsy and ID.

Methods: We performed a subanalysis of the PEROC study, an Italian multicenter, retrospective, longitudinal study assessing PER as the only add-on treatment [2]. Patients were stratified according to ID status into three groups: no ID (no-ID), mild ID (mID) and moderate/profound ID (mpID). Retention rate, responder rate ($\geq 50\%$ seizure reduction), seizure freedom and adverse events (AEs) were evaluated at 3, 6, and 12 months after PER introduction.

Results: The study included 356 patients: 277 no-ID, 44 mID, 35 mpID. Retention rates were slightly lower in mpID (57.1%) than mID (78.6%) and no-ID (72.7%), without significant differences ($p < 0.05$). Responder rates were comparable across groups, reaching 69.1%, in no-ID, 72% in mID, 75% in mpID at 12 months ($p = 0.848$). Seizure-freedom was higher in no-ID compared with mID and mpID at 3 (38.1%, 21.4%, 17.4%; $p = 0.046$) and 6 (42.3%,

20.7%, 19.2%; $p=0.011$) months, but no difference resulted at 12 months (46.3%, 40.5%, and 35%, respectively; $p=0.579$). AEs (mainly irritability, dizziness, drowsiness) occurred in 28.7% no-ID, 30.2% mID and 37.1% mpID patients, without significant difference ($p=0.585$).

Conclusion: Peramppanel as the only add-on therapy showed good effectiveness and tolerability in patients with ID, achieving meaningful seizure control and satisfactory retention rates in this challenging population.

Disclosure: Nothing to disclose.

OPR-117 | Performance of ictal electrical source imaging from routine clinical EEG to define the epileptogenic zone

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Background and Aims: Epilepsy surgery is the treatment of choice for drug-resistant focal epilepsy, provided the epileptogenic zone (EZ) is accurately identified. Electrical source imaging (ESI) allows three-dimensional cortical projection of EEG activity and may improve EZ localisation. Ictal analysis is more specific than interictal analysis. This study evaluates the diagnostic performance of ictal ESI using a standard clinical EEG montage in presurgical assessment.

Methods: Seventy-five patients underwent EEG recording with 37 electrodes. Individualised head models (BEM) were generated. Ictal ESI was performed using sLORETA and eLORETA. Seizure onset, time window (1–5 s), and frequency band were visually selected. Ictal power and dynamic functional connectivity (FC) were computed using Granger causality (sPDC). EZ was defined by the resection area or SEEG. Diagnostic performance was assessed using ROC analysis.

Results: A total of 213 seizures were analysed (median age 29 years); 73 patients underwent surgery, with 58.6% seizure-free at follow-up. MRI was positive in 54 cases, and 37 patients had mesiotemporal epilepsy. sLORETA and eLORETA showed comparable performance (AUROC 0.853 and 0.858, respectively). For eLORETA, sensitivity was 0.752, specificity 0.816, and accuracy 0.813. Performance was higher in seizure-free patients and in mesiotemporal epilepsy. Diagnostic accuracy varied with discharge morphology, highest for sinusoidal rhythmic patterns. FC metrics showed limited added value (AUROC 0.66). Patients with poor surgical outcome exhibited a more diffuse ictal power distribution.

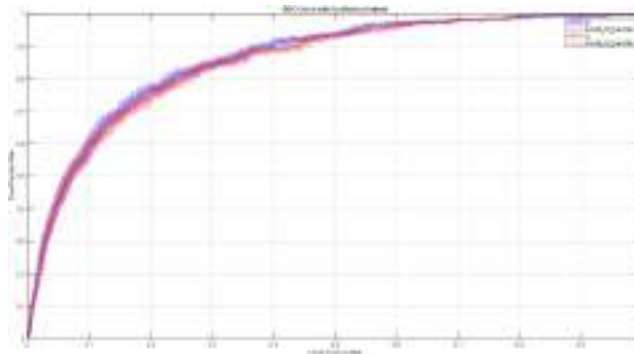


FIGURE 1 ROC curve eLORETA vs. sLORETA, ictal discharge power.

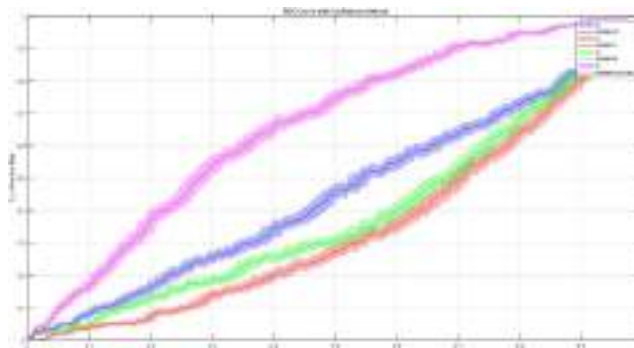


FIGURE 2 ROC curve for directional ictal connectivity (sPDC) using eLORETA.

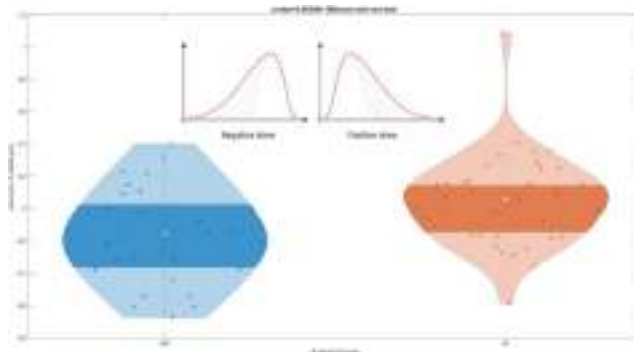


FIGURE 3 Violin plot, skewness of ictal frequency power distribution in eLORETA, depending on whether the patient is seizure-free or not.

Conclusion: Ictal ESI using standard EEG provides good accuracy for EZ localisation, particularly in seizure-free patients. Power-based ESI outperforms FC metrics, supporting its clinical relevance in presurgical evaluation.

Disclosure: Nothing to report.

OPR-118 | Discontinuation of ocrelizumab in multiple sclerosis: Re-occurrence of disease activity

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Background and Aims: Discontinuation of B-cell depleting therapies such as ocrelizumab in people with multiple sclerosis (pwMS) is increasingly discussed due to infection risk and hypogammaglobulinemia. However, prospective data on disease reactivation, disability progression, and optimal treatment duration after cessation are limited.

Methods: In this prospective two-centre observational cohort study, pwMS treated with ocrelizumab for ≥ 12 months without inflammatory disease activity in the preceding year were enrolled between 2018 and 2023. Patients who discontinued ocrelizumab were compared with continuers using 4:1 propensity score matching. Primary outcomes were time to combined inflammatory activity (CIA; relapse and/or new T2 lesions) and progression independent of relapse activity (PIRA). Receiver operating characteristic (ROC) analyses explored associations between treatment duration, discontinuation length, and disease activity.

Results: Among 655 eligible pwMS, 290 patients (58 discontinuers, 232 continuers) were included after matching. Median post-discontinuation follow-up was 28.5 months. Discontinuation was not associated with a significantly increased risk of CIA (hazard ratio [HR] 0.91, 95% CI 0.08–10.79) or PIRA (HR 4.8, 95% CI 0.38–60.2; Figure 1) compared with continuation. Disease activity remained largely stable during the first 24 months after discontinuation, with a numerical increase thereafter. ROC analyses suggested a plateau of anti-inflammatory benefit

after approximately 29–30 months of ocrelizumab treatment and a rising risk of disease reactivation beyond 30–32 months off-treatment.

Conclusion: In clinically stable pwMS, ocrelizumab discontinuation after about 30 months appears safe in the short term for 25–43 months. Careful monitoring is warranted beyond 2 years off-treatment to detect potential delayed disease reactivation.

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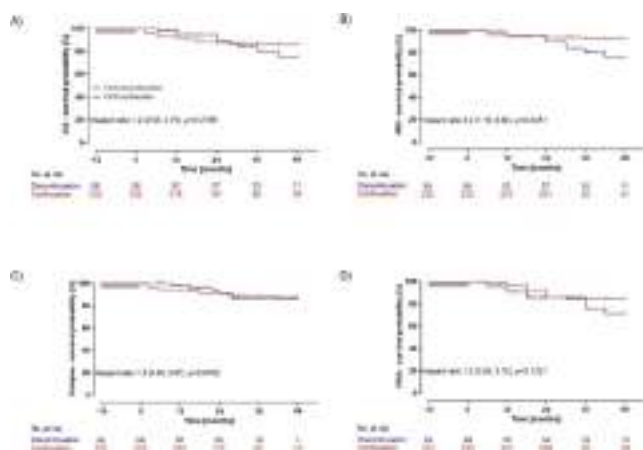


FIGURE 1 Effectiveness outcomes following ocrelizumab discontinuation. Depicted are propensity score matched patients with multiple sclerosis with discontinuation (blue lines) and with continuation (red lines) of ocrelizumab treatment. Onset of (A) combi.

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Background and Aims: Epstein–Barr virus (EBV) has been implicated in the pathogenesis of multiple sclerosis (MS). Elevated immunoglobulin G (IgG) titers against EBV nuclear antigen 1 (EBNA1) represent the most consistent serological marker of MS risk, with levels remaining persistently elevated following disease onset.

Methods: We conducted high-dimensional profiling of virus-specific antibody-associated immune features in 60 individuals with relapsing-onset MS followed over time and in 54 age- and sex-matched EBV-seropositive healthy controls.

Results: EBNA1-specific IgG in MS patients exhibited a distinct proinflammatory Fc signature, characterized by increased affinity for Fcγ receptors (FcγRs) and enhanced Fc-mediated effector functions, including antibody-dependent phagocytosis, complement activation, and natural killer cell engagement. This signature was not observed for antibodies against other common viral antigens and showed a strong association with MS. Increased FcγR binding by IgG specific for both EBNA1 and the

lytic EBV glycoprotein 350 correlated with disease activity, defined as clinical relapse and/or presence of contrast-enhancing brain lesions.

Conclusion: Both Fab- and pro-inflammatory functional Fc-mediated features contribute to the association between EBNA1-specific antibody responses and MS. Fc effector functions of EBV-specific antibodies may actively participate in promoting focal disease activity in MS.

Disclosure: Nothing to disclose.

OPR-120 | T-cell receptor excision circles and Kappa-deleting recombination excision circles as biomarkers of immune aging in multiple sclerosis

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Background and Aims: Kappa-deleting recombination excision circles (KRECs) and T-cell receptor excision circles (TRECs) are measures of newly generated B- and T-cells respectively. Given their potential as immune aging biomarkers, we evaluated their association with disease activity and disability in people with Multiple Sclerosis and assessed their relationship with Disease-Modifying Therapies (DMTs).

Methods: We included treatment-naïve relapsing–remitting MS patients from a 24-month prospective multicenter study

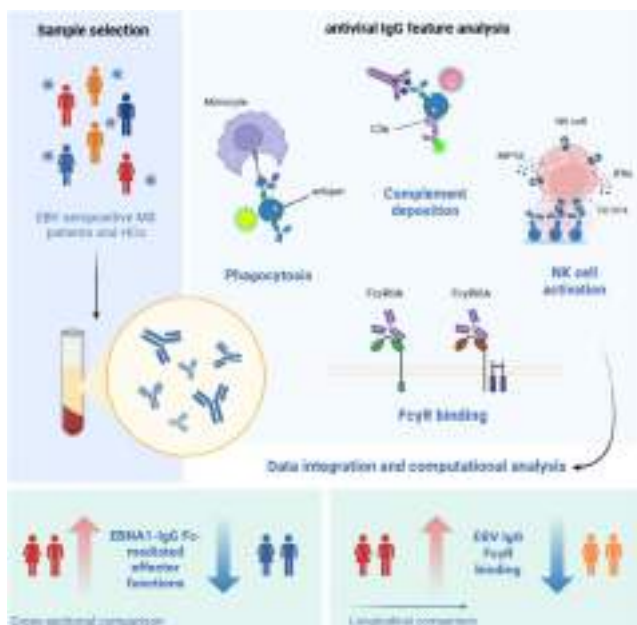


FIGURE 1 Project Overview.

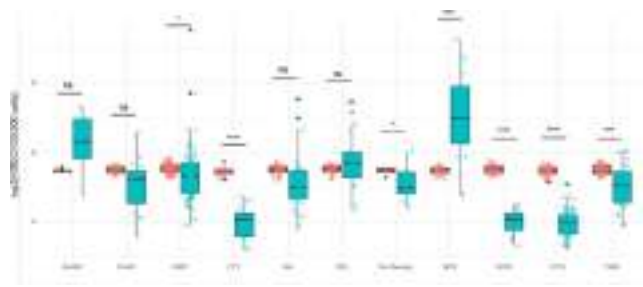


FIGURE 1 Longitudinal KRECs changes under different DMTs at baseline and after 12 months.

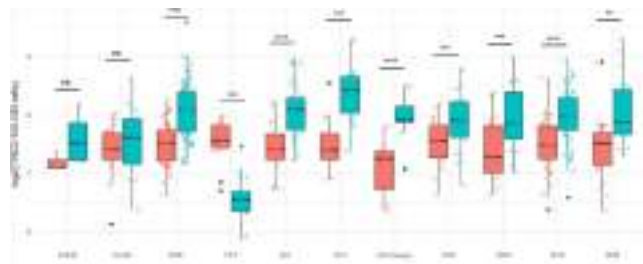


FIGURE 2 Longitudinal TRECs changes under different DMTs at baseline and after 12 months.

involving 9 European centers (MultipleMS). TRECs ($n=471$) and KRECs ($n=472$) levels were determined by qPCR at baseline and at 1-year. Associations with age, baseline Expanded Disability Status Scale (EDSS), and time to first relapse in the 24 months before and after baseline were assessed through linear models and Cox regression. Longitudinal TRECs and KRECs changes under different DMTs and in untreated patients were assessed by Wilcoxon tests on cell-count-adjusted values.

Results: Both TRECs ($p=4.65 \times 10^{-13}$) and KRECs ($p=0.004$) significantly decreased with age, with a stronger effect observed for TRECs. Neither TRECs nor KRECs correlated with baseline EDSS, although a negative trend was observed in patients with EDSS < 3.0. No associations were found with time to first relapse. DMTs had distinct effects on immune output: TRECs significantly decreased under fingolimod, remained stable with alemtuzumab and cladribine, and increased with other therapies at 1 year. KRECs declined markedly under fingolimod and anti-CD20 therapies, more mildly under dimethyl fumarate and teriflunomide, and increased under natalizumab.

Conclusion: TRECs and KRECs primarily reflect immune ageing in MS and are differentially modulated by DMTs. This study prompts future investigations for their role as markers of DMT response.

Disclosure: MS, AG, SM, PU, MK, MN, RO, MF, AG, BD, MS, DAS, NB, MT, AG, CB, DV, AG, SG, AGO, HBS, GA, TB, HFH, PV, JS, MJ and IK nothing to disclose FE received speaking honoraria from Merck and Genzyme. TG recipient of the Grant for Multiple Sclerosis Innovation (GMSI) funded by Merck, Darmstadt, Germany. FP has received research grants from Denka, Janssen, Merck KGaA, Novartis, Pfizer and UCB, and fees for serving on data monitoring committees in clinical trials with Lundbeck and Roche. FT is supported by Karolinska Institutet's doctoral education programme and has received a scholarship from the Blanceflor Foundation. FT has received

compensation from Novartis for serving as a meeting moderator. MR received speaker honoraria from Bayer, Biogen, Roche, and Teva and research support from the Canadian MS Society and Fondazione Italiana Sclerosi Multipla. MF is Editor-in-Chief of the Journal of Neurology and received compensation for consulting services and/or speaking activities from Alexion, Merck-Serono, Novartis, Roche and Teva Pharmaceutical Industries and receives research support from Biogen Idec, Merck-Serono, the Italian Ministry of Health, Fondazione Italiana Sclerosi Multipla, 770 and ARIsla (Fondazione Italiana di Ricerca per la SLA). BH has served on advisory boards for Novartis, Polpharma, and Hoffmann-La Roche, as well as on DMSC boards for AllergyCare, Polpharma, Sandoz, Biocom, and TG Therapeutics. He has received honoraria for consulting services provided to clients of the Gerson Lehrman Group and AlphaSights, expert advice for Wuesthoff&Wuesthoff and for educational activities organized by neuro.today and patients.today. His organization has received funding for research projects by Polpharma and Hoffmann-La Roche. All conflicts are not relevant to the topic of the study. He is associated with DIFUTURE (Data Integration for Future Medicine). He received funding for the study by the European Union's Horizon 2020 Research and Innovation Program and the Deutsche Forschungsgemeinschaft (DFG, German Research Foundation) under Germany's Excellence Strategy within the framework of the Munich Cluster for Systems Neurology. Chiristiane Gasperi received funding from the BMBF, DFG (German Research Foundation), the Hertie Foundation, and the Hans and Klementia Langmatz Stiftung. CG received funding from the BMBF and the DFG (German Research Foundation). SL received compensation for consulting services and speaker honoraria from Biogen Idec, Novartis, Johnson and Johnson, Sanofi, Merck, and Bristol-Myers Squibb, and holds grants from the Instituto de Salud Carlos III and AGAUR. MM has received research support from the German Research Foundation, the Bavarian State Ministry for Science and Art, the German Federal Ministry of Education and Research, and the National Institutes of Health; he has received honoraria for lecturing from Merck Serono. FMB received travel support from Roche, Novartis and Biogen.

OPR-121 | Treatment specific infection risk in multiple sclerosis patients: A Danish nationwide registry study

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Background and Aims: Disease-modifying therapies (DMTs) for multiple sclerosis (MS) vary in immunomodulatory and immunosuppressive effects. We compared infection risks across DMTs using nationwide registry data.

Methods: Patients starting DMTs (2017–2024) were identified in the Danish Multiple Sclerosis Registry. ICD-10 codes captured

hospital-treated infections, and antimicrobial (AM) prescriptions by ATC codes served as infection proxies. Risk windows covered treatment duration plus a treatment-specific period— infections within 6 months before treatment were excluded. Cox models estimated the risks of infection and AM use, accounting for repeated measures and adjusting for demographic and clinical covariates.

Results: The cohort included 11,140 patients and 24,403 treatment episodes. All DMTs, especially anti-CD20 agents (HR: 3.0, 95% CI: 2.7–3.5), were associated with higher infection risks than teriflunomide, except alemtuzumab. Most DMTs increased antiviral use, with alemtuzumab conferring the greatest risk (HR: 9.8, 95% CI: 7.1–13.3). Alemtuzumab and ocrelizumab raised antimycotic use, and alemtuzumab had the highest total AM use.

Conclusion: This nationwide registry study found that anti-CD20 DMTs conferred the highest infection risk, while alemtuzumab was associated with the greatest AM use. These findings underscore the heterogeneity of infection profiles and emphasize the need for tailored clinical management strategies across DMTs.

Disclosure: Rolf Pringler Holm has received speaker honoraria from Sanofi and Novartis, has served on an advisory board for Novartis, and has received a travel grant from The Danish Multiple Sclerosis Society. Frederik Elberling has received speaker honoraria from Sanofi and Roche. Susanne Dam Poulsen has received grants from the Novo Nordisk Foundation, the Independent Research Fund Denmark, and Svend Andersen Fonden, and reports advisory board activity for Gilead, Takeda, and GSK. Finn Sellebjerg has served on scientific advisory boards, served as a consultant, received support for congress participation, or received speaker honoraria from Biogen, Bristol Myers Squibb, H. Lundbeck A/S, Merck, Novartis, Roche, and Sanofi. His laboratory has received research support from Biogen, Merck, Novartis, Roche, and Sanofi. Additionally, FS serves as the section editor of the journal Multiple Sclerosis and Related Disorders, is the chairman of the research board of the Danish Multiple Sclerosis Society, and is a member of the scientific steering committee of the International Progressive MS Alliance. Melinda Magyari has served on scientific advisory boards, served as a consultant, received support for congress participation, or received speaker honoraria from Roche, Sanofi, Biogen, Merck, Novartis, Bristol Myers Squibb, Medscape, Alexion, and Moderna. Her research group has contracts with Biogen, Merck, Novartis, Roche, Sanofi, and Bristol Myers Squibb.

OPR-122 | The T cell repertoire of persons with multiple sclerosis is characterized by CD8+ T cells specifically targeting astrocytes

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Background and Aims: Clonally expanded CD8+ T cells are prevalent in CNS lesions of persons with MS (pwMS), suggesting antigen-driven proliferation. Beyond myelin, studies suggest that astrocytes and neurons may also be targeted. Since astrocytes and neurons upregulate HLA class I in active lesions, we sought to assess if pwMS harbor CD8+ T cells targeting these cells.

Methods: We generated human-induced pluripotent stem cell (hiPSC) derived astrocytes and neurons from 8 pwMS and 6 age-matched healthy donors (HD). We then cultured peripheral blood mononuclear cells (PBMC) with autologous hiPSC-derived astrocytes or neurons for 14 days. To identify proliferating clonotypes, CD8+ T cell TCR repertoires were compared before and after coculture. The TCRs of these clonotypes were then transfected into NFAT-luciferase Jurkat cells to validate for CNS autoreactivity. Public TCR databases from the blood/CSF of pwMS were screened for identical/similar TCR-β CDR3 sequences. Finally, scRNAseq was performed on expanded CD8+ T cells.

Results: We demonstrate that CD8+ T cell repertoires of pwMS are restricted after coculture with astrocytes only. Astrocyte-reactive clonotypes are exclusively found in pwMS (5/7 pwMS vs. 0/5 HD) with most clonotypes non-reactive against PBMC, indicating CNS specificity. Identified astrocyte-specific CD8+ T cell clonotypes share similar CDR3 sequences to blood/CSF clonotypes from pwMS from public databases. scRNAseq reveals that these clonotypes display a highly proliferative activated cytotoxic phenotype.

Conclusion: Our autologous CNS antigen presentation system demonstrates that astrocyte-specific CD8+ T cells are a frequent finding in pwMS whilst mostly absent in HD, reinforcing a potential pathogenic role for astrocyte-specific CD8+ T cells in MS.

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OPR-123 | Competing stroke etiologies and outcomes after breakthrough ischemic stroke on oral anticoagulants in patients with atrial fibrillation

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Background and Aims: Patients with atrial fibrillation (AF) experiencing ischemic stroke despite oral anticoagulation (OAC) face high risk of recurrence and bleeding. We compared 90-day outcomes between patients with and without competing stroke etiologies after breakthrough ischemic stroke on OAC for AF.

Methods: ASPERA-R retrospectively enrolled adults (>18 years) with AF experiencing breakthrough ischemic stroke on continuous OAC (≤ 48 h) across 35 centers (Feb 2020–Feb 2025). Eligibility required imaging-confirmed stroke, vessel imaging, and 90-day follow-up; inadequate dosing or poor adherence were excluded. Competing etiologies coexisting with cardioembolism were assessed. Primary outcome: 90-day recurrent stroke; secondary: mRS shift, myocardial infarction, vascular and all-cause death, and bleeding. IPW balanced baseline co-variables; Cox/regression models compared outcomes.

Results: Among 1649 patients (mean age 78, 47.8% male), 24.3% had competing etiologies, most commonly large-artery atherosclerosis (59.9%). Weighted analyses showed higher 90-day recurrent ischemic stroke in patients with competing etiologies (6.0% vs. 2.7%; aHR 2.62), particularly large-artery atherosclerosis and other determined causes. They also had worse disability (aOR 1.30), higher all-cause mortality (aHR 1.58), and increased moderate-to-severe bleeding (aHR 1.82), while 24-h

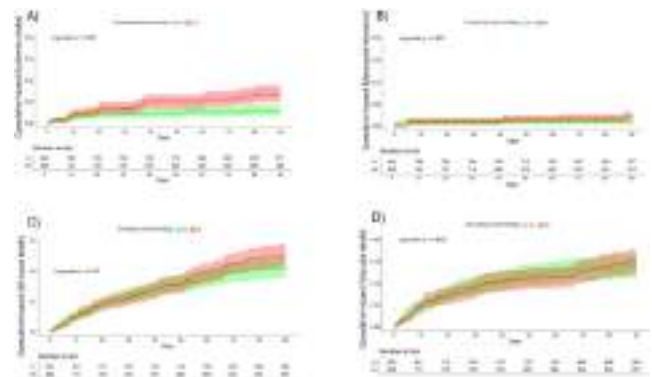


FIGURE 1

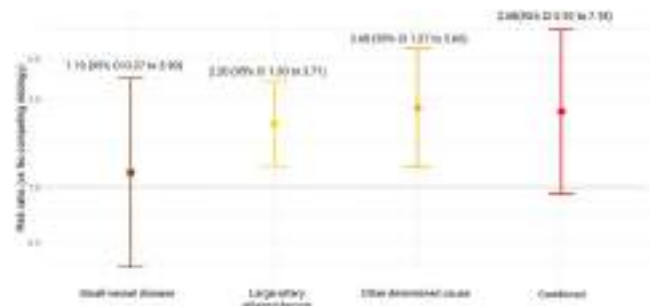


FIGURE 2

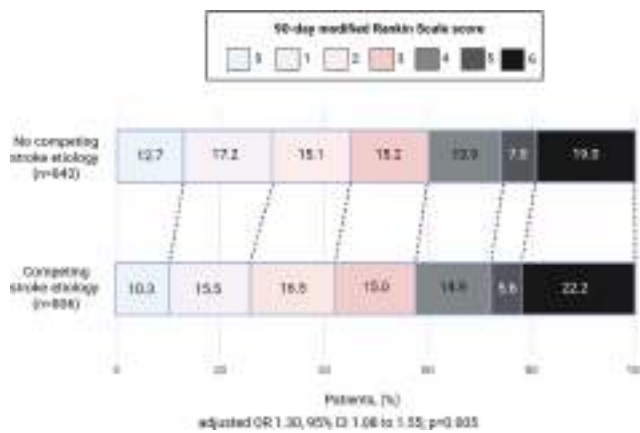


FIGURE 3

hemorrhagic transformation was less frequent (aRD -4.3%). Other outcomes did not differ

Conclusion: One in four patients with AF and breakthrough ischemic stroke on OAC had competing etiologies, most often large-artery atherosclerosis. These patients showed higher risk of recurrent stroke, functional dependence, mortality, and bleeding, highlighting the need for development of individualized secondary prevention. Main study limits include observational retrospective design and incomplete data on on-treatment anticoagulation quality.

Disclosure: Nothing to disclose.

OPR-124 | Continuation versus switching direct oral anticoagulant after breakthrough stroke: A non-inferiority target trial analysis from the ASPERA-R Study

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Background and Aims: Management of ischemic stroke occurring despite direct oral anticoagulant (DOAC) therapy for atrial fibrillation remains inconsistent due to limited evidence regarding the benefits of switching treatments. This study aims to evaluate the effectiveness and safety of continuing the current DOAC versus switching to an alternative oral anticoagulant, utilizing a predefined non-inferiority framework.

Methods: This multicenter, retrospective study involved 35 stroke centers across Europe and North Africa. We emulated a target trial employing a non-inferiority comparison of continuation versus switching to an alternative oral anticoagulant (different DOAC or VKA) after a breakthrough stroke. The primary outcome was 90-day net clinical benefit (recurrent ischemic stroke and moderate-to-severe bleeding). Secondary endpoints included all-cause mortality, vascular death, and specific recurrent ischemic or hemorrhagic events.

Results: We included 1006 consecutive adults (median age 80.4 years; 50% women). After IPTW adjustment, the 90-day net clinical benefit was similar between strategies (Figure 1): 5.1% in continuation vs. 4.9% in switching (Figure 2), with a risk difference of -0.3% (90% CI, -2.7% to 2.1%), satisfying non-inferiority. Recurrent ischemic events and bleeding outcomes were likewise comparable, with absolute differences within prespecified margins. Non-inferiority was not demonstrated for all-cause or vascular mortality.

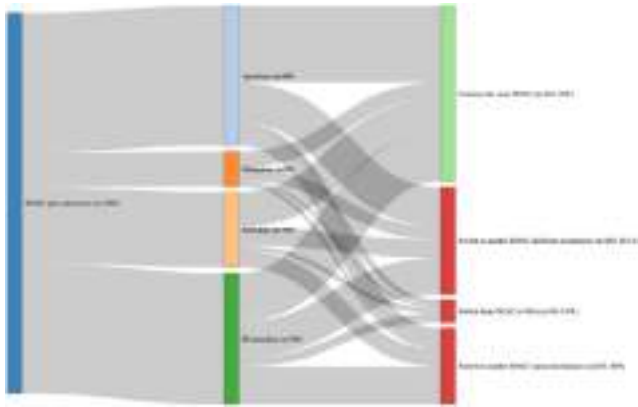


FIGURE 1 Sankey flow of pre-stroke DOAC agent and post-stroke anticoagulation strategy.

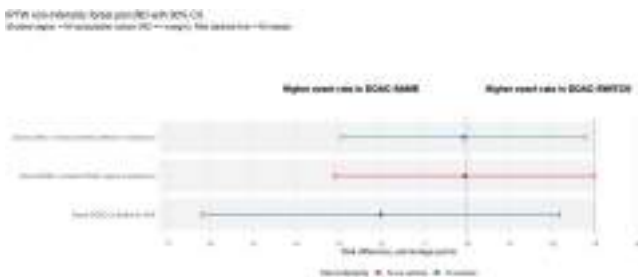


FIGURE 2 Strategy-specific comparison for net clinical benefit (IPTW, Risk Difference scale).

Conclusion: Continuing the same DOAC after a breakthrough ischemic stroke resulted in clinical outcomes comparable to switching anticoagulant therapy, with absolute differences within clinically acceptable margins for most outcomes. Our findings do not support routine switching to improve secondary prevention after a breakthrough event. Randomized controlled trials are needed to identify strategies to improve secondary prevention after a breakthrough ischemic stroke.

Disclosure: Nothing to disclose.

OPR-125 | Clinical outcome of MRI-negative acute ischemic stroke treated with and without intravenous thrombolysis

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Background and Aims: Intravenous thrombolysis (IVT) for acute ischemic stroke (AIS) is guided by clinical assessment and exclusion of radiological contraindications. While MRI protocols reduce mistreatment of stroke mimics, infra-radiologic strokes (IRS) on admission MRI remain diagnostically challenging. We aimed to characterize MRI-based IRS and evaluate their clinical and functional outcomes.

Methods: From our local prospectively-constructed stroke registry we identified all AIS undergoing acute MRI, without

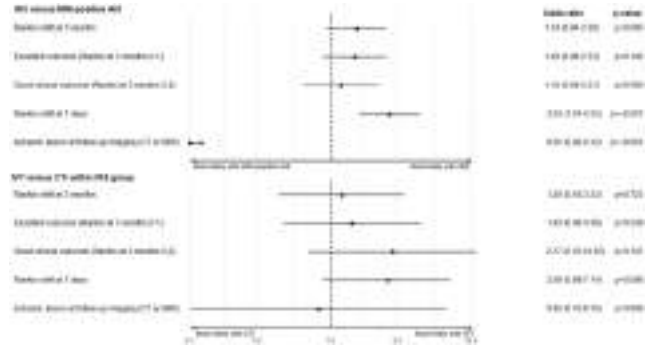


FIGURE 1 Forest plot of binary/categorical outcomes.

intracranial occlusion. IRS were defined as clinically diagnosed AIS with negative DWI and perfusion imaging. IRS were compared with radiologically confirmed (RC)-AIS. Within IRS-patients, IVT-treated patients were compared with those receiving conservative treatment. Outcomes included 3-month modified-Rankin-Scale (mRS) shift, 24-h-NIHSS-shift, lesion evidence on follow-up imaging (LFI). Analyses were adjusted using propensity score weighting.

Results: Among 1315 consecutive eligible AIS between 2018 and 2024, 58 (4.4%) had an IRS. Compared with RC-AIS in univariate analysis, IRS patients had similar baseline NIHSS, shorter onset-to-door times (96 vs. 246 min, $p < 0.001$), less paresis (44.8% vs. 63.4%, $p = 0.006$), more infra-tentorial localization (48.3% vs. 22.5%, $p < 0.001$), received more IVT (63.8% vs. 32.1%, $p < 0.001$), and had less LFI (17.8% vs. 73.3%, $p < 0.001$). After adjustment, IRS patients had greater 24-h-NIHSS improvement (beta = 1.68, 95% CI = 0.29–3.07), less LFI (psWOR = 0.05, 95% CI = 0.02–0.12), and similar 3-month mRS. Within the IRS group, IVT was associated with greater 24-h-NIHSS improvement (beta = 2.97, 95% CI = 1.17–4.78) without significant 3-month mRS changes.

Conclusion: IRS represent an uncommon AIS radiotype and overall similar medium-term outcomes as MRI-positive AIS. In IRS, IVT was associated with improved short but not medium-term outcomes.

Disclosure: Nothing to disclose.

OPR-126 | Exploring the contribution of cerebrovascular risk factors to glymphatic system impairment in multiple sclerosis

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Background and Aims: Cerebrovascular risk factors (cVRFs) associate with greater brain damage and disability in multiple sclerosis (MS) patients. The glymphatic system, contributing to brain waste clearance, is impaired in both MS and cerebrovascular diseases. We investigated whether choroid plexus volume (CPV) and the diffusion tensor imaging analysis along the perivascular spaces (DTI-ALPS) are influenced by cVRFs in MS.

Methods: Two-hundred nine MS patients and 123 age- and sex-matched healthy controls (HC) underwent cVRF, neurological evaluation, and brain 3T MRI acquisition. CP volume was quantified using a deep learning-based approach on 3D T1-weighted MRI scans; DTI-ALPS was used to assess glymphatic system function.

Results: At least one cVRF was present in 64% of MS patients and 41% of HC. MS patients with and without cVRFs showed no differences for disease duration, disability, clinical phenotype and treatment ($p \geq 0.738$). Compared to HC, MS patients had significantly higher T2-hyperintense white matter (WM) lesion volume, lower global and regional brain volumes, and larger CPV (FDR- $p \leq 0.003$), regardless of cVRFs (FDR- $p \geq 0.553$). Both MS groups with and without cVRFs exhibited significantly lower DTI-ALPS index compared to their respective HC counterparts (FDR- $p \leq 0.002$), with the coexistence of MS and cVRFs resulting in a significantly greater DTI-ALPS index reduction (FDR- $p = 0.013$). Lower DTI-ALPS and higher CPV correlated with higher T2-hyperintense WM lesion volume and lower brain volumes (FDR- $p \leq 0.007$), independently from cVRF presence (FDR- $p \geq 0.122$).

Conclusion: cVRFs may worsen glymphatic system dysfunction in MS, possibly contributing to aggravate neuroinflammation and neurodegeneration and supporting the importance of proactive cVRF management.

Disclosure: M. Rubin has nothing to disclose. P. Preziosa received speaker honoraria from Roche, Biogen, Novartis, Merck, Bristol Myers Squibb, Genzyme, Horizon and Sanofi. E. Pagani and A. Meani have nothing to disclose. M. Margoni reports grants and personal fees from Sanofi Genzyme, Merck Serono, Roche, Biogen, Amgen and Novartis. L. Storelli and M. Albergoni have nothing to disclose. M.A. Rocca received consulting fees from Biogen, Bristol Myers Squibb, Roche; and speaker honoraria from Alexion, Biogen, Bristol Myers Squibb, Celgene, Horizon Therapeutics Italy, Merck Serono SpA, Mitsubishi-Tanabe Pharma, Neuraxpharm, Novartis, Roche, Sandoz, and Sanofi. She receives research support from the MS Society of Canada, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. She is Associate Editor for Multiple Sclerosis and Related Disorders; and Associate Co-Editor for Europe and Africa for Multiple Sclerosis Journal. M. Filippi is Editor-in-Chief of the Journal of Neurology, Associate Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmed Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers

Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla.

OPR-127 | Endovascular therapy improves clinical outcomes in acute ischemic stroke due to isolated M2 occlusion: Real-world experience from the RES-Q registry

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Background and Aims: Indication for endovascular therapy (EVT) in acute ischemic stroke (AIS) caused by middle cerebral artery M2 segment (MCA/M2) occlusion remains controversial. The aim was to compare clinical outcomes in these patients treated with intravenous thrombolysis (IVT), EVT, and IVT + EVT.

Methods: In an observational multicenter cohort study, we analyzed data from AIS with causative MCA/M2 occlusion treated with IVT, EVT, and IVT+EVT, prospectively collected in the RES-Q registry in the Czech Republic (2022–2024). Clinical outcomes were assessed using utility-weighted 3-month modified Rankin Scale (mRS) and 3-month mortality. Baseline differences were adjusted using multivariable ordinal regression for utility-weighted 3-month mRS and multivariable logistic regression for 3-month mortality, including age, sex, National Institutes of Health Stroke Scale, pre-stroke mRS, and onset-to-treatment time. Missing 3-month outcomes were handled using multiple imputation.

Results: The IVT group consisted of 520 (248 males; median age 74 [65–82] years), the EVT group of 223 (117 males; median age 73 [61–81] years), and the IVT+EVT group of 321 patients (163 males; median age 73 [64–79] years). Adjusted utility-weighted 3-month mRS was significantly better in the IVT+EVT versus IVT group (OR 1.26, 95% CI 1.05–1.51), and in the IVT+EVT versus EVT group (OR 1.31, 95% CI 1.07–1.59); there was no difference between the EVT and IVT groups. There were no differences in 3-month mortality between the groups.

Conclusion: The addition of EVT to IVT was associated with better 3-month clinical outcomes compared with IVT alone in patients with AIS due to MCA/M2 occlusion.

Disclosure: Roman Herzig was supported by STROCZCH within the CZECRIN Large Research Infrastructure (No. LM2023049) funded by the state budget of the Czech Republic, Ministry of Health of the Czech Republic (Grant No. DRO-UHHK 00179906), and Charles University, Czech Republic (Cooperatio Program, research area NEUR). Szabolcs Szabó was supported by Charles University, Czech Republic (Cooperatio Program, research area NEUR). Geraldo Neto and Robert Mikulík were supported by STROCZCH within the CZECRIN Large Research Infrastructure (No. LM2023049) funded by the state budget of the Czech Republic.

OPR-128 | The EJS ACT-PD multi-arm, multi-stage platform trial: Assessing potential disease-modifying therapies for Parkinson's disease

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Background and Aims: The Edmond J. Safra Accelerating Clinical Trials in Parkinson's disease (EJS ACT-PD) trial uses a multi-arm, multi-stage (MAMS) design to accelerate the evaluation of potential disease-modifying therapies (DMTs) for Parkinson's disease (PD). MAMS designs enable simultaneous testing of multiple therapies; early identification of ineffective therapies for discontinuation; and efficient incorporation of new candidates. The primary objective is to detect a 30% reduction in the rate of increase in Movement Disorder Society-Unified PD Rating Scale (MDS-UPDRS) parts I and II combined within a representative UK population of people with PD.

Methods: Initially 1600 participants will be recruited across 40 UK sites using a self-referral online registration of interest process. Participants will be randomised to telmisartan, terazosin, or placebo, with a third treatment arm (ursodeoxycholic acid) opening in 2026. A hybrid delivery model enables 6-monthly visits to be conducted either in person or remotely. Trial medication will be delivered to participants homes to be taken for up to 3 years. Early-stage analyses will use the MDS-UPDRS parts I, II and remote III to inform discontinuation of ineffective treatments. Sub-studies include passive digital monitoring of progression and optional collection of blood and cerebrospinal fluid for biomarker research.

Results: Recruitment opened in September 2025, with site activation continuing until March 2026. Over 1300 individuals have already registered interest.

Conclusion: EJS ACT-PD is set to become the world's largest Parkinson's disease trial, providing an efficient platform to evaluate multiple DMTs while generating an extensive dataset to advance understanding of disease progression.

Disclosure: Nothing to disclose.

OPR-129 | PD vs. PSP-P: superior diagnostic biomarker performance for combining Tau-PET and MRI

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Background and Aims: Differentiating the parkinsonian variant of progressive supranuclear palsy (PSP-P) from Parkinson's disease (PD) remains clinically challenging due to substantial phenotypic overlap. Reliable imaging biomarkers are therefore essential to improve early diagnostic accuracy. This study evaluated whether structural MRI (midbrain-to-pons ratio) or Tau-PET ([¹⁸F]PI-2620) provides superior discriminatory power, and whether a multimodal approach enhances diagnostic performance.

Methods: Fourteen patients with PD and fifteen with PSP-P were diagnosed according to recent consensus criteria. All subjects underwent Tau-PET imaging (20–40 min p.i.). Midbrain-to-pons (M/P) ratios were derived from high-resolution T1-weighted MRI. ROC analyses were performed for M/P ratio, globus pallidus (GP) SUVRs, and a combined logistic-regression model including both biomarkers. AUCs were compared using DeLong tests. Binary logistic regression models assessed explained variance (Nagelkerke R²) and improvements in model fit via -2LL chi-square comparisons.

Results: AUCs were 0.79 for M/P ratio, 0.83 for GP SUVRs, and 0.91 for the combined model. DeLong testing showed no significant difference between M/P ratio and GP SUVRs ($p=0.73$), but the combined model outperformed M/P ratio alone ($p<0.05$). Logistic regression revealed explained variance of 38% for M/P

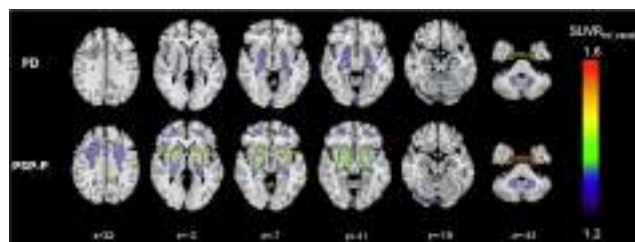


FIGURE 1 Mean [¹⁸F]PI-2620 SUVR images of the PD and PSP-P group.

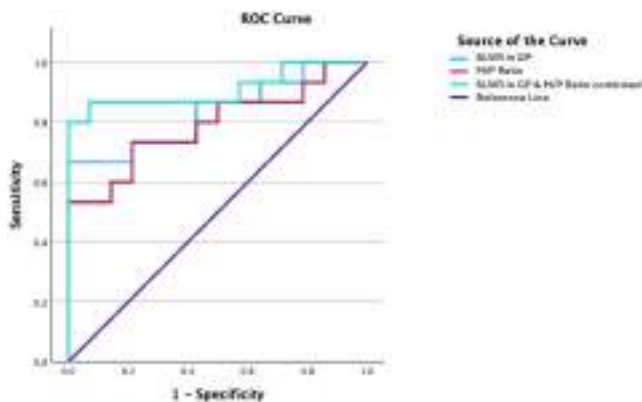


FIGURE 2 ROC curves for M/P ratio, globus pallidus (GP) SUVRs, and a combined logistic-regression model including both biomarkers.

ratio, 50% for GP SUVRs, and 72% for the combined model. Adding PET to MRI and adding MRI to PET improved model fit significantly ($p < 0.005$) in both directions.

Conclusion: Both MRI and Tau-PET serve as informative biomarkers for differentiating PD from PSP-P, with PET showing higher individual discriminatory power. However, combining structural MRI and Tau-PET provides the strongest diagnostic accuracy, supporting multimodal biomarker strategy for early and reliable differentiation of PD and PSP.

Disclosure: #Contributed equally GNB is a full time employee at Life Molecular Imaging, a Lantheus Company. N.F. has received speaker or consulting honoraria from Life Molecular Imaging, GE Healthcare, MSD, Eisai and Biogen. C.P. is inventor in a patent “Oral Phenylbutyrate for Treatment of Human 4-Repeat Tauopathies” (PCT/EP2024/053388) filed by LMU Munich. M.B. is a member of the Neuroimaging Committee of the EANM. M.B. has received speaker honoraria from Roche, GE Healthcare, Iba, Miltenyi, and Life Molecular Imaging; has advised Life Molecular Imaging, MIAC, Cenos, and GE healthcare; and is currently on the advisory or imaging review boards of AC Immune and ZRO Imaging. H.T. was supported by the Cologne Clinician Scientist Program (CCSP)/Faculty of Medicine/University of Cologne, funded by the DFG—Project ID 413543196.

OPR-130 | Digital biomarkers in normal pressure hydrocephalus: Instrumented timed up and go for assessing and predicting shunt response

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Background and Aims: Normal pressure hydrocephalus (NPH) is characterized by early manifestations of gait apraxia [1]. Wearable sensors have emerged as a promising approach to quantitatively capture motor impairments, with sensor-derived motor parameters demonstrating moderate to strong correlations with clinical measures [3]. This study aims to evaluate sensor-derived

motor parameters by assessing both their responsiveness to shunt surgery and their ability to predict shunt response.

Methods: A total of 254 NPH individuals from the Bologna PRO-Hydro cohort performed instrumented Timed Up and Go using a sensor worn on the lower back, at baseline (preTT), 3 days after (postTT) the cerebrospinal fluid tap test (CSF-TT), and 6 months following shunt surgery (postShunt) [4]. Responsiveness to shunt surgery was assessed by examining changes between preTT and postShunt. Two predictive models were developed using preTT values alone or combined with postTT–preTT changes. The prognostic value of CSF-TT for surgery outcomes was assessed via likelihood ratio testing.

Results: Most instrumental variables showed statistically significant improvements after surgery, including reduced duration ($p < 0.001$), more controlled sit-to-walk transitions (lower jerk, $p < 0.001$), and faster turns (higher velocity, $p < 0.01$). Most parameters showed a significant improvement in the predictive model when $\Delta(\text{postTT} - \text{preTT})$ was included as a predictor. Motor strategies during sit-to-walk were found to be key determinants in post-shunt prediction (adjusted $R^2 > 0.65$).

Conclusion: Sensor-derived parameters capture clinically meaningful motor changes provide significant predictive value for surgery outcomes. These findings support the use of instrumented motor tests as objective measures to complement traditional clinical assessments and guide candidate selection for shunt surgery.

Disclosure: Nothing to disclose.

OPR-131 | Tourette syndrome symptomatology associated with hyper-glutamatergic activity in the anterior cingulate cortex

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Background and Aims: In Tourette syndrome (TS), tics would result from a dysfunction in the cortico-basal ganglia-thalamo-cortical loops (Atkinson-Clement et al. 2020), related to an hyper-dopaminergic activity. Nonetheless, treatment by dopamine antagonists is not sufficient for every patients, and topiramate, a glutamatergic antagonist, is sometimes more efficient to reduce tics (Cs et al. 2013). Therefore, our objective was to study the glutamatergic system in vivo in TS patients by evaluating NMDA receptors (NMDAR) activity using PET (Positron emission tomography) imaging.

Methods: TS patients ($n = 12$) and matched healthy volunteers ($n = 12$) were included in the GlutaTour study (NCT03681795). PET was done using the [18F]-FNM (Fluoroethylnormemantine) radiotracer to study activated NMDAR. Tics severity was measured using the YGTSS (Yale Global Tic Severity Scale). Normalized PET activity was compared between TS patients

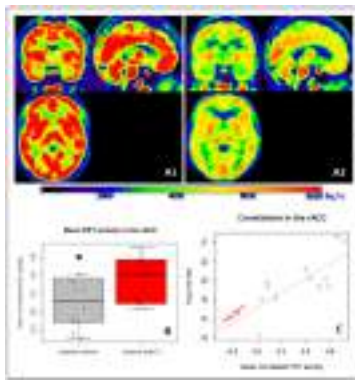


Figure 1. Comparisons of PET activity between patients with Tourette syndrome (TS) and healthy volunteers. **A.** Comparisons of PET activity between two paired subjects (A1, a patient with TS, and A2, a healthy control). A color scale bar indicates the intensity of ¹⁸F-NMBA activity in PET imaging (A1/A2). **B.** Comparisons of PET activity between groups. A color scale bar indicates the intensity of ¹⁸F-NMBA activity in PET imaging (B1/B2). **C.** Correlations between PET activity in the rACC and total PET activity in patients with TS (linear regression in dotted red lines; $r = \text{Pearson coefficient of correlation}$; and $**p < 0.01$).

FIGURE 1 Comparisons of PET activity between patients with Tourette syndrome (TS) and healthy volunteers.

and healthy volunteers (t -tests), and correlations were done between YGTSS scores and normalized PET activity in patients.

Results: Activated NMDAR were significantly higher in TS patients compared to healthy volunteers, mainly in the right anterior cingulate cortex (rACC) (p -value = 0.03), and their activity in the rACC correlated significantly and positively with the YGTSS scores (p -value = 0.005 and coefficient = 0.75).

Conclusion: This study is the first to have evaluated in vivo part of the glutamatergic system in TS patients, showing more activity within the NMDAR compared to controls. This supposed “hyper-glutamatergic activity” in the ACC of TS patients could highlight a disinhibition of direct striatal pathway in TS which would result in tics emergence.

Disclosure: I declare no conflict of interest for this study. This study was funded by the AFSGT (Association Française du Syndrome de Gilles de La Tourette/French Tourette Syndrome Association) and HoPeS (Institut des Handicaps Neurologiques, Psychiatriques et Sensoriels/Neurological, Psychiatric and Sensorial Disabilities Institute).

OPR-132 | Remote deep brain stimulation programming for Parkinson's disease: A prospective, randomized, controlled, multicentre, non-inferiority trial

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Background and Aims: Deep brain stimulation (DBS) is an effective therapy for advanced Parkinson's disease (PD), but outcomes depend on expert programming, usually requiring in-person visits to specialised centres. Remote DBS programming could improve access, yet controlled prospective evidence is limited.

Methods: We conducted a prospective, multicentre, randomised, open-label, non-inferiority trial at DBS centres in Germany. Patients with idiopathic PD requiring DBS programming were assigned (1:1) to standard in-person programming (standard of care [SoC]) or remote programming via a secure telemedicine platform. The primary endpoint was the MDS-UPDRS Part III score (medication-on/stimulation-on) at 3 months, with a non-inferiority margin of 5 points. Secondary outcomes included motor and non-motor symptoms, quality of life, cognition, levodopa equivalent daily dose (LEDD), and safety. Analyses were per protocol (ClinicalTrials.gov NCT05193825).

Results: Between March 2022 and December 2024, 52 patients were randomised; 48 were analysed (SoC $n = 26$; remote $n = 22$). Median MDS-UPDRS Part III improvement at 3 months was -23.0 points in the SoC group and -20.5 in the remote group. The median between-group difference was -2.5 (95% CI -9.0 to 2.0), meeting non-inferiority criteria. Improvements in total MDS-UPDRS, Hoehn and Yahr stage, and PDQ-39 were similar. MoCA scores improved more with remote programming, while LEDD reduction was greater with SoC. Adverse events and serious adverse events were comparable between groups.

Conclusion: Remote DBS programming was non-inferior to in-person programming for motor outcomes at 3 months, with similar safety and patient-reported benefits, supporting its integration into routine DBS care.

Disclosure: Abbott Medical Inc.

OPR-133 | Does DaTScan have a prognostic value for motor decline in Parkinson's disease patients? A multimodal deep learning study

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Background and Aims: Parkinson's disease (PD) trajectories vary widely among individuals, making it difficult to predict disease progression and to identify patients at risk of rapid decline. Among routinely collected data from PD patients there is DaTScan, whose prognostic value for motor complications remains debated. This study implements state-of-the-art Deep Learning (DL) architectures to investigate the prognostic value of DaTScan volumes and electronic health records (EHRs) data, separately and in multimodal combination, in predicting future motor decline.

Methods: A longitudinal cohort of 513 patients at different PD stages from the Parkinson's Progression Markers Initiative dataset with EHRs data, DaTScan volumes at baseline and UPDRS Part III evaluation at 3-year follow-up was used to develop a dual-branch DL architecture. The first branch employs a tabular foundation model for EHRs data, while the second extracts compressed embeddings from DaTScan volumes using a 3D Convolutional Neural Network. The embeddings are then

TABLE 1 Mean Performance Metrics. Averaged scores across the three folds for the uni- and multimodal architectures.

Mean Metric	EHRs-Only	DaTscan-Only	Multimodal
MAE	6.51 ± 0.03	8.82 ± 0.03	6.49 ± 0.09
R ²	0.414 ± 0.003	0.049 ± 0.019	0.423 ± 0.013

merged for regression of the UPDRS Part III evaluation at 3-year follow-up. This modular design facilitated an ablation study to quantify the contribution of each modality.

Results: Model performances were evaluated using Mean Absolute Error (MAE) and R2 on a three-fold cross validation scheme (Table 1). The unimodal EHRs-based model achieved a MAE of 6.51 ± 0.03 (mean ± SD), comparable to the complex multimodal architecture.

Conclusion: Despite its diagnostic relevance, DaTscan provides limited prognostic benefits beyond standard clinical features. The EHRs-only model offers comparable performance while reducing model complexity and computational demands. Future prognostic modeling should prioritize richer clinical characterization over striatal imaging features.

Disclosure: Nothing to disclose.

Epilepsy 2

OPR-134 | Dysregulated CX3CR1 signaling in mesial temporal lobe epilepsy: Evidence from human brain tissue and monocyte-derived microglia

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Background and Aims: CX3CR1, the fractalkine (CX3CL1) receptor, is expressed by CNS-resident microglia and mediates microglia–neuron communication. Chronic microglial activation is a hallmark of mesial temporal lobe epilepsy with hippocampal sclerosis (MTLE-HS) and contributes to neuroinflammation and neuronal hyperexcitability. Seizure-induced release of CX3CL1 may activate microglial CX3CR1, amplifying inflammatory signaling. We studied this pathway by analyzing

CX3CR1 expression in human epileptic brain tissue and in an in vitro model of monocyte-derived microglia-like cells (MMG). **Methods:** MMG cells were differentiated from peripheral blood monocytes of 15 MTLE-HS patients and 5 healthy donors. CX3CR1 mRNA levels were assessed under basal conditions and following lipopolysaccharide (LPS) stimulation. The findings were validated in hippocampal and temporal neocortex samples from 19 MTLE-HS (8 males, 43.5 ± 10.0 years) patients and 10 controls (8 males, 67.0 ± 10.0 years).

Results: MMG cells derived from MTLE-HS patients showed higher basal CX3CR1 expression compared to controls, with further upregulation after inflammatory stimulation. In parallel, CX3CR1 transcripts were significantly increased in both the hippocampus and the temporal neocortex of MTLE-HS patients (1.96-fold, $p=0.004$; 2.23-fold, $p<0.01$, respectively).

Conclusion: Together, these concordant in vitro and in vivo findings suggest a primed, maladaptive microglial activation state in MTLE-HS. Although CX3CR1 is typically associated with microglial homeostasis, its sustained upregulation in chronic epilepsy may contribute to persistent neuroinflammation. These results highlight the CX3CL1/CX3CR1 axis as a potential therapeutic target to modulate microglia–neuron interactions and reduce seizure susceptibility in drug-resistant epilepsy.

Disclosure: UMIB is funded by FCT Portugal (UID/215/2025; UID/PRR/00215/2025). Work funded by a FCT grant 2022. 10372.PTDC. No other disclosures.

OPR-135 | Pharmacokinetics of cenobamate in focal epilepsy: Influence of sex and concomitant antiseizure medications

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Background and Aims: Cenobamate (CNB) is a recently approved antiseizure medication (ASM) for drug-resistant focal epilepsy. Despite its proven efficacy, real-world pharmacokinetic data, particularly regarding sex-based variations and interactions with concomitant ASMs, remain limited. This study aimed to investigate the pharmacokinetic profile of CNB in clinical practice and evaluate its relationship with demographic and clinical variables.

Methods: We conducted an exploratory study on 17 adult responder patients receiving add-on CNB. Plasma concentrations were measured at each dose titration step using a validated method. Pharmacokinetic parameters, including concentration-to-dose ratio, incremental slope (delta C/delta D), and area under the dose-concentration curve (AUC), were calculated. Patients were stratified into three exposure clusters (low, medium, and high) based on AUC distribution.

Results: A significant association was observed between sex and AUC exposure clusters ($p=0.026$), with females predominating in the high-exposure group. The analysis revealed a non-linear dose-concentration relationship, characterized by steeper

incremental slopes at lower doses (12.5–50 mg) and increased variability at higher dosages (100–200 mg). Regarding drug interactions, higher CNB concentrations were observed in patients co-treated with lacosamide ($p=0.0335$), while concomitant topiramate was associated with lower exposure ($p=0.0044$).

Conclusion: The pharmacokinetics of cenobamate appear highly variable and are influenced by sex and concomitant ASMs. These findings highlight the importance of therapeutic drug monitoring and individualized titration strategies to optimize efficacy and safety in real-world clinical practice.

Disclosure: Nothing to disclose.

OPR-136 | The effect of subacute neocortical stimulation during intracranial EEG on seizure frequency

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Background and Aims: Neurostimulation is an alternative therapy for patients with drug-resistant epilepsy (DRE) who are not candidates for resective surgery. This study evaluates the impact of Subacute Neocortical Stimulation (SNCS) during intracranial presurgical assessment on seizure frequency.

Methods: This study was approved by the HRA ethical committee (IRAS 187072). Patients undergoing intracranial EEG with subdural or depth electrodes for epilepsy surgery evaluation were recruited. SNCS was initiated after sufficient seizures were captured for clinical purposes and reinstatement of all antiseizure medications. Following a 24-h baseline, SNCS was applied for approximately 48 h using different stimulation parameters (varying electrode pairs and cortical locations), separated by periods without stimulation. Each stimulation period involved one electrode pair. Stimulation targeted electrode pairs located within MRI-visible lesions, the seizure onset zone and/or areas with high interictal epileptiform discharge rates, or delayed responses to single-pulse electrical stimulation (450 μ s pulse width, up to 2 V or 2.5 mA, 130 Hz). Seizure rate ratio per hour (SRR/h) was calculated as the seizure rate during SNCS divided by the baseline rate.

Results: Twenty-eight patients were included (11 females; mean age 25 ± 44). Median SRR/h during baseline was $0.296 (\pm 3.57)$ and decreased to $0.0947 (\pm 4.27)$ during SNCS ($p=0.0179$). Twenty-three patients showed a reduction in SRR/h (median reduction 77%), with 17 demonstrating reductions greater than 50%.

Conclusion: SNCS is safe and significantly reduces seizure frequency. It may represent a therapeutic alternative for patients unsuitable for resective surgery and could serve as an additional marker of epileptogenic tissue, warranting further investigation.

Disclosure: This study was funded by Epilepsy Research UK and by a jointly grant from both Action Medical Research and Great Ormond Street Hospital Children's Charity.

OPR-137 | Integrative approach to transient loss of consciousness: Guided protocol of differential diagnosis

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Background and Aims: Transient loss of consciousness (TLOC) represents a spontaneous loss of consciousness with complete resolution being a common and serious presentation in the emergency department (ED). Despite the existence of evidence-based recommendations, the diagnostic process is frequently fragmented, discipline-specific and sequential. This leads to a significant proportion of cases remaining either unexplained or misclassified. The objective of this study is to develop an integrated neuro-cardiology protocol to reduce "unexplained" TLOC after initial ED assessment.

Methods: We conducted a structured review of the literature (PubMed/Embase/Cochrane) from 2000 to 2025 to identify existing diagnostic frameworks approaches for TLOC and their limitations in practice. In parallel, we performed a prospective, single-centre pilot study including adult patients, presenting with unexplained TLOC after an initial standard assessment. We applied a standardised diagnostic pathway involving the targeted use of neuroimaging, prolonged simultaneous ECG, arterial tension and EEG monitoring, echocardiography and tilt-table testing. Final diagnoses were adjudicated using ESC syncope guidelines and ILAE seizure definitions, with clinical follow-up.

Results: No fully operationalised, integrated diagnostic protocol for TLOC applicable to the neurological and cardiological settings was identified in the literature revising 1470 publications. Implementation of the structured multidisciplinary pathway in our cohort substantially reduced the proportion of unexplained TLOC cases and clarified the diagnostic yield of individual investigations when applied within a coordinated framework.

Conclusion: The evaluation of TLOC should evolve from isolated neurological or cardiological reasoning towards structured, synchronised diagnostic pathways. Translating the existing guidance in the form of a standardised protocol improves diagnostic accuracy, reduces uncertainty and enhances patient safety.

Disclosure: The authors declare no conflicts of interest.

OPR-138 | Mortality and SUDEP in developmental and epileptic encephalopathies: A bayesian meta-analysis of rcts and open-label extensions

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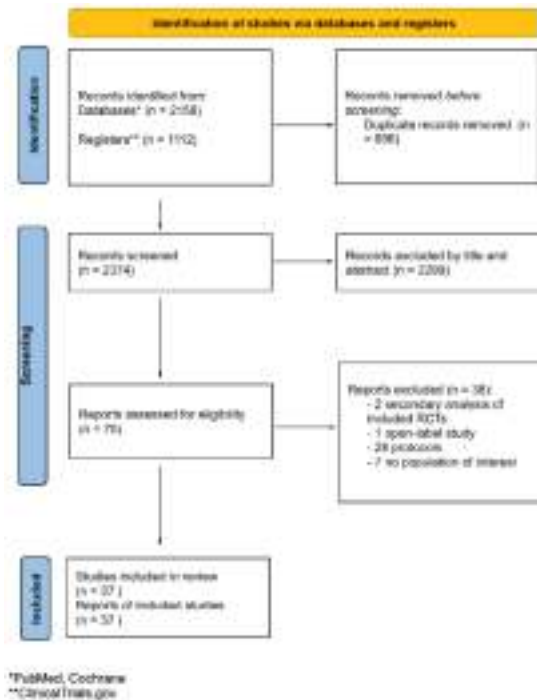


FIGURE 1 PRISMA 2020 flow diagram for new systematic reviews which included searches of databases and registers only.

Background and Aims: Mortality and Sudden Unexpected Death in Epilepsy (SUDEP) are major concerns in Developmental and Epileptic Encephalopathies (DEEs), particularly Lennox-Gastaut (LGS) and Dravet syndromes (DS). Accurate incidence estimates from prospectively monitored populations are limited. We aimed to estimate long-term rates of all-cause mortality and SUDEP by pooling data from RCTs and their corresponding long-term open-label extensions (OLEs) using a Bayesian framework.

Methods: We systematically reviewed pharmacological trials in DEEs and performed a Bayesian random-effects Poisson meta-analysis with informative priors derived from published literature. Sensitivity analyses were conducted using weakly informative priors and frequentist generalized linear mixed models. Results are expressed as incidence per 1000 person-years (PY) with 95% Credible Intervals (CrI).

Results: The analysis included 37 RCTs (3757 patients) and 16 OLE studies (2210 patients). In the overall population, all-cause mortality was 8.17 per 1000 PY (95% CrI 4.77–11.84), and SUDEP was 3.96 (2.24–6.12). These estimates were consistent across sensitivity analyses. In LGS, all-cause mortality was 7.77 (3.58–13.31) and SUDEP 3.73 (1.73–6.49) per 1000 PY. In DS, all-cause mortality was comparable (8.97; 3.84–16.99), whereas SUDEP incidence appeared higher at 7.29 (2.88–14.42).

Conclusion: This study provides robust long-term estimates of mortality and SUDEP in DEEs by leveraging pooled trial data. The consistency between Bayesian and frequentist approaches validates these findings, highlighting a substantial mortality burden, with SUDEP particularly elevated in DS. These results emphasize the need for proactive risk management and counseling in this vulnerable population.

Disclosure: Nothing to disclose.

OPR-139 | Cognitive impairment and cardiovascular autonomic failure as independent and combined predictors of progression and phenoconversion in iRBD

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Background and Aims: REM sleep behaviour disorder (RBD) is a parasomnia caused by loss of physiological muscle atonia during REM, resulting in dream-enactment behaviours. Isolated RBD (iRBD) is recognized as a prodromal manifestation of α -synucleinopathies, including Parkinson's disease (PD), dementia with Lewy bodies (DLB), and multiple system atrophy (MSA). Although cardiovascular autonomic failure (cAF) and cognitive impairment (CI) are established prodromal biomarkers, their combined prognostic significance remains unclear. This study investigated the interaction between cAF and CI in iRBD at baseline and longitudinally, evaluating their individual and combined phenoconversion predictive value.

Methods: Seventy-four iRBD (73% male; age 68.61 ± 7.23 years) were prospectively enrolled and followed-up for a mean of 3.17 ± 1.88 years. Cardiovascular autonomic function was assessed using a standardized reflex testing battery alongside a comprehensive neuropsychological battery. Cross-sectional and longitudinal cognitive and autonomic changes were analyzed, conversion risk was assessed using survival analyses.

Results: At baseline, 17 patients (25.4%) had mild CI (MCI) and 12 (17.9%) had neurogenic orthostatic hypotension (nOH), with no significant association between them ($p=0.484$). Longitudinally, multidomain MCI increased significantly ($p=0.038$), driven by visuospatial ($p=0.001$) and memory decline ($p=0.050$), and nOH prevalence also rose ($p=0.026$). Fifteen patients (20.3%) converted to an α -synucleinopathy (6 DLB, 5 PD, 2 MSA, 2 parkinsonism). Both MCI (HR=3.71; 95% CI: 1.22–11.22; $p=0.020$) and nOH (HR=3.64; 95% CI: 1.20–10.98; $p=0.022$) independently predicted conversion, while their coexistence markedly increased risk (HR=12.12; 95% CI: 2.76–53.29; $p=0.001$).

Conclusion: In iRBD, CI and cAF evolve largely independently, but their coexistence strongly increases the risk of phenoconversion, supporting multidimensional biomarker-based risk stratification.

Disclosure: Nothing to disclose.

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Background and Aims: Are gestures in dreams produced symmetrically or as an inverted mirror image of real-life gestures? Evidence from actigraphy and analyses of nocturnal movements in sexual parasomnia during NREM sleep suggests that motor activity during sleep may shift toward the non-dominant hand. We aimed to investigate whether a similar state-dependent inversion of motor lateralisation occurs in REM sleep behaviour disorder (RBD), a condition in which dream-enacting movements can be directly observed.

Methods: Consecutive patients with RBD underwent a structured interview to assess handedness, and their video-polysomnography recordings were visually analysed. Upper limb movements during wakefulness and REM sleep were annotated according to laterality (right, left, bilateral), movement

type (simple, complex, jerk-like), and duration. Linear mixed-effects models were used to examine the interaction between behavioural state (wakefulness vs. REM sleep) and motor lateralisation.

Results: Seventy-one patients were included: 33 with isolated RBD and 38 with RBD associated with parkinsonism (13 Parkinson's disease, 25 multiple system atrophy). Most participants were right-handed (65/71, 91.5%). A total of 22,066 movements were analysed. During wakefulness, right-handed individuals showed longer right-arm movements, consistent with normal motor dominance. In contrast, during REM sleep this pattern was reversed: right-sided movements became shorter and less frequent, while left-sided movements predominated, particularly for complex gestures. Jerks were largely symmetrical. Among right-handed patients, 72% exhibited leftward motor predominance during REM sleep. Importantly, the side affected by parkinsonism did not abolish this inversion.

Conclusion: Dream-enacting movements in RBD are preferentially expressed through the non-dominant hand, suggesting a transient reversal of motor lateralisation during REM sleep.

Disclosure: Nothing to disclose.

OPR-141 | Mass spectrometry-based quantification of hypocretin-1 and its fragment_1-16 following immunoprecipitation and solid-phase extraction

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Background and Aims: Loss of hypocretin-1/orexin-A (Hcrt-1) immunoreactivity in cerebrospinal fluid is a biomarker for type 1 narcolepsy (NT1). However, the current radioimmunoassay (RIA) does not allow for precise quantification, which limits the assessment of disease severity and progression. Three previous mass-spectrometry studies showed mixed results. Following the identification of RIA antibody targets by immunoprecipitation (IP), this study aimed to compare the ability of two purification methods (IP and solid phase extraction (SPE)) followed by nano-liquid chromatography coupled to high-resolution mass spectrometry (nLC-HRMS) to identify patients with NT1.

Methods: IP with magnetic beads coupled to the RIA antibody (Phoenix Pharmaceuticals) was performed, and the resulting peptides were identified by MS using data-dependent acquisition. Full-length Hcrt-1 and Hcrt-1 fragment_1-16 levels were analyzed in 4 NT1 patients and 4 controls after both IP and SPE by nLC-HRMS.

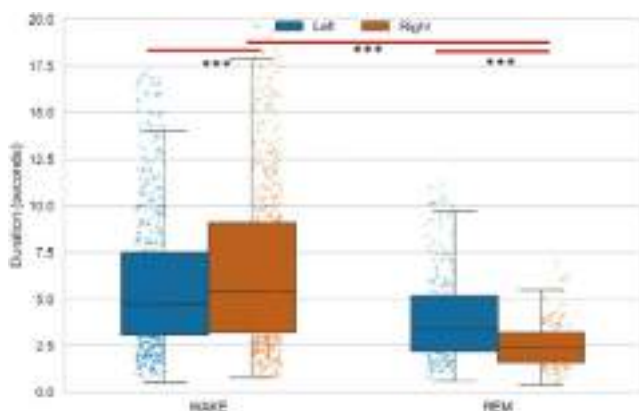


FIGURE 1 Boxplot of mean simple and complex movement duration per subject(s) across wakefulness and REM sleep. (*): $p < 0.05$, (***): $p < 0.001$ Each box represents the distribution of subject-level mean durations for movements performed with the left (blue) or right (orange).

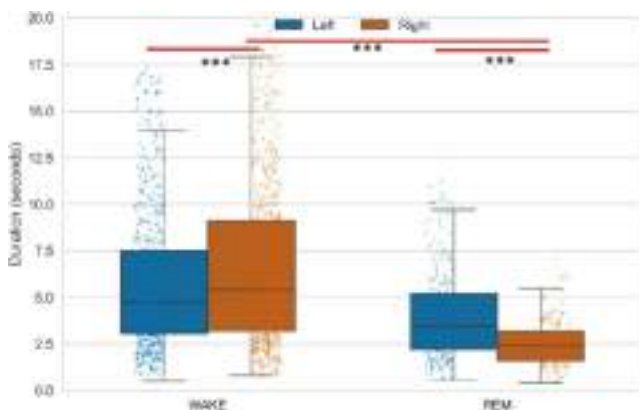


FIGURE 2 Distribution of individual complex movement durations during wakefulness and REM sleep. Each dot represents a single movement performed with the left (blue) or right (orange) arm, while boxes indicate the interquartile range (IQR) and whiskers extend to 1.

Results: The discovery experiment with IP most reliably revealed Hcrt-1 fragment_1-16. SPE generally revealed much higher concentrations than IP. Hcrt-1_1-16 levels enabled the distinction between patients and controls after both IP (controls 0.94 pg/mL [0.71–1.21] NT1 0.15 pg/mL [0.04–0.30], $p=0.029$) and SPE (controls 29.19 pg/mL [21.67–37.55], NT1 1.66 pg/mL [0.49–2.57], $p=0.029$), whereas full-length Hcrt-1 levels after IP (controls 1.48 pg/mL [0.89–2.33], NT1 2.26 pg/mL [1.86–2.89], $p=0.2$) did not.

Conclusion: Hcrt-1_1-16 appears stable and abundant in the CSF of controls and allows the identification of patients with NT1, making it a promising surrogate marker and confirming results of a recent study. SPE seems to be a more suitable method for further validation in clinical practice.

Disclosure: The authors declare no conflict of interest. The international Swiss Primary Hypersomnolence and Narcolepsy Cohort study is supported by the Swiss National Science Foundation (SNF 320030_185362; SNF 32003B_215721).

OPR-142 | Brain and heart cross-talk: Cortical topography of slow-wave–heart rhythm coupling is associated with cognition

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Background and Aims: Cognitive dysfunction is associated with both cardiac autonomic dysfunction and reduced slow-wave (SW) activity. Slow-wave–heart rhythm (SW–HR) coupling reflects the interaction between SW and autonomic activity and is largely underexplored. We investigated its association with cognition.

Methods: “Sleep and cognitive functioning” ($n=63$) included volunteers in good or excellent health condition. Demographics, medical history, cognition (11 tests), sleep architecture by polysomnography were assessed at study inclusion (Figure 1). SW–HR coupling was quantified using previously described (Fabus et al., 2024) and newly conceptualized metrics in terms of temporal coherence, SW amplitude, and SW globality (Figure 2). Associations between SW–HR coupling (dependent variables) and cognition (independent variables) were explored using multivariate linear regression, adjusted for age and arousal index at the level of individual electrodes for all parameters except globality. The significance level was set at $\alpha=0.05$.

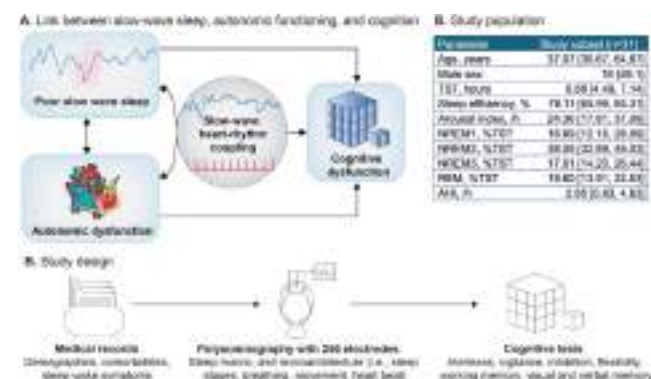


FIGURE 1

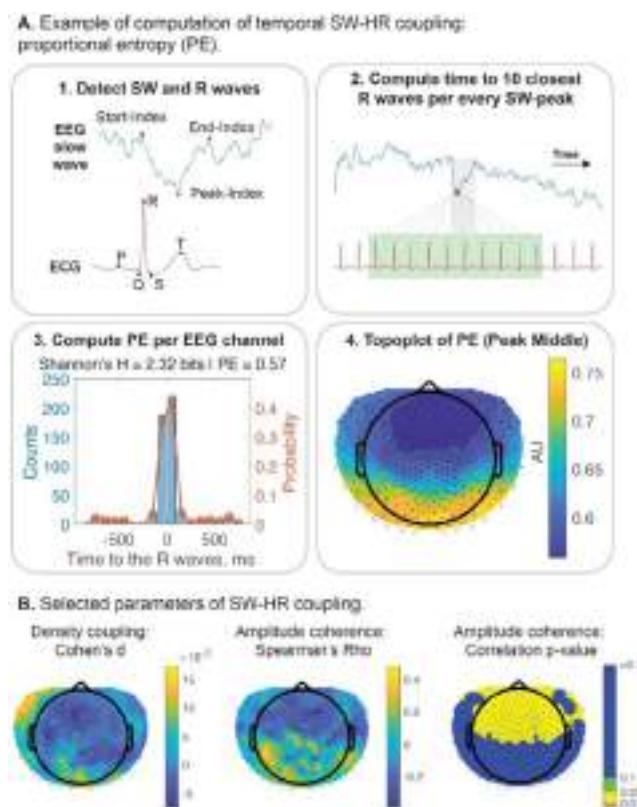


FIGURE 2

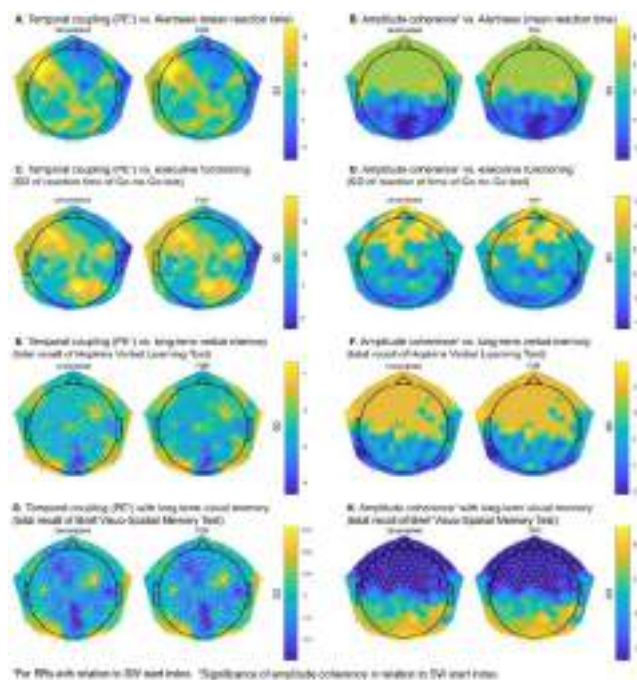


FIGURE 3

Results: A subset of 31 participants with 256-electrode electroencephalography was analyzed. SW–HR coupling was bi-directional and mostly involved fronto-central cortical areas. Stronger temporal and amplitude coupling was associated with alertness (frontal, left-lateralized), response inhibition (frontal and occipital, left-lateralized), verbal memory (frontal and occipital), and visual memory (central regions; Figure 3). Stronger

globality coupling was associated with the same domains (e.g., significance with visuospatial memory recall: $\beta = 5.84$ SD, $p < 0.008$). No associations were found for density coupling.

Conclusion: This is the first description of the cortical topography of SW-HR coupling and its association with multiple cognitive domains. SW-HR coupling may represent a promising marker of brain health.

Disclosure: Nothing to disclose.

OPR-143 | Behind the mirror: Clinical and neuroradiological significance of mirror movements in idiopathic REM sleep behavior disorder

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Background and Aims: Idiopathic REM Sleep Behavior Disorder (iRBD) is a prodromal stage of synucleinopathies such as Parkinson's disease (PD). Mirror movements (MM) are involuntary contralateral mirroring of voluntary actions, considered para-physiological in childhood and described in stroke, multiple sclerosis, and movement disorders, including PD. Their presence and significance in iRBD remain unexplored.

Methods: We enrolled 49 polysomnography-confirmed iRBD patients, divided into MM-positive ($n = 33$) and MM-negative ($n = 16$) groups. MM were further classified as monolateral ($n = 10$) or bilateral ($n = 23$). All underwent neurological, cognitive (MMSE, FAB, Rey and Benson figures, Rey Word List, Corsi, Token Test), motor (UPDRS I–III, 9HPT, 5TSTS, 10MWT), and MRI evaluations. FLAIR, 3DT1, and 3DT2 sequences quantified juxtacortical, deep grey matter (DGM), periventricular, and infratentorial hyperintensities.

Results: MM-positive patients showed lower upper limb dexterity (9HPT, $p = 0.029$) and reduced visuospatial-executive function (Corsi, $p = 0.004$, $p = 0.01$). Monolateral MM were associated with younger age and worse visuospatial, memory, and language

performance. Bilateral MM showed higher DGM hyperintensity volume ($p = 0.049$) and number ($p = 0.029$), with a trend toward higher periventricular burden ($p = 0.071$). Periventricular lesion volume and number correlated with worse visuospatial performance (Rey copy: $r = -0.668$, $p = 0.002$), mood symptoms (BDI-II: $r = 0.572$, $p = 0.013$), and gait impairment (10MWT steps: $r = 0.555$, $p = 0.014$).

Conclusion: Mirror movements are common in iRBD and show distinct clinical and radiological patterns. Monolateral MM may indicate early neurodegenerative changes, while bilateral MM may reflect a less specific pattern. MM assessment could help stratify RBD patients.

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OPR-144 | CSF proteomic alterations in spontaneous intracranial hypotension

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Background and Aims: Spontaneous intracranial hypotension (SIH) results from spinal CSF leaks, but there is sparse knowledge about metabolic sequelae, and no fluid biomarkers exist. We investigated the CSF proteome of SIH patients, aiming to identify disease signatures and potential biomarkers.

Methods: We analyzed CSF from 26 patients (9 with type I ventral dural leaks and 17 controls). Samples were analyzed by label-free liquid chromatography-tandem mass spectrometry after depletion and SP3-based preparation. Data were evaluated using differential expression, weighted correlation network analysis (WGCNA), gene set variation analysis (GSVA) with 50 Hallmark pathways, and protein–protein interaction network centrality. Classification performance was assessed with cross-validation and permutation testing.

Results: A total of 918 proteins were quantified, 311 were included in the final analysis, of which 31 were significantly dysregulated (FDR<0.05). A 31-protein panel discriminated leaks from controls with an AUC of 0.93 ($p<0.001$). LYVE1, a regulator of extracellular matrix and fluid balance, was the most stable biomarker, potentially implicating meningeal lymphatic dysfunction in patients with CSF leaks. Network analysis highlighted ENPP2, GOT1, CTSD, and RELN as central hubs within the disease network. Functional clustering pointed out the downregulation of extracellular matrix remodeling (e.g., FAT2, CD44) and aberrant tissue repair proteins, coupled with

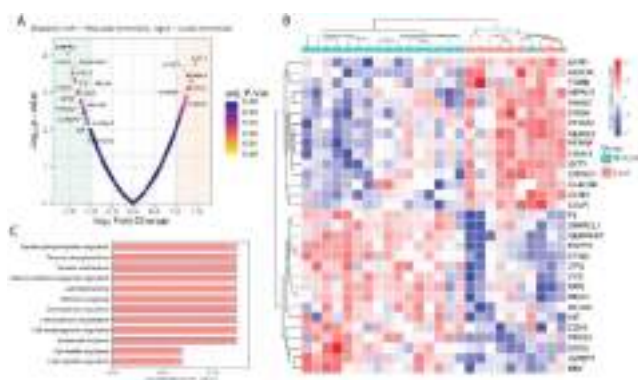


FIGURE 1 CSF-leak proteomic signature showing a leak-associated proteomic fingerprint.

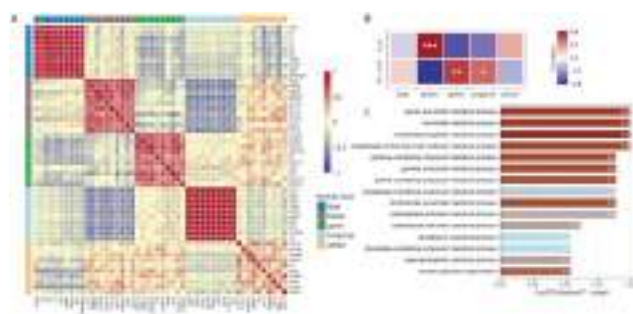


FIGURE 2 Protein co-expression network analysis of ventral CSF leaks reveals a specific leak-associated protein module.

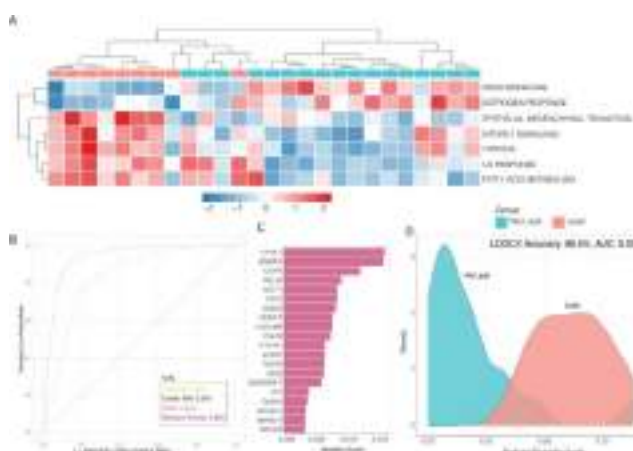


FIGURE 3 Integrated stratification of leak and control samples, enabling group stratification with high accuracy.

the downregulation of metabolic and neuroprotective pathways (CYST C, ENPP2, GOT1, ALDOA).

Conclusion: CSF proteomics revealed that CSF leaks induced a distinct proteomic biological signature. Interestingly, we identified proteomic patterns commonly associated with tissue remodeling, lymphatic dysfunction, and cognitive disturbance. CSF proteomics may identify biomarkers for CSF leaks.

Disclosure: Nothing to disclose.

OPR-145 | MRI biomarker predicts headache burden in spontaneous intracranial hypotension

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Background and Aims: Spontaneous intracranial hypotension (SIH) is caused by spinal cerebrospinal fluid (CSF) leaks and is classically characterized by orthostatic headache. Objective: To evaluate whether headache intensity is associated with clinical, anatomical, or dynamic MRI parameters.

Methods: Patients with SIH and a confirmed spinal CSF leak (12/2021–07/2024) were analyzed. Data included demographics, symptom duration, leak type, Bern score, HIT-6, and phase-contrast MRI-derived measures of CSF and spinal cord velocities at C2/C3. From these, derivatives as intradural volume

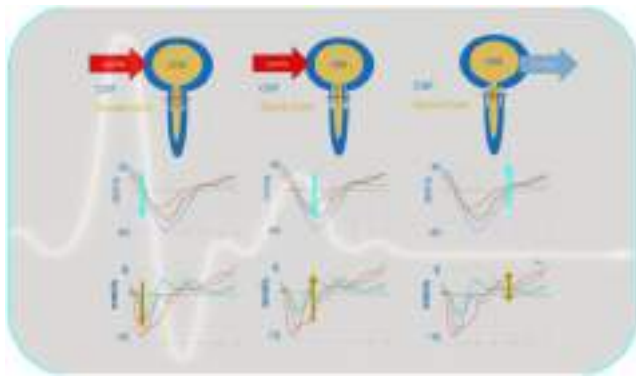


FIGURE 1 Example of cardiac dynamic phase-contrast MRI measurements of CSF and spinal cord velocities.

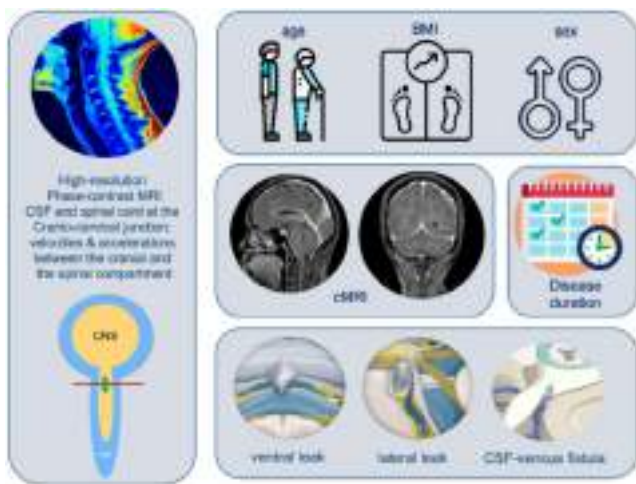


FIGURE 1 Data assessed per patient.



FIGURE 2 Results on demographics, leak types and headache intensity.

displacements and accelerations as compliance surrogates ($C = \Delta V / \Delta P$) were calculated.

Results: 150 patients were included (57% female; mean age 46.6 ± 12 years). Leak types were ventral (60%), lateral (17.3%), CSF–venous fistula (21.4%), and sacral (1.3%). Median symptom duration was 8 months (IQR 3–21). Substantial to severe headache burden was reported by 81%, and 75% had predominantly orthostatic headaches. Conventional MRI findings and clinical variables did not predict headache severity. However, in patients with dural tears (ventral&lateral leaks), increased cranially directed CSF acceleration was strongly associated with orthostatic headache burden within the first 12 months ($R = 0.574$, adj. $R^2 = 0.268$, $p = 0.012$), particularly in early disease (<3 months: $R = 0.909$, adj. $R^2 = 0.786$, $p < 0.001$). These associations became diffuse in chronic disease, and in CSF–venous fistulas.

Conclusion: SIH is associated with a high headache burden not explained by standard imaging or demographics. In early and intermediate disease, dynamic MRI biomarkers identify patients with greater orthostatic headache burden, whereas chronic SIH shows increasing heterogeneity, suggesting evolving pathophysiology.

Disclosure: none in relation to this study. KW is supported by the Margarete-Von-Wrangell-Fellowship, Ministry of Science, Research and Arts, Baden-Württemberg, Germany.

OPR-146 | Machine learning-based statistical interpretation of headache pattern data: Insights from the myclusters application

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Background and Aims: Management of cluster headache (CH) remains a challenge due to its variable presentation patterns, idiosyncratic nature of attacks and the rarity of the disease. Most patients with CH experience a diagnostic delay and effective treatment strategies are limited. The MyClusters application collects real-world global patient-reported data on, among others, attack frequency, timing and patient characteristics. The aim of the app is to aid pattern recognition, both on an individual as well as population level.

Methods: As of now, 2060 patients with (cluster) headaches have input 21,048 data points on attack timing, duration of the attack, intensity, symptoms, medications and trigger factors. An unsupervised machine learning model was trained for automated statistical analyses, to identify and classify clinically relevant headache attack patterns, predict bout periods, and identify personalised risk factors with trigger fingerprinting.

Results: The model successfully classified five distinct attack pattern subtypes with significant differences in treatment response. Personalised trigger identification showed 82% concordance with patient-reported associations, revealing temporal relationships between lifestyle factors and attack occurrence.

Conclusion: This machine learning-based tool provides objective, automated statistical analysis of MyClusters data, enabling early bout detection, pattern classification and personalised trigger identification. Integration of artificial intelligence with patient-reported outcomes offers promising applications for improving management of CH, potentially informing treatment decisions. This shows promise of further analysis by adding more data points by integrations with wearables and weather data to establish more pattern based sub-types.

Disclosure: Nothing to disclose.

OPR-147 | Beyond the aura: Is burden measurable in migraine?

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Background and Aims: Migraine is traditionally considered a benign disorder; however, it can be sorely disabling. Its pathogenesis includes multiple mechanisms, notably neurogenic

inflammation and neuroaxonal involvement. Therefore, a measurable biomarker to demonstrate subclinical disease burden is needed.

Methods: We included 86 patients with migraine (62 with aura [MWA]; 24 without aura [MWOA]) (classified according to ICH 3rd beta Version), and 126 (age- and sex-matched) control subjects (tension-type headache, dizziness, functional disorder [Controls]). Individuals with comorbidities associated with neuroaxonal damage, obesity, or renal insufficiency were excluded. By the selection of Controls, we aimed to strengthen the clinical relevance of the study. Serum neurofilament light chain (sNfL) and glial fibrillary acidic protein (sGFAP) were analyzed on a sensitive immunoassay platform. Age- and sex-adjusted biomarker levels were derived using linear regression residuals. Comprehensive demographic and clinical characteristics were considered.

Results: sNfL levels differed significantly between patients with migraine groups and Controls (Kruskal–Wallis, $p=0.004$). In contrast, sGFAP levels did not differ between the groups. The subgroup analysis revealed higher sNfL levels in patients with MWOA (median 0.58; IQR 4.22) compared to Controls (median -0.40 ; IQR 2.16) ($p=0.001$), whereas no statistically significant difference was found between MWA (median -0.26 ; IQR 2.00) and Controls ($p=0.307$).

Conclusion: These findings point to neuroaxonal involvement rather than astroglial injury and suggest that axonal burden is mainly dominant in MWOA. Furthermore, this study highlights the need to search for neurobiological differences between migraine phenotypes, to enable diagnostic and therapeutic stratification.

Disclosure: H.T.: Received honoraria as a consultant/speaker and/or for events sponsored by Alexion, Bayer, Biogen, Bristol–Myers Squibb, Celgene, Diamed, Fresenius, Fujirebio, GlaxoSmithKline, Horizon, Janssen–Cilag, Merck, Novartis, Roche, Sanofi–Genzyme, Siemens, Teva and Viatrix. No conflicts are relevant to the present study. F.B.: Nothing to disclose. P.K.: Nothing to disclose. Ö.S.: Nothing to disclose.

Neuroimaging 2

OPR-148 | Functional classification of thalamic nuclei in myotrophic lateral sclerosis: Contribution of limbic and associative nuclei to behavioural symptoms and disease progression

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TABLE 1 Classification of thalamic nuclei according to their functional specialization

Functional Group	Abbreviation	Full Name
Sensory nuclei	VPL	Ventral posterolateral
	VPM	Ventral posteromedial
	LGN	Lateral geniculate nucleus
	MDN	Medial geniculate nucleus
	L-SS	Lateral subgenicular state
Motor nuclei	VA	Ventral anterior
	VAmc	Ventral anterior, macular nucleus
	VLa	Ventral lateral anterior
	VLD	Ventral lateral posterior
Associative nuclei	LP	Lateral posterior
	PuA	Pulvinar anterior
	PuI	Pulvinar inferior
	PuL	Pulvinar lateral
	PuM	Pulvinar medial
Limbic nuclei	AV	Anteromedial
	LD	Lateral dorsal
	MDL	Mediodorsal lateral
	MDm	Mediodorsal medial
Intralaminar nuclei	CeM	Central medial
	CL	Central lateral
	CM	Centromedian
	Pc	Paracentral
	PI	Parasagittal
	Pl	Paralateral
	MY-Ra	Reunions (medial ventral)

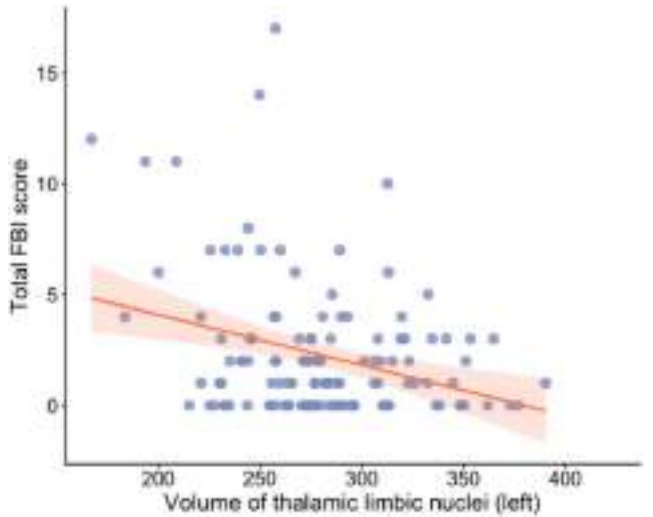


FIGURE 1 Scatter plots with regression lines showing inverse association between FBI total score and volume of limbic thalamic nuclei. FBI, frontal behaviour inventory.

Background and Aims: Although amyotrophic lateral sclerosis (ALS) has traditionally been regarded as a disease primarily affecting cortical structures, growing evidence highlights the significant involvement of subcortical regions in the emergence and progression of clinical symptoms. In this study, we investigated whether thalamic subnuclei, grouped according to their

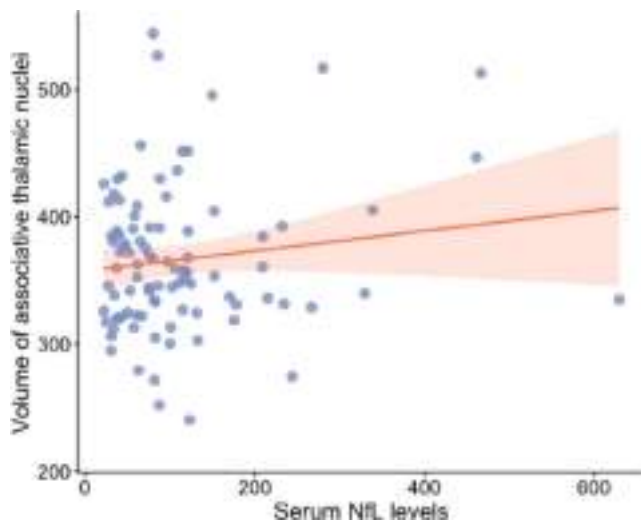


FIGURE 2 Scatter plots with regression lines showing positive association between serum NFL levels and volume of associative thalamic nuclei. NFL, neurofilament light chain.

functional specialization, contribute to the motor and neuropsychological phenotype of ALS patients.

Methods: We retrospectively analyzed 289 ALS patients who underwent 3-Tesla MRI with a 3D T1-weighted acquisition. Thalamic subnuclei volumes were estimated using FreeSurfer based on the probabilistic atlas of human thalamic nuclei (Iglesias et al., 2018) and subsequently grouped according to their functional classification into sensory, motor, limbic, associative, and diffuse projection nuclei.

Results: Thalamic subnuclei volumes were not significantly associated with the motor or cognitive profiles of the cohort. In contrast, limbic nuclei volumes (specifically mediodorsal medial and lateral) were inversely associated with behavioral symptom burden ($\beta = -0.28$; $p < 0.001$) and plasma neurofilament light chain (NFL) levels ($\beta = -0.26$; $p = 0.009$). Notably, volumes of associative (specifically pulvinar and lateral posterior) thalamic nuclei were positively correlated with disease progression rate ($\beta = 0.17$; $p = 0.001$) and plasma NFL levels ($\beta = 0.31$; $p < 0.001$).

Conclusion: Findings from our work suggest that limbic thalamic nuclei may contribute to the emergence of behavioural symptoms and to the rate of neurodegeneration. In contrast, associative nuclei appeared to be relatively spared in patients with rapid disease progression, supporting a putative protective or compensatory role of the pulvinar–lateral posterior nuclei in the progression of ALS.

Disclosure: A. Maranzano, G. Scacciatella, A. Cocuzza, V. Patisso, S. Pierro, M. Passaretti, S. Torre, E.N. Aiello, A. Doretti, C. Morelli, C.M. Cinnante, J.B. Pereira, F. Verde report no disclosure. Barabara Poletti. received compensation for consulting services and/or speaking activities from Liquidweb S.r.l. and she is associated Editor for Frontiers in Neuroscience Vincenzo Silani received compensation for consulting services and/or speaking activities from AveXis, Cytokinetics, Italfarmaco, LiquidWeb, Srl and Novartis Pharma AG. He receives or he has received research support from the Italian Ministry of Health, AriSLA, and E-Rare Joint Translational Call. He is on. the Editorial Board of Amyotrophic Lateral Sclerosis and Frontotemporal Degeneration, European Neurology, American Journal of Neurodegenerative Disease and Frontiers

in Neurology. Nicola Ticozzi received compensation for consulting services from Amylyx Pharmaceutical, Biogen, Italfarmaco and Zambon Biotech SA. He received research funding from the Italian Ministry of Health, the Italian Ministry of University and Research and AriSLA. He is associate editor of Frontiers in Aging Neuroscience.

OPR-149 | Structural brain changes in Alzheimer's disease: Relationship with cerebrospinal fluid pTAU181 and clinical implications

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Background and Aims: Alzheimer's disease (AD) is a neurodegenerative disorder characterized by cognitive decline and brain atrophy. Neuroimaging biomarkers reflecting underlying neuropathological processes are essential for diagnosis and prognosis. This study aims to investigate the relationship between cerebrospinal fluid (CSF) biomarkers, atrophy of relevant brain regions and cognitive decline in patients with AD.

Methods: Ninety-four patients with mild cognitive impairment and mild dementia due to AD, diagnosed according to the 2024 NIA-AA criteria, were enrolled [1]. Available data included CSF biomarkers, MRI-derived brain volumes, demographic information at diagnosis, and Mini-Mental State Examination (MMSE) scores at 1-year follow-up (56/94 patients). Patients were classified according to: pTAU181 levels above or below 78 pg/mL [2] (54/94 vs. 40/94); MMSE outcome at 1-year (stable vs. worsened). Brain volumes were adjusted for age, sex, magnetic field strength, and total intracranial volume. Analyses of covariance and binomial regression models were applied.

Results: Forty-six patients were recruited in Vrije Universiteit Brussels (mean age 72.17; 21 males/25 females; education 12.75) and forty-eight in University of Siena (mean age 74.14; 25males/23females; education 9.34). Higher pTAU181 levels were associated with lower volumes of the hippocampal head ($p = 0.005$), body ($p = 0.04$), and tail ($p = 0.03$), the CA1 hippocampal subfield ($p = 0.004$), the medial-mediodorsal thalamic nucleus ($p = 0.02$), total amygdala volume ($p = 0.003$), and basal ($p < 0.001$) and cortical ($p = 0.02$) amygdala nuclei. Only

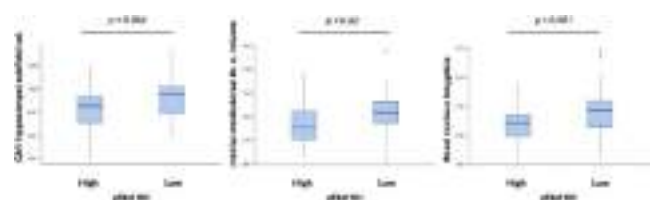


FIGURE 1 Boxplots showing the comparison of regional brain volumes in subjects with high and low pTAU181 levels. Subjects with high pTAU181 levels exhibit significantly reduced volumes in all the brain regions shown.

hippocampal head ($p=0.02$) and CA1 volumes ($p=0.03$) predicted cognitive performance at 1-year follow-up.

Conclusion: Elevated CSF pTAU181 levels are associated with selective atrophy of hippocampal, thalamic, and amygdala regions. Hippocampal head and CA1 subfield volumes are linked to cognitive outcome.

Disclosure: Nothing to disclose.

OPR-150 | The developmental dynamics of cortical grey matter in children with self-limiting epilepsy and centrotemporal spikes

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Background and Aims: Self-limited epilepsy with centrotemporal spikes (SeLECTS) has long been considered to be a benign condition that resolves without lasting consequences. However, evidence has emerged linking epileptiform activity in SeLECTS to impairments in language, attention, working memory and socio-emotional processing, challenging this assumption. Recurrent centrotemporal spikes may disrupt critical phases of cortical maturation. In this context, the longitudinal impact of epileptiform activity on grey matter development in SeLECTS remains insufficiently explored.

Methods: We conducted a 5-year longitudinal 1.5 T MRI study (Siemens) in children with SeLECTS and matched controls. 23 patients and 28 healthy participants (mean age: ~9–14 years) were included in the analyses. Cortical thickness trajectories were analysed using CAT12/SPM12, with vertex-wise non-parametric permutation testing of linear and quadratic effects adjusted for age, age², and sex with family-wise error correction, used for quantitation of thickness and thickness change.

Results: Vertex-wise longitudinal analyses revealed the expected progressive cortical thinning in the control group across the frontal, cingulate, parietal, occipital and insular cortices. In the SeLECTS group cortical maturation followed distinct trajectories, with delayed linear thinning observed in the fronto-central and paracentral regions alongside steeper thinning in the posterior associative cortices. Quadratic effects further distinguished between the two groups, indicating nonlinear deviations in cortical thickness trajectories over time, particularly within the medial frontal, cingulate and temporo-parietal regions.

Conclusion: SeLECTS shows subtle, region-specific alterations in cortical thinning trajectories, suggesting a

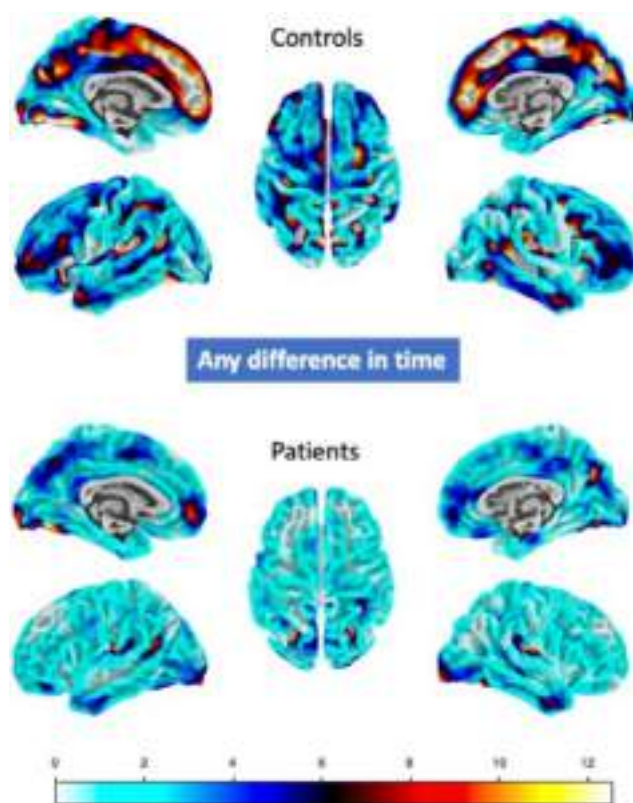


FIGURE 2 Differences in each group over time.

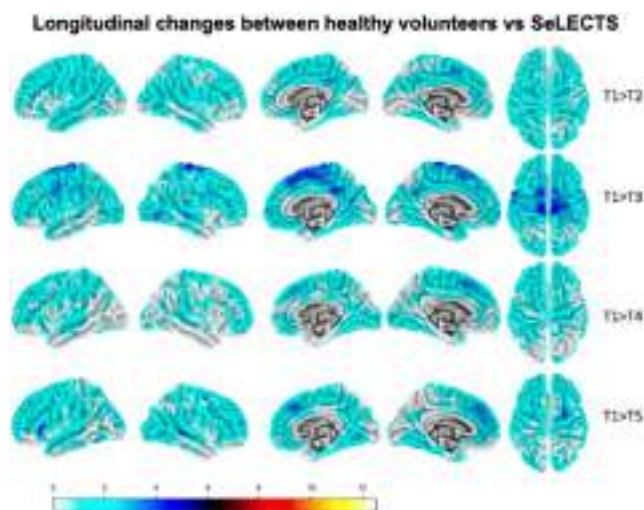


FIGURE 3 Statistically significant differences between groups over 5 years period.

neurodevelopmental impact that challenges its traditional characterization as a benign condition.

Disclosure: Nothing to disclose.

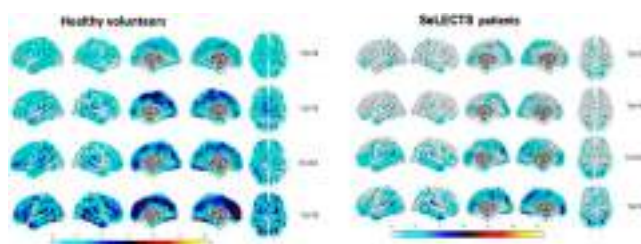


FIGURE 1 Within group difference from T1 to T5.

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Background and Aims: Physiological aging involves brain structural changes. Understanding these changes is essential to distinguish normal aging from disease-related alterations in multiple sclerosis (MS). This study modeled normative age-related trajectories of brain and grey matter (GM) volumes in healthy controls (HC) and applied them to a large multicenter MS cohort to identify disease-specific atrophy patterns.

Methods: We analyzed 3D T1- and T2-weighted MRI scans from 995 HC and 2016 MS patients from the Italian Neuroimaging Network Initiative (INNI). After image pre-processing, normalized brain, cortical, and deep GM volumes were estimated using FSL-SIENAX and FSL-FIRST. Age-related volumetric trajectories were modeled in HC using regression analyses accounting for linear and non-linear age effects, sex, scanner, and interactions. These models were applied to MS patients to compute z-scores reflecting disease-related atrophy beyond normal aging, with analyses stratified by sex and age at disease onset.

Results: In HC, brain volume declined non-linearly with age, while cortical and deep GM decreased linearly. In MS patients, z-scores declined linearly with age ($p < 0.01$), indicating progressively greater MS-related brain and GM atrophy. Cortical atrophy was more pronounced in men after age 40 ($p = 0.03$), while deep GM atrophy was greater in women after age 30 ($p < 0.001$). Pediatric-onset MS showed faster brain and cortical atrophy than adult-onset MS, whereas late-onset MS exhibited greater brain and cortical atrophy than adult-onset MS with aging.

Conclusion: In conclusion, these results highlight the need to account for both physiological aging and patient-specific factors, such as sex and age at onset, when evaluating brain atrophy in individuals with MS.

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SUPPLEMENT ABSTRACTS

ePoster

Saturday, June 27 2026

Ageing and Dementia 1

EPO-0001 | Sodium channel blockers are associated with reduced dementia risk in late-onset unexplained epilepsy

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Background and aims: Late-onset unexplained epilepsy is associated with increased dementia risk. Network hyperexcitability, central to seizure generation, has been implicated in amyloid- β release and tau propagation, suggesting a mechanistic link. Under this model, antiseizure medications that more effectively suppress excitability may modify dementia risk, but comparative neuroprotective effects across commonly used ASMs remain unclear.

Methods: We emulated a target trial using real-world healthcare data from the TriNetX platform to compare dementia outcomes across ASM monotherapy strategies in patients with newly diagnosed LOUE. Confounding addressed with propensity-score matching. Cox proportional hazards models estimated hazard ratios (HRs) with 95% confidence intervals (CIs) for incident all-cause dementia and Alzheimer's disease. The cohort included 24,262 patients treated with SV2A ligands, sodium channel

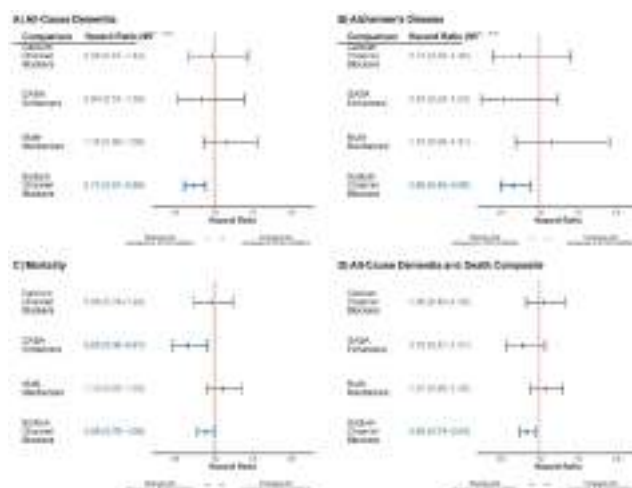


FIGURE 1 Hazard ratios (95% CI) for dementia and mortality outcomes comparing mechanism-based antiseizure medication groups versus SV2A inhibitors. Panels show: (A) All-cause dementia, (B) Alzheimer's disease, (C) Mortality, (D) All-cause dementia + death. Diamond.

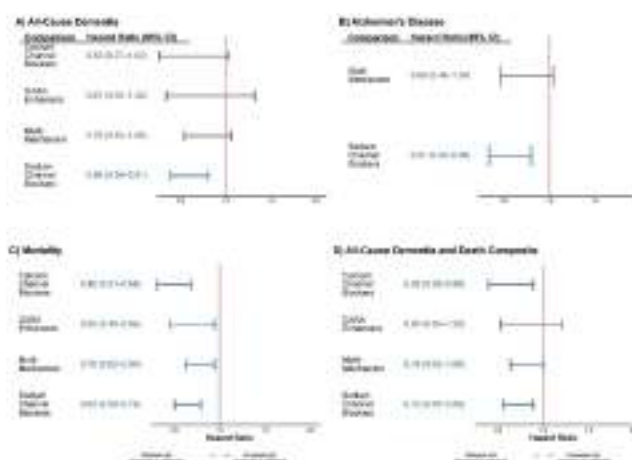


FIGURE 2 Hazard ratios (95% CI) for dementia and mortality outcomes in the Down syndrome cohort comparing mechanism groups vs SV2A inhibitors. Panels show: (A) All-cause dementia, (B) Alzheimer's disease, (C) Mortality, (D) All-cause dementia + death. Diamond mark.

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blockers, multi-mechanism agents, calcium channel blockers, or GABAergic agents. Findings were externally validated in a high-risk Down syndrome cohort and the National Alzheimer's Coordinating Center (NACC) dataset.

Results: Sodium channel-blocking ASMs were associated with lower dementia risk compared with SV2A ligands, including a 27% reduction in all-cause dementia (HR 0.73, CI 0.61–0.88) and a 36% reduction in Alzheimer's disease (HR 0.66, CI 0.49–0.88). Phenytoin and carbamazepine showed the strongest inverse associations with dementia, lamotrigine was associated with lower mortality. Associations were consistent across earlier cognitive endpoints, absent for negative controls, and directionally concordant in external validation.

Conclusion: Sustained suppression of neuronal hyperexcitability by specific ASMs appears to modify downstream neurodegenerative risk. These findings nominate sodium channel blockers as candidates for repurposing in dementia prevention and support prospective randomized trials targeting early network excitability.

Disclosure: Nothing to disclose.

EPO-0002 | Hearing aid use and risk of dementia in adults with late-onset epilepsy, stroke, or diabetes and comorbid hearing loss

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Background and aims: Hearing loss is a major modifiable risk factor for dementia, yet its impact on high-risk neurological populations remains poorly characterized. We investigated whether hearing aid (HA) use reduces dementia incidence in adults with late-onset epilepsy (LOE), stroke, or diabetes with comorbid hearing loss.

Methods: Using TriNetX, a federated electronic health record network, we conducted retrospective cohort studies emulating target trials. We analyzed propensity score-matched cohorts of 1,093 LOE pairs, 2,363 stroke pairs, and 14,934 diabetes pairs (all aged ≥ 55 years). Primary outcomes included incident Alzheimer's disease (AD) and all-cause dementia over a median follow-up of 4.2–5.1 years.

Results: HA use was associated with significant reductions in AD across all cohorts: LOE (HR 0.52, 95% CI 0.38–0.71), stroke (HR 0.59, 0.37–0.95), and diabetes (HR 0.69, 0.53–0.90). All-cause dementia risk was also significantly reduced in LOE (HR 0.50, 0.32–0.79) and diabetes (HR 0.81, 0.70–0.94). Negative control outcomes (glaucoma, frailty) showed no consistent protective patterns. Sensitivity analyses in LOE patients indicated that significant protective effects for AD persisted even when HA intervention was initiated after five years of hearing loss onset.

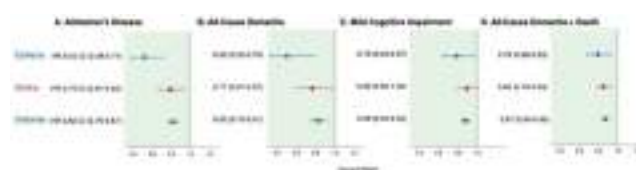


FIGURE 1 Hazard ratios (95% CI) for outcomes across the epilepsy, stroke and diabetes cohorts.

Conclusion: Auditory intervention is a scalable, low-risk strategy for dementia prevention in neurologically vulnerable populations. The protective effects in these groups may exceed those observed in the general population, likely by reducing cognitive load and potentially interrupting shared neurodegenerative pathways.

Disclosure: Nothing to disclose.

EPO-0003 | Sleep-related experiences inflate hallucination scores across neurodegenerative disorders

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Background and aims: Hallucinations are widely used to support diagnosis, prognosis, and treatment decisions in neurodegenerative disorders. However, standard clinical scales do not distinguish hallucinations occurring during full wakefulness from perceptual experiences emerging at sleep–wake transitions (hypnagogic and hypnopompic phenomena), which are non-psychotic by definition.

Methods: We developed the Hypno/Awake Hallucination Assessment (HAHA), a two-item extension of the MDS-UPDRS hallucination item, and the Sleep-Related Experiences Questionnaire (SREQ), a 7-item scale capturing dream and sleep–wake boundary phenomena. These were administered in 73 patients with Parkinson's disease and in 177 consecutive patients from a tertiary neurodegenerative disorders clinic. Bayesian multiple regression tested whether sleep-related hallucinations contributed to standard hallucination scores independently of awake hallucinations, and whether they were specifically associated with sleep-related experiences.

Results: Across both cohorts, sleep-related hallucinations showed extreme evidence of contributing to standard hallucination scores even after accounting for awake hallucinations. Sleep-related hallucinations, but not awake hallucinations, were strongly associated with the frequency of sleep-related experiences. Sleep-related experiences differed across diseases with extreme evidence, being more frequent in Lewy-body disorders than in Alzheimer's disease and frontotemporal lobar degeneration syndromes and correlated with REM sleep behavior disorder.

Conclusion: Standard hallucination scales conflate awake hallucinations with sleep-related perceptual phenomena, leading to systematic inflation of hallucination estimates, particularly in Lewy-body disorders. Disentangling these mechanisms

is essential for accurate diagnosis, appropriate treatment, and mechanistic interpretation of hallucinations.

Disclosure: Nothing to disclose.

EPO-0004 | Volumetric analysis of hippocampal subfields and cognitive change in Alzheimer's dementia and mild cognitive impairment: A prospective cohort study

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Background and aims: Hippocampal subfield atrophy demonstrates differential vulnerability in Alzheimer's disease (AD), but the clinical relevance of specific subfield volume ratios for predicting cognitive decline remains unclear. This study examined associations between hippocampal subfield volume ratios

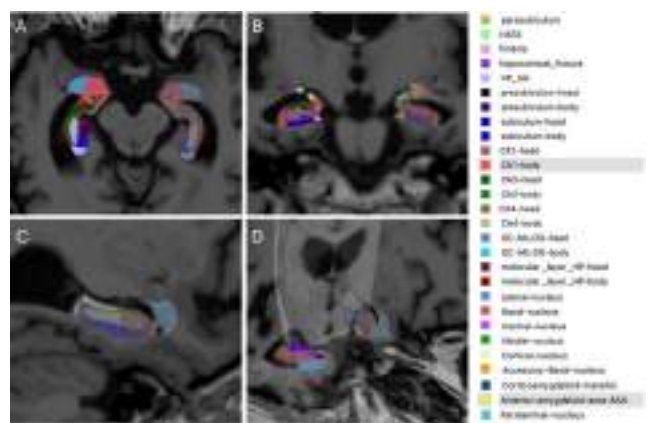


FIGURE 1 Representative segmentation of hippocampal and amygdala subfields on high-resolution T1-weighted MRI. (A) Axial, (B) coronal, (C) sagittal, and (D) three-dimensional views with color-coded subregions.

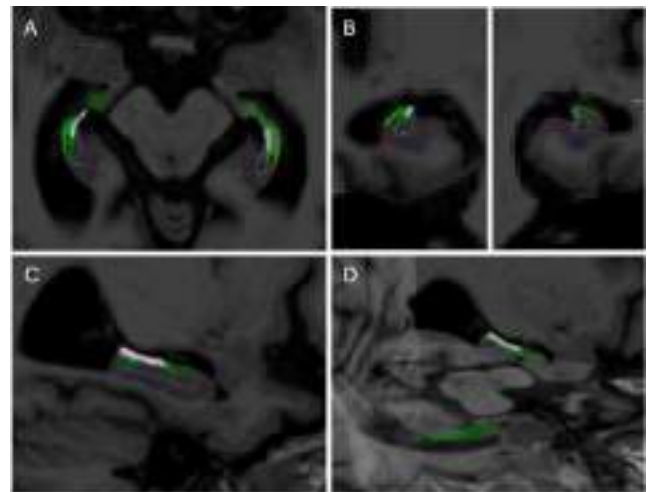


FIGURE 2 Hippocampal subfields demonstrating associations with cognitive decline. (A-D) Multiple viewing planes showing color-filled regions with statistically significant associations (green for CA3, pink for fimbria).

TABLE 1 Multivariable associations of bilateral CA3 and fimbria volume ratios (per 1-SD) with changes in cognitive scores (MMSE and MoCA).

		Associations to cognitive ratio to total hippocampal volume			Associations to cognitive ratio to TIV		
		B	SE	P-value	B	SE	P-value
MMSE (scores)	Model 1	-1.827	0.579	0.001**	-1.558	0.589	0.010*
	Model 2	-1.828	0.580	0.001**	-1.559	0.603	0.013*
	Model 3	-1.829	0.580	0.001**	-1.560	0.612	0.017*
MoCA (scores)	Model 1	-2.081	0.648	0.001**	-1.860	0.657	0.004**
	Model 2	-2.082	0.649	0.001**	-1.861	0.668	0.006**
	Model 3	-2.083	0.650	0.001**	-1.862	0.679	0.008**
TIV (cm³)	Model 1	-0.037	0.003	0.001**	-0.039	0.004	0.002*
	Model 2	-0.038	0.004	0.001**	-0.040	0.004	0.001*
	Model 3	-0.039	0.004	0.001**	-0.041	0.005	0.001*
Ratio of fimbria	Model 1	2.178	0.528	0.000**	2.128	0.566	0.000**
	Model 2	2.182	0.529	0.000**	2.132	0.578	0.000**
	Model 3	2.183	0.530	0.000**	2.133	0.589	0.000**

and longitudinal cognitive changes in AD dementia and amnesic mild cognitive impairment (aMCI).

Methods: Fifty-four participants with AD dementia or aMCI underwent baseline cognitive assessments, high-resolution T1-weighted MRI, and follow-up cognitive evaluations at two years. Hippocampal subfield volumes were automatically segmented and normalized to total hippocampal volume and total intracranial volume (TIV). Linear regression analyses examined associations between standardized subfield ratios and changes in Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment (MoCA) scores.

Results: The volume ratios of the fimbria and cornu ammonis 3 (CA3) demonstrated the strongest associations with changes in MMSE (fimbria: B = 1.95, SE = 0.51, $p < 0.001$; CA3: B = 1.75, SE = 0.52, $p = 0.002$) and MoCA (fimbria: B = 1.818, SE = 0.559, $p = 0.002$; CA3: B = 1.58, SE = 0.57, $p = 0.008$) scores. Similar patterns emerged with TIV normalization. Associations remained significant after adjusting for age, education, baseline cognition, ApoE4 genotype, and medication use.

Conclusion: Among hippocampal subfields, the fimbria and CA3 regions demonstrate stronger associations with longitudinal cognitive decline in AD and aMCI compared to other subfield structures. These in vivo findings may serve as useful biomarkers for risk assessment, though further validation studies are needed to confirm their clinical utility.

Disclosure: Nothing to disclose.

EPO-0005 | A 48-month analysis of the lecanemab clarity AD open-label extension in the ApoE ε4 non-carriers or heterozygotes early Alzheimer's disease population

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Background and aims: Lecanemab, an Aβeta-directed antibody, showed meaningful delay in disease progression and biological effects consistent with disease modification in Clarity AD. Herein, we present the 48-month results in ApoE ε4 non-carriers or heterozygotes population from the ongoing Clarity AD open-label extension (OLE) study.

Methods: Clarity AD is an 18-month, randomized study (Core) with a subsequent OLE phase in individuals with early AD.

Safety and clinical efficacy outcomes (CDR-SB, ADAS-Cog14, and ADCS-MCI-ADL) were assessed from OLE data out to 48 months. Continued lecanemab treatment in the OLE beyond the 18 months from the Core study was compared to matched historical placebo controls from Alzheimer's Disease Neuroimaging Initiative (ADNI) data and the Swedish BioFINDER1 study. Subgroup analyses were conducted for participants with no/low baseline tau.

Results: In the ApoE ϵ 4 non-carriers or heterozygotes population, 1521 participants were treated with lecanemab across the Clarity AD Core and OLE. No new safety signals were observed. ARIA rates were low and similar on placebo after 6 months. Lecanemab-treated participants continued to accrue benefit in CDR-SB through 48 months, with continued separation through 36 months relative to the historical controls. Results were similar for ADAS-Cog14 and ADCS-MCI-ADL. Consistent rates of clinical stability or improvements were observed across assessments regardless of baseline tau levels. Lecanemab reduced relative risk of progression to next dementia stage by 55% and 62% vs ADNI and BioFINDER, respectively.

Conclusion: In the ApoE ϵ 4 heterozygotes or non-carrier subgroup of Clarity AD, lecanemab slowed decline in disease progression with expanding benefit over time.

Disclosure: L reports a relationship with Anavex, Avanir/Otsuka, Biogen, BioVie, Bristol-Myers-Squibb, DerCampus, Eisai, Eli Lilly, FOMF, Araclon/Grifols, Janssen Cilag, Johnson & Johnson, Medical Tribune, Medfora, Medscape, Neurimmune, Neuroscios, Noselab, NovoNordisk, Pharmatrophix, Hoffmann-La Roche, TauRX, Schwabe, StreamUp, Vivoryon that includes: personal consulting fees. SD, DL, MK, AG, MH, ST, SH, MI, and LK are employees of Eisai.

EPO-0006 | Electric field modulation of amyloid- β fibril propagation: Molecular insights supporting neuromodulation approaches in Alzheimer's disease

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Background and aims: Amyloid- β 42 (A β 42) fibril accumulation is a central pathological hallmark of Alzheimer's disease and a primary target of disease-modifying therapies. Non-invasive brain stimulation techniques, such as transcranial direct current stimulation (tDCS), are increasingly explored as complementary interventions, but their direct biological targets remain unclear. This study aimed to investigate whether electric fields, conceptually analogous to those induced by tDCS, can modulate A β 42 fibril structure and propagation at the molecular level.

Methods: We performed molecular simulations on a brain-derived A β 42 fibril model composed of 66 monomers arranged in 33 layers, reconstructed from cryo-EM data and including

disordered N-terminal regions. Static electric fields ranging from 0 to 0.2 V/nm were applied under different structural restraint conditions to separately assess effects on fibril surface properties and fibril elongation. Simulations were run for 1–2 μ s in triplicate.

Results: Electric fields induced a displacement of the disordered N-terminal regions that saturated at $\sim$ 0.08 V/nm, reducing their interactions with the fibril core and modifying surface properties associated with amyloid proliferation. In addition, increased structural fluctuations were observed at fibril ends, particularly in residues involved in elongation, indicating a destabilizing effect on fibril growth. These effects were systematic and increased with field strength.

Conclusion: These findings provide mechanistic evidence that electric fields can influence molecular processes underlying amyloid fibril propagation. Although the applied field strengths exceed those generated by clinical tDCS, the results support the biological plausibility of neuromodulation approaches as potential modifiers of amyloid pathology and encourage further experimental and clinical investigation.

Disclosure: Nothing to disclose.

EPO-0007 | OCTARIA-AD: Optical coherence tomography angiography as a non-invasive biomarker for ARIA in early Alzheimer's disease

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Background and aims: Alzheimer's disease (AD), the leading cause of dementia worldwide, is characterized by progressive cognitive decline driven by amyloid- β and tau pathology. Anti-amyloid monoclonal antibodies show clinical benefit in early AD but are limited by amyloid-related imaging abnormalities (ARIA). Current monitoring relies on resource-intensive MRI, which may miss early microvascular changes. Optical coherence tomography (OCT) and OCT angiography (OCT-A) offer non-invasive assessment of retinal neuroaxonal and microvascular integrity and may reflect cerebral small-vessel pathology.

Methods: OCTARIA-AD is a monocentric, prospective observational study enrolling 60 participants aged 50–90 years: 30 patients initiating anti-amyloid therapy and 30 age- and sex-matched controls with mild cognitive impairment or mild AD. OCT and OCT-A are performed at baseline and four follow-ups up to 52 weeks. Outcomes include peripapillary retinal nerve fiber layer (pRNFL) and ganglion cell-inner plexiform layer (GCIPL) thickness, vessel density and foveal avascular zone area. ARIA is assessed using routine MRI.

Results: As of January 25, 2026, 8 and 7 patients receiving lecanemab and donanemab have been enrolled, respectively; two were excluded due to insufficient baseline OCT image quality. The preliminary cohort comprises 13 patients (mean age 71 years [4.7], 83.3% female, 66.7% APOE heterozygotes, median MMSE 27 [range 23–30]). Mean baseline pRNFL and GCIPL thickness were 98.8 μ m (8.3) and 67.2 μ m (7.1), respectively. After a

median follow-up of 33 days (range 0–50), no ARIA events were observed.

Conclusion: OCTARIA-AD seeks to establish OCT/OCT-A as accessible biomarkers for ARIA risk stratification in early AD, potentially enhancing patient safety and complementing MRI monitoring.

Disclosure: Nothing to disclose.

EPO-0008 | Higher planetary health diet index scores are associated with better executive function and processing speed in older adults

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Background and aims: The EAT–Lancet Planetary Health Diet integrates human health with environmental sustainability. However, evidence linking its adherence, measured by the Planetary Health Diet Index (PHDI), to domain-specific cognitive performance remains sparse.

Methods: We analyzed 2,636 participants aged ≥60 from the National Health and Nutrition Examination Survey (2003–2010). Dietary intake was assessed via two 24-hour recalls to calculate the PHDI. Cognitive outcomes included the CERAD battery, Animal Fluency, and the Digit Symbol Substitution Test (DSST). Survey-weighted linear regression estimated β coefficients, adjusting for sociodemographic, lifestyle, and cardiometabolic factors.

Results: Higher PHDI adherence was significantly associated with superior cognitive performance, characterized by a distinct domain-specific sensitivity. In the fully adjusted model, each 10-point higher PHDI was associated with higher scores on the DSST (β=0.9746; p=0.0012) and Animal Fluency (β=0.3811; p=0.0003). Strong dose–response patterns were observed for both executive function (p-trend=0.0008) and semantic fluency (p-trend=0.0001). While the PHDI was positively associated with CERAD immediate recall (Trials 1–3: β=0.1767; p=0.0410), associations with delayed recall were not statistically significant.

Conclusion: Greater adherence to a planetary health-aligned dietary pattern was associated with significantly better executive function and processing speed in older adults. These findings support the “co-benefit” of sustainable diets for brain health, suggesting that sustainability-aligned dietary frameworks may serve as an effective strategy for preserving cognitive resilience in an aging population.

Disclosure: Nothing to disclose.

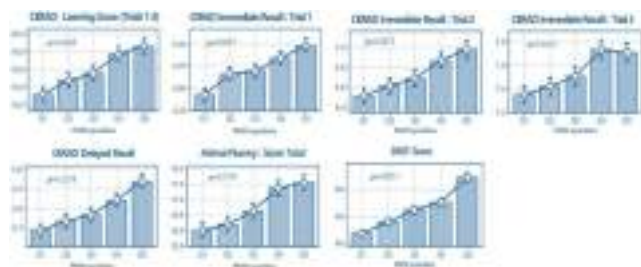


FIGURE 1 Cognitive test scores across quintiles of the PHDI Bars show the mean cognitive test score within each PHDI quintile (Q1–Q5).

TABLE 1 PHDI (per 10-point increase) and cognitive performance.

Cognitive outcome	Model 0			Model 1			Model 2		
	β	95% CI	p-value	β	95% CI	p-value	β	95% CI	p-value
CERAD Learning Recall (Trials 1–3)	0.1806	(0.141, 0.220)	0.0001	0.1777	(0.138, 0.216)	0.0001	0.1767	(0.137, 0.215)	0.0001
CERAD Immediate Recall (Trial 1)	0.1492	(0.09, 0.208)	0.0001	0.1477	(0.088, 0.206)	0.0001	0.1470	(0.088, 0.206)	0.0001
CERAD Immediate Recall (Trial 2)	0.1344	(0.08, 0.188)	0.0001	0.1334	(0.079, 0.187)	0.0001	0.1334	(0.079, 0.187)	0.0001
CERAD Immediate Recall (Trial 3)	0.0217	(0.001, 0.042)	0.0001	0.0216	(0.001, 0.042)	0.0001	0.0216	(0.001, 0.042)	0.0001
CERAD Delayed Recall	0.1127	(0.02, 0.205)	0.0001	0.1116	(0.02, 0.202)	0.0001	0.1117	(0.02, 0.202)	0.0001
Animal Fluency (Total)	0.3811	(0.15, 0.604)	0.0001	0.3806	(0.15, 0.604)	0.0001	0.3803	(0.15, 0.604)	0.0001
DSST Total	0.9746	(0.49, 1.459)	0.0001	0.9737	(0.49, 1.458)	0.0001	0.9736	(0.49, 1.458)	0.0001

PHDI, Planetary Health Diet Index; β, beta coefficient; 95% CI, 95% confidence interval; p-value, p-value; Model 0, unadjusted model; Model 1, adjusted for age, sex, education, and lifestyle; Model 2, adjusted for age, sex, education, lifestyle, and cardiometabolic factors.

EPO-0009 | NPTX1 differentiates idiopathic normal pressure hydrocephalus from Alzheimer's disease

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Background and aims: Normal pressure hydrocephalus (NPH) is a neurological syndrome characterised by dementia, gait dysfunction, urinary incontinence, and ventriculomegaly. Differentiating NPH from other dementias, including Alzheimer's disease (AD), remains challenging, and there are no cerebrospinal fluid (CSF)-based diagnostic biomarkers specific to NPH.

Methods: CSF samples from 20 NPH patients, 30 AD patients, and 25 cognitively normal (CN) controls were subjected to mass spectrometry-based proteomic analysis. Differential expression analysis was performed for all pairwise group comparisons, and differentially expressed proteins in NPH were analysed with the Gene Ontology enrichment tool. A random forest model with protein inputs was trained to assess its ability to differentiate NPH from AD patients. Feature selection algorithms were applied to identify individual and complementary candidate biomarkers. Results were evaluated with immunological testing in both discovery and verification cohorts.

Results: Proteomic profiling revealed upregulated immune-related proteins, including components of the complement cascade, and many downregulated synaptic proteins in NPH patients (Figure 1). The random forest model achieved a mean classification accuracy of 92% over 150 re-runs. Neuronal pentraxin 1 (NPTX1) and gamma-enolase (ENO2) emerged as top biomarker candidates (Figure 2). NPTX1 concentration was lower in NPH patients than in AD patients, resulting in a diagnostic accuracy of 0.94 (95% CI 0.89–0.99) (Figure 3). Additionally, NPTX1 concentrations correlated positively with total tau, phosphorylated tau181, and amyloid β42 in NPH patients, but not in AD patients.

Conclusion: Together, our results highlight synaptic alterations and neuroinflammation as key characteristics of NPH and identify NPTX1 as a promising diagnostic biomarker.

Disclosure: Nothing to disclose.

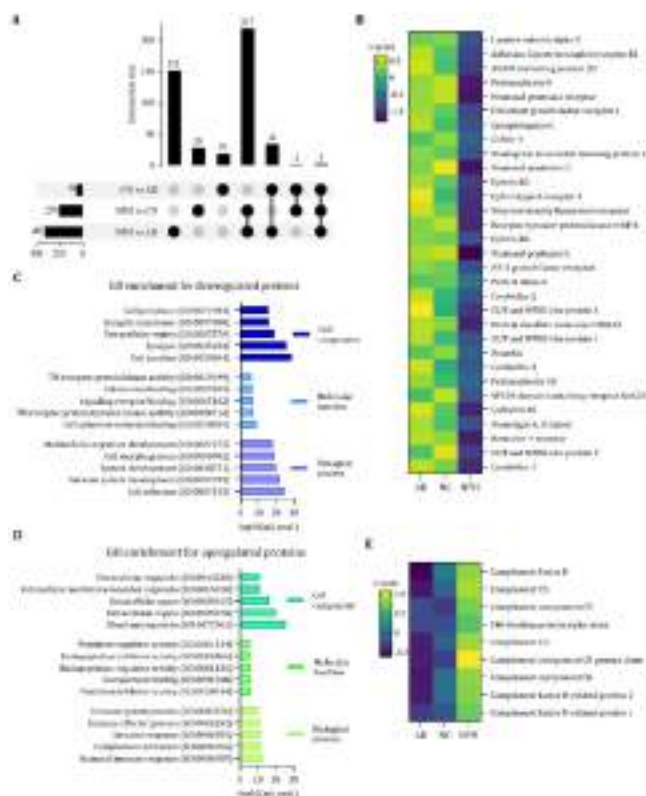


FIGURE 1 Analysis of differentially expressed proteins specific for NPH in comparison to AD and CN.

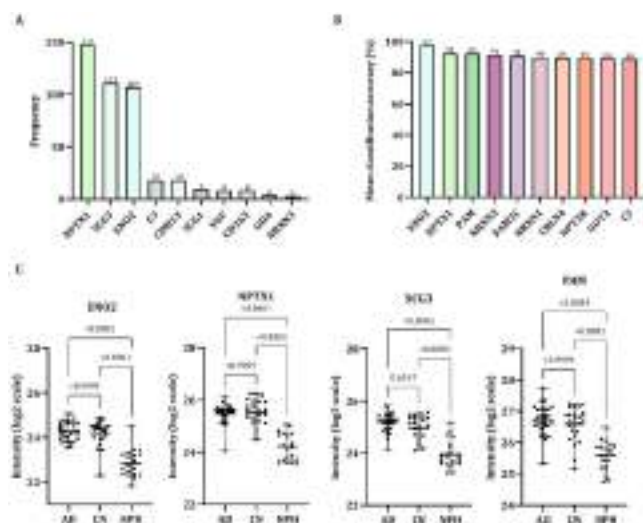


FIGURE 2 Individual and complementary proteins identified with feature selection algorithms as potential diagnostic biomarkers.

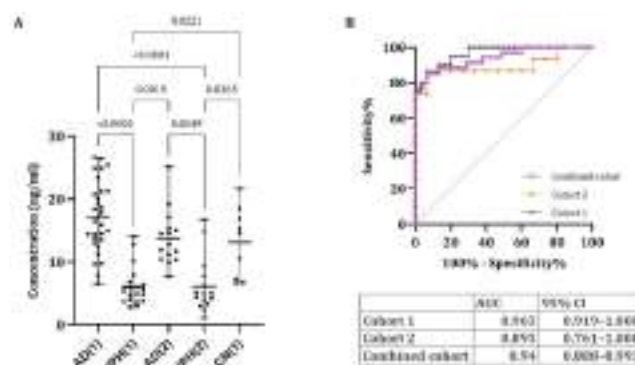


FIGURE 3 Diagnostic performance of NPTX1 as a potential biomarker for NPH.

EPO-0010 | CAA in Alzheimer's disease: What does CSF A β 40 really tell us?

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Background and aims: Cerebral amyloid angiopathy (CAA) commonly co-occurs with Alzheimer's disease (AD), reflecting shared amyloid- β (A β) pathology. Definitive CAA diagnosis requires histopathological confirmation, while in vivo evaluation uses Boston criteria. Cerebrospinal fluid (CSF) biomarkers, particularly A β 40, support in vivo CAA diagnosis; utility in AD populations needs further study.

Methods: We enrolled patients with AD with available MRI and CSF data. Patients were categorized using Boston criteria versions 1.0, 1.5, and 2.0, as absent, possible, and probable CAA. CSF concentrations of A β 40, A β 42, total tau, and phosphorylated tau were measured using standardized chemiluminescent immunoassays. Univariate Bayesian linear regression models were used to evaluate differences in CSF biomarkers according to CAA status and specific MRI markers.

Results: 173 patients were included in the study. CSF A β 40 and A β 42 were reduced in CAA patients when applying criteria v1.5 (posterior probabilities of reduction of 95.95% and 97.28% respectively). No significant differences between the CAA and non-CAA groups according to the v2.0 criteria (posterior probabilities of reduction of 27.4% and 33.6%). Regarding MRI markers, the reduction in CSF A β 40 and A β 42 was confined to hemorrhagic markers (HMs), associated with a decrease of -12.51% (95%CrI -22.69 to -0.78) and -14.50% (-24.12 to -3.07), respectively. No significant correlations were found between CSF biomarkers and non-hemorrhagic markers (nHMs).

Conclusion: CSF β -amyloid reduction was observed in comorbid CAA-AD patients only with v1.5 criteria; criteria v2.0 made such association improbable. This reflects the association between CAA radiological marker and A β confined to HMs, while nHMs showed no association

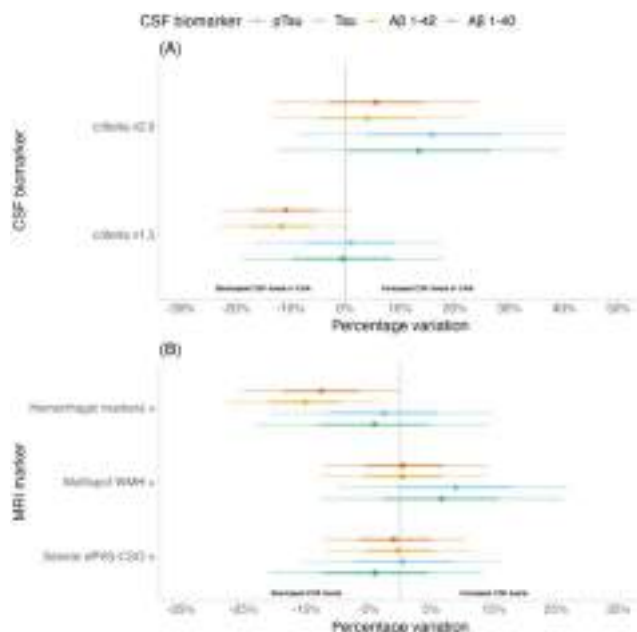


FIGURE 1 Impact of Boston criteria versions and MRI markers on CSF biomarker levels.

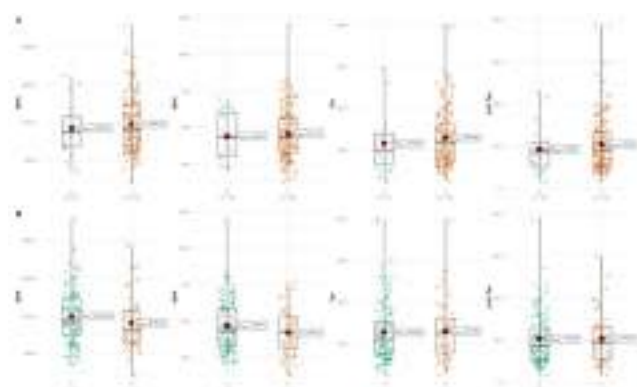


FIGURE 2 CSF biomarker levels according to CAA diagnostic status and associations with MRI markers.

Disclosure: Nothing to disclose.

EPO-0011 | Dual-task walking unmasks inter-joint coordination instability in amnesic mild cognitive impairment

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Background and aims: Gait disturbances in amnesic mild cognitive impairment (MCI) may remain subtle during normal walking and become evident only under increased cognitive load. Examining both single-task and dual-task walking may clarify how cognitive demands affect motor coordination and stability.

Methods: Older adults with amnesic MCI and cognitively normal controls performed level walking under single-task and dual-task conditions, including a cognitive task (serial subtraction) and a motor task (tray carrying). Three-dimensional kinematic data were analysed to derive phase angles and continuous relative phase, characterising hip-knee and knee-ankle inter-joint coordination. Coordination stability, pattern similarity, and temporal alignment were quantified using deviation phase, cross-correlation coefficients, and root-mean-square error across key gait phases.

Results: During single-task walking, spatiotemporal parameters and overall inter-joint coordination patterns were largely comparable between groups, with only mild increases in coordination variability in amnesic MCI. Under dual-task conditions, coordination deficits became more pronounced, particularly in knee-ankle coupling during single-limb support and swing. Individuals with amnesic MCI showed increased variability, reduced waveform similarity, and greater phase-position displacement, despite preserved gait timing. Cognitive dual-task walking induced larger effects than motor dual-task walking.

Conclusion: While basic gait patterns appear relatively preserved during single-task walking, dual-task conditions reveal impaired inter-joint coordination in amnesic MCI. Increased instability and altered temporal organisation of knee-ankle coordination under cognitive load suggest reduced adaptability of motor control. Dual-task gait assessment may provide a sensitive approach for detecting early cognitive-motor interference in pre-dementia stages.

Disclosure: This study was supported by public research funding from the Ministry of Science and Technology, Taiwan (Grant Nos. 110-2628-B-038-025). The authors declare no commercial or financial conflicts of interest.

EPO-0012 | Behavioral dysexecutive syndrome at the intersection of autoimmunity and genetically predisposed frontotemporal lobar degeneration

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Background and aims: Autoimmune encephalitis can present with slowly progressive cognitive and movement disorders mimicking neurodegenerative diseases. Emerging evidence, including immunoglobulin-like cell adhesion molecule 5 (IgLON5)-associated disease, suggests that immune-mediated inflammation may trigger or accelerate latent neurodegenerative processes in genetically predisposed individuals.

Methods: A 55-year-old male patient with gradual behavioral impairment characterized by apathy and compulsive behaviors underwent comprehensive clinical, neuropsychological and neurophysiological assessment, multimodal neuroimaging, extensive laboratory, including immunological and genetic testing. **Results:** Examination revealed frontal disinhibition, asymmetric upper-limb and truncal rigidity and REM sleep behavior disorder. Neuropsychological evaluation demonstrated executive dysfunction, mainly impaired cognitive flexibility. Neuroimaging showed frontal-predominant cortical atrophy,

electroencephalography revealed diffuse background slowing with intermittent theta activity and dopamine transporter imaging demonstrated asymmetric reduction of striatal uptake. Cerebrospinal fluid (CSF) analysis showed markedly elevated protein (330 mg/dL) without pleocytosis or intrathecal immunoglobulin synthesis. Neuronal antibody panels were negative; however, tissue-based immunofluorescence demonstrated strong neuronal reactivity in serum and CSF. Neurodegeneration biomarkers were inconsistent with Alzheimer's disease. Whole-body fluorodeoxyglucose positron emission tomography (FDG-PET) excluded occult malignancy. Genetic analysis identified a pathogenic sequestosome 1 (SQSTM1) missense mutation (Pro392Leu) associated with frontotemporal dementia and a glucocerebrosidase (GBA) variant (Asn409Ser) conferring synucleinopathy risk. To target the autoimmune inflammatory response, the patient received high-dose corticosteroids followed by plasmapheresis. Partial improvement and stabilization were observed during six months of follow-up.

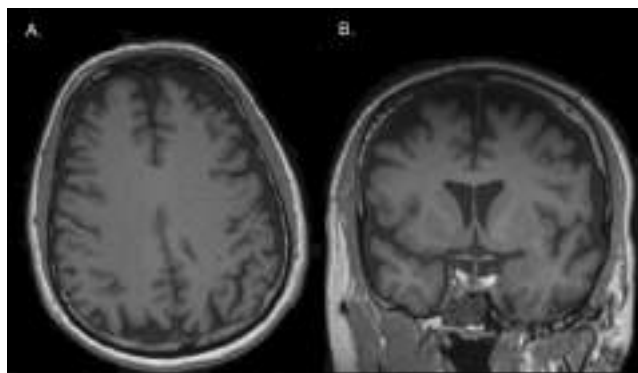


FIGURE 1 Axial (A) and coronal (B) T1-weighted brain MRI demonstrating frontal-predominant cortical atrophy.

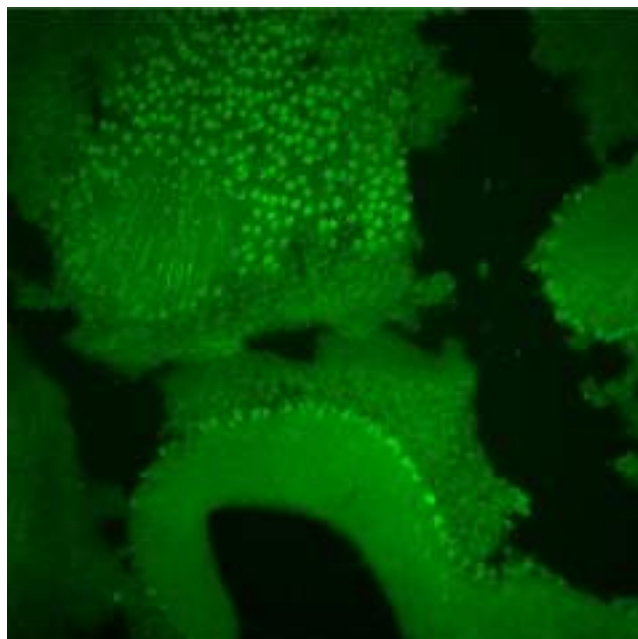


FIGURE 2 Tissue-based immunofluorescence on mice brain sections: patient serum demonstrating a strong reaction against cerebellar neurons.

Conclusion: This coexistence of neurodegeneration-linked mutations alongside inflammatory CSF findings, neuronal immunoreactivity and response to immunotherapy suggests a bidirectional interaction between autoimmune mechanisms and genetically programmed neurodegeneration, thereby blurring diagnostic boundaries and encouraging integrated diagnostic strategies.

Disclosure: Nothing to disclose.

Autonomic Nervous System Diseases

EPO-0013 | When symptoms outweigh criteria:

Dizziness and autonomic burden across the orthostatic intolerance spectrum

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Background and aims: Orthostatic dizziness is common, yet symptoms may not match hemodynamic response during the active stand-test. This study aimed to assess whether dizziness in daily life (DDL) and during orthostatic testing (DOT) is associated with autonomic symptom burden and dizziness-related disability.

Methods: In this cross-sectional study, 21 inpatients at the Diomid Gherman Institute of Neurology and Neurosurgery underwent orthostatic testing, with blood pressure and heart rate assessed supine and serially after standing (at 0–10 minutes). Orthostatic intolerance (OI) spectrum phenotype: orthostatic hypotension (OH), orthostatic hypertension (OHT), POTS, OI. Patient-reported outcomes: DHI, COMPASS-31 (OI domain), BDI. Descriptive and inferential statistics were used.

Results: The cohort (mean age 67.9 ± 11.4 years; 61.9% male) reported frequent DDL (66.7%; provoked 64.3%, episodic 71.4%). Most had cerebral small vessel disease (81%); no χ^2 association with DDL or DOT ($p > 0.05$). OI predominated (52.4%), whereas classical orthostatic phenotypes were less common (OH 19.0%, OHT 14.3%, POTS 4.8%); 9.5% had normal responses. Daily-life dizziness was associated with higher total COMPASS-31 (33.56[16.67–51.15] vs 11.27[1.78–22.55], $p < 0.001$), OI domain scores (20[10–28] vs 0[0–8], $p < 0.001$), yet showed no significant association with DOT ($p > 0.05$). DHI did not differ across OI spectrum phenotypes ($p = 0.571$), but correlated strongly with total COMPASS-31 ($\rho = 0.811$, $p < 0.001$), COMPASS-31-OI ($\rho = 0.533$, $p = 0.05$) and BDI ($\rho = 0.686$, $p = 0.007$).

Conclusion: Symptoms outweighed hemodynamic criteria, as DDL tracked autonomic and depressive burden rather than orthostatic phenotype, supporting symptom-led stratification and repeat or extended autonomic evaluation.

Disclosure: Nothing to disclose.

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Background and aims: Autonomic nervous system dysfunction has been linked to outcomes following intracerebral hemorrhage (ICH). Heart rate entropy (HRE) reflects autonomic complexity and may provide prognostic information. This study aimed to assess the association between HRE and mortality after ICH and to compare its predictive value with established autonomic markers and the ICH score.

Methods: This prospective study was conducted at Al-Azhar University Hospitals and Cairo Fatemic Hospital and included 110 consecutive patients with spontaneous ICH admitted between October 2023 and November 2024. Sample HRE, heart rate variability parameters, and baroreflex sensitivity were assessed early after admission. Hematoma volume, intraventricular extension, infratentorial location, level of consciousness, and age were integrated into the standard ICH score. Predictive performance for mortality was evaluated using receiver operating characteristic (ROC) curve analysis and multivariable logistic regression.

Results: Among the studied patients (mean age 60.8 ± 11.2 years), HRE demonstrated the highest accuracy for mortality prediction (AUC 0.88), outperforming individual autonomic markers and showing comparable performance to the ICH score (AUC 0.84). Both HRE and ICH score were independently associated with mortality. Combining HRE with the ICH score significantly improved prognostic accuracy, yielding an AUC of 0.94

Conclusion: Heart rate entropy provides substantial prognostic information regarding mortality after ICH and performs comparably to the ICH score. Incorporating HRE into conventional clinical scoring systems may enhance mortality prediction following intracerebral hemorrhage.

Disclosure: Nothing to disclose.

TABLE 1 Baseline characteristics of ICH patients (n = 110).

Variable	Value
Total patients	110
Age (years), mean $\pm$ SD	60.8 ± 11.2
Male sex	67 (60.9%)
Median hematoma volume (mL)	36 (IQR 24–52)
Intraventricular hemorrhage	41 (37.3%)
Infratentorial hemorrhage	29 (26.4%)
Impaired consciousness on admission	58 (52.7%)

TABLE 2 Predictive performance of autonomic markers for mortality.

Predictor	AUC for mortality
Heart rate entropy (HRE)	0.88
ICH score	0.84
Normalized LF power	0.77
LF/HF ratio	0.75
Normalized HF power	0.73
Baroreflex sensitivity (BRS)	0.71

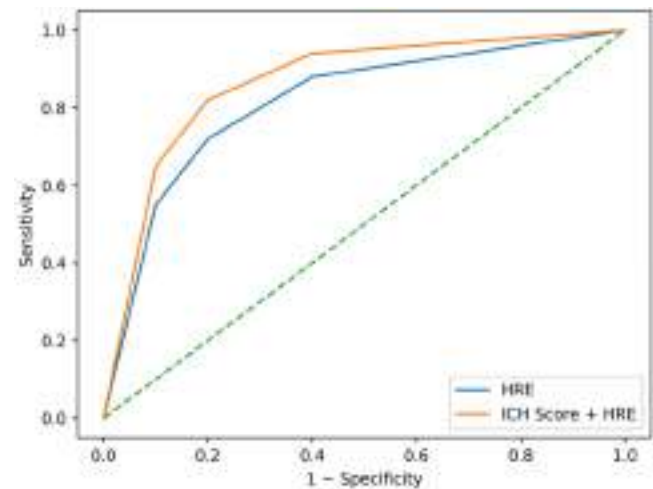


FIGURE 1 ROC curves for mortality prediction after ICH.

EPO-0015 | Characterization and staging of cardiovascular autonomic failure in light-chain amyloidosis

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Background and aims: Light-chain amyloidosis (AL amyloidosis) is a systemic disorder characterized by the deposition of monoclonal light-chain as amyloid fibrils, leading to multiorgan dysfunction. Cardiovascular autonomic failure (cvAF) represents a major contributor to morbidity, yet its natural history and staging remain poorly characterized.

Methods: This retrospective study included 38 patients with AL amyloidosis who underwent comprehensive autonomic testing. cvAF was classified as stage 1 when mild parasympathetic and/or sympathetic impairment was present without neurogenic orthostatic hypotension (nOH), and as stage 2 when both domains were affected with nOH.

Results: At onset, about one-third of patients reported autonomic symptoms. At the time of testing (median disease duration = 3 years), nearly all showed evidence of cvAF. Half were classified as stage 1, with predominant parasympathetic involvement, and 47% as stage 2. Stage 2 patients more frequently exhibited gastrointestinal ($p=0.032$) and urogenital symptoms ($p=0.004$), and impaired norepinephrine response during head-up tilt ($p<0.001$). Mortality rate and overall survival did not differ between stages. Patients with autonomic onset were more likely to have developed somatic polyneuropathy ($p=0.036$), nOH ($p=0.012$), and higher orthostatic intolerance ratios at T1 ($p=0.001$). Autonomic onset was independently associated with progression to stage 2 cvAF (OR = 7.600, 95% CI 1.373–42.063, $p=0.020$).

Conclusion: Cardiovascular autonomic failure is a prevalent and impactful manifestation of AL amyloidosis, often presenting early and progressing with the disease course. Our findings highlight the importance of systematic autonomic assessment and increased awareness of management strategies.

Disclosure: Nothing to disclose.

EPO-0016 | The burden of neurogenic orthostatic hypotension across autonomic disorders: Real-world management outcomes

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Background and aims: Neurogenic orthostatic hypotension (nOH) is a major cause of morbidity in autonomic failure, associated with falls, hospitalisations, and reduced quality of life, yet remains challenging to manage. We conducted a prospective real-world study to evaluate symptom burden, quality of life, and treatment response in patients with established nOH across all aetiologies.

Methods: Patients with established nOH were prospectively followed within a tertiary referral centre management programme. All patients attended a dedicated clinic, where non-pharmacological measures were implemented, and individualised combinations of anti-hypotensive medications were optimised. Outcomes included validated patient-reported measures of symptom burden and quality of life (Orthostatic Hypotension Questionnaire, OHQ; Patient Global Impression, PGI), alongside prospective recording of falls and hospital admissions. Orthostatic intolerance severity was assessed using standardised autonomic testing, including head-up tilt, active stand, and 24-hour blood pressure monitoring. Data are presented as mean $\pm$ SD.

Results: A total of 142 patients were enrolled (10 Lewy body dementia, 23 multiple system atrophy, 35 Parkinson's Disease, 62 pure autonomic failure, 12 autonomic neuropathy). Mean falls and hospital admissions were 1.6 ± 4.8 and 0.5 ± 2.6 respectively. OHQ composite-score was 5.9 ± 2.0 , and PGI score was 4.6 ± 1.5 .

Conclusion: In real-world tertiary-centre cohort with long-term follow-up, patients with autonomic failure and nOH across all aetiologies experience high symptom burden, frequent falls, and impaired quality of life despite individualised combination therapy. These findings highlight nOH as a major contributor to morbidity, demonstrating limitations of existing management strategies.

Disclosure: Nothing to disclose.

EPO-0017 | Can SUDOSCAN replace QSART in the composite autonomic severity scale for evaluating sudomotor function in people with multiple sclerosis?

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Background and aims: The Quantitative Sudomotor Axon Reflex Test (QSART) is the gold standard for evaluating sudomotor function within the Composite Autonomic Scoring Scale (CASS). SUDOSCAN, a quick and noninvasive electrochemical test of sweat gland function, has been suggested as a potential substitute. This study investigated whether SUDOSCAN-derived indices could replace QSART within the CASS framework and provide reliable sudomotor evaluation in people with multiple sclerosis (pwMS).

Methods: This cross-sectional study involved 99 pwMS. Subjective autonomic symptoms were evaluated using the Composite Autonomic Symptom Score-31 (COMPASS-31), while objective autonomic testing was conducted with the CASS, which includes QSART for sudomotor assessment. Additionally, all pwMS underwent SUDOSCAN testing, and a modified CASS score was derived based on adrenergic, cardiovagal, and SUDOSCAN-based sudomotor measures.

Results: A strong correlation was observed between CASS and modified CASS ($r=0.795$, $p<0.001$). However, no correlation was found between QSART-based and SUDOSCAN-based sudomotor indices ($r=0.043$, $p=0.672$). CASS did not correlate with COMPASS-31 or its subdomains, whereas modified CASS showed a correlation with the gastrointestinal domain of COMPASS-31 ($r=0.228$, $p=0.027$). Additionally, modified CASS correlated with age ($r=0.204$, $p=0.048$) and the Expanded Disability Status Scale (EDSS) score ($r=0.207$, $p=0.043$).

Conclusion: The modified CASS including SUDOSCAN showed a strong overall correlation with the standard CASS and meaningful associations with age and disability level. Although SUDOSCAN did not correlate with QSART-based sudomotor indices, it could serve as a helpful supplement when QSART is unavailable. More long-term studies are needed to assess its effectiveness in monitoring sudomotor dysfunction over time.

Disclosure: Funding: Croatian Science Foundation (IP-2022-10-8200) KT: Nothing to disclose. MS: Nothing to disclose. KJ: Nothing to disclose. MKS: Received consultation and/or speaker fees from Biogen, Novartis, Roche. MH: Received consultation and/or speaker fees from Biogen, Merck, Novartis, Roche, Astra Zeneca, Amgen.

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Background and aims: Autonomic failure is a common feature in hereditary transthyretin amyloidosis (ATTRv), but has not been systematically described in other genetic subtypes including gelsolin (AGel), apolipoprotein A1 (AApoA1), and fibrinogen A α -chain amyloidosis (AFib). This study aimed to quantitatively investigate cardiovascular autonomic dysfunction in these rare entities, and to compare cardiovascular autonomic dysfunction to patients with ATTRv.

Methods: This retrospective study enrolled patients with genetically confirmed diagnosis of AGel, AApoA1, and AFib. Age- and sex-matched control patients with ATTRv were included. All participants underwent standardised cardiovascular autonomic function testing (AFT), a subgroup had ambulatory 24h blood pressure monitoring (ABPM) with autonomic patient diary. Stages of autonomic failure were defined according to AFT results: stage 0 (normal AFT), stage I (mild parasympathetic and/or sympathetic impairment), stage II (neurogenic orthostatic hypotension (nOH)).

Results: A total of 12 patients were included. Overall, 9/11 (82%) patients had abnormal AFT. 8/11 (73%) presented with stage I, and only 1/11 (9%) patient with AApoA1 had nOH. In the matched ATTRv cohort, 4/12 (33%) presented with nOH. There were no significant differences in cardiovascular autonomic parameters when comparing ATTRv versus non-ATTRv patients. ABPM was available in two patients: one individual with AFib (stage I) had normal circadian BP rhythm, while one patient with AApoA1 (stage II) was a reverse dipper.

Conclusion: Cardiovascular autonomic dysfunction is very prevalent in genetic non-TTR amyloidosis, but is usually mild. Quantitative AFT could help to monitor disease progression. Prospective studies are needed to investigate dysfunction of other autonomic domains in these patients.

Disclosure: LS has been supported by the Universität Basel, Switzerland, and the Freiwillige Akademische Gesellschaft Switzerland. GC, MMR and CJM: nothing to disclose. VI is supported by the National Institute for Health Research, University College London Hospitals Biomedical Research Centre and by the Autonomic Charitable Trust (ACT) (Lord Bagri) Research

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EPO-0019 | Mucosal denervation and markers of immune activation in the gut of patients with chronic inflammatory diseases

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Background and aims: Chronic intestinal inflammation is a condition characterized by immune activation, increased epithelial permeability, and altered neuro-immune interactions. These features make it a suitable model to investigate mechanisms of mucosal denervation and impaired interoceptive signaling within the gut-brain axis, implicated in neuropsychiatric conditions, including opioid use disorder (UOD).

Methods: To address this objective, we analyzed ileal biopsies from 10 patients with chronic inflammatory conditions (4M/6F; 47.3 $\pm$ 8.6) and matched healthy controls (5M/5F; 55.2 $\pm$ 10.9). Using indirect immunofluorescence, immune activation was assessed by quantifying CD68⁺ macrophages, IBA-1⁺ activated myeloid cells, CD8⁺ lymphocytes, and IL-24-expressing cells, while mucosal innervation was evaluated by measuring VIP⁺ enteric fibers.

Results: Compared to controls, patients with inflammatory conditions showed a marked increase in CD68⁺ macrophages (755.0 $\pm$ 338.1 vs 141.1 $\pm$ 70.5; p < 0.01), IBA-1⁺ myeloid cells (862.3 $\pm$ 257.7 vs 239.9 $\pm$ 116.4; p < 0.01), and IL-24-expressing cells (339.7 $\pm$ 151.4 vs 32.0 $\pm$ 44.6; p < 0.01). CD8⁺ lymphocytes were also increased, although the difference did not reach statistical significance (1608.6 $\pm$ 568.6 vs 1391.6 $\pm$ 392.3; p = 0.504). Concomitantly, a significant reduction in VIP-positive enteric fibers was observed in inflammatory conditions compared to controls (0.036 $\pm$ 0.025 vs 0.069 $\pm$ 0.025; p < 0.01).

Conclusion: Overall, the findings reveal a distinct neuro-immune signature characterized by chronic inflammation and enteric denervation. This pattern may underlie impaired interoceptive signaling and disrupted gut-brain axis communication in inflammatory bowel conditions, with potential relevance to neuropsychiatric disorders such as opioid use disorder.

Disclosure: Nothing to disclose.

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Background and aims: Gastrointestinal autonomic symptoms are frequently reported by people with MS (pwMS) but remain insufficiently characterized, with prevalence estimates varying widely across studies. The aim of this study was to conduct a systematic review with meta-analysis of the available scientific evidence on the prevalence of gastrointestinal autonomic symptoms in people with multiple sclerosis.

Methods: A systematic review of the literature (PubMed, Scopus, and Web of Science until October 2025) identified 21693 studies, of which 15 reported frequency of gastrointestinal autonomic symptoms in pwMS compared to healthy controls. Of these, 5 evaluated dysphagia, 5 evaluated bowel dysfunction in general, and a single study evaluated constipation, fecal incontinence, abnormal gastric emptying, dyspepsia, anorectal dysfunction, and intestinal symptoms.

Results: Meta-analysis showed a significantly higher prevalence of gastrointestinal autonomic symptoms in pwMS than in healthy controls (0.283, IQR 0.026–0.715 in pwMS vs. 0.022, IQR 0.000–0.185 in healthy controls; OR 3.40, 95% CI 1.79–6.44; $p=0.0002$) (Figure 1). Substantial heterogeneity was observed ($I^2=99\%$, $\tau^2=1.37$). Among symptoms with more than one study, the prevalence of dysphagia was significantly higher (OR 20.65, 95% CI 3.20–133.34; $p=0.001$), whereas the prevalence of bowel dysfunction did not differ (OR 1.21, 95% CI 0.65–2.24; $p=0.55$).

Conclusion: Gastrointestinal autonomic symptoms are significantly more frequent in people with multiple sclerosis than in healthy controls, with dysphagia showing a particularly strong association. Greater awareness and systematic screening of gastrointestinal autonomic symptoms are warranted in clinical

practice to improve recognition and management of these common and impactful manifestations of MS.

Disclosure: MS: Nothing to disclose. CH: Consultant for Coloplast, Convatec, Bbraun. IA: Received consultation and/or speaker fees from Biogen, Merck, Novartis, Roche. CC: Nothing to disclose. MKS: Received consultation and/or speaker fees from Biogen, Novartis, Roche, Merck. MH: Received consultation and/or speaker fees from Biogen, Merck, Novartis, Roche, Astra Zeneca, Amgen.

EPO-0021 | Developing a pain scale for multiple system atrophy (MSA)

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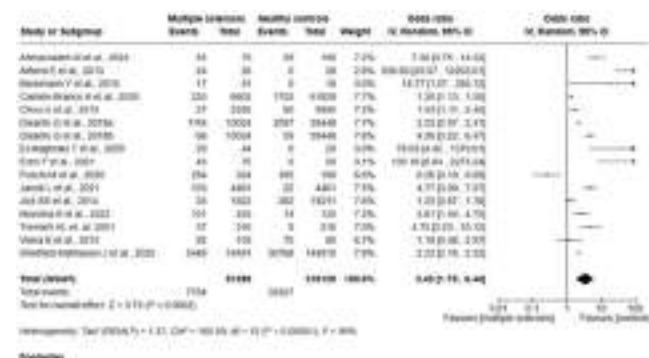


FIGURE 1 Forest plot showing the pooled odds ratio for gastrointestinal autonomic symptoms in people with multiple sclerosis compared with healthy controls.

Background and aims: Pain affects up to 87% of persons with multiple system atrophy (MSA), impacting core activities of daily living, but remains under-recognized in clinical practice. In the absence of disease-specific pain assessment tools, we aim at developing a patient-reported pain scale for MSA.

Methods: A taskforce of movement disorder and pain experts will develop a first draft of the MSA-pain scale. The scale development will be informed by a systematic literature review

and by the results of targeted surveys addressing patients, caregivers, and healthcare professionals (ongoing) on current standards and unmet needs on pain assessment and management in MSA. The questionnaire will be developed in English and undergo a preliminary field test ($n = 5$ English-speaking persons with MSA) with subsequent item reduction and refinement. The Delphi methodology will be followed to reach consensus on the final version of the questionnaire.

Results: The questionnaire development is expected to last 12 months. Once finalized, the final version of the questionnaire will be translated into other languages (German, Italian, French) and validated in a large multi-center study.

Conclusion: The MSA-pain questionnaire is expected to assist in the screening, systematic assessment and follow-up of different types of pain in MSA. The validation of the first disease-specific patient reported outcome measure (PROM) for pain in MSA will also hopefully pave the way for interventional trials targeting pain in MSA.

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EPO-0022 | Increased infarct volume is associated with a shift towards cardiac sympathetic predominance in patients with ischemic stroke

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Background and aims: Ischemic stroke often causes cardiovascular autonomic dysfunction. We previously showed associations between higher National Institutes of Health Stroke Scale scores and more severe cardiovascular autonomic dysfunction. This study evaluated associations between infarct-volume and parameters of cardiac autonomic modulation in patients with acute ischemic stroke in the middle cerebral artery (MCA) territory.

Methods: In 48 patients with acute MCA stroke (68 ± 15 years old, 23 men and 25 women), we monitored RR intervals (RRI), systolic, diastolic blood pressures (BPsys, BPdia), and respiration (RESP) at rest within 24 hours after stroke-onset. We calculated parameters of total cardiac autonomic modulation (RRI-standard-deviation (RRI-SD), RRI-coefficient-of-variation (RRI-CV), RRI-total-powers), sympathetic (RRI-low-frequency-powers (RRI-LF), normalized (nu) RRI-LF-powers, BPsys-LF-powers) and parasympathetic modulation (root-mean-square-of-successive-RRI-differences (RMSSD), RRI-high-frequency-powers (RRI-HF), RRI-HFnu-powers), sympathetic-parasympathetic balance (RRI-LF/HF-ratios), and baroreflex-sensitivity (BRS). The infarct-volume was calculated

using the Ellipsoid model ABC/2 (where A = longest dimension in axis x, B = longest perpendicular dimension to axis x (y), and C = total length in z dimension) from the diffusion-weighted Magnetic Resonance image (DWI) within 24 hours upon stroke-onset or Computed Tomography (CT) taken within 48 hours upon stroke-onset.

Results: Values of infarct-volumes correlated positively with RRI-LF-nu-powers (correlation-coefficient: 0.297, $p = 0.038$) and RRI-LF/HF-ratios (correlation-coefficient: 0.414, $p = 0.003$), negatively with RRI-HFnu-powers (correlation-coefficient: -0.297 , $p = 0.038$). Patients with infarct-volumes above 50 mL had higher RRI-LF-nu-powers and RRI-LF/HF-ratios but lower RRI-HF-nu-powers than patients with smaller infarct-volumes.

Conclusion: In our patients with acute ischemic MCA-stroke, increased infarct-volume was associated with a shift towards increased sympathetic and reduced parasympathetic cardiac modulation. These changes bear an increased risk of secondary complications.

Disclosure: Nothing to disclose.

EPO-0023 | Lower urinary tract symptoms in Parkinson's disease: Prevalence and aetiology

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Background and aims: Lower urinary tract symptoms (LUTS) are common autonomic manifestations of Parkinson's disease (PD), frequently preceding or accompanying motor progression and significantly impairing quality of life. However, the relationship between LUTS, motor severity, and disease characteristics remains incompletely defined. The aim of this study was to assess the prevalence and pattern of LUTS in PD and to explore their associations with motor severity, disease duration, and quality of life.

Methods: We conducted a cross-sectional observational study including 153 patients with idiopathic PD (mean age 67.4 ± 8.9 years, disease duration 6.1 ± 4.2 years, 58% male). LUTS were evaluated using the Overactive Bladder Questionnaire short form (OAB-qSF) and the International Prostate Symptom Score (IPSS). Quality of life was assessed using disease-specific patient-reported measures. Correlation and multivariate linear regression analysis were performed.

Results: Overall, 107 patients (69.93%) reported at least one LUTS. Storage symptoms predominated (66.35%) with nocturia (61.68%) and urgency (48.59%) being most frequent. Mean OAB-q symptoms score was 38.7 ± 17.4 , and mean IPSS total score was 12.4 ± 6.3 . OAB-q scores correlated significantly with UPDRS-III ($r = 0.35$, $p < 0.001$), H&Y stage ($r = 0.33$, $p < 0.001$), age ($r = 0.21$, $p = 0.001$), and disease duration. In multivariate analysis, UPDRS-III and age independently predicted LUTS severity. Higher LUTS burden was independently associated with poorer quality of life ($p < 0.001$).

Conclusion: LUTS are highly prevalent in PD, with storage symptoms predominating, and are strongly associated with motor severity and disease progression.

Disclosure: Nothing to disclose.

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Background and aims: While normal metaiodobenzylguanidine (MIBG) uptake on cardiac scintigraphy and sweat function on dynamic sweat test (DST) in pure autonomic failure (PAF) are predictors of phenoconversion to multiple system atrophy (MSA) over parkinson's disease (PD) or dementia with lewy bodies (DLB), they can become abnormal in late-stage MSA, consistent with a top-down model of disease. However, no study has evaluated the sequence of peripheral denervation across cardiovascular and sudomotor domains in MSA. We examined sweat loss in patients with normal versus abnormal MIBG uptake and considered implications for predicting phenoconversion trajectory.

Methods: As part of our ongoing natural history study, consecutive patients with an initial diagnosis of PAF were systematically phenotyped and followed longitudinally until phenoconversion or death. Participants underwent cardiovascular autonomic testing, cardiac MIBG scintigraphy, dynamic sweat testing (DST), and thermoregulatory sweat testing (TST).

Results: 24 PAF patients (median follow up 5.5 years) underwent both scintigraphy and sudomotor testing. 4/24 patients (17%) had normal MIBG uptake, of whom 1/4(25%) had pre-ganglionic sweat loss. This patient and 2 others (75%) later phenoconverted into MSA, while the remaining patient retained a PAF diagnosis. Among the 20/24 (83%) patients with abnormal MIBG uptake, all 20/20 (100%) had evidence of post-ganglionic sudomotor dysfunction and retained a PAF diagnosis or phenoconverted to PD/DLB.

Conclusion: In PAF patients phenoconverting to MSA, cardiac sympathetic innervation may remain preserved even in established peripheral sudomotor denervation, possibly reflecting differing neuronal vulnerability to alpha-synuclein deposition. These findings support a multimodal biological framework for

PAF to improve prediction of phenoconversion trajectory across disease stages.
Disclosure: Nothing to disclose.

Cerebrovascular Diseases 1

EPO-0025 | Ethical barriers to time-critical stroke care: Family consent, delayed treatment, and the need for presumed consent in Armenia

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Background and aims: Acute ischemic stroke is a leading cause of mortality and long-term disability worldwide. Intravenous thrombolysis and mechanical thrombectomy are evidence-based, time-critical interventions that significantly improve survival and functional outcomes. Despite their established efficacy, ethical-legal barriers may impede timely treatment in certain healthcare systems. This study examines the ethical implications of consent requirements in acute stroke care, comparing European practice with the current regulatory framework in Armenia.

Methods: A comparative ethical and policy analysis was conducted, reviewing European stroke guidelines and ethical frameworks (ESO, NICE) alongside Armenian Ministry of Health regulations governing informed consent in neurological emergencies.

Results: In most European countries, acute stroke treatment is guided by the principle of presumed consent, allowing immediate initiation of life-saving therapy when patients lack decision-making capacity and no prior advance directive / living will is documented. Family members serve an advisory rather than decisional role. In contrast, Armenian practice permits family members to refuse thrombolysis and thrombectomy even when the patient is conscious, and mandates written family consent when treatment is approved. This requirement, formally regulated at the ministerial level, introduces critical delays and contradicts the ethical principles of beneficence and non-maleficence by effectively transforming time-dependent care into a preventable source of harm.

Conclusion: Current consent practices in Armenia represent a significant ethical and clinical barrier to optimal stroke care. Addressing this issue requires legislative reform, implementation of presumed consent for emergency stroke treatment, and enhanced legal protection for physicians to ensure timely, ethical, and evidence-based intervention.

Disclosure: Nothing to disclose.

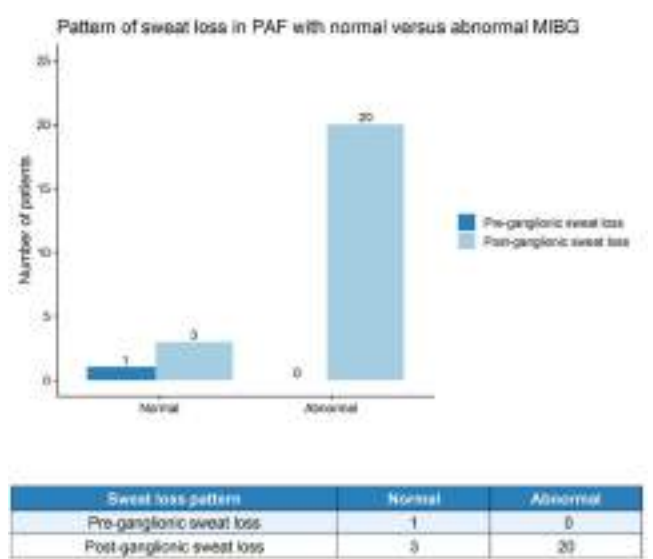


FIGURE 1 Pattern of sweat loss in normal vs abnormal MIBG.

TABLE 1 Comparison of demographics and clinical characteristics of patients with mRS 0–2 vs patients with mRS 3–6 at 3 months. Data are presented as medians (IQR) and frequencies.

	mRS 0–2 at 3 months n = 403	mRS 3–6 at 3 months n = 246	Sig.
Demographics			
Age (years) [median (IQR)]	74 (63–83)	80 (73–87)	<0.001
Females (n (%))	169 (44)	182 (55)	0.004
STROKE			
Comorbidities (n (%))			
Hypertension	112 (11)	281 (81)	0.000
Diabetes mellitus	36 (20)	91 (26)	0.036
Hypocholeraemia	257 (60)	186 (54)	0.100
Chronic heart failure	76 (8)	10 (3)	0.002
Coronary artery disease	10 (11.8)	62 (18)	0.013
peripheral vascular disease	80 (21)	77 (22)	0.760
Atrial fibrillation	116 (27)	147 (42)	<0.000
Stroke status	75 (17)	87 (26)	0.732
Previous cerebrovascular event	64 (15)	59 (17)	0.405
Brain failure	44 (10)	56 (16)	0.013
Stroke severity [median (IQR)]			
NIHSS at baseline	4 (2–7)	12 (6–19)	<0.000
Rankin at baseline	0 (0–0)	0 (0–2)	<0.000
Stroke (n (%))			
Ischaemic	160 (99)	223 (79)	<0.000
Haemorrhagic	46 (11)	73 (21)	<0.000
Type of intervention (n (%))			
Reperfusion treatment	232 (60)	170 (52)	0.151
Thrombolysis (IVT)	176 (44)	10 (3)	<0.000
Thrombectomy (MT)	7 (2)	22 (6)	<0.000
IVT + MT	47 (11)	18 (5)	0.013
Clinical outcomes [median (IQR)]			
Intrahospital mortality (n (%))	0 (0)	28 (9)	<0.000
NIHSS at discharge	1 (0–2)	19 (4–19)	<0.000
Rankin at discharge	1 (0–2)	5 (4–7)	<0.000
mRS at 3 months	1 (0–1)	4.5 (3–6)	<0.000
EPILEPSY			
Early seizures and status epilepticus			
Early seizures (n (%))	13 (3)	27 (8)	0.003
Type of seizure (n (%))			
Partial	7 (54)	16 (59)	
Generalized	5 (38)	11 (41)	
Partial to generalized	1 (8)	0 (0)	
Days seizure status (median (IQR))	1 (0–2)	1 (0–2)	0.002
Early status epilepticus (n (%))	3 (6.7)	14 (21)	
Type of SE			
Convulsive	0 (0)	2 (11)	
Non convulsive	3 (16.6)	12 (88)	
Days (seizure status) [median (IQR)]	2 (1.5–4)	2 (1–4)	
Drug treatment (n (%))			
Stroke	161 (99)	229 (77)	<0.000
Pre-stroke stroke	124 (29)	119 (34)	0.095
Post-stroke stroke	157 (60)	129 (40)	<0.000

TABLE 2 Logistic multivariate regression for variables associated with poor functional outcome in terms of mRS 3–6 at 3 months.

Variables	OR	CI - Lower	CI - Upper	Sig.
Age	1.006	1.004	1.009	<0.001
NIHSS at admission	1.056	1.032	1.081	<0.001
Haemorrhagic stroke	1.391	1.022	1.774	0.014
ES/SE	1.342	1.021	1.777	0.036
Statin treatment	0.879	0.813	0.952	0.001

clinical predictors of unfavorable functional outcomes, defined as a modified Rankin Scale (mRS) score of 3–6 at 3 months, with a focus on the potential impact of statin therapy.

Methods: The clinical data of 777 consecutive patients with acute stroke were retrospectively analyzed. Patients were stratified with favorable outcomes defined as an mRS score of 0–2 and unfavorable outcomes (mRS score of 3–6). Univariate and multivariate logistic regression analyses were then performed to identify variables independently associated with poor clinical outcomes.

Results: Patients in the mRS 3–6 group were older on average (80 vs. 74 years, $p < 0.001$) and had a significantly higher median NIHSS score at admission compared to those in the mRS 0–2 group (12 vs. 4, $p < 0.001$). Multivariate analysis showed that older age (OR=1.006, $p < 0.001$), higher NIHSS scores at admission (OR=1.036, $p < 0.001$), haemorrhagic stroke (OR=1.101, $p = 0.014$), and the occurrence of early seizures or status epilepticus (OR=1.142, $p = 0.020$) were independently associated with a

poor functional outcome (mRS 3–6). Conversely, statin therapy was associated with a reduced risk of poor outcome (OR=0.879, $p = 0.001$).

Conclusion: In a consecutive cohort of patients with acute stroke, statin therapy emerged as an independent protective factor against poor functional outcome at three months, highlighting its potential pleiotropic effects even in patients with haemorrhagic stroke. Further studies are warranted to elucidate the underlying pharmacological mechanisms.

Disclosure: Nothing to disclose.

EPO-0028 | Impact of statin therapy on post-stroke epilepsy (PSE): An emerging anti-epileptogenic strategy

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Background and aims: Growing evidence support the hypothesis that statin therapy in patients with acute stroke may reduce

TABLE 1 Comparison of demographics and clinical characteristics of patients with PSE vs patients without PSE. Data are presented as medians (IQR) and frequencies.

	No PSE n = 729	PSE n = 48	Sig.
Demographics			
Age (years) [median (IQR)]	77 (67–83)	78 (63–75–81–25)	0.400
Females (n (%))	181 (48)	29 (60)	0.181
STROKE			
Comorbidities (n (%))			
Hypertension	336 (76)	40 (81)	0.142
Diabetes mellitus	161 (22)	14 (29)	0.234
Hypocholeraemia	603 (77)	28 (58)	0.071
Chronic heart failure	87 (12)	2 (4)	0.182
Coronary artery disease	186 (15)	6 (12)	0.099
peripheral vascular disease	159 (22)	19 (21)	0.677
Atrial fibrillation	241 (33)	29 (42)	0.238
Stroke status	123 (17)	7 (15)	0.400
Previous cerebrovascular event	127 (16)	6 (12)	0.506
Brain failure	94 (13)	6 (12)	0.940
Stroke severity [median (IQR)]			
NIHSS at baseline	4 (3–12)	12 (3.75–18.5)	<0.001
Rankin at baseline	0 (0–0)	0 (0–0)	0.781
Type of Stroke (n (%))			
Ischaemic	626 (85)	38 (81)	0.681
Haemorrhagic	103 (15)	9 (19)	0.681
Type of intervention (n (%))			
Reperfusion treatment	376 (60)	20 (60)	0.771
Thrombolysis (IVT)	238 (41)	9 (23)	0.009
Thrombectomy (MT)	24 (4)	5 (13)	0.013
IVT + MT	94 (15)	12 (31)	0.008
TOAST classification (n (%))			
Cardioembolic (CE)	176 (76)	15 (38)	0.266
Large Arterial Atherosclerosis (LAA)	79 (15)	4 (10)	0.589
Small Arterial Occlusion (SAO)	131 (22)	3 (8)	0.033
Cryptogenic	194 (32)	16 (41)	0.187
Other cause	31 (5)	1 (3)	0.644
Outcomes [median (IQR)]			
Intrahospital mortality (n (%))	28 (3.8)	0 (0)	0.187
NIHSS at discharge	2 (0–7)	7 (2–16–25)	<0.001
Rankin at discharge	2 (1–5)	5 (2–5)	<0.001
mRS at 3 months	2 (1–4)	3.5 (2–5)	0.007
mRS at 3 months 0–2 (n (%))	406 (57)	16 (33)	0.001
mRS at 3 months 3–6 (n (%))	304 (42)	32 (67)	0.001
Epilepsy (n (%))			
Early seizures (ES)	36 (4)	16 (33)	<0.001
Early status epilepticus (ESE)	20 (3)	2 (4)	<0.001
Drug treatment (n (%))			
Stroke	687 (93)	37 (88)	0.013
Pre-stroke stroke	233 (32)	16 (33)	0.199
Post-stroke stroke	374 (51)	21 (44)	0.656

TABLE 2 Logistic multivariate regression for variables associated with the onset of post-stroke epilepsy.

Variables	OR	CI - Lower	CI - Upper	Sig.
NIHSS at admission	1.004	1.002	1.007	0.001
ES/ESE	1.112	1.042	1.187	<0.001
Statin treatment	0.987	0.946	0.999	0.048

the risk of post-stroke epilepsy through pleiotropic mechanisms. This study aimed to evaluate factors associated with the development of post-stroke epilepsy (PSE), with a focus on the potential role of statin therapy, in a real-world cohort of stroke patients.

Methods: Clinical and radiological data of 777 consecutive patients with acute stroke were retrospectively analyzed. Data of patient with PSE and without PSE were compared. Univariate and multivariate logistic regression analysis was conducted to identify variables associated with the development of PSE.

Results: The two groups did not differ significantly with respect to demographic characteristics or comorbidities, the median NIHSS score at admission was significantly higher in the post-stroke epilepsy (PSE) group than in the non-PSE group (12 vs. 6, $p < 0.001$). Multivariate analysis showed that higher NIHSS scores at admission (OR = 1.004, 95% CI: 1.002–1.007; $p = 0.001$) and the occurrence of early seizures or early status epilepticus (ES/ESE) (OR = 1.112, 95% CI: 1.042–1.187; $p < 0.001$) were independently associated with an increased risk of PSE, while statin therapy emerged as an independent protective factor against PSE development (OR = 0.948, 95% CI: 0.915–0.999; $p = 0.048$).

Conclusion: In a consecutive cohort of acute stroke patients, statin therapy emerged as an independent protective factor against the development of PSE, underscoring its potential role in antiepileptogenic processes.

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EPO-0029 | Early versus late starting of direct oral anticoagulants after breakthrough ischemic stroke: A target trial analysis from the ASPERA-R study

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Background and aims: Randomized trials support early initiation of direct oral anticoagulants (DOACs) after atrial fibrillation (AF)-related ischemic stroke. However, patients with breakthrough ischemic stroke occurring despite ongoing anticoagulation have been largely under-represented. We evaluated the effectiveness and safety of early versus delayed DOAC initiation in these patients.

Methods: We performed a target trial emulation comparing early versus delayed DOAC initiation in patients with AF experiencing ischemic stroke on anticoagulation from the retrospective

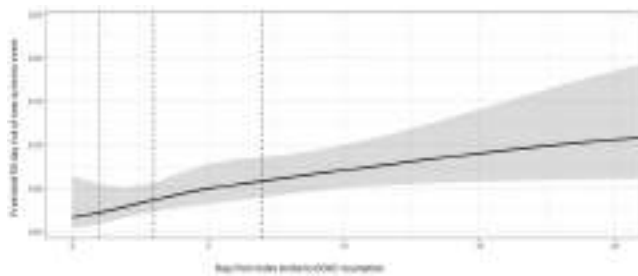


FIGURE 1 Predicted risk of new ischemic event at 90 days based on days to DOAC resumption.

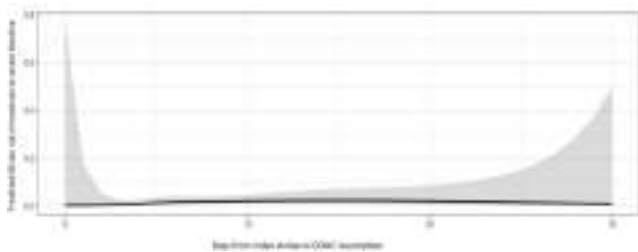


FIGURE 2 Predicted risk of moderate-severe bleeding at 90 days based on days to DOAC resumption.

arm of the international, multicentre ASPERA study. Treatment strategies were defined using severity-adapted timing thresholds based on baseline National Institutes of Health Stroke Scale (NIHSS) scores. A cloning-censoring-weighting approach with inverse probability weighting was applied to emulate random assignment and avoid immortal time bias. Primary outcomes were 90-day new ischemic events and moderate-to-severe bleeding. Risk ratios (RRs), absolute risk differences (RDs), and hazard ratios (HRs) were estimated using weighted regression and Cox models.

Results: Among 833 patients (median age 81 years), 336 were assigned to early and 497 to delayed DOAC initiation. At 90 days, early initiation was associated with a lower risk of new ischemic events (RR 0.44, 95% CI 0.21–0.90; RD –3.64%, 95% CI –6.40 to –0.87; HR 0.43, 95% CI 0.21–0.91), reduced moderate-to-severe bleeding (RR 0.10, 95% CI 0.01–0.76) and decreased all-cause and vascular mortality (FIGURE 1 and 2). A Net Early Benefit Score integrating ischemic and bleeding risks was positive across all NIHSS strata.

Conclusion: Early severity-adapted DOAC initiation was associated with lower risks of recurrent ischemic events and mortality at 90 days without increasing major bleeding.

Disclosure: AZ received consulting and speaker fees from Angels Initiative, Boehringer-Ingelheim, Alexion, Daiichi Sankyo, Pfizer, Amgen, Aurora Biopharma, and fees for Advisory Board from Bayer, Daiichi Sankyo, Astra Zeneca, and Boehringer-Ingelheim, none relevant to this work. The other authors report no conflicts.

EPO-0030 | Prophylactic induced hypertension in deep perforator infarction: Impact on early neurological deterioration and blood pressure variability

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Background and aims: Early neurological deterioration (END) is a frequent and disabling complication in acute ischemic stroke. We evaluated whether prophylactic pharmacologically induced hypertension (PPIHT) could reduce the risk of END and improve clinical outcomes in high-risk population.

Methods: Among 424 patients, 118 (27.8%) received PPIHT. After PSM, 260 patients were retained (118 PPIHT, 142 non-PPIHT). END occurred significantly less often with PPIHT. Within 3 months after stroke onset, the PPIHT group had lower mRS, lower incidence of recurrent stroke, and no increase in hemorrhagic complications. The PPIHT group had a lower incidence of END compared to patients with initial SBP ≥ 180 mmHg.

Results: Among 424 patients, 118 (27.8%) received PPIHT. After PSM, 260 patients were retained (118 PPIHT, 142 non-PPIHT). END occurred significantly less often with PPIHT. Within 3 months after stroke onset, the PPIHT group had lower mRS, lower incidence of recurrent stroke, and no increase in hemorrhagic complications. The PPIHT group had a lower incidence of END compared to patients with initial SBP ≥ 180 mmHg.

Conclusion: PPIHT was associated with a substantial reduction in END and improved functional outcomes in lacunar infarction. Compared with the patients with initial high SBP, PPIHT had better outcomes, suggesting that it is the drug-induced increase in SBP, rather than the SBP level itself, that is beneficial.

Disclosure: Nothing to disclose.

EPO-0031 | Machine Learning models for 3-month functional outcome prediction after mechanical thrombectomy in acute ischaemic stroke patients

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Background and aims: Acute ischaemic stroke (AIS) treated with mechanical thrombectomy (MT) carries variable prognosis. Early identification of patients at risk of poor prognosis is crucial for personalised management and family counseling. This study evaluates and compares the performance of various Machine Learning (ML) algorithms in predicting the 3-month functional outcome using admission clinical and laboratory data.

Methods: Data from 843 AIS patients treated with MT were analysed. The primary endpoint was poor functional outcome, defined as a modified Rankin Scale (mRS) score of 3–6 at 3 months. Predictors included demographics, admission National Institutes of Health Stroke Scale (NIHSS) score, comorbidities, laboratory parameters, and recanalisation status. Five classifiers were developed and validated: Logistic Regression (LR), Random Forest (RF), Support Vector Machine (SVM), K-Nearest Neighbors (KNN), and Multilayer Perceptron (MLP). Model performance was assessed using Accuracy, Area Under the Curve (AUC), and F1-score.

Results: LR demonstrated the best overall performance (Accuracy 0.76, AUC 0.80, F1-score 0.83). RF and SVM showed comparable results (Accuracy 0.75, AUC 0.80). Feature importance analysis in RF highlighted age and admission NIHSS score as the strongest predictors. More complex models, including MLP and KNN, achieved lower performance (Accuracy

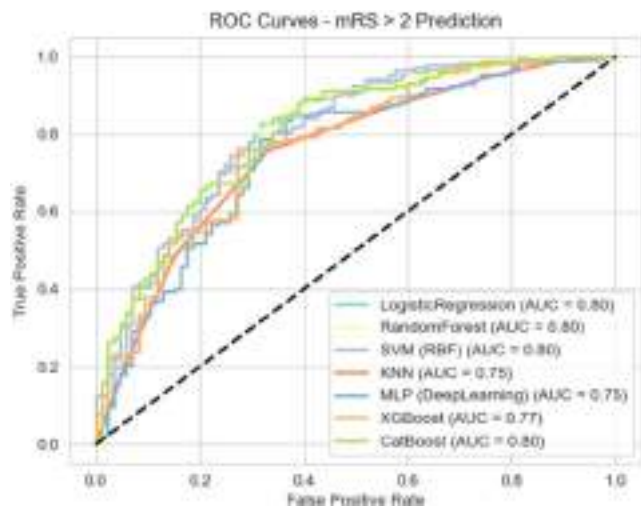


FIGURE 1 Receiver Operating Characteristic (ROC) curves comparing the performance of seven training ML models in predicting poor functional outcome (mRS > 2) at 3 months.

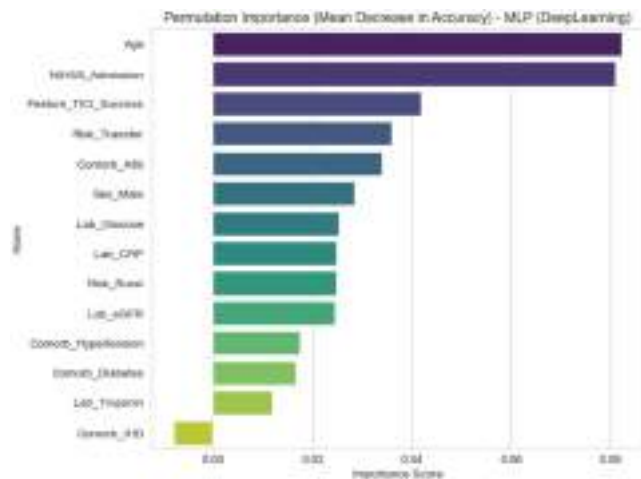


FIGURE 2 Feature importance ranking from the MLP model, showing NIHSS score on admission and age as the strongest predictors of functional outcome, followed by successful recanalisation defined by the Thrombolysis in Cerebral Infarction (TICI) score.

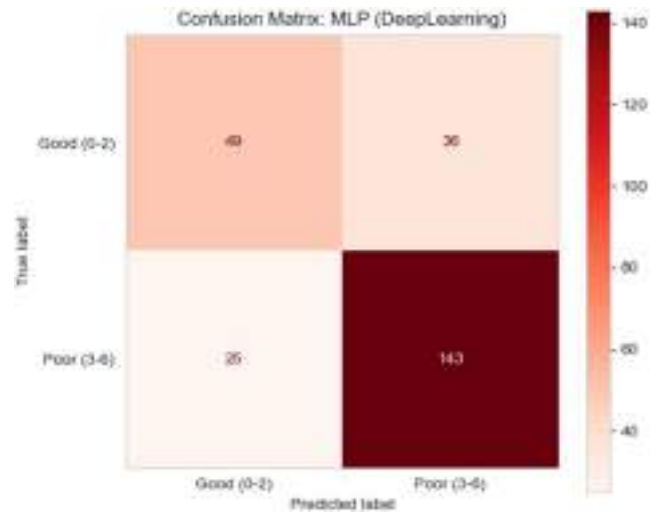


FIGURE 3 Confusion Matrix for the MLP model on the test dataset. The matrix displays the classification performance in distinguishing between good (mRS 0–2) and poor (mRS 3–6) outcomes.

~0.70, AUC ~0.75), indicating limited advantage over linear approaches for this tabular dataset.

Conclusion: ML models based on admission data can effectively predict functional outcomes after AIS and can provide valuable prognostic support in acute clinical conditions. LR outperformed more complex algorithms, suggesting that key clinical predictors exhibit predominantly linear associations with 3-month functional outcomes.

Disclosure: Nothing to disclose.

EPO-0032 | RNF213 variants and angiographic progression in Korean patients with Moyamoya disease and intracranial arterial stenosis

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Background and aims: RNF213 p.R4810K is a major susceptibility variant for moyamoya disease (MMD) and has been linked to intracranial artery atherosclerosis. We investigated whether RNF213 variants are associated with angiographic progression of intracranial arterial stenosis in Korean patients with MMD and intracranial artery stenosis (ICAS).

Methods: We retrospectively analyzed 57 Korean patients diagnosed with MMD or ICAS who underwent RNF213 genotyping and serial angiographic follow-up (MRA or TFCA). RNF213 p.R4810K status was classified as carrier (heterozygous) or non-carrier. Angiographic change was categorized as 1) progression 2) no progression 3) improvement; progression was the primary outcome. Baseline clinical characteristics, vascular phenotype, and medical treatments were compared by genotype. Multivariable logistic regression adjusted for age, sex, hypertension, diabetes, dyslipidemia, smoking, and statin use.

Results: Among 57 patients, 31 (54.4%) were RNF213 carriers. Progression occurred in 15/31 (48.4%) carriers and 7/26 (26.9%) non-carriers during mean 5.0 ± 2.8 years (range 2.0–14.2). The

3-category distribution of angiographic outcomes differed by genotype ($p=0.017$). In multivariable analysis, RNF213 carrier status was independently associated with progression (adjusted OR 4.06, 95% CI 1.05–15.66; $p=0.042$). New stroke/TIA during follow-up occurred in 5/57 (8.8%).

Conclusion: In Korean patients with MMD and intracranial artery stenosis, RNF213 p.R4810K carrier status was associated with a higher risk of angiographic progression. These findings suggest that RNF213-associated vasculopathy may represent a progressive phenotype and warrant closer longitudinal monitoring.

Disclosure: Nothing to disclose

EPO-0033 | Nutritional risk assessed by CONUT independently predicts 90-day outcome after mechanical thrombectomy

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Background and aims: Malnutrition may worsen recovery after large-vessel occlusion stroke, yet its prognostic value in patients treated with mechanical thrombectomy remains insufficiently explored. We aimed to assess whether nutritional status measured by the Controlling Nutritional Status (CONUT) score predicts 90-day functional outcome and mortality after mechanical thrombectomy.

Methods: This observational cohort study included 385 patients treated with mechanical thrombectomy. Nutritional status at admission was assessed using the CONUT score. Outcomes were poor functional outcome at 90 days (modified Rankin Scale 3–6) and 90-day mortality. Univariable and multivariable logistic

regression analyses were performed, and model discrimination was evaluated using receiver operating characteristic curves.

Results: Patients were distributed across CONUT categories as follows: normal 96 (21%), mild 169 (37%), moderate 99 (21%), and severe 21 (5%). Increasing CONUT scores were associated with progressively worse 90-day modified Rankin Scale distributions. In multivariable analysis, each one-point increase in the CONUT score was independently associated with poor functional outcome (adjusted odds ratio 1.20; 95% confidence interval 1.05–1.36; $p=0.008$) and with 90-day mortality (adjusted odds ratio 1.26; 95% confidence interval 1.11–1.42; $p<0.001$). Discrimination for poor functional outcome was good (area under the curve 0.85).

Conclusion: Nutritional risk assessed by the CONUT score independently predicts functional disability and mortality at 90 days after mechanical thrombectomy and may support early risk stratification using routine laboratory data.

Disclosure: Nothing to disclose.

EPO-0034 | The cost of acute stroke: A detailed analysis focusing on sex differences

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Background and aims: Stroke has significant economic impact due to high prevalence and disability. Studies across Europe show disparities in costs among countries, underscoring the necessity for country-specific data. Few studies have explored cost differences between sexes. Using a microcosting approach, we analysed a cohort of ischemic stroke survivors to estimate costs, comparing expenses across levels of disability and between sexes.

Methods: Demographic data and healthcare resource utilization information were collected from ischemic stroke patients, at two tertiary hospitals in Portugal, over two years, employing validated questionnaires. We report mean costs (euros) at 3 months and 1 year post-stroke, per patient, with 95% confidence intervals (95% CI). Disability levels were classified by the modified Rankin Scale (mRS). Cost comparisons employed non-parametric tests, non-linear regression, and covariance analysis.

Results: We included 206 patients, median age 75 years, 52% male, and 24% employed at stroke onset. The average hospitalization cost was €11,149 (95% CI 9,970–12,328), while total outpatient costs were €4,792 (95% CI 3,970–5,614) at 3 months and €13,195 (95% CI 10,602–15,788) at 1 year. At 1 year, outpatient costs per disability level peaked at mRS 4 (€3,549 to €23,543; $p<0.001$). Male patients exhibited average higher costs (€15,065 vs. €11,087, $p>0.05$), driven mainly by non-medical and indirect costs, and they increased exponentially with disability ($R^2=0.51$ in males versus 0.27 in females; $p<0.030$).

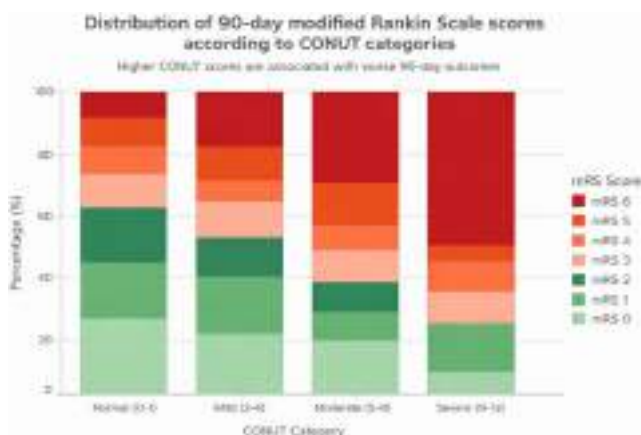


FIGURE 1 Distribution of 90-day modified Rankin Scale scores according to CONUT categories (normal, mild, moderate and severe malnutrition).

Conclusion: We showed annual outpatient stroke costs amounting to 16 minimum wages. The escalation of cost with disability is notable, and consistent with prior studies. This increase was significantly more pronounced in males, emphasizing the importance of sex-specific healthcare studies.

Disclosure: CUF scholarship.

EPO-0035 | **HBOC-mediated pre-reperfusion oxygen delivery freezes ischemic penumbra via suppression of ferroptosis and neuroinflammation**

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Background and aims: Overcoming the challenges of delayed intervention and single-target mechanisms in stroke neuroprotection, we propose a paradigm-shifting “pre-reperfusion” therapy using a hemoglobin-based oxygen carrier (HBOC) for diffusive oxygen delivery. This study aims to validate the efficacy of HBOC in preserving the ischemic penumbra and elucidate its integrated mechanism involving the mitochondria-ferroptosis-inflammation axis.

Methods: In a rat transient middle cerebral artery occlusion (tMCAO) model, a single intravenous dose of HBOC or saline was administered 5 minutes post-ischemia, followed by reperfusion at 1 hour. Assessment included: 1) safety and dose-escalation (mortality, hemorrhagic transformation, systemic toxicity); 2) multi-timepoint outcomes (infarct volume by MRI/TTC, neurobehavior from 4h to 30d); 3) mechanistic dissection (mitochondrial ultrastructure/ATP, ferroptosis markers GPX4/ACSL4/GSH/Fe²⁺, neuroinflammation via cytokine array and microglial polarization).

Results: Optimal-dose HBOC was safe, with no increased bleeding risk or systemic toxicity. It conferred dose-dependent neuroprotection, significantly reducing infarct volume at 24h with progressive resolution up to 30days, alongside robust and sustained neurological improvement. Mechanistically, HBOC rapidly stabilized mitochondrial ultrastructure, suppressed lipid

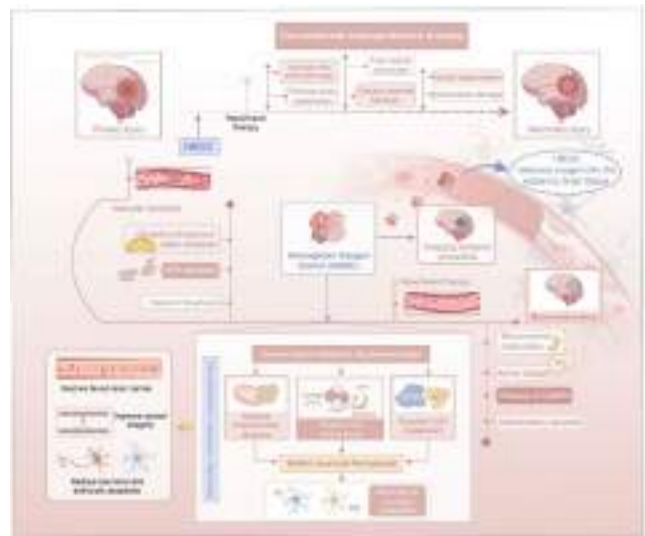


FIGURE 1 Schematic illustrating the proposed multi-target cytoprotective mechanism of pre-reperfusion HBOC therapy.

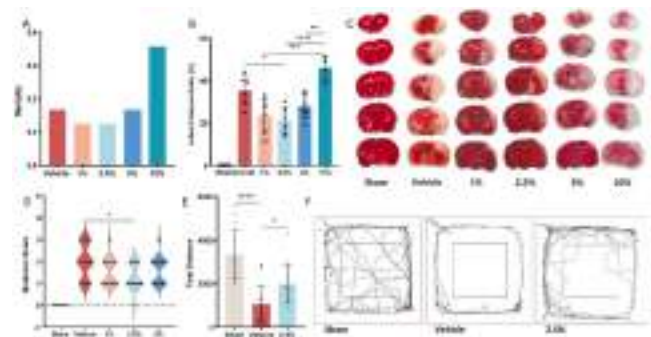


FIGURE 2 Dose-dependent neuroprotection and safety profile of HBOC in tMCAO rats.

peroxidation, and orchestrated a coordinated anti-ferroptotic response. This cascade drove a sustained shift in microglial polarization from pro-inflammatory M1 to reparative M2 phenotype, attenuating neuroinflammation.

Conclusion: Pre-reperfusion diffusive oxygen delivery via HBOC robustly preserves the ischemic penumbra through a multi-target mechanism centered on mitochondrial stabilization, ferroptosis inhibition, and immunomodulation. Our findings establish HBOC as a novel, practical “bridge therapy” for the hyper-acute phase, offering a transformative strategy to extend the therapeutic window in ischemic stroke.

Disclosure: Nothing to disclose.

Child Neurology/Developmental Neurology

EPO-0036 | **Therapeutic hypothermia for neonatal hypoxic-ischemic encephalopathy: A meta-analysis of neurodevelopmental and mortality outcomes**

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Background and aims: Neonatal Hypoxic-Ischemic Encephalopathy (HIE) stills a leading cause of mortality and morbidity in newborns. While Therapeutic Hypothermia (TH) has been used widely for moderate to severe HIE management; however, uncertainty persists regarding the consistency of its benefits, particularly for long-term neurodevelopmental outcomes. This study aimed to synthesize current evidence on the impact of TH on mortality and neurodevelopment in term and near-term infants with HIE.

Methods: We conducted a meta-analysis including 10 primary studies that compared TH with standard care in neonates diagnosed with HIE. Data from over 4,600 infants were combined for analysis. The primary outcome was all-cause mortality, which was estimated using risk ratio. We measured Neurodevelopmental outcomes with validated assessment tools such as Bayley Scales. Additional subgroup analyses were conducted according to HIE severity and World Bank income categories.

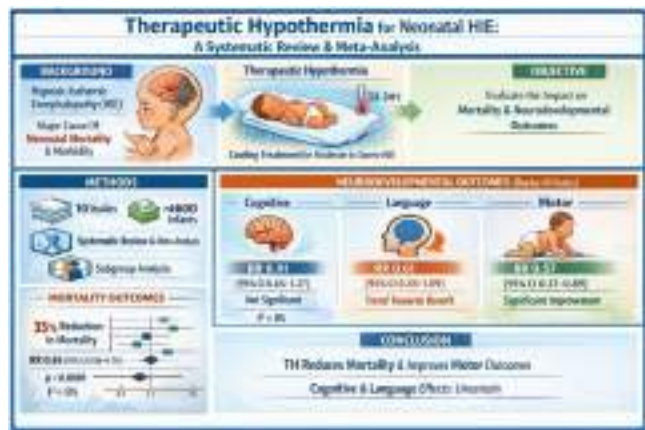


FIGURE 1 Therapeutic hypothermia for neonatal HIE: A systematic review & meta-analysis.

Figure 1. Mortality



Figure 3. Bayley III Motor Domain



Figure 2. Bayley III Cognitive Domain



Figure 4. Bayley III Language Domain



Results: The combined findings showed that therapeutic hypothermia led to a meaningful reduction in all-cause mortality, with about a 35% lower risk compared with standard treatment. The results were consistent across studies, with little variation observed. According to neurodevelopment outcomes, infants who received TH showed significantly better motor outcomes. Improvements in language and cognitive outcomes were also noted, but these effects did not always reach statistical significance and less consistent.

Conclusion: TH is effective in decreasing mortality and improving motor outcomes in infants with HIE. However language and cognitive outcomes suggest some benefit, more standardized follow-up and long-term studies are needed to confirm these effects.

Disclosure: Nothing to disclose.

EPO-0037 | Overcoming the “Tunnel of fear”: A systematic review of interventions for MRI-related anxiety and phobia in pediatrics

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Background and aims: Pediatric MRI often fails due to anxiety-induced motion or requires risky general anesthesia. This review synthesizes evidence on strategies to improve compliance and diagnostic quality. The study aims to evaluate the effectiveness of behavioral, digital, and pharmacological interventions in reducing anxiety in pediatric patients (ages 4–18).

Methods: Following PRISMA guidelines, we analyzed 41 primary studies, including RCTs and cohorts. Outcomes included

TABLE 1 Summary of Evidence: Pediatric MRI Compliance strategies (n = 41).

Intervention Category	Specific Strategies & Tools	Duration / Dosage	Primary Clinical Outcomes
Behavioral Preparation	Storytelling Training, "Statue Game", and puppets (statue puppets)	30-60 min	Decreased anxiety, reduced need for sedation, increased compliance
Engaged MR	Virtual Reality (VR) for Mock MRI, augmented reality, and interactive games (e.g., "Ollie the Elephant")	15-20 min	Reduced anxiety, improved image quality, decreased need for sedation
Multimodal Prep	Integrated story, interactive games, and PCC (see Table 1 for details)	30-60 min	Significant reduction in sedation rates and improved compliance
Human Support	Child Life Specialist (CLS) or Pediatric Nurse (PN) presence, and Parental Support (PCC)	30-60 min	Reduced anxiety, improved image quality, decreased need for sedation
Pharmacology	Oral sedation (e.g., Midazolam) and Intranasal Dexmedetomidine (ID) or Propofol	As needed	Effective for patients with failed behavioral prep



anxiety scores (e.g., mYPAS), image quality, and sedation reduction rates.

Results: Mock MRI sessions (30–60 minutes) led by Child Life Specialists or pediatricians successfully converted “sedation-required” patients to “awake-successful” status through “statue games” and sound simulation. Digital innovations, such as the “Pengonaut Trainer” VR app and in-bore systems like “Ollie the Elephant,” provided progressive desensitization and real-time guidance. Creative approaches, including therapy dogs, hospital clowns, and live music, significantly improved emotional status. Additionally, brief 15-minute Patient-Centered Communication (PCC) sessions built effective cooperation. When behavioral methods were insufficient, intranasal Dexmedetomidine (3 µg/

kg) emerged as a superior, less invasive rescue sedation compared to Midazolam.

Conclusion: The most effective strategy for managing pediatric MRI phobia is Multi-modal preparation combining Mock MRI and digital distraction. Hospitals should prioritize these low-risk protocols to decrease anesthesia reliance.

Disclosure: Nothing to disclose.

EPO-0038 | Clinical and demographical profile of pediatric MOGAD patients: Portuguese national data

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Background and aims: Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an autoimmune demyelinating disease affecting both adults and children. Despite its clinical heterogeneity, the prognosis is often favorable. This study aimed to describe demographic and clinical characteristics of pediatric MOGAD patients in Portugal and to identify potential risk factors for relapsing disease.

Methods: We conducted a retrospective and multicenter study including pediatric patients (age below 18 years-old) with MOGAD diagnosed between 2011 and 2025. Data were collected from clinical records from participant centers.

Results: Seventy-seven patients met the inclusion criteria; 49.7% were male and the mean age was 7.8 years-old. A preceding infection was reported in 45.5% of cases. ADEM was the most frequent phenotype (61%) and was associated with the age at presentation ($p=0.005$). The EDSS at onset was of 4.0 (0–9). Anti-MOG antibodies were positive at diagnosis in 97.4% of patients with seroreversion occurring in 49.5%, which was not correlated with relapsing disease ($p=0.5$). Most of the patients received intravenous methylprednisolone (97.4%); nine patients with more severe disease had add-on treatment with plasma-pheresis ($p=0.003$). Complete recovery was observed in 81.6% of patients, while 26% had a relapsing disease. A univariate analysis showed a positive correlation between EDSS at presentation

and at follow-up ($p=0.009$) and time of follow-up and relapsing disease ($p=0.001$).

Conclusion: Portuguese national data confirm previously described features of MOGAD. Therapeutic approaches appear consistent throughout centers and guided by disease severity ab initio even though standardized treatment guidelines are missing.

Disclosure: Nothing to disclose.

EPO-0039 | **Transitioning from pediatric to adult care in neuromuscular diseases: A management model in the Tuscany region, Italy**

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Background and aims: The improvement of standards of care and the availability of effective, disease-modifying treatments for neuromuscular diseases has expanded patients' lifespan and improved their quality of life. This translates into a higher number of subjects reaching the "transition" phase to the adult care centers from the pediatric ones. With this concept in mind, the Italian region of Tuscany developed a program for transition from two pediatric reference centers for rare and neuromuscular disorders to the adult reference center.

Methods: This study presents retrospective data acquired longitudinally in a 2-years period after the start of the collaborative Tuscany region program to promote the transition process to the adult care center from pediatric institutions.

Results: 70 patients transitioned from the pediatric centers to the adult center. Most of the patients had previously received a genetic and clinical diagnosis. All patients were firstly seen in the pediatric center in presence of the adult neurologist and pediatrician/childhood neurologist and then re-evaluated after 6 months at the adult center; after that visit, the ordinary schedule of follow-up was re-established according to the disease progression and patients' needs. Regarding multidisciplinary care, 25 patients were referred to specialized adult services for cardiology and pulmonology.

Conclusion: The transition passage is in most cases fragmented and is subject to data loss or even discontinuation of care. To ensure a smooth and complete transitional passage, it must be based on epidemiological awareness and an established

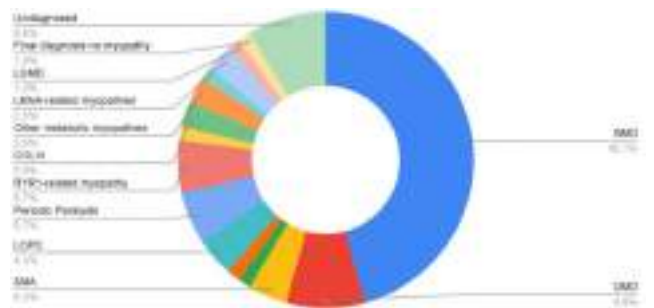


FIGURE 1 Graphical representation of the different diagnoses included in the study cohort.

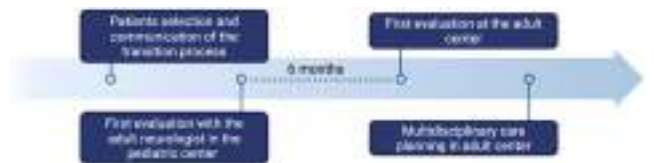


FIGURE 2 Graphical representation of the transition process organization.

workflow including all the necessary specialists. Tuscany region successfully provided a pre-organized and well-defined process for transition.

Disclosure: Nothing to disclose.

EPO-0040 | **CUL1 variants underlie severe neurodevelopmental disorders: Insights from human genetics and a zebrafish microcephaly model**

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Background and aims: Microcephaly is a neurodevelopmental anomaly defined by reduced head circumference and impaired brain growth, frequently accompanied by intellectual disability, developmental delays, and seizures. Although numerous genes have been linked to this condition, the role of CUL1—a core component of the SCF (Skp1-Cul1-F-box protein) ubiquitin ligase complex—remains poorly elucidated. This study aims to explore the association between CUL1 and severe neurodevelopmental disorders (NDDs).

Methods: First, comprehensive clinical evaluations were performed on four unrelated affected families, confirming all patients exhibited severe microcephaly, intellectual disability, and developmental delay phenotypes. Whole-exome sequencing (WES) was subsequently conducted to detect pathogenic variants, followed by screening and validation of heterozygous de

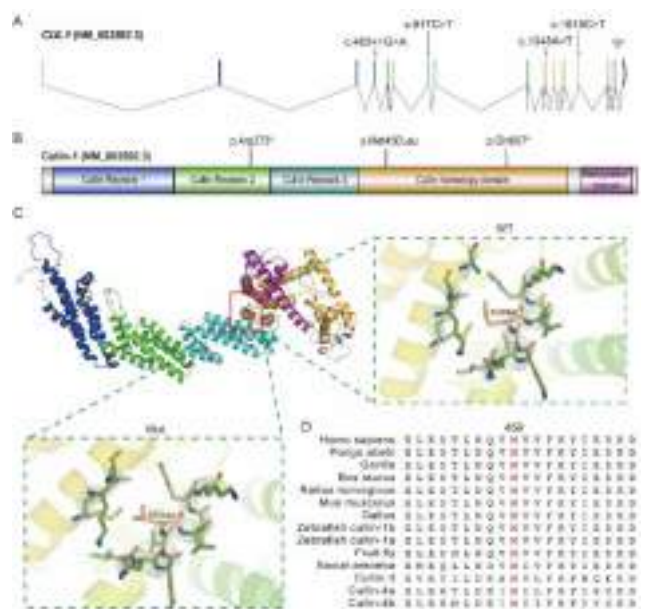


FIGURE 1 Genetic and structural analysis of CUL1 variants.

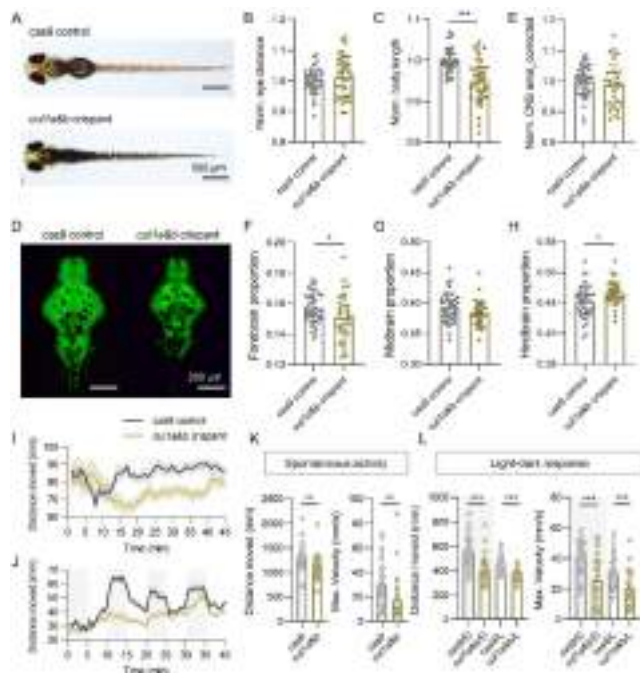


FIGURE 2 CRISPR/Cas9 knockdown of zebrafish cul1a and cul1b results in abnormal embryonic development and neurodevelopmental deficits.

TABLE 1 Clinical features of five patients with CUL1 variants (NM_003592.3).

Patient	Family 1 (TR12)	Family 1 (TR12)	Family 2 (TR13)	Family 2 (TR13)	Family 4 (TR11)
Sex	Female	Female	Female	Male	Male
HGS	<1000<1, p<0.000007	<1000<1, p<0.000007	<1000<1, p<0.000007	<1000<1, p<0.000007	<1000<1, p<0.000007
Age	12	12	12	8	10
Height (cm)	130	130	130	127	129
Weight (kg)	36	36	28.1	20.4	41.3
IQ (mean, SD)	46, <1<1 percentile	46, <1<1 percentile	46, <1<1 percentile	44, <1<1 percentile	52, <1<1 percentile
EEG/EEG	Yes	Yes	Yes	Yes	Yes
Feeding difficulties	No	No	No	Yes	No
Epilepsy type (A/N/A/age)	No	No	Interictal spikes (12)	Interictal spikes (10)	No
Seizure type	-	-	Yes	Yes	-
Language ability	Yes, single words	Yes, single words	Yes, single words	Yes, single words	Yes, single words
Motor delay	Yes	Developmental Delay, mild	Yes	Yes	No
Genotypic consequences	No	No	Yes	No	No
Associated features	Epilepsy, mild	No	No	No	Aggressive behavior

Abbreviations: IQ, Intelligence Quotient; EEG, Electroencephalogram; SD, Standard Deviation; EEG, Electroencephalogram; EEG, Electroencephalogram; EEG, Electroencephalogram.

novo and inherited variants in the CUL1 gene. To further verify the functional consequences of CUL1 loss-of-function, a zebrafish model with double knockdown of cul1a and cul1b was established for subsequent functional analysis to corroborate clinical findings.

Results: WES identified heterozygous de novo and inherited CUL1 variants in all four unrelated families, with these variants showing a clear correlation with the patients' severe phenotypes (microcephaly, intellectual disability, and developmental delays). The cul1a&b double-knockdown zebrafish model exhibited significant central nervous system hypoplasia and abnormal behaviors, which closely recapitulated the clinical manifestations observed in human patients.

Conclusion: These findings establish CUL1 as a novel gene associated with severe NDDs and highlight its critical role in brain development. Our study further delineates genotype-phenotype correlations for CUL1 in NDDs, expands the genetic spectrum of disorders linked to the SCF complex, and underscores the complex's pivotal importance in neurodevelopment.

Disclosure: Nothing to disclose.

EPO-0041 | HMGB1-TREM2 axis mediates microglial activation in neonatal sepsis-associated encephalopathy

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Background and aims: The objective of this study is to investigate the High-mobility group box 1 (HMGB1) - Triggering Receptor Expressed on Myeloid Cells 2 (TREM2) axis in microglia during neonatal sepsis-associated encephalopathy (nSAE).

Methods: An nSAE mouse model was established in 3-day-old C57 mice via intraperitoneal injection of lipopolysaccharide (LPS, 4 mg/kg; n=3), littermates receiving saline as controls (n=3). Brain tissues were collected 12 hours after modeling for analysis. Microglial activation, HMGB1 levels in brain tissue homogenates, and total HMGB1 protein expression were assessed by immunofluorescence (IF), enzyme-linked immunosorbent assay (ELISA), and Western blot, respectively. Immunoprecipitation-Mass Spectrometry (IP-MS) was used to identify TREM2-interacting proteins in microglia, and then validated by co-immunoprecipitation (Co-IP) and IF co-localization.

Results: Compared with the control group, the number of Iba1-positive microglia in the brain tissue of nSAE models was significantly increased, with a shift in cell morphology towards an amoeboid appearance. Both the concentration of HMGB1 in brain tissue homogenates and its total protein expression were significantly elevated in nSAE models. IP-MS analysis indicated that HMGB1 was co-precipitated with TREM2 antibodies in LPS-stimulated BV2 cells. This interaction was confirmed by Co-IP in 293T cells co-transfected with Flag-tagged HMGB1

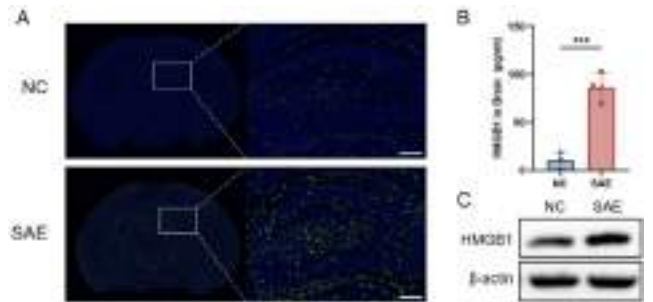


FIGURE 1 (A) Immunofluorescence of Iba1+(green) microglia in hippocampus of nSAE mice brain slice (Scale bar, 400µm). (B) HMGB1 levels in brain were determined by ELISA (p < 0.001). (C) Western blot bands of the HMGB1 expression levels in brain of mice.

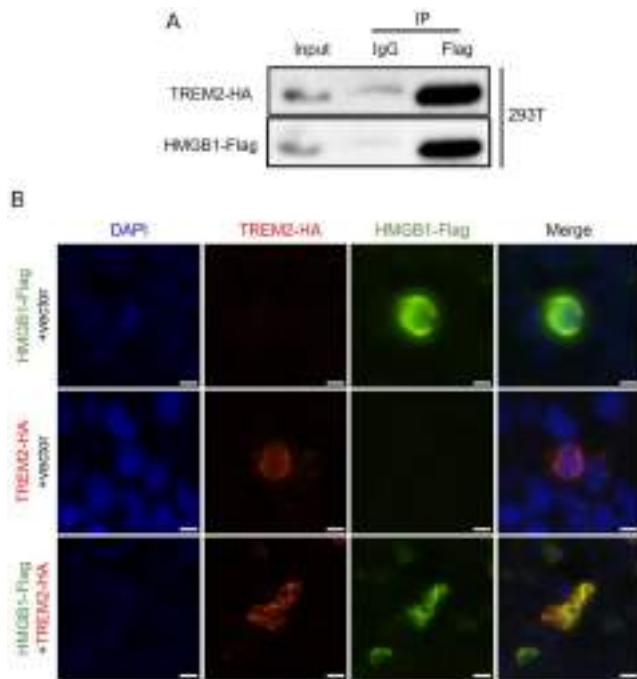


FIGURE 2 293T cells were transfected with pcDNA3.1-HMGB1-Flag and pcDNA3.1-TREM2-HA plasmids. (A) Anti-Flag antibody was employed for exogenous Co-IP experiments. (B) The co-localization between Flag and HA was examined using confocal microscopy (Scale bar, 5um).

and HA-tagged TREM2 plasmids, and further validated by IF demonstrating their co-localization.

Conclusion: This study revealed an interaction between HMGB1 and TREM2 in microglia, suggesting that HMGB1 may regulate microglial activation by binding to TREM2, thereby mediating neuroinflammation associated with nSAE. Targeting the HMGB1-TREM2 axis may represent a novel therapeutic strategy for intervening in nSAE-related neuroinflammation.

Disclosure: Nothing to disclose.

EPO-0042 | Speech, language, social communication, and communication assessments in Rett syndrome: A systematic review

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Background and aims: Rett syndrome (RTT) is a neurodevelopmental disorder caused by pathogenic MECP2 variants, characterised by early regression, including loss of purposeful hand use and spoken language. Communication impairment is among its most disabling features; however, speech, language,

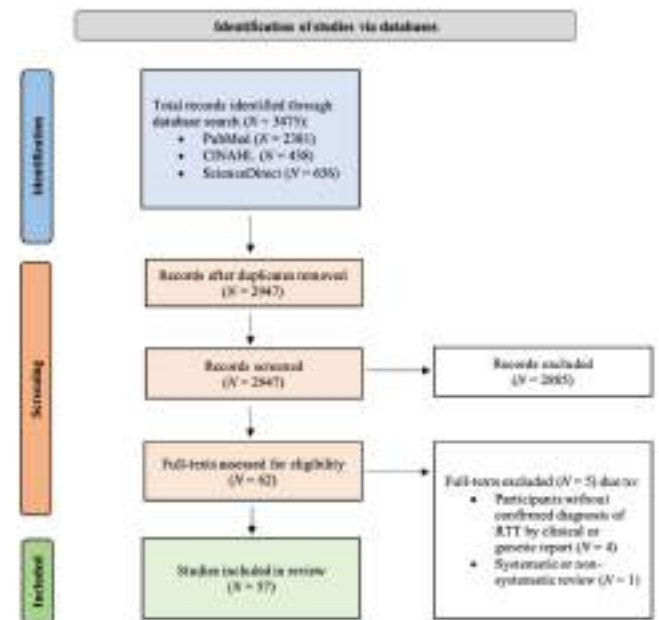


FIGURE 1 Overview of Systematic Search Results.



FIGURE 2 Key Insights of the Current Systematic Review

and social communication phenotypes have not been systematically synthesised. This review aimed to (a) characterise each domain in RTT and (b) identify the most common communication measures.

Methods: A systematic search was conducted per PRISMA guidelines and registered on PROSPERO (CRD420251076117). PubMed, CINAHL, and ScienceDirect databases were searched from January 1985 to February 2025. Studies including individuals with genetically/clinically confirmed RTT and subjective/objective communication data were eligible. Two reviewers independently screened studies. Data were synthesised descriptively due to methodological heterogeneity. Studies were appraised using Joanna Briggs Institute tools. A supplementary search of ClinicalTrials.gov was conducted for therapeutic interventions.

Results: 57 studies met inclusion criteria. Communication impairment was universal but heterogeneous. Expressive language and physical speech were most severely affected, with near-total speech loss post-regression (median onset: 18–24 months). Pre-regression atypicalities (e.g. high-pitched vocalisations) were reported. Receptive abilities were relatively preserved, namely in atypical forms. Eye gaze was the predominant post-regression

communicative modality. Social motivation often persisted despite autistic features. Genetic and epigenetic factors (e.g. age) modulated severity. Most studies (63%) relied on caregiver report. No gold-standard battery was identified.

Conclusion: RTT is associated with profound communication impairment, disproportionately affecting expressive language and speech. Incorporating objective and subjective tools may improve phenotype accuracy and clinical monitoring.

Disclosure: Adam Vogel is an employee of Redenlab Ltd, a speech neuroscience company. All other authors do not have a conflict of interest to declare.

EPO-0043 | Disrupted time-varying brain connectivity associates with expressive language impairment in pediatric multiple sclerosis

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Background and aims: Multiple sclerosis (MS) onset during developmental age makes pediatric (ped) patients vulnerable to cognitive impairment. As cognitive processes rely on dynamic reconfiguration of functional brain networks, we explored time-varying (TV) functional connectivity (FC) in pedMS patients and its association with clinical disability and structural brain damage.

Methods: Ninety-five pedMS and twenty-four matched healthy controls (HC) underwent neurological, neuropsychological and 3.0T brain MRI assessments. Expanded Disability Status Scale (EDSS) and z-scores of verbal and visuospatial memory, attention, receptive language, and expressive language were obtained. Brain T2 white matter (WM) lesion volume (LV) and atrophy were assessed. TVFC was assessed using sliding-window correlation analysis; FC variability was quantified as the standard deviation of degree centrality (DC) and seed-based correlation within basal ganglia, executive, language, sensorimotor, and default-mode networks.

Results: PedMS patients showed widespread reduction of DC TVFC vs. HC, particularly involving the right middle temporal, inferior parietal and superior frontal cortices ($p < 0.05$, corrected), and extending to bilateral parahippocampal, basal ganglia, fronto-insular and cerebellar regions ($p < 0.001$,

uncorrected). PedMS patients exhibited reduced TVFC ($p < 0.05$, corrected) within basal ganglia, executive and language networks vs. HC. No associations were observed between TVFC abnormalities and EDSS score, brain T2 LV and atrophy. Reduced TVFC of the right inferior parietal lobule, right middle superior frontal gyrus and right middle temporal gyrus associated with worse expressive language z-scores ($r \leq 0.30$; $p \leq 0.04$).

Conclusion: PedMS patients experience widespread reductions in TVFC, with selective regions showing associations with expressive language impairment, suggesting that altered brain network dynamics contribute to cognitive dysfunction.

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EPO-0044 | Non-epileptic paroxysmal events in Rett syndrome: A systematic review of case-based and observational evidence

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Background and aims: Rett syndrome (RTT) is a severe neurodevelopmental disorder frequently complicated by epilepsy. Many individuals also experience non-epileptic paroxysmal events that mimic seizures and risk inappropriate treatment. The spectrum and phenomenology of these events remain poorly defined. We aimed to systematically characterise non-epileptic paroxysmal events in RTT.

Methods: We performed a PRISMA-guided systematic review of observational studies from PubMed, Embase, and OVID (1962–2024). Eligible studies included case reports, case series, cohort studies, and small clinical trials describing non-epileptic events in individuals with clinically or genetically confirmed RTT. Data were extracted on study design, participant characteristics, and event type. Events were categorised into respiratory, neuromotor, and behavioural domains. Due to heterogeneity, results were synthesised descriptively.

Results: Sixty-two studies were included. Respiratory events predominated, with apnoea reported in 1,092 clinician-reported cases (26 studies) and 507 family-reported cases (9 studies). Breathing abnormalities persisted into adulthood in 30/50 adults (apnoea 54%, hyperventilation 24%). Neuromotor events, including dystonia and involuntary movements, were reported in 51 patients, with three life-threatening episodes involving airway compromise; vacant spells frequently accompanied respiratory events. Behavioural and autonomic disturbances were common. Video-EEG and telemetry reclassified up to 80% of events labelled as seizures as non-epileptic. The term “Rett episodes” was used inconsistently and by a minority of authors.

Conclusion: Non-epileptic paroxysmal events are common and heterogeneous in RTT. Frequent misclassification as epilepsy risks unnecessary treatment and harm. Greater clinician awareness, precise phenomenological description, and objective recording are needed, alongside clearer diagnostic frameworks to optimise care and quality of life.

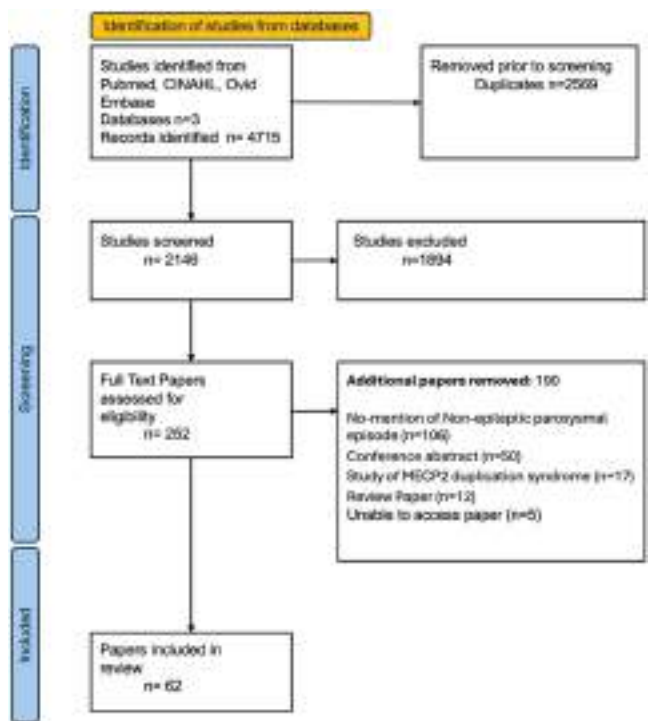


FIGURE 1 Literature search and selection flow diagram.

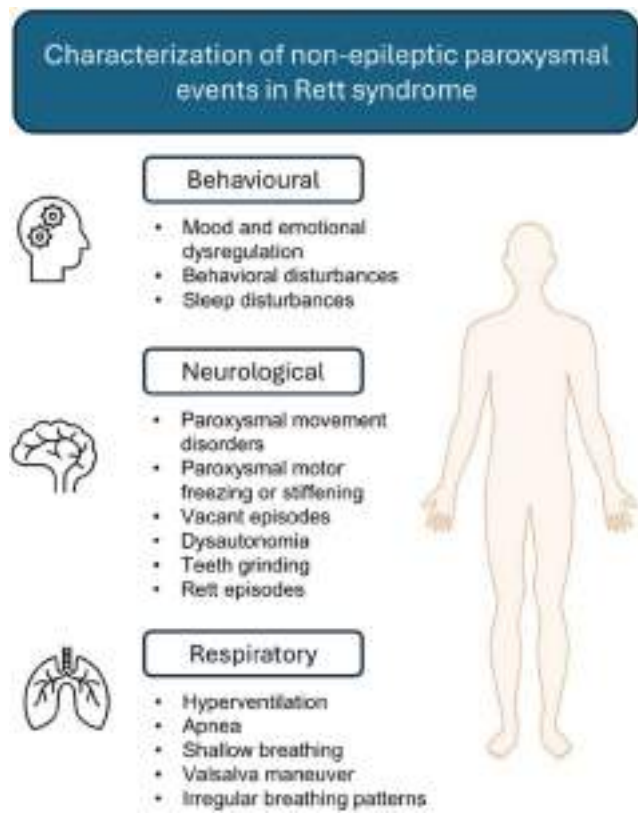


FIGURE 2 Classification of non-epileptic paroxysmal events in Rett syndrome.

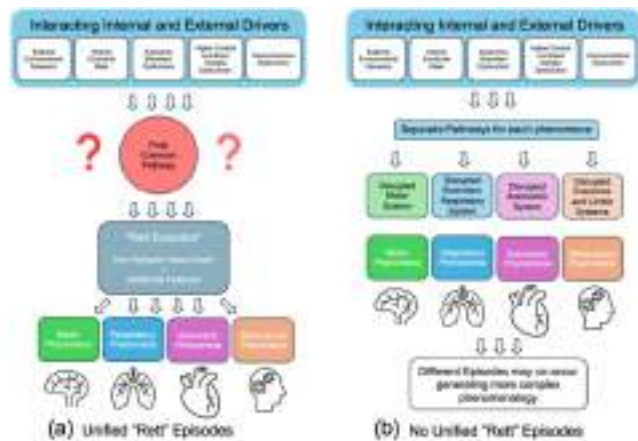


FIGURE 3 Potential pathways for Rett episodes with an assumption that events arise from a single common pathway (a) and a model of separate pathways independently giving rise to different episodes (b), which may temporally coincide.

Disclosure: NB has nothing to disclose DEL has undertaken consultancy work for Acadia and Neurogene.

EPO-0045 | iPSC-derived cellular models and extracellular vesicles: A translational pipeline for biomarker identification in neurodevelopmental disorders

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Background and aims: Neurodevelopmental disorders (NDDs), including Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD), show complex etiopathogenesis and substantial clinical heterogeneity, limiting early diagnosis and personalized treatment. The identification of reliable biomarkers is therefore a major unmet clinical need. Induced pluripotent stem cell (iPSC) models and circulating extracellular vesicles (EVs) represent promising translational tools to investigate neurodevelopmental mechanisms and identify disease-related molecular signatures.

Methods: A pediatric cohort (3–17 years) with NDDs diagnosed according to DSM-5/DSM-5-TR criteria is being recruited, together with a typically developing control group. Participants undergo standardized neuropsychiatric, cognitive, and behavioral assessments. Peripheral blood and urine samples are collected from patients and controls. Plasma EVs are isolated by differential ultracentrifugation and characterized using immunoblotting for exosomal markers, and nanoparticle tracking analysis or transmission electron microscopy. Then plasma EVs are investigated with a multiomic approach. In parallel, urinary stem cells and peripheral blood cells are reprogrammed into patient-specific iPSCs and differentiated into neuronal and glial precursors to generate three-dimensional brain organoid models.

Results: Sixty-one patients have been enrolled to date, including ASD, ADHD, and ASD/ADHD overlap groups, with a marked male predominance and predominantly preserved cognitive functioning. Patient-specific iPSC lines have been successfully generated, and neuro-glial differentiation protocols have yielded initial astrocyte cultures. Multi-omic profiling of plasma-derived EVs is currently ongoing.

Conclusion: The combined analyses of clinical phenotypes, EV molecular cargo may enable the identification of biomarker signatures supporting stratification and precision medicine approaches in NDDs. Creating diseases-specific brain organoids models may shed a light on the underlying neurobiological pathways of these disorders.

Disclosure: Nothing to disclose.

EPO-0046 | Impact of early seizure onset on language development in genetic epilepsy with febrile seizures plus

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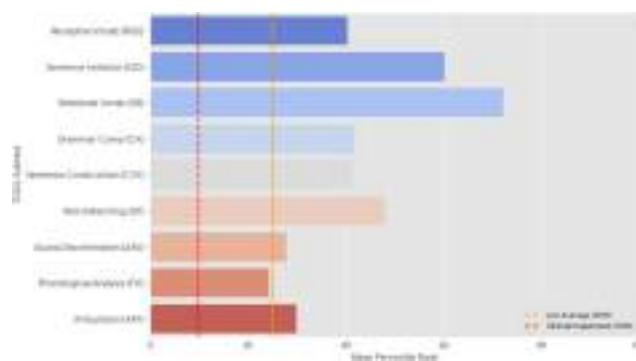


FIGURE 1 Mean Percentile Ranks Across TODIL Subtests in Children with GEFS+ (N=51). The dashed red line indicates the 10th percentile clinical impairment threshold, and the orange line represents the 25th percentile “at-risk” threshold. Note the profound.

Background and aims: Genetic Epilepsy with Febrile Seizures Plus (GEFS+) involves early-onset seizures during critical neurodevelopmental windows. While seizure semiology is well-documented, the impact on linguistic acquisition remains unexplored. This study evaluates the prevalence and characteristics of language impairments in GEFS+ using the Turkish Test of Preschool Language Development (TODIL).

Methods: A cohort of 51 pediatric patients (31 male, 20 female) with GEFS+ was evaluated. The mean age was 71.36 +/- 16.38 months. Linguistic performance was assessed across nine domains using the TODIL. Raw scores (HP) were converted to percentile ranks based on national norms. Clinical impairment was defined as a score falling below the 10th percentile.

Results: Impairment was most prevalent in Sound Discrimination (45.10%), Phonological Analysis (37.25%), Grammar Comprehension (33.33%), and Articulation (33.33%). Relational Vocabulary was a relative strength (1.96% impairment). Mean percentile ranks for Phonological Analysis (24.31%) and Articulation (29.97%) were significantly low. Gender-based comparisons revealed no statistically significant differences across subtests (all $p > 0.05$), suggesting a gender-independent linguistic phenotype.

Conclusion: Early-onset seizures in GEFS+ are associated with high rates of language impairment, a correlation previously under-reported. The specific phenotype involves significant phonological and structural deficits despite preserved conceptual vocabulary. These findings underscore the urgent need for speech-language assessment immediately following diagnosis to improve long-term communicative outcomes.

Disclosure: The authors declare that they have no competing financial interests or personal relationships that could have appeared to influence the work reported in this paper. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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Background and aims: Growing evidence indicates that neuroinflammatory pathways contribute to epileptogenesis and pharmacoresistance in epilepsy. The main impact on clinical practice is the development of new targeted therapies for epilepsy. We studied the importance of neuroinflammation in the development of epileptogenesis and pharmacoresistance in childhood epilepsy patients.

Methods: A cross-sectional study conducted at two epilepsy centers in the Czech Republic compared 22 pharmacoresistant patients and 4 pharmacodependent patients to 9 controls. We analyzed the ProcartaPlex™ 9-Plex immunoassay panel consisting of interleukin (IL)-6, IL-8, IL-10, IL-18, CXCL10/IP-10, monocyte chemoattractant protein 1 (CCL2/MCP-1), B lymphocyte chemoattractant (BLC), tumor necrosis factor- α (TNF- α), and chemokine (C-X3-X motif) ligand 1 (fractalkine/CXC3CL1) to determine their alterations in cerebrospinal fluid (CSF) and blood plasma, concurrently

Results: The analysis of 21 paired CSF and plasma samples in pharmacoresistant patients compared to controls revealed a significant elevation of CCL2/MCP-1 in CSF ($p < 0.000512$) and plasma ($p < 0.00017$). Higher levels of fractalkine/CXC3CL1 were revealed in the plasma of pharmacoresistant patients than in controls ($p < 0.0704$), and we determined an upward trend in CSF IL-8 levels ($p < 0.08$). No significant differences in CSF and plasma levels were detected between pharmacodependent patients and controls.

Conclusion: Our results confirm that CCL2/MCP-1, IL-8, and fractalkine/CXC3CL1 are potential biomarkers of epileptogenesis and pharmacoresistance in epilepsy. These findings provide new insights into processes of epileptogenesis and development of pharmacoresistance in epilepsy with potential impact on developing disease-modifying drugs in epilepsy.

Disclosure: Nothing to disclose.

EPO-0048 | Antibodies against proteolipid protein-1 are identified in patients with inflammatory neuropathies

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Background and aims: Conformational antibodies to proteolipid protein-1 (PLP1-IgG), a major myelin protein, have been reported in CNS demyelinating disorders with peripheral involvement, but their prevalence and clinical significance in isolated inflammatory peripheral neuropathies remain unclear.

Methods: A validated live cell-based assay for PLP1-IgG detection was implemented. We tested 135 patients with inflammatory neuropathies [94 chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), 34 Guillain-Barré syndrome (GBS), 7 autoimmune nodopathies (AN)] and 48 controls. Clinical data were retrospectively extracted from medical records.

Results: PLP1-IgG were detected in 13/135 (9.6%) inflammatory neuropathy patients and in none of the controls. Seropositivity occurred in 11/94 CIDP (11.7%), 1/72 GBS (1.4%), and 1/7 AN-CNTN1 (14.3%). Among seropositive CIDP patients, six were male (54.5%) and mean age at onset was 53.2 ± 16.4 years. Eight (72.7%) had a typical phenotype and three (27.3%) a multifocal variant; onset was acute/subacute in eight (72.7%). Ataxia occurred in nine (81%), tremor in three (27.3%), and cranial nerve involvement in three (27.3%). Electrophysiology confirmed demyelinating neuropathy in all cases, including conduction block in seven (63.6%); one patient had GM1-IgG. CSF analysis (8 patients) showed albuminocytological dissociation in seven (87.5%); mean protein concentration 104.7 ± 62.9 mg/dL. Treatment response was satisfactory in 7/11 to IVIg (63.6%) and in 5/9 to corticosteroids (55.6%). Two refractory patients improved partially with plasma exchange; one subsequently underwent HSCT with substantial improvement.

Conclusion: PLP1-IgG identify a subset of inflammatory neuropathy patients characterized by frequent acute/subacute onset, ataxia, and generally favorable response to immunomodulatory therapy. Their pathogenic role remains unclear, particularly given co-existing pathogenic antibodies in two cases.

Disclosure: No.

EPO-0049 | Health literacy and patient activation as modifiable determinants of perceived quality of life in adults living with ATTRv amyloidosis in Portugal

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Background and aims: Hereditary transthyretin (ATTRv) amyloidosis is a progressive disease that affects daily functioning and overall wellbeing. Identifying modifiable determinants that enhance perceived quality of life (QoL) is essential for patient-centred interventions. Health literacy and patient activation shape individuals' ability to understand their condition and engage with care management. This study examined how these competencies relate to perceived QoL among adults with ATTRv amyloidosis.

Methods: A cross-sectional analysis was conducted using data from adults (≥ 18 years) enrolled in the LANTERN study in Portugal, a national observational study of adults living with ATTRv amyloidosis in Portugal. Health literacy (HLS-EU-Q16), patient activation (PAM-13), and perceived QoL (EQ-5D VAS) were assessed. Descriptive statistics were performed, and associations between these measures were examined using Spearman correlations, with statistical significance set at $p < 0.05$.

Results: Among 282 participants (mean age 48 years; 42.9% male), the mean EQ-5D VAS score was 66.31 (0–100 scale). Health literacy (0–50 scale) averaged 32.16, with 18.5% classified as inadequate, 31.1% problematic, 35.2% sufficient, and 15.2% excellent. Patient activation (0–100 scale) averaged 68.83, with most participants in level 3 (47.8%) or level 4 (40.8%). Higher EQ-5D VAS scores were significantly associated with higher health literacy and higher patient activation ($p < 0.001$).

Conclusion: Despite high activation levels, nearly half of people living with ATTRv amyloidosis have suboptimal health literacy. Health literacy and patient activation were both positively associated with perceived QoL, representing modifiable intervention targets. Enhancing these competencies may support better self-management and contribute to improved patient-centred outcomes.

Disclosure: This work was supported by AstraZeneca – Produtos Farmacêuticos, Lda. NOVA National School of Public Health Research Grant: LANTERN|951114|2025).

EPO-0050 | Semaglutide is associated with preserved motor axon function in type 2 diabetic peripheral neuropathy

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Background and aims: The motor axon dysfunction reflects the peripheral neuropathy severity in patients with Type 2 Diabetes (T2DM). The aim of this cross-sectional observational study was to investigate the effect of the Glucagon-like peptide-1 receptor agonist semaglutide on motor neuropathy in T2DM patients referred for electrodiagnostic investigations.

Methods: We compared consecutive T2DM patients treated with semaglutide and metformin ($n=8$) with gender and age-matched T2DM patients receiving metformin only. The dominant median nerve was stimulated at the wrist. The evoked compound motor axon potential (CMAP) was recorded from the abductor pollicis brevis muscle. Multiple measures of motor axon excitability were obtained by threshold-tracking. Motor unit number estimation (MUNE) was carried out by MScanFit-2. The clinical sensory-motor involvement was assessed by the modified Toronto Clinical Neuropathy Score (mTCNS).

Results: The patients (14 men and 2 women, mean age of 65.3 years) had a BMI of 30.9, a HbA1c of 53.2 (nmol/mol), and a mTCNS of 12.4. The semaglutide group had a CMAP amplitude of 7.9 mV. The metformin-only group had a smaller CMAP of 6 mV ($p < 0.05$), and a MUNE of 73.8 which was reduced compared to normal controls (94.2, $n=52$, $p < 0.05$). The motor axon excitability of the metformin-only group showed depolarizing changes ($p < 0.05$). In the semaglutide group, these excitability measures did not differ from normal controls ($n=99$).

Conclusion: Our data suggest that the addition of semaglutide to the treatment of T2DM preserves motor axon function, possibly by improving the hyperpolarizing Na⁺/K⁺ ATPase which is critical for maintaining the ionic homeostasis during conduction.

Disclosure: Nothing to disclose.

EPO-0051 | Toward standardized multidisciplinary assessment of Charcot-Marie-Tooth: Integrating MUNIX, clinical scales and plasma biomarkers

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Background and aims: Charcot-Marie-Tooth disease (CMT) is a spectrum disorder with variable progression and severity, but reliable biomarkers for predicting severity are lacking. We assessed whether combining neurophysiological measures, plasma biomarkers, and clinical scales could identify predictors of severity across CMT subtypes.

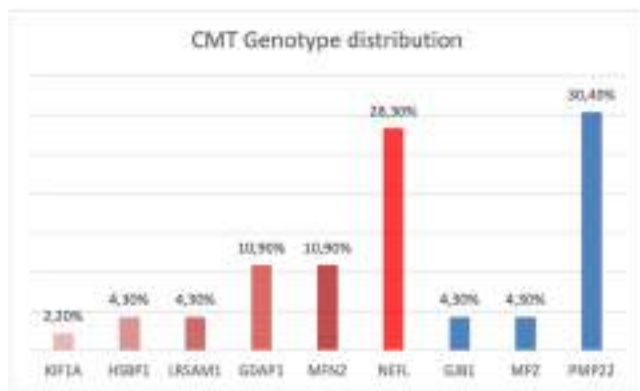


FIGURE 1 Distribution of genetic variants in a cohort of CMT patients: red bars represent axonal genotypes (HSBP1, LRSAM1, KIF1A, GDAP1, MFN2, NEFL), while blue bars indicate demyelinating ones (GJB1, MPZ, PMP22).

Methods: Forty-six genetically confirmed CMT patients performed standardized assessment at two Italian referral centres: clinical scales (CMTNSv2, NIS, ONLS, MRC, SF-36, MAM-16), neurophysiology (MUNIX, MUSIX, ulnar CMAP, radial SNAP), and plasma biomarkers (NfL, p-tau181 in $n=22$). Patients were stratified by electrophysiological phenotype (axonal 56.5%, demyelinating 43.5%). Correlations and multivariable regression identified independent predictors of clinical severity.

Results: MUNIX score was significantly lower compared to normal reference (57.7 ± 34.2 , $p < 0.001$) and negatively correlated with clinical disability measures. Axonal phenotype had higher MUNIX (69.7 ± 33.2) than demyelinating phenotype (40.2 ± 28.4 , $p = 0.005$). NfL and p-tau181 were elevated in 17.4% and 22.7% patients, respectively, but did not correlate with severity. Multivariable regression showed MUNIX and disease duration as independent severity predictors ($R^2 = 0.650$, $p = 0.011$). Phenotype-stratified models found strong prediction in demyelinating forms ($R^2 = 0.862$, $p = 0.037$) in contrast to axonal ones ($R^2 = 0.530$, $p = 0.450$).

Conclusion: MUNIX emerges as the most robust objective neurophysiological predictor of clinical severity in CMT. Phenotype-specific assessment is essential: demyelinating forms benefit from MUNIX-based monitoring with robust predictive models, more than axonal phenotypes. Plasma biomarkers (NfL, p-tau181), although elevated, probably represent acute rather than chronic disease pathophysiology and merit longitudinal evaluation alongside serial MUNIX measurements. This integrated phenotype-stratified multimodal approach requires prospective validation for individualized outcome assessment and monitoring in genetically diverse CMT.

Disclosure: Nothing to disclose.

EPO-0052 | Unmasking POEMS syndrome: The diagnostic value of clinical red flags, PEST criteria and nerve conduction studies

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Background and aims: POEMS syndrome is a rare paraneoplastic disorder frequently misdiagnosed as Chronic Inflammatory Demyelinating Polyneuropathy (CIDP). We aim to distinguish both entities by analysing PEST criteria (Papilloedema, Extravascular volume overload, Sclerotic bone lesions, Thrombocytosis), the Nerve Conduction Study (NCS) pattern and specific clinical features.



FIGURE 1 Papilloedema.



FIGURE 2 Tc-99m scintigraphy demonstrates axial osteoblastic lesions.

Methods: We conducted a descriptive retrospective study of five patients with confirmed POEMS syndrome, initially misdiagnosed as CIDP in four of them. We assessed mandatory criteria, Vascular Endothelial Growth Factor (VEGF) levels, PEST domains and NCS pattern.

Results: Mean age was 45.6 ± 15.8 years. All patients fulfilled mandatory criteria of polyneuropathy and monoclonal gammopathy (IgG lambda-restricted in 80%), alongside elevated VEGF (>1000 pg/mL). Clinically, all patients showed refractoriness to immunoglobulins and 80% ultimately required autologous stem cell transplantation. Motor predominance was distinctively observed (40%) with predominant distal lower limbs weakness. NCS revealed a demyelinating background consistently associated with an axonal component, which was the predominant feature in one patient (20%). Regarding PEST criteria, papilloedema was present in 40% (Figure 1), whereas the triad of extravascular volume overload, sclerotic bone lesions (axial predominance) (Figure 2) and thrombocytosis was universally present. Hypoalbuminaemia was identified as a frequent supportive biochemical finding (40%).

Conclusion: In patients with suspected CIDP, evaluation of PEST criteria is crucial, particularly in treatment-refractory cases. Furthermore, motor-predominant polyneuropathy or an associated axonal component on NCS should prompt VEGF testing and serum protein electrophoresis to rule out POEMS syndrome.

Disclosure: Nothing to disclose.

EPO-0053 | Distinctive characteristics of acute-onset chronic inflammatory demyelinating polyneuropathy: A multicenter cohort study

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Background and aims: Approximately 16% of patients with chronic inflammatory demyelinating polyneuropathy (CIDP)

present with a rapidly progressive course, reaching clinical nadir within eight weeks (acute-onset CIDP, A-CIDP). Limited data exists regarding the pathophysiological and clinical differences compared with classical-onset CIDP. This study aimed to compare these two entities.

Methods: National, multicenter, retrospective study. Demographic, clinical, laboratory, and neurophysiological data were collected from electronic medical records of patients with A-CIDP and CIDP and compared between groups.

Results: Eighty-three patients with typical, multifocal, or motor-predominant CIDP were included; 57.8% were male, median age was 51 (IQR 41–61) years and median follow-up was 72.0 (IQR 40.0–138.5) months. Twenty of these patients (24%) had A-CIDP. Median age at presentation did not differ between groups (50[30–60] vs 53[43–61] years; $p=0.28$). A-CIDP patients more frequently exhibited hypo/areflexia at presentation (95.0% vs 29.4%, $p=0.02$) and fewer sensory symptoms (at presentation: 75.0% vs 93.4%, $p=0.06$; at last visit: 45.0% vs 73.8%, $p=0.04$). At diagnosis, total leukocyte counts ($8.30 \times 10^9/L$ [7.12–9.45] vs $6.72 \times 10^9/L$ [5.40–7.90], $p=0.01$) and neutrophil counts ($5.24 \times 10^9/L$ [4.70–6.89] vs $3.84 \times 10^9/L$ [3.01–5.30], $p=0.02$) were higher in A-CIDP. No significant differences were observed in cerebrospinal fluid protein levels or nerve conduction studies' parameters. Immunoglobulin therapy was most frequently used in both groups, followed by oral corticosteroids and rituximab. Overall, A-CIDP patients more often achieved an objective treatment response, which was significant for oral corticosteroid therapy (88.90% vs 50.00%, $p=0.04$).

Conclusion: In our cohort A-CIDP is characterized by more frequent hypo/areflexia, fewer sensory symptoms, higher inflammatory hematological markers, and a better treatment response, particularly to oral corticosteroids.

Disclosure: Nothing to disclose.

EPO-0054 | Phase 2 trial of Riliprubart in CIDP: Patient-reported quality-of-life, pain and fatigue outcomes at week-76

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Background and aims: People living with chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) experience fatigue, weakness and sensory abnormalities, affecting quality-of-life (QoL). Riliprubart, a first-in-class, humanized, IgG4-monoclonal antibody, selectively inhibits activated-C1s within the classical complement pathway and is self-administered subcutaneously via auto-injector. A Phase-2 trial (NCT04658472) evaluating riliprubart in CIDP demonstrated decreased fatigue-severity and improved QoL at week-48. Here we assess effect of riliprubart on fatigue, pain and QoL up to week-76.

Methods: Open-label trial evaluating riliprubart across three groups: Standard-of-Care(SoC)-Treated ($N=48$), SoC-Refractory ($N=18$), and SoC-Naïve ($N=12$). Participants underwent 24-week treatment (Part-A), followed by optional treatment-extension(Part-B:52-weeks). At week-76, changes from baseline in health-related QoL were descriptively analyzed using EuroQol-Visual Analogue Scale(EQ-VAS; range 0–100, higher score indicate better outcomes) and health utility index value(range <0[as bad or worse than dead] to 1[full-health]), pain through EQ-5D-5L Dimension(range 1[no pain/discomfort] to 5[extreme pain/discomfort]), and fatigue using Modified Rasch-built Fatigue Severity Scale (R-FSS; range: 0–21, higher score indicate greater fatigue-severity).

Results: At week-76, QoL, pain and fatigue were assessed in 37 SoC-Treated, 11 SoC-Refractory and 6 SoC-Naïve participants. Mean[SD] change from baseline in EQ-VAS(SoC-Treated:12.1[18.4], SoC-Refractory:19.1[21.8], SoC-Naïve:8.5[16.2]) and health utility index value(SoC-Treated:0.18[0.28], SoC-Refractory:0.21[0.18], SoC-Naïve:0.05[0.22]) suggested QoL improvement across all groups. Higher proportion reported no pain versus baseline(SoC-Treated:27.0%[10/37] vs 12.8%[6/47], SoC-Refractory:45.5%[5/11] vs 16.7%[3/18], SoC-Naïve:33.3%[2/6] vs 16.7%[2/12]). Mean[SD] R-FSS changes indicated reduced fatigue across SoC-Treated (–3.7[7.6]), SoC-Refractory (–2.2[9.5]), and SoC-Naïve (–3.3[4.2]) groups.

Conclusion: Riliprubart indicated sustained decrease in fatigue and pain, and improved QoL in people with CIDP, along with clinical improvement. Two randomized, placebo-controlled Phase-3 studies (MOBILIZE, VITALIZE) are ongoing to confirm these findings.

Disclosure: Shahram Attarian: Consultant with Alexion, argenx, UCB, Janssen, Hansa Biopharma, Roche, Sanofi, Amicus, LFB, Alnylam, Astrazeneca, Pfizer and Biogen. Hans-Peter Hartung: Consultant with Sanofi and Octapharma. He has received fees for serving on Steering and Data Monitoring Committees from Biogen, BMS Celgene, GeNeuro, Merck, Novartis, Octapharma, Roche, and TG Therapeutics. Yi Lu, Miguel Alonso-Alonso, Asif Paker: Employees of Sanofi and may hold shares and/or stock options in the company.

EPO-0055 | Riliprubart in CIDP: Time and magnitude of response analysis from a phase 2 trial

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Background and aims: Riliprubart, a first-in-class humanized IgG4-monoclonal antibody, selectively inhibits activated-C1s in classical complement pathway and can be

self-administered subcutaneously via auto-injector. In Phase-2 trial (NCT04658472), riliprubart demonstrated favorable safety and encouraging efficacy in chronic inflammatory demyelinating polyradiculoneuropathy (CIDP). Here, we present time and magnitude of response analyses to characterize clinical benefits.

Methods: The open-label Phase-2 trial evaluated riliprubart across three groups: Standard-of-care (SoC)-Treated, SoC-Refractory, and SoC-Naïve. Participants received 24-week treatment (Part-A), followed by an optional 52-week treatment-extension (Part-B). Primary endpoint (Part-A) evaluated percentage of participants relapsing in SoC-Treated (≥ 1 -point increase in adjusted Inflammatory Neuropathy Cause and Treatment [INCAT] disability score) and percentage of participants responding in SoC-Refractory/Naïve groups (≥ 1 -point decrease in adjusted INCAT score). This post-hoc analysis focused on INCAT responders (≥ 1 -point decrease) in Part-A, and evaluated time-to-first-response, and proportion of participants with deep response (≥ 2 -point decrease), overall, and by baseline INCAT score (3–4 versus ≥ 5).

Results: Among 43 INCAT responders in Part-A, first INCAT response was observed as early as Week-2 with $\geq 44\%$ of first responses achieved by Week-4, and $\geq 89\%$ by Week-12 in all three groups. At the last INCAT assessment through Week-24, 44% (19/43) responders achieved deep response with higher rates in participants with severe baseline impairment (INCAT ≥ 5 : 69% [11/16]) versus moderate impairment (INCAT 3–4: 38% [8/21]).

Conclusion: Riliprubart provided rapid and substantial improvement in INCAT score with majority of responders achieving first response by Week-12 and nearly half of responders achieving deep response (≥ 2 -point decrease); supporting Phase-3 development of riliprubart as a potential treatment for people with CIDP.

Disclosure: Richard A. Lewis: Consultant with CSL Behring, Grifols, Pfizer, Sanofi (Steering Committee), argenx, Pharnext, Roche, Johnson & Johnson, Takeda, Boehringer Ingelheim (DSMB), and Momena. He is also part of the scientific advisory boards Alnylam and Akcea and medical advisory board The GBS-CIDP Foundation International. Jie Lin: Nothing to disclose. Annie Dionne: Received honoraria from argenx and Alexion for conference and ad board. Yi Lu, Miguel Alonso-Alonso, Asif Paker: Employees of Sanofi and may hold shares and/or stock options in the company. Luis Querol: Received research grants from Instituto de Salud Carlos III – Ministry of Economy and Innovation (Spain), CIBERER, Fundació La Marató, GBS-CIDP Foundation International, UCB and Grifols. He received speaker or expert testimony honoraria from CSL Behring, Novartis, Sanofi, Merck, Annexon, Alnylam, Biogen, Janssen, Lundbeck, argenx, UCB, Dianthus, LFB, Avilar Therapeutics, Octapharma and Roche. He serves at Clinical Trial Steering Committee for Sanofi and was Principal Investigator for UCB's CIDP01 trial.

EPO-0056 | GM1 neuropathies from antibody to phenotype

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Background and aims: Multifocal motor neuropathy (MMN) is a chronic immune-mediated, purely motor neuropathy

associated with GM1 antibodies. The underlying immune mechanisms, particularly the origin, clonality, and pathogenic role of GM1-specific B cells, remain incompletely understood. This study aims to characterize the cellular origin and functional relevance of GM1 antibodies to inform more targeted therapeutic strategies.

Methods: Thirty-five MMN patients were clinically assessed using MRC-SS, ONLS, and MMN-RODS scores. Paraproteins and light-chain restriction were evaluated by serum protein electrophoresis, ELISA, and high-resolution mass spectrometry. Peripheral blood mononuclear cells were analysed by flow cytometry, followed by bulk and single-cell sorting and in vitro culture to assess GM1-specific IgM production. The pathogenic potential of patient-derived antibodies was evaluated using human iPSC-derived neuromuscular organoids.

Results: MMN patients showed an initial response to treatment, followed by functional decline after approximately seven years. GM1 antibodies were detected in 57% of patients, and evidence of B-cell clonality was observed in 35%. Mass spectrometry identified additional IgM paraproteins in MMN sera. GM1 reactivity was present in both IgD⁺ and IgD[−] memory B-cell subsets, suggesting an early origin of autoreactivity involving defects in central and early peripheral tolerance rather than germinal-centre-dependent somatic hypermutation. Single-cell B-cell receptor sequencing is ongoing to further test this hypothesis. In neuromuscular organoids, patient-derived IgM impaired muscle contraction, supporting a direct pathogenic effect.

Conclusion: MMN is characterized by a clonally expanded, GM1-reactive B-cell response with direct pathogenic potential. These findings provide insight into disease initiation and persistence and support the development of targeted B-cell-directed therapies.

Disclosure: Nothing to disclose.

EPO-0057 | Efficacy and safety of Tafamidis in hereditary TTR amyloidosis and mixed phenotypes—the effect of the highest dose in the progression of neuropathy

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Background and aims: Hereditary transthyretin amyloidosis (ATTRv) may present with neuropathic, cardiac, or mixed phenotypes. Tafamidis, a transthyretin stabilizer, is approved at a dose of 20 mg/day for the treatment of stage 1 ATTRv neuropathy. The 61 mg/day dose has been approved for the cardiac phenotype, although its impact on neuropathic progression in mixed phenotype has not yet been established.

Methods: To evaluate the rate of neuropathy progression and changes in nutritional status in patients with a mixed phenotype previously treated with Tafamidis 20 mg, after dose escalation to Tafamidis 61 mg. An ambispective observational study was conducted, including 18 patients with a mixed phenotype who were treated with Tafamidis 61 mg for at least 12 months and had previously received Tafamidis 20 mg for the same minimum

period. Four patients were excluded due to temporal criteria. Neuropathy progression was assessed using the Neuropathy Impairment Scale (NIS), while nutritional status was monitored using body mass index (BMI).

Results: Patients treated with Tafamidis 61 mg showed slower neuropathy progression than those treated with Tafamidis 20 mg, although no statistically significant difference was reached (annual change in total NIS = 1.41 vs. 2.71; $p = 0.55$). Additionally, a potential benefit in nutritional status was observed in patients treated with Tafamidis 61 mg (annual change in BMI = 0.86 vs. −0.04; $p = 0.02$).

Conclusion: Dose escalation to Tafamidis 61 mg did not result in a statistically significant reduction in neuropathy progression. However, nutritional status appears to be better preserved at this dosage. Interpretation is limited by the small sample size.

Disclosure: Nothing to disclose.

EPO-0058 | Immunological profiling of skin biopsies in patients with idiopathic small fiber neuropathy

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Background and aims: Small fibre neuropathy (SFN) is a heterogeneous group of disorders characterized by selective involvement of thinly myelinated A-delta and unmyelinated C fibres. Although multiple aetiologies have been described, up to 50% of cases remain classified as idiopathic (iSFN). Immunological characterisation of skin biopsies in iSFN may help define disease subtypes and clarify underlying pathogenic mechanisms.

Methods: A pilot study analysed distal and proximal skin biopsies from 10 biopsy-confirmed SFN patients and distal biopsies from 5 healthy controls. Immunohistochemistry was performed for IgG, IgM, IgA, CD45, and C5b9; CD45-positive biopsies were further stained for CD4, CD8, and CD20. Double immunofluorescence was used to assess Langerhans cells (CD207) and intraepidermal nerve fibre density (IENFD) using PGP9.5.

Results: Most patients were male (70%), with a mean age of 47 years (32–64). The majority had iSFN, while three patients

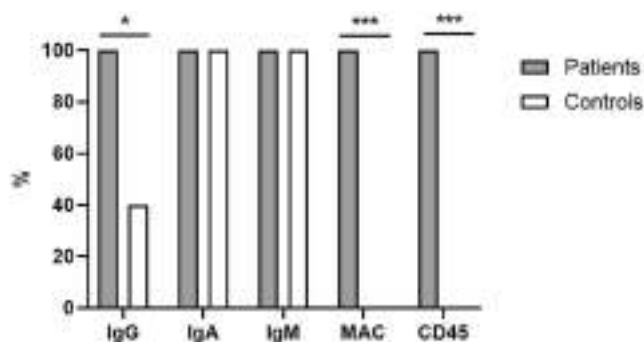


FIGURE 1 Immunohistochemistry findings in patients and healthy controls.

TABLE 1 Immunohistochemistry characterisation in CD45+ patients.

Marker	Distal+	Proximal+	Pattern (Distal)
CD4+	9/9	9/9	Diffusely, T of B cells
CD8+	9/9	9/9	All infiltrative
CD20+	3/10	0/9	All infiltrative

had secondary SFN (monoclonal gammopathy, diabetes with rheumatoid arthritis, and Sjögren). All SFN patients showed reduced distal IENFD. C5b9 deposits and leukocyte infiltration, predominantly CD8+ T cells, were observed exclusively in SFN patients. Leukocyte infiltration was present in both distal and proximal skin, suggesting immune activation beyond distal sites of fibre loss. IgG deposition was significantly increased in SFN patients, whereas no differences were found for IgM, IgA, or Langerhans cell density.

Conclusion: Consistent immune alterations in proximal and distal skin biopsies from both idiopathic and autoimmune-associated SFN patients support a primary role for localized immune dysregulation in SFN pathogenesis. The ongoing study includes 50 patients, and final results will be presented at the congress.

Disclosure: Nothing to disclose.

Cognitive Neurology/Neuropsychology 1

EPO-0059 | The impact of cognitive reserve on neuropsychological profiles in different etiologies of dementia

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Background and aims: Dementia is characterized by a progressive decline in cognitive functions. Different lifelong activities contribute to build a cognitive reserve (CR), which can influence cognitive decline trajectory. This study aims to investigate the effect of CR on neuropsychological performances in patients with dementia.

Methods: We enrolled 61 patients affected by Alzheimer's disease (AD) dementia, Vascular dementia (VaD) and Mixed forms. The sample included 28 Females (46%). Mean ($\pm$ SD) age was 73.96 ± 7.00 years, while mean ($\pm$ SD) education years were 11.38 ± 3.75 . Neuropsychological assessment included the Mini Mental State Examination (MMSE), Digit Span, Trial Making Test (TMT-A and B), Rey Auditory Verbal Learning Test (RAVLT; Immediate and Delayed) and Verbal Fluency (Phonemic and Semantic). Cognitive reserve was assessed using the Cognitive Reserve Index Questionnaire (CRI-q). Patients were classified as High CR (HCR) or Low CR (LCR) according to an established cut-off value.

Results: Significant differences between the two groups were observed in Immediate-RAVLT ($U=262.5$; $p=0.043$, $HCR=30.316 \pm 9.417$; $LCR=24.450 \pm 7.891$) and in Phonemic Fluency ($U=133.0$; $p=0.044$; $HCR=35.571 \pm 11.501$; $LCR=25.462 \pm 12.231$). Spearman's correlation revealed

positive correlations between the total score of CRIq and MMSE, Immediate-RAVLT and Phonemic Fluency.

Conclusion: HCR group showed better performances in short-term memory and language. These findings emphasize the modulating and protective role of cognitive reserve on cognitive functioning and suggest that specific neuropsychological tests may be particularly sensitive in detecting its effects.

Disclosure: Nothing to disclose.

EPO-0060 | Predicting neurocognitive disorder development in multiple sclerosis using artificial intelligence on MRI and clinical data

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Background and aims: Neurocognitive disorders (NDs) are often under-recognized symptoms of multiple sclerosis (MS). An early prediction of ND in people with MS is crucial for implementing effective early interventions. This study aimed to develop an artificial intelligence (AI) neural network to predict ND in MS patients after at least one year, using baseline demographic, clinical, and MRI data.

Methods: 3D T1-weighted brain MRIs of 223 MS patients were collected at baseline with demographics, clinical data, and cognitive reserve proxies. At baseline and after a median follow-up of 1.5 years, each patient underwent cognitive assessment and was classified as having mild ND or major ND according to the DSM-5-TR manual. Patients whose cognitive status decayed over time were classified as worsened. A deep learning model used a 10-layer ResNet to extract MRI features, combined with non-imaging data (demographics, clinical details, lesion load, brain volumes) to classify patients as cognitively preserved or impaired. Performance was evaluated using 5-fold cross-validation.

Results: At follow-up, 27 out of 224 MS (12%) experienced cognitive worsening. AI model achieved a mean validation accuracy of 90%, F1-score of 81% and average AUC of 0.89. Grad-CAM heatmaps identified the frontal, occipital, middle temporal gyri, and hippocampus as key brain regions, while important tabular features included cortical gray matter volume, age, thalamic and hippocampal volumes, T2 lesion volume, and cognitive reserve score.

Conclusion: AI, mainly based on MRI data, can effectively predict development of NDs, even within a short follow-up period, enabling timely identification and management of MS patients at risk of worsening.

Disclosure: D. Mistri has nothing to disclose. L. Storelli received speakers' honoraria from Biogen. A. Mastropasqua, M. Grosselle have nothing to disclose. P. Preziosa received speaker honoraria from Roche, Biogen, Novartis, Merck, Bristol Myers Squibb, Genzyme, Horizon and Sanofi. G. Mazzetti and L. Rossi have nothing to disclose. M. Filippi is Editor-in-Chief of the Journal of Neurology, Associate Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmed Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. M.A. Rocca received consulting fees from Biogen, Bristol Myers Squibb, Roche; and speaker honoraria from Alexion, Biogen, Bristol Myers Squibb, Celgene, Horizon Therapeutics Italy, Merck Serono SpA, Mitsubishi-Tanabe Pharma, Neuraxpharm, Novartis, Roche, Sandoz, and Sanofi. She receives research support from the MS Society of Canada, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. She is Associate Editor for Multiple Sclerosis and Related Disorders; and Associate Co-Editor for Europe and Africa for Multiple Sclerosis Journal.

EPO-0061 | Cognitive impairment in multiple sclerosis: Correlation between brain atrophy measures and Raos's brief repeatable battery

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Background and aims: Multiple sclerosis (MS) is a chronic, neurodegenerative disease of the central nervous system that progressively leads to increasing disability. The prevalence of cognitive impairment (CI) in people with MS (pwMS) ranges from 40% to 65%. Structurally, both brain atrophy and lesion load, together with disease duration, are associated with CI. Our objective was to analyze the relationship between MRI brain atrophy measures and performance on the neuropsychological tests of Rao's Brief Repeatable Battery (BRB) in pwMS.

Methods: We studied 95 pwMS with cognitive complaints, including both relapsing and secondary progressive MS phenotypes. Brain atrophy measures on MRI included the corpus callosum index (CCI), third ventricle width (3VW), and the bi-caudate ratio (BCR). T2 lesion load was also quantified. The BRB consisted of the following tests: the Selective Reminding Test (SRT), the 10/36 Spatial Recall Test (SPART), the Symbol Digit Modalities Test (SDMT), the Paced Auditory Serial Addition Test (PASAT), and the Word List Generation Test (WLG).

TABLE 1 Correlations between brain atrophy indices and neuropsychological tests.

Table. Age-controlled partial correlations between brain atrophy indices and neuropsychological tests.			
	3VW	CCI	BCR
	(p value)	(p value)	(p value)
SRT-LTS	0.002	- 0.081	0.011
	(0.989)	(0.607)	(0.943)
SRT-CLTR	- 0.084	- 0.053	- 0.027
	(0.593)	(0.734)	(0.862)
SRT-D	- 0.168	0.064	- 0.087
	(0.283)	(0.685)	(0.578)
SPART	-0.111	0.212	- 0.225
	(0.477)	(0.173)	(0.146)
SPART-D	0.064	0.068	0.031
	(0.683)	(0.664)	(0.844)
SDMT	- 0.340	0.382	- 0.418
	(0.026)	(0.011)	(0.005)
PASAT-3	- 0.170	0.315	- 0.323
	(0.277)	(0.040)	(0.035)
WLG	- 0.040	0.023	- 0.267
	(0.801)	(0.884)	(0.083)

LTS: Long Term Storage. CLTR: Consistent Long Term Retrieval. D: Delayed

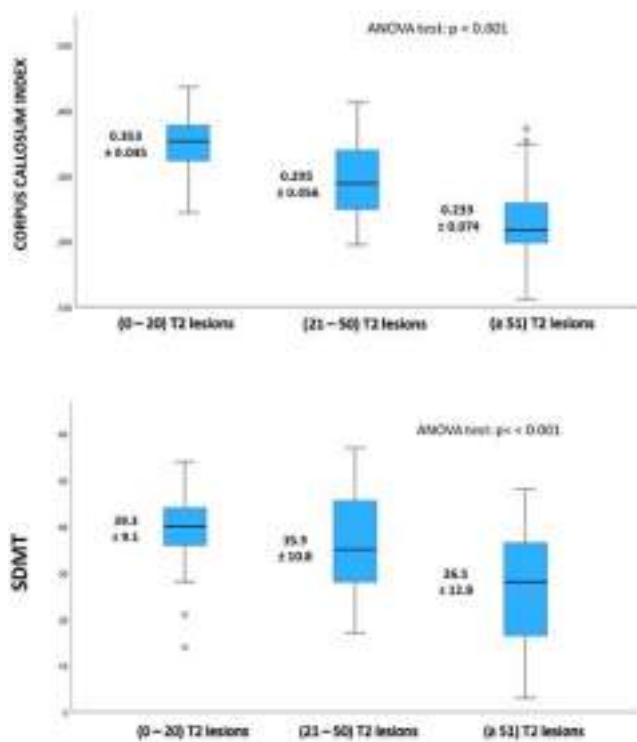


FIGURE 1 T2 Lesion Load, brain atrophy and SDMT.

Results: Sex: 69F/26M. Age (years): 50.1 ± 8.5. EDSS: 3.2 ± 2.1. Age was significantly correlated with the brain atrophy indices. The Table 1 shows the partial correlations, controlling for the effect of age, between the brain atrophy indices and the BRB. Brain atrophy measures were significantly higher in progressive forms. Lesion load was associated with greater atrophy levels and poorer cognitive performance (Figure 1).

Conclusion: In our cohort of pwMS, brain atrophy measures were inversely correlated with performance on the SDMT and

PASAT test, both reflecting information processing speed. Cognitive impairment was influenced by brain atrophy, lesion load, and progressive disease phenotypes.

Disclosure: Nothing to disclose.

EPO-0062 | Functional brain activation and reorganization before and after tailored speech language therapy in PPA: A fMRI study

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Background and aims: This study investigated functional MRI (fMRI) activation in language tasks before and after tailored speech and language therapy (SLT) targeting lexical access deficits in semantic (svPPA) and logopenic (lvPPA) variants of Primary Progressive Aphasia (PPA), and articulation deficits in nonfluent/agrammatic (nfvPPA) cases.

Methods: Ten PPA patients (5 lvPPA, 3 svPPA, 2 nfvPPA) underwent pre-treatment MRI, and post-treatment scans after five weeks of SLT. fMRI tasks were tailored to treatment: svPPA and lvPPA performed a silent confrontation naming (CN) and nfvPPA completed a speech-observation (SO). Eight healthy controls (HC) underwent fMRI tasks at baseline only. Baseline group-level comparisons and longitudinal analyses examined post-treatment changes in patients were conducted.

Results: At baseline, during naming and compared to HC, PPA patients showed reduced activation in bilateral superior temporal regions, right supplementary motor area (SMA), and right superior frontal cortex, and increased activation in right anterior cingulate cortex and in left inferior temporal and parietal

regions, mainly in lvPPA. Longitudinally, naming-related activation increased post-treatment in bilateral superior temporal cortex. During the SO task at baseline, compared to HC, nfvPPA cases showed reduced activation in bilateral cingulate and inferior frontal cortices, and right sensorimotor regions, alongside increased activation in right inferior occipital cortex. Qualitative single-case analyses revealed a greater recruitment of bilateral SMA after treatment.

Conclusion: Findings suggest that tailored SLT in PPA induces changes in functional brain activation, reflecting treatment-related neuroplasticity. Furthermore, SLT may enhance compensatory activation patterns already evident at baseline. Task-based fMRI may help capture differential treatment responsiveness and monitor SLT effects in PPA.

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EPO-0063 | Mirroring emotional faces in the developing brain: Functional connectivity of the mirror neuron system

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Background and aims: The Mirror Neuron System (MNS) underlies important neurocognitive functions, such as imitation learning, social cognition, and empathy, and may facilitate the recognition of emotional expressions via motor simulation. However, its development remains poorly understood. This study investigated MNS functional connectivity maturation between early childhood and adulthood during an emotional faces task.

Methods: Using the innovative magnetoencephalography approach of optically pumped magnetometers (OPMs), we assessed whole-brain connectivity of 8 bilateral regions of interest (ROIs) comprising the MNS in 57 typically developing children aged 3-to-5 years and 27 adults. We applied both classical network-based statistic (NBS) and its more recent approach incorporating machine learning, NBS-Predict.

Results: We observed networks of increased connectivity after face presentation in both adults and children in broadband, as well as in the alpha and beta bands; only adults recruited a theta band network. The ROIs were functionally linked to key limbic, prefrontal, parietal and occipital areas. Noteworthy, adults exhibited stronger right-hemisphere broadband and theta connections than children. Results were validated by NBS-Predict, with a comparable subnetwork exhibiting predictive features for classification between children and adults.

Conclusion: Results suggest consolidation of right-sided MNS connections through to adulthood, consistent with right-hemisphere specialization in emotional processing.

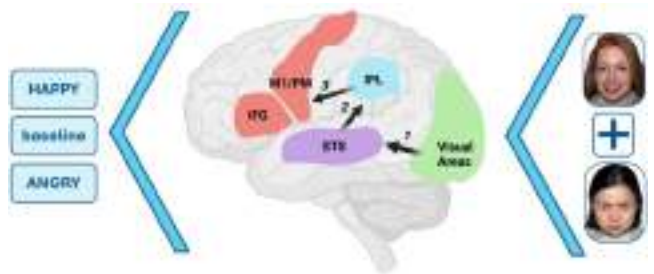


FIGURE 1 Processing of visual inputs through the key hubs of the MNS facilitates the recognition of others' emotions via motor simulation of the observed faces. STS: superior temporal sulcus. IPL: inferior parietal lobe. M1/PM: primary motor and premotor cortices.

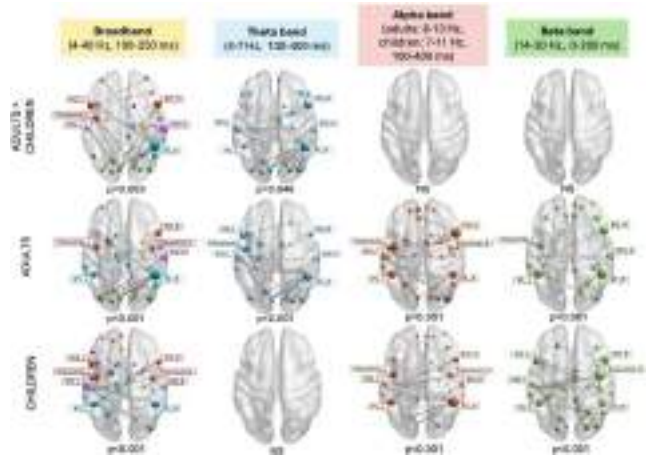


FIGURE 2 Total NBS output. Both the within-group analysis (Adults or Children; pcorrchildren; pcorr=0.003 in the broadband).

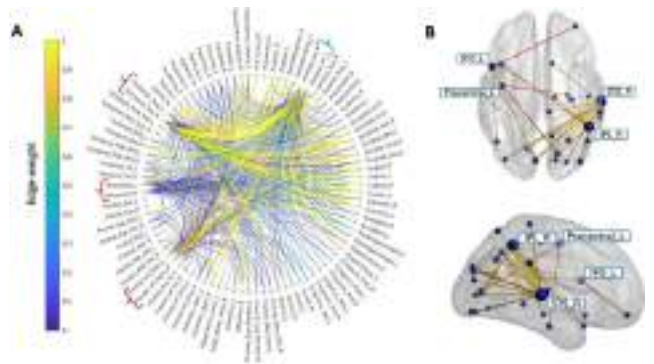


FIGURE 3 A. Overall NBS-Predict output (300-edge network). Colours, from blue (=0) to yellow (=1), indicate the increasing relevance of the edge in the model prediction performance ($p=0.005$). B. 27-edge sub-network with weight-thresholded connections (>0.95), in ax.

Disclosure: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work. Funding was provided by the Canadian Institutes of Health Research (PJT-178370) and the Simons Foundation Autism Research Initiative (SFARI; 2021 Human Cognitive and Behavioural Science award). FF was supported by the Sant'Anna School of Advanced Studies, Pisa, Italy.

EPO-0064 | Multilingual co-creation of aphasia topics priorities through patient and public involvement

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Background and aims: People living with chronic aphasia face persistent challenges. It is essential to involve people with aphasia (PWAs) directly in the identification of thematic priorities concerning their quality of life. The aim is to share research agenda with PWA and health professionals across multiple countries and languages. i) evaluate the participation and adherence of strongly involved PWA and ii) collaboratively identify and prioritize topics of greatest importance to PWA through a multilingual PPI process for orienting future research.

Methods: The project involved more than 100 PWA from 14 countries, with 11 countries contributing to voting. The three-phase process included (1) an online consultation phase to generate initial topics, (2) a development phase through national-level codesign sessions to refine and rank topics and that concluded with international voting of four top priorities, and (3) a multilingual dissemination phase. Aphasia-friendly materials and real-time translation ensured accessibility.

Results: PWA played key roles in proposing topics, organizing and summarizing national and international voting and promoting dissemination. Eleven out of the 14 participating countries (78%) voted. Four priorities emerged: (1) raising awareness of aphasia among families and society; (2) psychological changes, including impacts on intimacy and relationships; (3) rebuilding

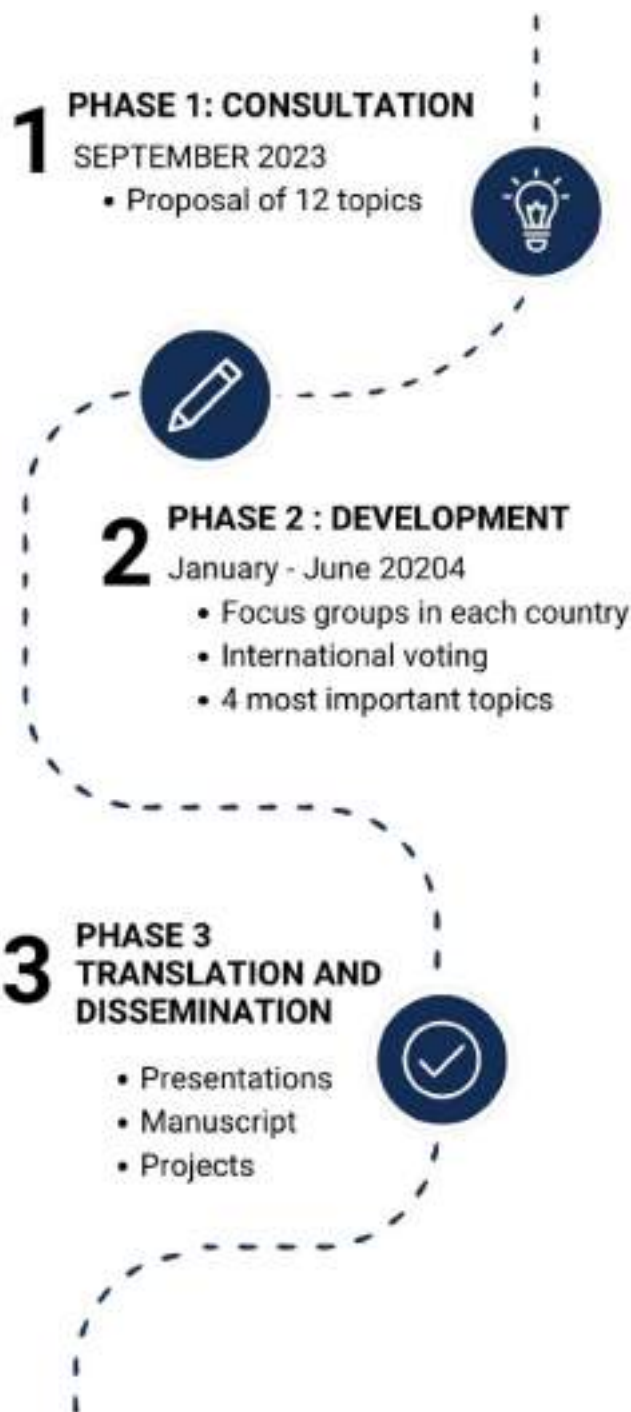


FIGURE 1 Roadmap timeline.



FIGURE 2 The twelve initial topics.



FIGURE 3 The four important topics.

self-confidence after aphasia; and (4) improving therapy and hospital attitudes towards treatment.

Conclusion: This study shows that with the right help, people with aphasia can clearly share what matters to them. Their voices highlight not only medical needs but also the full picture of living with aphasia. They can guide researchers, health professionals and policy-makers in their decisions.

Disclosure: Nothing to disclose.

EPO-0065 | Non-motor features: Distinguishing dementia with Lewy bodies from Alzheimer's and amyloid co-pathology

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Background and aims: Dementia with Lewy bodies (DLB) presents a higher prevalence of non-motor symptoms than Alzheimer's disease (AD). This study aimed to compare non-motor symptoms between DLB and AD, assess the impact of comorbidity and examine correlations between AD biomarkers and symptoms.

Methods: This cross-sectional study examined patients with DLB, AD and healthy controls. Data collected included demographic information, clinical characteristics, CSF AD

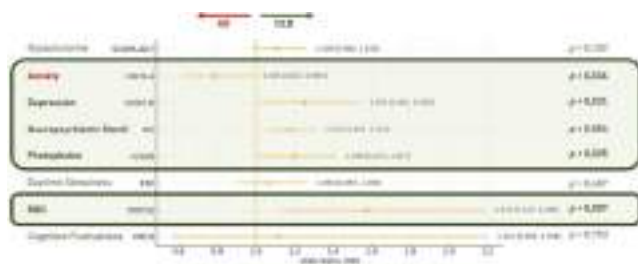


FIGURE 1 Non-motor clinical predictors for differentiating DLB from AD.

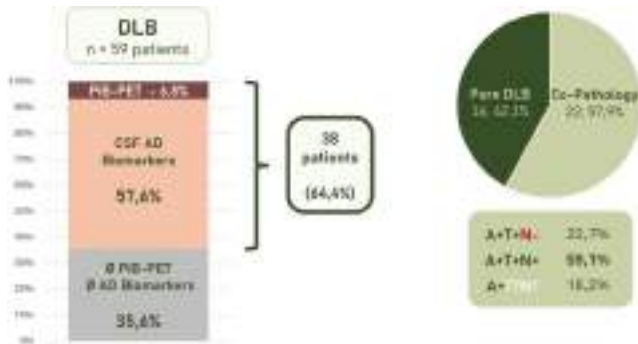


FIGURE 2 Characterization of AD co-pathology in a DLB cohort using CSF and PiB-PET biomarkers.

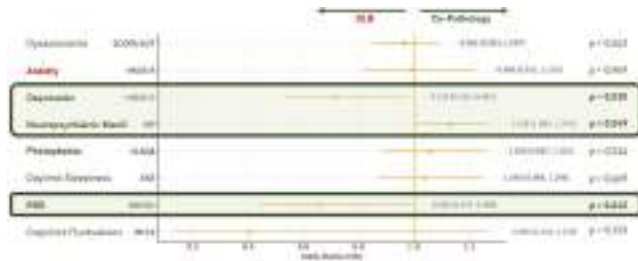


FIGURE 3 Non-motor clinical predictors for differentiating DLB from myloid co-pathology.

biomarkers and results from evaluations using scales to assess non-motor symptoms.

Results: A total of 134 participants were included (44.0% DLB, 40.3% AD, and 15.7% controls). After adjusting for confounders, DLB patients scored significantly higher than AD patients across all scales, except for anxiety. Logistic regression identified the following independent predictors of DLB versus AD: REM Sleep Behavior Disorder (RBD) (OR 1.574), depression (OR 1.253), photophobia (OR 1.198), and psychopathology (OR 1.174), while anxiety was associated with AD (OR 0.783). Regarding co-pathology, it was present in 57.9% of DLB cases. Patients with co-pathology exhibited lower depression scores ($p=0.029$). While psychopathology predicted co-pathology (OR 1.128), depression (OR 0.038) and RBD (OR 0.042) were more strongly associated with pure DLB. Regarding biomarkers, the Aβ42/40 ratio correlated positively with depression scores, whereas Tau and p-Tau181 levels correlated positively with psychopathology and negatively with depression scores.

Conclusion: DLB patients present a higher burden of non-motor symptoms compared to AD. Although photophobia is not currently a supportive clinical feature for DLB diagnosis, it emerged

as a significant predictor. Amyloid co-pathology appears to be linked to fewer depressive symptoms but increased psychiatric manifestations.

Disclosure: Nothing to disclose.

EPO-0066 | Right temporal variant frontotemporal dementia due to FTL-D-FET confirmed by neuropathology

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Background and aims: Frontotemporal lobar degeneration with FET protein (fused in sarcoma [FUS], Ewing sarcoma breakpoint region 1 [EWSR1], TATA-binding protein-associated factor 15 [TAF15]) inclusions (FTLD-FET) is a rare subtype of frontotemporal dementia, usually presenting with early-onset behavioural changes. Right temporal variant frontotemporal dementia (rtvFTD) is typically associated with TDP-43 type C pathology. We report a well-characterized case of a rtvFTD with post-mortem confirmation of FTLD-FET and distinctive neuroimaging features that may inform pre-mortem diagnosis.

Methods: Retrospective review of the medical history, neuroimaging and neuropathological study of a 51-year-old brain donor with atypical frontotemporal lobar degeneration with ubiquitin-positive inclusions (aFTLD-U) with FET pathology (FTLD-FET).

Results: A 45 odd-year-old man presented with early, rapidly progressive behavioural changes including apathy, disinhibition, stereotyped behaviors, altered eating habits, prosopagnosia and anosognosia with executive, language, and memory deficits. Neurological examination showed no parkinsonism or motor neuron signs. MRI revealed right-predominant frontal and temporal atrophy. FDG-PET showed bilateral frontal and anterior temporal hypometabolism, more extensive on the right, with marked right caudate hypometabolism. The patient died 5 years after the onset of the symptoms. Neuropathology revealed cytoplasmic inclusions positive for FET proteins (FUS, TAF15), transportin, as well as ubiquitin, consistent with atypical FTLD with ubiquitin-positive inclusions (aFTLD-U). Right caudate total degeneration correlated with imaging. Mild motor neuron involvement and hippocampal sclerosis were present.

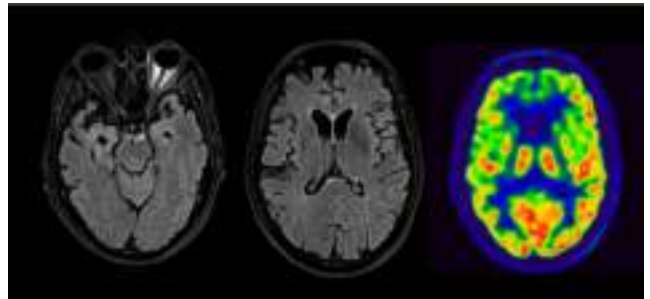


FIGURE 1 Structural MRI and FDG-PET reveal right-predominant frontotemporal atrophy and corresponding hypometabolism, with additional impairment of the caudate nucleus.



FIGURE 2 Macroscopic brain examination showing marked right-predominant frontotemporal atrophy, with noticeable volume loss.

Conclusion: This case illustrates rtvFTD with prominent striatal involvement due to FTLN-FET. Caudate degeneration on imaging may help suggest this molecular subtype and emphasize the importance of clinicopathological correlation in early-onset dementia.

Disclosure: The authors report no relevant financial or institutional conflicts of interest.

EPO-0067 | Alzheimer's disease (AD), mild cognitive impairment (MCI) and predictors of driving Cessation: A 7-year longitudinal prospective study

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Background and aims: Cognitive impairment has a detrimental effect on driving ability. We longitudinally assessed drivers with MCI or mild AD Dementia to identify diagnostic and prognostic factors of unsafe driving.

Methods: Our study sample consisted of 109 participants: 32 Controls (Mean Age 65.8years, 57% Women), 47 MCI patients (Mean Age 69.1years, 40% Women) and 30 AD patients (Mean Age 72.8years, 7% Women). Baseline evaluation included neurological and neuropsychological assessment and a driving simulator test. We performed two follow-up evaluations after a mean period of 48 and 84 months. Re-evaluations included a structured interview with patients and caregivers. Primary endpoints were driving cessation and death. We also performed a systematic review of prospective studies regarding driving cessation among patients with cognitive impairment.

Results: 21/47 MCI patients (45%, Mean time to cease: 35 months) and 25/30 AD patients (83%, Mean time to cease: 15 months) ceased driving during the follow-up period (Figure 1). 45% and 59% of the MCI and AD patients respectively had at least one dangerous driving event during follow-up, while only 12% of NC participants had a similar event. Age (HR = 1.080, $p = 0.007$),

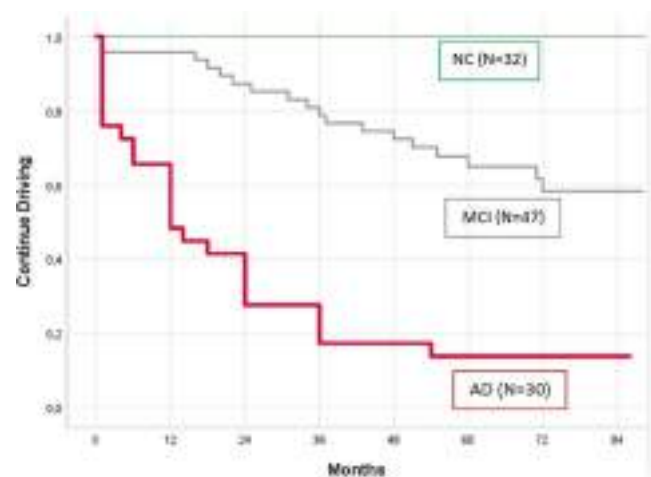


FIGURE 1 Kaplan-Meier plot for driving cessation among controls (green line), MCI (gray line) and AD patients (red line).

Semantic Verbal Fluency (HR=0.822, $p=0.001$) and modified Tandem Walking Test (HR=1.099, $p=0.043$) were significant predictors of driving cessation among the cognitively impaired participants (Cox multivariate regression analysis). Our systematic review of the literature identified nine prospective studies.

Conclusion: A multi-disciplinary evaluation (neurological, neuropsychological, driving simulator test) is crucial to individualize driving risk among patients with cognitive impairment. Large-scale, longitudinal studies are needed to establish a standard protocol of driving assessment.

Disclosure: This study is part of the Dr Stamatelos' PhD project with title "Evaluation of driving behavior of patients with MCI, Dementia or Parkinson's Disease: Diagnostic and Prognostic Markers", funded and supported by Onassis Foundation.

EPO-0068 | Lactoferrin-Quercetin-ORMOSIL nanoparticles confer neuroprotection in scopolamine-induced cognitive deficits

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Background and aims: Introduction This study aimed to develop lactoferrin (LF)-conjugated organically modified silica (ORMOSIL) nanoparticles (LF-Q-ORMOSIL) for brain-targeted delivery of quercetin (Q), evaluating their ability to enhance bio-availability, cross the blood-brain barrier (BBB), and improve cognitive outcomes in scopolamine-induced amnesia.

Methods: ORMOSIL nanoparticles loaded with quercetin were synthesized and conjugated with LF, followed by physicochemical characterization (size, zeta potential, encapsulation efficiency, release profile). In vivo behavioural assessment was done using morris water maze and novel object recognition tests, biochemical assays and acetylcholinesterase (AChE) inhibition study was performed. Histological analysis for hippocampal morphology was done and in vivo imaging for nanoformulation biodistribution was performed by IVIS imager.

Results: Results LF-Q-ORMOSIL nanoparticles were synthesized with an optimal particle size (~150 nm), high encapsulation (>80%), and sustained release. Compared to free quercetin and ORMOSIL, LF-Q-ORMOSIL significantly ameliorated scopolamine-induced cognitive deficits, reduced oxidative markers (including MDA and ROS), and restored antioxidant levels (of GSH and SOD), potentially inhibiting AChE and showing preserved hippocampal neurons. Biodistribution revealed many-fold higher brain accumulation and retention at 24 h post-administration.

Conclusion: Conclusion LF-Q-ORMOSIL nanoparticles enhance quercetin's brain delivery, neuroprotective effects, and therapeutic efficacy against cognitive impairment, and shows promise as a nanotherapeutic for Scopolamine-induced amnesia.

Disclosure: Disclosures The authors declare no conflicts of interest and received no specific funding for this study.

EPO-0069 | Longitudinal trajectories of cognitive function in heart failure and associated risk factors: A systematic review and meta-analysis

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Background and aims: We aimed to characterise temporal trajectories of cognitive outcomes and identify factors associated with cognitive functioning in heart failure (HF).

Methods: We conducted a systematic review and meta-analysis, searching four databases from inception to 6 February 2025. We included longitudinal observational studies for trajectory analyses and longitudinal observational, cross-sectional, case-control studies, post hoc analyses of randomised controlled trials (RCTs), and RCTs for risk-factor analyses. Study quality was assessed using Joanna Briggs Institute checklists. We pooled prevalence and incidence of cognitive outcomes at comparable follow-up intervals and synthesised risk estimates from cross-sectional studies with random-effects meta-analysis. Risk factors from longitudinal studies were summarised using direction-based vote counting due to limited number of studies. The protocol was registered in PROSPERO (CRD42025645235).

Results: We identified 35 longitudinal articles from 33 studies and 66 cross-sectional studies. Studies with short-term follow-up (≤ 1 year) often reported cognitive improvement, whereas longer follow-up generally showed gradual decline. Consistent modifiable risk factors across study designs included sleep problems, depression, hypertension, atrial fibrillation, inflammation, and anaemia. Other behavioural, cardiac, and systemic factors (e.g. physical capacity, perceived stress, ischaemic aetiology, cardiac index, HbA1c, serum albumin) were also associated with cognitive outcomes, although supported by limited evidence. Substantial heterogeneity was observed in quantitative analyses.



FIGURE 1 Conceptual model of associated factors for cognitive outcomes in heart failure patients.

Conclusion: Our results demonstrate an increased risk of cognitive decline during follow-up among HF patients, highlighting the need to integrate cognitive assessment into cardiac management. Sleep problems, depression, hypertension, atrial fibrillation, inflammation, and anaemia emerged as consistent modifiable risk factors and should be prioritised in interventions.

Disclosure: The authors declare no conflicts of interest.

EPO-0070 | TUSC3-deficient intellectual disability fails Mg²⁺ uptake for synaptic function and neurodevelopment

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Background and aims: Intellectual disability (ID) is characterized by deficits in cognition and adaptive behavior, with few treatment options. Tumor Suppressor Candidate 3 (TUSC3) has been genetically linked to autosomal recessive ID, but its molecular mechanism and therapeutic potential remain unclear.

Methods: TUSC3 knockout mice were generated and analyzed for their behavior, including memory function and social interaction etc. Cellular magnesium was measured with Mag-FRET assay. TUSC3 knockout mice were supplemented with magnesium or chelated magnesium for recovery of the pathology. ID- and Non-ID fibroblasts were analyzed for their ability to modulate ER magnesium. ER stress and synaptic proteins were analyzed with western blot.

Results: We show that TUSC3 is essential for endoplasmic reticulum (ER) Mg²⁺ homeostasis and neuronal function. Using a TUSC3 knockout (KO) mouse model, we find ID-like phenotypes including impairments in learning, memory, stress adaptation, and social behavior. Mechanistically, TUSC3 forms an ER-localized Mg²⁺ transport complex with ERMA and its loss leads to ER Mg²⁺ depletion, PERK-eIF2 α pathway activation, synaptic dysfunction, and neuronal vulnerability. Fibroblasts from TUSC3 mutant patients similarly exhibit ER Mg²⁺ deficiency and heightened ER stress. Magnesium supplementation restores ER Mg²⁺ levels, reduces ER stress, and rescues cognitive deficits.

Conclusion: Our findings establish ER Mg²⁺ dysregulation as a key driver of neurodevelopmental dysfunction and a promising therapeutic target for TUSC3-deficient ID.

Disclosure: Nothing to disclose.

Epilepsy 1

EPO-0071 | Gut microbiota alteration in Angelman syndrome: A case-control 16S rRNA sequencing study

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Background and aims: Angelman syndrome (AS) is a rare neurogenetic disorder caused by impaired maternal UBE3A expression and is characterized by severe intellectual disability, motor impairment, absent speech, epilepsy and behavioral abnormalities. Evidence supports a role of the gut microbiota in various neurological disorders, particularly epilepsy, through the gut-brain axis. This study aimed to investigate gut microbiota alterations, microbial diversity and metabolic profiles in AS patients.

Methods: Pediatric and adult patients with AS and age- and sex-matched healthy subjects (HS) were enrolled. Fecal samples were collected and analyzed using 16S rRNA gene sequencing to assess microbial composition, relative abundance and diversity. Short-chain fatty acids (SCFAs) were quantified to evaluate metabolomic profile by gas chromatography-mass spectrometry.

Results: Seventy-three subjects were included, comprising 42 AS patients and 31 HS. At the phylum level, AS patients showed reduced Bacteroidota and increased Firmicutes and Proteobacteria, with a higher Firmicutes/Bacteroidota ratio. Alpha diversity was comparable between groups, whereas beta diversity differed significantly between AS and HS. At the genus level, *Parasutterella*, *Tyzzelerella*, *Eubacterium hallii*, and *UBA1819* were significantly more abundant in AS, while *Eubacterium ventriosum* was more abundant in HS. Venn diagram analysis revealed greater microbial heterogeneity in AS, with 2986 exclusive taxa. SCFA concentrations were significantly reduced in AS.

Conclusion: Angelman syndrome is associated with a distinct gut microbiota alteration characterized by different taxonomic composition, increased microbial heterogeneity and reduced SCFA production, supporting a contributory role of microbiota in AS pathophysiology and suggesting microbiota modulation as a biomarker and promising adjunctive therapeutic strategies.

Disclosure: This work was supported by Angelman Syndrome Alliance (ASA), 2024 Research Grant.

EPO-0072 | Temporal encephaloceles, epilepsy and idiopathic intracranial hypertension: Toward a pressure-mediated pathogenetic model?

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Background and aims: Temporal encephaloceles (TE) are often overlooked in the context of temporal lobe epilepsy (TLE). In parallel, radiological signs of idiopathic intracranial hypertension (IIH) are increasingly described in TLE, often without typical IIH symptoms. We hypothesized that chronic intracranial pressure (ICP) may contribute causally to encephalocele formation and epileptogenesis.

Methods: A retrospective multimodal series of consecutive TLE ($n=21$) was conducted. High-resolution MRI and skull-base CT characterized TE morphology and bony defects, while scalp EEG and FDG-PET aided in lateralization. Prespecified IIH markers included empty/partially empty sella, optic nerve sheath distension, and Meckel's cave enlargement. When serial imaging became available, volumetric analysis quantified TE progression. Primary outcome was IIH marker prevalence; secondary analyses explored correlations with TE multiplicity/bilaterality, and clinical phenotype.

Results: Signs of IIH were identified in 8 of 21 patients (38%). Within this subgroup, TE more frequently demonstrated multiplicity/bilaterality, with a prevalence of 62.5% among IIH+ patients (5/8) compared with 33.3% overall (7/21). One patient showed progression in TE burden, with lesion volume increasing from ~2.4 to ~6.8 cm³. IIH+ patients had later epilepsy onset (51.5 vs 35.0 years) and a higher proportion of epigastric/autonomic features (50% vs 38.5%). TE were predominantly left-lateralized (88.2%).

Conclusion: A radiologic IIH endophenotype is frequent in TLE and correlates with lesion multiplicity, progression, and delayed seizure onset, supporting a pressure-mediated model. TLE may represent a subclinical IIH phenotype with seizures as the main clinical expression. This hypothesis opens avenues for future research, and therapeutic ICP-lowering strategies.

Disclosure: Nothing to disclose.

EPO-0073 | Features of endothelial dysfunction and vascular glycocalyx in patients with epilepsy

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Background and aims: In recent years, it has been established that endothelial dysfunction and damage to the blood-brain barrier play an important role in maintaining the epileptic process. However, molecular markers of endothelial damage in epilepsy remain insufficiently studied. In this regard, the study of endothelial and stress-associated biomarkers is of significant interest

for deepening understanding of epilepsy pathogenesis and improving diagnostic approaches.

Methods: 51 patients with an established diagnosis of epilepsy were examined, from whom venous blood collection was carried out during the period of epileptic activity and in the early post-sexual stage. The control group consisted of 20 healthy individuals without signs of neurological pathology. In blood serum samples using enzyme-linked immunosorbent assay, the concentrations of endothelial damage markers and systemic stress were determined: endocane, asymmetric dimethylarginine, differentiation growth factor-15, and syndecan-1.

Results: Analysis of the obtained data showed that in patients with epilepsy, a statistically significant increase in the levels of endocane and ADMA was registered compared to the control group, which indicates a pronounced disruption of endothelial function and a decrease in the bioavailability of nitric oxide. The concentration of GDF-15 in epilepsy patients was also significantly higher ($p < 0.005$), reflecting the activation of stress-realizing and inflammatory mechanisms. In addition, an increase in the level of syndecan-1 indicated a degradation of endothelial glycocalyx and a disruption of the vascular barrier apparatus.

Conclusion: The identified changes in biomarkers allow us to consider epilepsy as a condition accompanied by systemic endothelial damage and disruption of the integrity of the vascular barrier. Increased levels of endocane.

Disclosure: Nothing is disclosed.

EPO-0074 | Induction of ecstatic phenomena via insular direct electrical stimulation: A case series in non-ecstatic focal epilepsy

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Background and aims: Stereo-electroencephalography (SEEG) has recently advanced our understanding of ecstatic epilepsy. While the epileptogenic zone is frequently temporal, the symptomatogenic zone for ecstatic symptoms often resides in the anterior insula. However, inducing these phenomena via direct electrical stimulation (DES) in people with non-ecstatic epilepsy is exceptionally rare, with only two cases previously reported thus far in the literature. This study presents three new cases identified across multiple centers.

Methods: We retrospectively analyzed data from three patients with drug-resistant, non-ecstatic focal epilepsy undergoing pre-surgical evaluation. In these patients, 50 Hz DES of the insula induced ecstatic phenomena without triggering afterdischarges.

Results: The identified patients had reproducible ecstatic phenomena during 50 Hz DES of the insula. Patient 1 (left anterior insula, 1–1.5 mA) described a fantastic feeling like “being with her daughter a sunny summer day on a meadow full of flowers

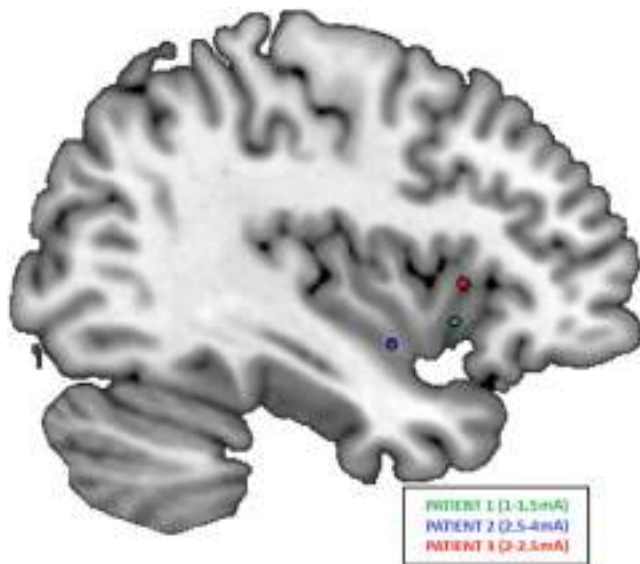


FIGURE 1 Representation of the midway location of the bipolar SEEG electrode pair evoking reproducible ecstatic symptoms for the 3 patients, projected onto a standard sagittal T1-MRI of the MNI atlas brain.

and everything is well. Patient 2 (very anterior part of the right posterior long gyrus, 2.5–4 mA) experienced being « as one with the whole universe ». Patient 3 (right dorsal anterior insula, 2–2.5 mA) reported intense serenity and happiness. The second patient had a hippocampal sclerosis while the two others were MRI-negative.

Conclusion: These three additional cases provide further evidence for the insula's pivotal role in the genesis of ecstatic symptoms. The induction of these states in patients with non-ecstatic epilepsy suggests that the insular “ecstatic network” may be a universal functional pathway rather than one unique to patients with ecstatic seizure semiology.

Disclosure: Nothing to disclose.

EPO-0075 | Predictive factors of nonadherence to antiseizure medication in epileptic patients

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Background and aims: Poor adherence to antiseizure medications (ASM) remains a major barrier to optimal epilepsy control. Data on determinants of nonadherence in Tunisia are lacking.

Methods: A prospective analytical study was conducted in the Adult Neurology Department of Habib Bourguiba University Hospital, Sfax Tunisia. Adult patients meeting ILAE 2017 criteria were included. Adherence was assessed using the MMAS-8 scale and categorized as adherent, minimally nonadherent, or majorly nonadherent. Sociodemographic, clinical, psychosocial (Stigma Scale for Epilepsy, Hospital Anxiety and Depression Scale, Acceptance of Illness Scale), therapeutic, and healthcare-related variables were analyzed.

Results: Sixty-two patients were included (mean age 43.6 ± 11.5 years; 51.6% female). According to MMAS-8, 37.1% were adherent, 27.4% minimally nonadherent, and 35.5% majorly nonadherent. Higher education level ($p=0.014$) and better

socioeconomic status ($p=0.002$) were significantly associated with adherence. Seizure freedom for more than two years ($p=0.033$) and normal EEG findings ($p=0.011$) were also predictive of better adherence. Stigma was reported by 54.8% of patients and was markedly higher among major nonadherents (81.8%, $p < 0.001$). Depression affected 37% of the cohort, predominating in major nonadherents (50%, $p < 0.001$). Therapeutic factors associated with nonadherence included higher daily dosing frequency ($p=0.028$) and perceived negative impact of treatment on daily life ($p=0.001$). Medication unavailability ($p=0.005$) and disadvantaged social coverage ($p=0.032$) were additional contributors.

Conclusion: ASM nonadherence in Tunisia is frequent and multifactorial, involving socioeconomic, clinical, psychosocial, therapeutic, and health system determinants, underscoring the need for integrated, context-adapted interventions.

Disclosure: Nothing to disclose.

EPO-0076 | Cumulative hippocampal seizure-related burden impairs long-term memory consolidation in focal epilepsy

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Background and aims: Memory impairment is a frequent and disabling comorbidity of focal drug-resistant epilepsy (fDRE), yet its relationship to epileptic activity is incompletely explained by seizure frequency or structural pathology alone. This study introduces the concept of cumulative hippocampal seizure-related burden (c-HipSZB) and examines its association with long-term memory consolidation in patients with fDRE.

Methods: Twenty patients undergoing stereo-electroencephalography (SEEG) were prospectively included. Continuous SEEG recordings covering the interval between two memory assessments (30 minutes and one week) were analysed. Hippocampal ictal involvement was identified using quantitative epileptogenicity markers and visual SEEG analysis. Permutation entropy was applied to SEEG signals to quantify postictal recovery dynamics, yielding a Postictal Alteration Time. Cumulative hippocampal ictal burden (c-HipIC), postictal burden (c-HipPIC) and their sum (c-HipSZB) were computed by integrating durations across all hippocampus-involving ictal events (clinical and subclinical). Verbal and visual memory consolidation was assessed using standardized recall and recognition paradigms. Associations were examined using correlation analyses and univariate and multivariate regression models adjusted for relevant clinical variables.

Results: Higher seizure-related burden metrics in the dominant-hemisphere hippocampus were consistently associated with poorer long-term verbal memory performance and reduced retention between 30 minutes and one week. The strongest effects were observed for c-HipSZB. These associations remained significant after adjustment for epilepsy duration or hippocampal lesion status. Clinical covariates were not independently associated with outcome. In contrast, seizure-related burden metrics

in the non-dominant hippocampus showed no robust associations with visual memory outcomes.

Conclusion: Cumulative ictal–postictal hippocampal dysfunction emerges as a key determinant of impaired long-term memory consolidation in fDRE.

Disclosure: None.

EPO-0077 | Newcastle's first experience with EASEE® epicranial neuromodulation for drug-refractory focal epilepsy: A two-patient case series

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Background and aims: Many patients with focal epilepsy remain drug-resistant and are unsuitable for resective surgery because of non-lesional imaging, widespread epileptogenic networks, or overlap with important brain regions. The Precisis EASEE® system is an epicranial neuromodulation device delivering cortical stimulation.

Methods: We report the first two patients in Newcastle implanted with the EASEE® device. Both had long-standing drug-resistant focal epilepsy and were not candidates for resection. Patient 1 had MRI-negative sleep-related hypermotor epilepsy with a right parietal–frontal network. Patient 2 had left fronto-parietal focal cortical dysplasia. Epicranial electrodes were placed over the epileptogenic zone and connected to a chest-wall pulse generator.

Results: Implantation and activation were uncomplicated, taking place on the 01/04/25 and the 03/09/25 respectively. Both patients experienced mild local discomfort that resolved. Patient 1 had early improvement with variable seizures. Patient 2 showed a reduction in nocturnal seizures from 3–4 per week to 1–2 per week and aborted an aura using patient-activated stimulation. EASEE® programming was adjusted over time to balance seizure control and tolerability.

Conclusion: Epicranial focal neuromodulation with the EASEE® device appears feasible and acceptable in selected patients with non-resectable focal epilepsy and may offer a practical alternative when resection is unsafe. Further follow-up is required to assess durability.

Disclosure: Nothing to disclose.

EPO-0078 | Structure-function coupling of interictal epileptiform discharges to predict surgical outcomes in epilepsy

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Background and aims: One third of patients with drug-resistant epilepsy remains non-seizure-free after surgery. This work studies brain structure-function coupling (SFC) during interictal epileptic discharges (IEDs) through the graph signal processing framework, to distinguish between good and poor surgical outcomes in temporal lobe epilepsy (TLE).

Methods: IEDs during high-density EEG recordings of 30 TLE patients (N=17 with good post-surgery outcome, ILAE 1–2) were source-reconstructed and then summarised into 118 region-of-interest (ROI) time-series. Network harmonics of a template structural connectome, defined as the graph Laplacian's eigenvectors, were used to decompose the ROI time-series. The transformed signal's energy spectrum was divided into low-frequency (LF) content (first harmonics, more coupled to the structure) and high-frequency (HF) one (last harmonics, more decoupled). The LF and HF signal norms along the IED were compared with a cluster-based permutation test. SFC at the IED peak was quantified as the ratio of LF to HF signal norm and compared between outcome groups with a Wilcoxon rank-sum test.

Results: Findings replicated previously published results, confirming an increased SFC during the IED ($p < 0.05$) and decreased SFC before the IED's start ($p < 0.001$). Additionally, a SFC decrease was observed after the IED ($p < 0.05$). Patients with poor surgical outcomes presented a significantly lower SFC at the IED's peak than those with good outcomes ($p < 0.05$).

Conclusion: SFC during IEDs differs according to the surgical outcome: IEDs of patients with good outcome are better explained by the LF network harmonics, meaning that the spatial pattern of the IED is smoother on the SC.

Disclosure: Nothing to disclose.

EPO-0079 | Efficacy of repetitive transcranial magnetic stimulation in drug-resistant epilepsy: A systematic review and meta-analysis

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Background and aims: Drug-resistant epilepsy (DRE), defined as failure of at least two anti-seizure medications, affects one-third of patients and poses major therapeutic challenges. Repetitive transcranial magnetic stimulation (rTMS), a non-invasive neuromodulation technique, has been proposed for DRE. This systematic review assessed the efficacy of rTMS in reducing seizures in these patients.

Methods: We searched PubMed, Scopus, Web of Science, and Cochrane Central through October 31, 2025. Randomized controlled trials (RCTs) comparing low-frequency (≤ 1 Hz)

rTMS with sham stimulation in DRE patients were included. Continuous outcomes were pooled using a random-effects model and reported as standardized mean differences (SMD) with 95% confidence intervals (CI).

Results: Five RCTs comprising 172 participants (88 active rTMS, 84 sham rTMS) were included. The mean participant age ranged from 8.9 to 40 years. Active rTMS significantly reduced seizure frequency at two weeks (SMD = -0.57, 95% CI: -0.96 to -0.18; $p=0.004$), four weeks (SMD = -0.62, 95% CI: -1.03 to -0.21; $p=0.002$), and eight weeks (SMD = -0.65, 95% CI: -0.98 to -0.32; $p<0.0001$). A subgroup analysis based on stimulation frequency showed comparable effects for 1 Hz (SMD = -0.33, 95% CI: -0.97 to 0.31) and 0.5 Hz (SMD = -0.79, 95% CI: -1.18 to -0.4). Additionally, active rTMS showed a significant reduction in interictal epileptiform discharge (SMD = -0.44, 95% CI: -0.84 to -0.04; $p=0.03$).

Conclusion: Low-frequency rTMS is an effective therapeutic intervention for reducing seizure frequency and interictal epileptiform discharge in patients with DRE, with sustained benefits observed over 8 weeks.

Disclosure: Nothing to disclose.

TABLE 1 Change in seizure frequency across different time points following low-frequency rTMS.

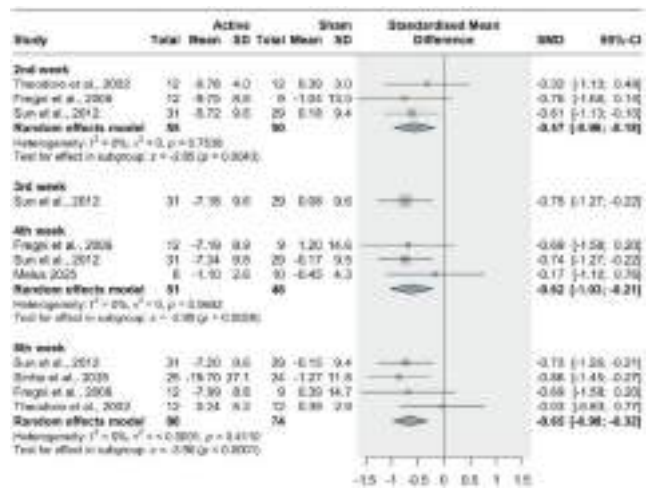


TABLE 2 Subgroup analysis based on stimulation frequency.

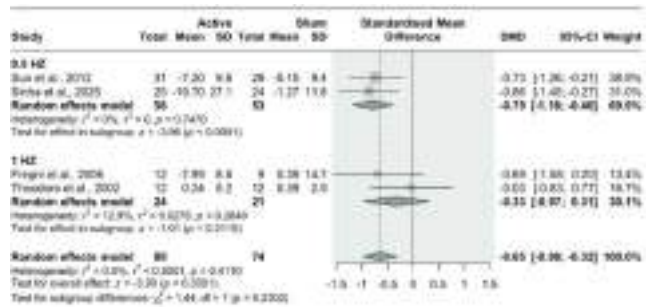
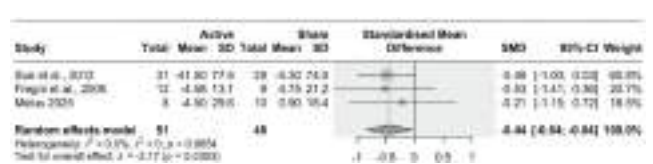


TABLE 3 Change in interictal epileptiform discharge.



EPO-0080 | Beyond visual analysis: Uncovering the electrographic signature of genetic focal epilepsy via AI-driven gamma power quantification

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Background and aims: Focal cortical dysplasia (FCD) and other structural epilepsies are increasingly recognized as mTOR-driven genetic disorders (mTORopathies). While these conditions are characterized by cellular hyper-excitability, standard visual EEG analysis often fails to detect interictal background abnormalities. This study utilizes Machine Learning (ML) to identify quantitative EEG (qEEG) biomarkers—specifically high-frequency activity—that correlate with this “invisible” genetic phenotype.

Methods: A retrospective cohort analysis was conducted using the CHB-MIT Scalp EEG database. A total of 23,760 interictal EEG epochs (5-second segments) were extracted from 20 pediatric patients with intractable focal epilepsy. Signal processing included a 0.5–100 Hz bandpass filter to preserve high-frequency oscillations. A Random Forest classifier was trained to characterize patient-specific background phenotypes, using 5-fold cross-validation to assess statistical reliability.

Results: The ML model demonstrated a mean classification accuracy of 92.91% ± 0.95% in blind testing. The results were statistically significant against random chance ($p < 0.05$), indicating the presence of distinct neurophysiological signatures in the interictal background. Crucially, Feature Importance analysis indicated that Gamma Power (30–80 Hz) was the most significant discriminator across the cohort (Importance Score > 0.28), consistently outperforming traditional biomarkers such as Delta slowing.

Conclusion: These findings suggest that patients with intractable focal epilepsy exhibit a ubiquitous “Hyper-Excitable Background Phenotype” driven by high-frequency activity. This supports the potential of AI-driven qEEG to serve as a non-invasive screening tool for genetic etiologies (mTORopathies), potentially guiding early targeted therapy and genetic panel selection.

Disclosure: Nothing to disclose.

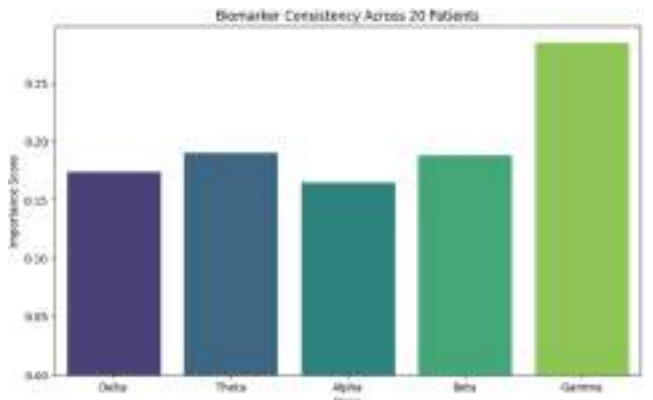


FIGURE 1 Feature Importance analysis showing Gamma Power (30–80 Hz) as the most consistent biomarker across the 20-patient cohort compared to other frequency bands.

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Background and aims: Sleep staging is critical in EEG interpretation. However, current models are developed using polysomnography (PSG) data, which are unsuitable for standard EEG recordings. This study aims to construct a sleep staging recognition model for achieving objective and accurate sleep structure assessment.

Methods: The sleep staging model vEpiSleepNet consists of four modules: sleep feature extraction with transfer learning, cascaded multi-layer dilated convolution encoding, temporal context encoding, and MLP classification (Figure 1). All signals from 19 electrodes along with A1/A2 were used for model development, distinguishing from previous models employing <10 channels. This study used Peking Union Medical College Hospital (PUMCH) dataset, containing 117 patients and 140 h EEG data, dividing into 30-second segments. Patient-wise split five-fold cross-validation was applied.

Results: vEpiSleepNet achieves 70.5% accuracy and 0.700 F1-score on PUMCH dataset, with a wake-sleep binary classification accuracy of 87.6%. vEpiSleepNet exhibited nearly 12% improvement in F1 scores, 10% in accuracy, and 15% in Kappa, compared to the best-performing comparative models among

the five published classic models (Figure 2). On two public PSG datasets, vEpiSleepNet also showed superior performance, with 81.9% and 80.1% accuracies, respectively. Ablation experiments demonstrated the efficacy of transfer learning and cascaded dilated convolution. Additionally, the more electrodes, the better mode performance.

Conclusion: We have developed vEpiSleepNet which is specifically designed for EEG monitoring, filling the gap in the lack of dedicated automatic sleep staging models for EEG monitoring. Accurate automated sleep staging of EEG data has the potential to advance research on epileptic discharges and improve clinical diagnostic capabilities for sleep-related epilepsy.

Disclosure: Nothing to disclose.

EPO-0082 | VIDÉPÉ: A web-app to interactively visualize multimodal electrophysiological and imaging data to improve epilepsy surgery evaluation

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Background and aims: Drug-resistant epilepsy affects a substantial proportion of the more than 80 million people living with epilepsy worldwide. For these patients, resective surgery offers the highest likelihood of seizure freedom, but its success critically depends on accurate delineation of the epileptogenic zone while preserving eloquent cortex. This process requires the joint interpretation of heterogeneous data, including structural, functional and nuclear imaging, and electrophysiological recordings (EEG). In current clinical practice, these modalities are typically explored in separate software environments, leading to fragmented, largely static workflows that limit interactive, multidisciplinary discussion and restrict the use of advanced analyses.

Methods: We developed a web-based application for integrated visualization of multimodal presurgical epilepsy data. Volumetric neuroimaging (MRI and PET) is rendered using Niivue.js, while electrophysiological signals are displayed with Plotly.js. The application is deployed through GitHub Pages, allowing access from any modern web browser without local installation. The system supports simultaneous visualization

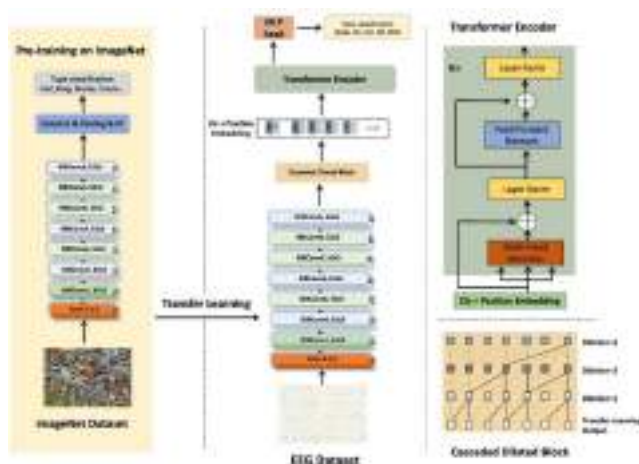


FIGURE 1 The architecture of vEpiSleepNet.



FIGURE 2 Confusion matrix on XIEHE-SLEEP-16K. vEpiSleepNet showed the best performance on all wake and sleep stages, compared to the other networks, MMASleepNet and SleepTransformer.

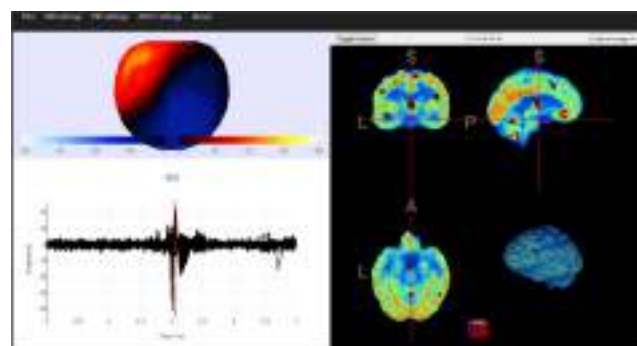


FIGURE 1 Web-app interface with EEG (right) and MRI/PET imaging (left). Time navigation is performed in the EEG panel, slice and view selection in the imaging panel, and a central menu bar controls independent opacity and thresholding of each volume.

of imaging volumes, functional maps, and time-resolved EEG within a unified interface.

Results: The application is publicly available online (<https://ziply.pk/2E853M>) and operates across operating systems and devices. A demonstration page combining MRI, PET, and EEG is provided for interactive exploration (<https://ziply.pk/Kf3H50>).

Conclusion: This web-based platform integrates multimodal imaging into a single interactive environment to support coherent data interpretation potentially improving clinical decision-making in epilepsy surgery. Its browser-based design ensures easy and broad accessibility. Ongoing work will add modalities such as intracranial EEG and will evaluate usability and clinical utility with clinicians and patients.

Disclosure: Nothing to disclose.

Headache 1

EPO-0083 | Changes in anxiety and depression symptoms in patients with chronic migraine and medication-overuse headache in the RESOLUTION trial

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Background and aims: These analyses explored the impact of eptinezumab versus placebo on anxiety and depression symptoms in adults with chronic migraine (CM) and medication-overuse headache (MOH).

Methods: The phase 4 RESOLUTION (NCT05452239) trial included a 28-day screening (baseline) period and 12-week double-blind, placebo-controlled period. Adults (18–75 yrs) with CM and MOH were randomized 1:1 to IV eptinezumab 100 mg or placebo at baseline; all participants received a brief educational intervention on MOH before infusion. Secondary endpoints included the changes from baseline to Weeks 4 and 12 in Hospital Anxiety and Depression Scale (HADS) scores for anxiety (HADS-A) and depression (HADS-D) in the full-analysis set, with post hoc analyses in subgroups with elevated baseline scores (> 7). *p*-values are descriptive.

Results: Of 608 participants randomized, 596 (98%) completed the placebo-controlled period. In the full-analysis set (*n* = 602), eptinezumab resulted in greater improvements from baseline

than placebo for HADS A (Week 4: between-group difference [95% CI] −0.8 [−1.4, −0.3], *p* = 0.0017; Week 12: −1.0 [−1.5, −0.5], *p* = 0.0001) and HADS D (Week 4: −1.0 [−1.5, −0.4], *p* = 0.0006; Week 12: −1.2 [−1.8, −0.6], *p* < 0.0001). Subgroups with elevated baseline HADS-A (226/602 [38%] participants) and HADS-D (201/602 [33%] participants) scores showed greater reductions from baseline with eptinezumab vs placebo in the respective HADS scores, with similar between-group differences as the full-analysis set.

Conclusion: Participants with CM and MOH who also received patient education at baseline experienced greater reductions with eptinezumab than placebo in anxiety and depression scores at Weeks 4 and 12 after treatment. Similar differences between eptinezumab and placebo were seen in participants with elevated baseline anxiety and/or depression symptoms.

Disclosure: CT in the past 3 years has received support (financial or drugs) from AbbVie and Novartis for an investigator-initiated trial; consulting fees for the participation in advisory boards for AbbVie, Dompé, Eli Lilly, Ipsen, Lundbeck, Organon, Pfizer, and Teva; honoraria for scientific lectures and presentations from AbbVie, Eli Lilly, Lundbeck, Pfizer, and Teva; support for attending meetings from AbbVie, Dompé, Eli Lilly, Ipsen, Lundbeck, Pfizer, and Teva; has been Principal Investigator in clinical trials sponsored by AbbVie, Biohaven, Chordate, Eli Lilly, Ipsen, Lundbeck, Pfizer, and Teva; and received grants from the European Commission, the Italian Ministry of Health, and the Italian Ministry of University. RHJ has given lectures for Allergan, ATI, Eli Lilly, Lundbeck, Merck, Novartis, Pfizer, and Teva; served as investigator in clinical trials with ATI, Eli Lilly, Lundbeck, Novartis, and Novo Nordisk; is the director of the Danish Headache Center, Lifting The Global Burden of Headache, and Founder of Master of Headache Disorders at University of Copenhagen; and has received research funding from ATI, Lundbeck Foundation, Rigshospitalet, The Medical Society in Copenhagen, Novo Nordisk Foundation, Tryg Foundation, and University of Copenhagen. CL has participated on an advisory board and received payment for lectures arranged by AbbVie, Lundbeck, Novartis, Pfizer, and Roche, and has received research sponsorship from AbbVie. HWS has received personal fees from AbbVie, Eli Lilly, Lundbeck, Novartis, and Teva, and has received research grants from Novartis and Novo Nordisk Foundation. FV has received financial support from AbbVie, Angelini, and Lundbeck for investigator-initiated trials; consulting fees for the participation in advisory boards from AbbVie, Angelini, Eli Lilly, Lundbeck, Organon, Pfizer, and Teva; honoraria for scientific lectures and presentations from AbbVie, Eli Lilly, Lundbeck, Pfizer, and Teva; support for attending meetings from AbbVie, Eli Lilly, Lundbeck, Pfizer, and Teva; and has been Principal Investigator in clinical trials sponsored by AbbVie, Eli Lilly, Lundbeck, Pfizer, and Teva. GMT reports grants or consultancy support from AbbVie, Eli Lilly, Lundbeck, Novartis, Organon, Pfizer, and Teva, and independent support from the Clayco Foundation, Dioraphte Foundation, Dutch Research Council, Dutch Heart Foundation, Dutch Brain Foundation, IRRF, and the European Community. [Disclosures for the remaining authors will be included in the presentation.]

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Background and aims: Secondary clinical efficacy and functional endpoints from the TEMPLE trial modified-intent-to-treat (mITT) population were evaluated in treatment completers.

Methods: TEMPLE was a phase 3b, randomized, multicenter, double-blind, double-dummy, parallel-group, active-controlled trial to evaluate tolerability, safety, and efficacy of atogepant compared with topiramate for the preventive treatment of migraine. Post-hoc analyses in the mITT and completers populations assessed the proportion of participants with $\geq 50\%$ reduction in mean monthly migraine days (MMDs) during Months 4–6 and change from baseline in Headache Impact Test-6 (HIT-6) at Week 24 and Patient-Reported Outcomes Measurement Information System Cognitive Function (PROMIS-CF)–Abilities Subset–Short Form 6a(v2.0) at Week 6.

Results: Of 527 (atogepant = 270; topiramate = 257) mITT participants, 372 (atogepant = 209; topiramate = 163) were treatment completers. During Months 4–6, participants in the atogepant group were more likely to experience a $\geq 50\%$ reduction in mean MMDs compared with topiramate in both the mITT population (64.1% vs. 39.3%; $p < 0.0001$) and in treatment completers (73.7% vs. 48.5%; nominal $p < 0.0001$) (Figure 1). At Week 24, participants in the atogepant group showed a greater improvement in HIT-6 in both the mITT population (–11.7 vs. –7.4; $p < 0.0001$) and in treatment completers (–13.2 vs. –9.2; nominal $p < 0.0001$) than in the topiramate group (Figure 2). At Week 6, atogepant demonstrated greater improvement in PROMIS-CF in the mITT population (5.2 vs. 0.2, $p < 0.0001$) and in treatment completers (5.4 vs. 1.8, $p \leq 0.01$) compared to topiramate (Figure 3). Results were consistent across other secondary endpoints. Atogepant was generally safe and well-tolerated as reported previously.

Conclusion: TEMPLE treatment completers randomized to atogepant demonstrated similar improvements in clinical efficacy and functional endpoints to the overall mITT population; both populations demonstrated greater improvements in clinical efficacy and functional endpoints over those randomized to topiramate.

Disclosure: Andrew Blumenfeld has served on advisory boards for, consulted for, and/or been a speaker or contributing author for Aeon, Allergan/AbbVie, Alder, Amgen, Axsome, BMS, Biohaven, Eli Lilly, Impel, Lundbeck, Novartis, Revance, Teva, Theranica, and Zosano. He has received grant support from AbbVie, Amgen. Jan Versijpt received personal fees and non-financial support from Teva, Pfizer and Medtronic, personal fees from Novartis and Lundbeck, and grants and nonfinancial support from Allergan/AbbVie. Umer Najib has served on advisory boards for, consulted for, and received honoraria from AbbVie, Lundbeck, and Pfizer. Annelies Van Dycke has served

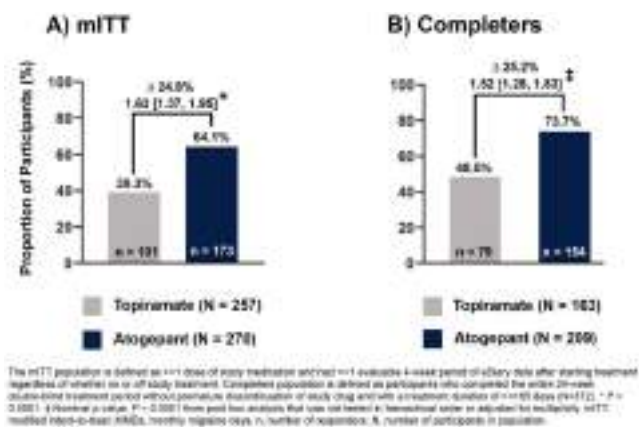


FIGURE 1 Proportion of participants achieving $\geq 50\%$ reduction in MMDs across months 4 to 6.

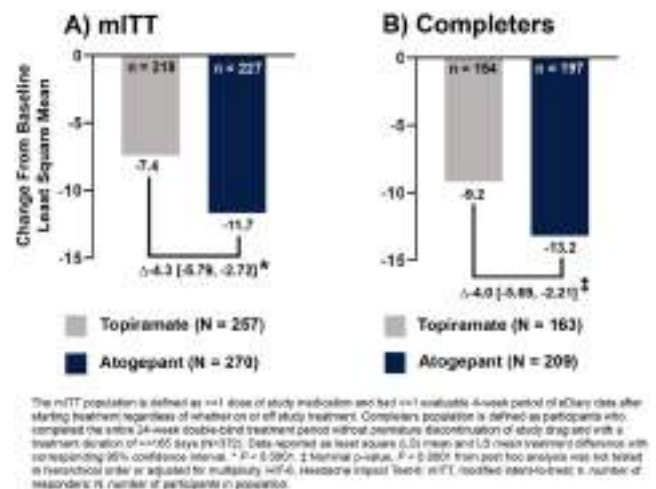


FIGURE 2 Change from baseline in HIT-6 total score at week 24.

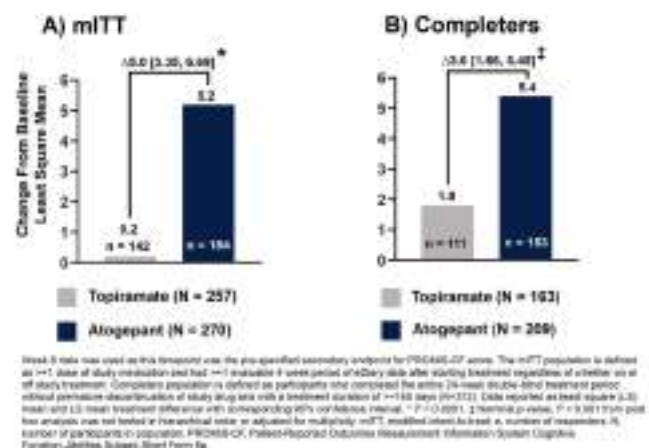


FIGURE 3 Change from baseline in PROMIS-CF score at week 6.

on advisory/scientific boards and/or given lectures for AbbVie, Allergan, Angelini Pharma, Eli Lilly, Lundbeck, Neuraxpharm, Novartis, Organon, Pfizer, TEVA and UCB, for which she has received honoraria. Uwe Reuter has served on advisory boards for Amgen, Allergan, AbbVie, Eli Lilly, Lundbeck, Novartis,

Pfizer, and Teva and has received institutional honoraria for lectures from Amgen, Allergan, AbbVie, Eli Lilly, Lundbeck, Novartis, electroCore, Medscape, StreaMedUp, Springer, and Teva, received institutional honoraria for consulting services from Lundbeck, Pfizer, and AbbVie; received research funding from Novartis (CHERUB01) and the German Federal Ministry of Education and Research; and is an associate editor of the Journal of Headache and Pain. PG, YL, KN, BD, and SM are employees of AbbVie and may hold AbbVie stock.

EPO-0085 | Efficacy of Candesartan for migraine prevention: A systematic review and meta-analysis

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Background and aims: Migraine is a major cause of disability, and many patients don't tolerate or respond to first-line preventive therapies. Candesartan has shown preventive benefit in randomized controlled trials, but its overall efficacy remains uncertain. This study aimed to evaluate the efficacy and safety of candesartan for migraine prevention.

Methods: A systematic review was performed according to PRISMA guidelines, using PubMed, Scopus and Web of Science for studies published until January 11,2026. Risk of bias was assessed using RoB 2.0 The primary outcome was migraine days per 4 weeks. For crossover trials, within-participant correlation was accounted for by variance adjustment. Pooled effects were calculated using mean difference (MD) with 95% confidence intervals (CI) under a random-effects model. Sensitivity analyses were conducted using different correlation assumptions.

Results: Three RCTs including 425 participants were analyzed. Mean age was 39.1 years±10.9 and 82.9% were females. Most

studies evaluated at dose 16 mg. Candesartan significantly reduced migraine days per 4 weeks compared with placebo (MD: -1.11 days, 95% CI: -1.95 to -0.28; *p*=0.009;). Monthly headache days showed a non-significant reduction (MD: -1.33 days, 95% CI: -2.73 to 0.07; *p*=0.06;). Results remained consistent across sensitivity analyses. A total of 453 adverse events were reported, occurring more frequently during candesartan exposure periods; most were mild, mainly dizziness and fatigue.

Conclusion: Candesartan demonstrates statistically significant but modest efficacy in reducing migraine days and a trend toward reduction in headache days, with acceptable tolerability. It may represent a useful preventive option, particularly for patients who don't respond to standard preventive therapies.

Disclosure: Astrid Geraldine Carrion Cuellar: Nothing to disclose Elizabeth Luisa Cuellar Jauregui: Nothing to disclose Thalia del Pilar Huiman Caro: Nothing to disclose.

EPO-0086 | Digital education for migraine self-management: A Systematic review of app-based interventions

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Background and aims: Effective migraine management requires patient education to improve self-management, but traditional methods lack scalability. Digital tools like smartphone apps offer new educational platforms. This review evaluates their impact on clinical and self-management outcomes.

Methods: A systematic review followed PRISMA guidelines. We searched PubMed, Embase, PsycINFO, Cochrane Central, and ClinicalTrials.gov up to December 2024 for randomised controlled trials (RCTs). Eligible RCTs assessed digital tools with educational or behavioural components for adult migraine. Primary outcome: change in monthly migraine days (MMD). Secondary outcomes: headache intensity, disability (MIDAS/HIT-6), adherence. Risk of bias was assessed using the Cochrane RoB 2 tool.

Results: From 1,124 screened records, 16 RCTs (*n*=2,736 participants) were included. A meta-analysis showed digital educational interventions significantly reduced MMD by a mean of -2.3 days/month (95% CI: -3.1 to -1.5) versus control. Interventions with cognitive behavioural therapy (CBT) modules were 35% more effective than basic ones. Significant improvements occurred in headache intensity (standardised mean difference, SMD: -0.41) and disability (SMD: -0.38). Intervention groups reported high engagement and improved disease knowledge.

Conclusion: Digitally delivered educational interventions are effective adjuncts to standard migraine care, leading to clinically meaningful improvements. Efficacy is strongest when tools include interactive, theory-based components like CBT. These apps represent a scalable new paradigm for patient education in neurology, directly empowering self-management. Future development must prioritise pedagogical design.

Disclosure: Nothing to disclose.

TABLE 1 Baseline demographic and clinical characteristics.

Study	Type of study	n	Age, mean (SD)	Female %	Migraine type	Blind procedure, scoring
Sonnen 2013	RCT crossover	81	38 (11)	82.8%	Acut: 6 No acut: 22; Both: 24 Chronic: 1	PR
Trovik 2003	RCT crossover	97	43.3 (7.4)	76.8%	PR	Placebo: 126/77 Candesartan: 115/75
Trovik 2005	RCT parallel	367	38.0 (10.7)	84.4%	Acut: 49 No acut: 186; Both: 66	Placebo: 126/82 Candesartan: 126/81
PR: not reported						

TABLE 2 Migraine days per 4 weeks: candesartan vs placebo.



TABLE 3 Headache days per 4 weeks: candesartan vs placebo.



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Background and aims: Atogepant is an oral CGRP receptor antagonist approved for migraine prevention. The aim of this study was to investigate the neurophysiological changes induced by atogepant in individuals with high-frequency episodic migraine (HFEM).

Methods: In this prospective study, 40 participants (age 35.5 ± 21.9 , 30 females) underwent interictal neurophysiological testing at baseline (T0) and after 3 months of daily atogepant 60 mg (T1). The assessment included: i) the temporal summation threshold (TST) of the nociceptive withdrawal reflex (nWR); and ii) the habituation of the nociceptive blink reflex (nBR) and of visual evoked potentials (VEPs). Habituation was assessed by means of the habituation index (HI), namely the percentage decrease of area under curve at the end of validated stimulation protocols.

Results: At T1, the TST increased compared to T0 (T0: 7.9 ± 4.5 mA vs. T1: 9.2 ± 4.8 mA, $p=0.021$). At T1, the HI of the nBR decreased compared to T0 (T0: 47.4 ± 25.6 vs T1: 38.3 ± 29.5 , $p=0.025$) and the habituation deficit became more pronounced (TIME $\times$ BLOCKS: $p=0.045$). VEPs showed a mild habituation, without changes at T1 ($p>0.050$). At T1, 70% of participants qualified as Responders ($>50\%$ in monthly migraine days). Being a Responder was not associated with any of the previously presented findings.

Conclusion: After a 3-month atogepant treatment, we described a reduction in central sensitization and a modulation of habituation mechanisms at the brainstem level, without changes at the cortical level. These findings suggest that atogepant induced direct or indirect effects on key neurophysiological mechanisms involved in migraine pathophysiology.

Disclosure: The ATOM project was funded by a CORE - AMD Resident Research Grant (application ID#: 1273396)

EPO-0088 | MIDAS and HIT-6 questionnaires versus headache diaries for monitoring treatment response to Erenumab in migraine: A REFORM study

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Background and aims: Efficacy of migraine preventive treatment is often assessed based on daily headache diaries, which can be burdensome for patients. Therefore, patient-reported outcome measures (e.g. Migraine Disability Assessment (MIDAS) and Headache Impact Test (HIT-6)) offer alternatives for monitoring treatment response. The effectiveness of these questionnaires, however, remains uncertain. Here, we aimed to establish cut-off scores for MIDAS and HIT-6 corresponding with a $\geq 50\%$ reduction in monthly migraine days (MMDs) to determine treatment responders.

Methods: Adults with migraine were enrolled in a prospective, open-label study and received 140 mg erenumab for 24 weeks. A $\geq 50\%$ reduction in MMD was defined as treatment success based on diary data. MIDAS and HIT-6 were completed at baseline, Week 12, and Week 24. Optimal cut-off values and corresponding sensitivity and specificity were determined using receiver operating characteristic curve analysis.

Results: Among 582 participants, 300 individuals (52%) achieved treatment success. MIDAS and HIT-6 showed fair discriminative ability in identifying treatment responders, with area under the curve values ranging from 0.71 to 0.77. The optimal cut-offs for MIDAS were a 16-point absolute reduction (78% sensitivity, 60% specificity) or a 49% relative reduction (82% sensitivity, 64% specificity). For HIT-6, optimal cut-offs were an 8-point reduction (53% sensitivity, 84% specificity) or a 12% relative reduction (54% sensitivity, 84% specificity). Sensitivity analyses with alternative definitions yielded consistent results.

Conclusion: While MIDAS and HIT-6 provide meaningful information on treatment-related disability reduction, they are not interchangeable with prospective headache diaries for assessing treatment response to erenumab.

Disclosure: JT reports receiving personal fees from Lundbeck and Pfizer, outside of the submitted work. RHC reports receiving personal fees from Pfizer, outside of the submitted work. HMA reports receiving personal fees from Pfizer and Lundbeck, outside of the submitted work. ZK reports receiving honoraria from AbbVie, Lilly, Merck, Novartis, TEVA and a research grant from Novartis, outside of the submitted work. MA reports receiving personal fees from AbbVie, Amgen, Astra Zeneca, Eli Lilly, GlaxoSmithKline, Lundbeck, Novartis, Pfizer and Teva Pharmaceuticals outside of the submitted work. MA received institutional grants from Lundbeck Foundation, Novo Nordisk Foundation, Lundbeck A/S, and Novartis. MA reports serving as associate editor of The Journal of Headache and Pain, and associate editor of Brain. HA reports receiving personal fees from AbbVie, Lundbeck, Pfizer, and Teva Pharmaceuticals, outside of the submitted work. Funding: This work was supported by research grants from the Lundbeck Foundation (R403-2022-1352 to H.A. and R310-2018-3711 to M.A.).

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Background and aims: Pituitary adenylate cyclase-activating polypeptide-38 (PACAP-38) is a key neuropeptide in migraine pathophysiology and an emerging therapeutic target, with monoclonal antibodies currently under clinical development. PACAP-38 has been measured mainly in blood, yielding inconsistent results. Novel, non-invasive biological sources closely related to trigeminal activation, such as tear fluid, remain unexplored. Here, we investigated PACAP-38 levels in tear fluid of patients with migraine compared with healthy controls (HCs) using the Schirmer test.

Methods: In this cross-sectional study, we included patients with migraine fulfilling ICHD-3 criteria and HCs. Tear fluid was collected with Schirmer strips and ELISA measured PACAP-38 concentrations. Clinical and demographic data were recorded. Between-group comparisons, correlation analyses, and receiver operating characteristic (ROC) analyses were performed.

Results: We included 26 migraine patients (76.9% female, mean age 38.0 ± 11.6 years) and 12 HCs (58.3% female, mean age 27.1 ± 5.2 years). Tear PACAP-38 concentrations were significantly higher in patients with migraine compared with HCs (22.2 ± 33.1 vs 4.5 ± 8.4 pg/mL, $p = 0.041$). ROC analysis showed good discriminative ability ($AUC = 0.71$, $p = 0.02$), with an optimal cut-off of 2.0 pg/mL (73% sensitivity, 67% specificity). PACAP-38 levels were not associated with monthly headache days, acute medication number, ictal vs interictal sampling, or migraine subtype.

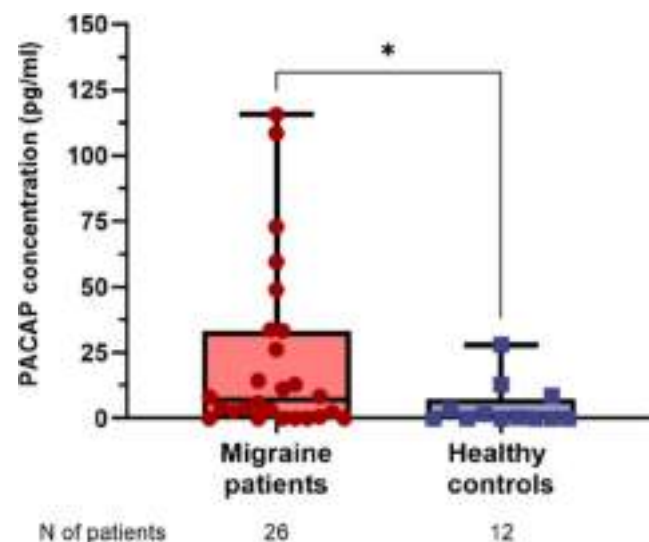


FIGURE 1 PACAP-38 levels in tear fluid of migraine patients compared to healthy controls.

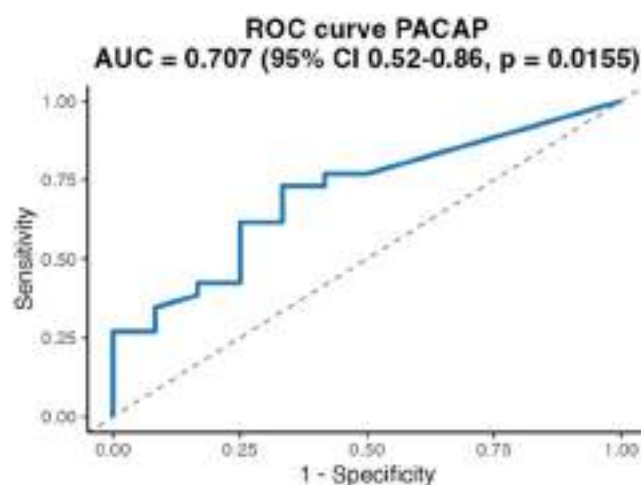


FIGURE 2 Receiver Operating Characteristic (ROC) curve for PACAP-38 levels in tears.

Conclusion: This is the first study to assess PACAP-38 concentrations in the tear fluid of patients with migraine, demonstrating significantly elevated levels compared with HCs. Schirmer strip-based sampling represents a rapid, non-invasive, and patient-friendly approach for neuropeptide detection, potentially providing a biomarker that reflects migraine-specific mechanisms and supporting PACAP-38 as an emerging therapeutic target.

Disclosure: Nothing to disclose.

EPO-0090 | Triple combination therapy with onabotulinumtoxinA, fremanezumab and atogepant in chronic migraine: A 52-week prospective case series (TRIPLE study)

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Background and aims: A substantial proportion of patients with chronic migraine (CM) persist with high disease burden despite treatment with advanced preventive monotherapies,

including onabotulinumtoxinA (BoNTa) and calcitonin gene related peptide (CGRP)-targeted drugs. Evidence supporting preventive combinations, particularly multiple regimens, is limited. This study evaluated the long-term effectiveness and safety of combining BoNTa, fremanezumab, and atogepant in a real-world CM population with persistent residual burden.

Methods: This was a 52-week, prospective, single-center case series including 13 consecutive adults with CM treated with BoNTa, then fremanezumab and followed by add-on atogepant 60 mg. Monthly headache days (MHDs) and monthly migraine days (MMDs) were assessed using electronic diaries. Secondary outcomes included acute medication days, responder rates, Headache Impact Test (HIT-6), Hospital Anxiety and Depression Scale (HADS), Patient Global Impression of Change (PGIC), and adverse events.

Results: At atogepant initiation, patients had severe residual disease with mean MHDs 25.8 ± 4.8 and MMDs 19.7 ± 4.2 . During triple therapy, MHDs decreased progressively to 19.1 ± 9.4 at week 52 ($p < 0.001$), and MMDs to 10.4 ± 6.2 ($p < 0.001$). $\geq 50\%$ MMD reduction was achieved by 30.8% of patients at week 24 and 53.8% at week 52. Acute medication days and HIT-6 scores significantly improved, while anxiety and depression scores remained stable. PGIC consistently indicated moderate-to-great improvement. No serious adverse events or treatment discontinuations occurred, and all adverse events were mild.

Conclusion: In this CM cohort, long-term triple preventive therapy with BoNTa, fremanezumab, and atogepant was associated with progressive and clinically meaningful reductions in migraine frequency and disability, with favorable tolerability.

Disclosure: MR: received financial support, consulting fees for the participation in advisory boards and support for attending meetings from: Lundbeck, Pfizer and AbbVie. LFI received financial support, consulting fees for the participation in advisory boards and support for attending meetings from: Teva, Eli Lilly, Lundbeck, Pfizer and AbbVie; he is Associate Editor for Frontiers in Neurology and junior editor of Cephalalgia and Cephalalgia report. AR: has received speaker honoraria from Allergan, Lilly, AbbVie, Pfizer, Novartis and Teva and serves as an associate editor of Frontiers in Neurology (Headache Medicine and Facial Pain session). IF: Grants/honorarium/Consulting fees: Allergan, Amgen, Eli Lilly, J and J, Merck, Purdue, Teva, Aralez, Novartis, Lundbeck, Pfizer. AHA: has received personal fees for lectures/advisory boards: Novartis, Abbvie, TEVA, Roche, Lundbeck, Pfizer, Boehringer Ingelheim. Non-personal research grants from several sources, including KlinBeForsk, EU, Boehringer Ingelheim, Medtronic, BMS. EC: has received honoraria from Novartis, Chiesi, Lundbeck, Medscape, Lilly; his salary has been partially funded by Río Hortega grant Acción Estratégica en Salud 2017–2020 from Instituto de Salud Carlos III (CM20/00217) and Juan Rodés fellowship, Subprograma Estatal de Incorporación de la Acción

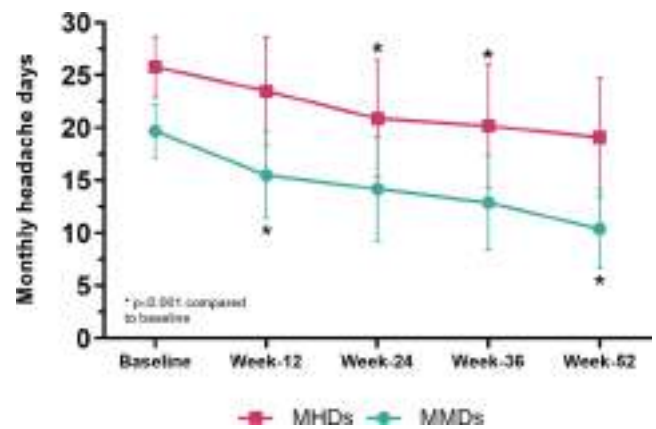


FIGURE 2 Change in monthly headache days and migraine days during triple combination treatment.

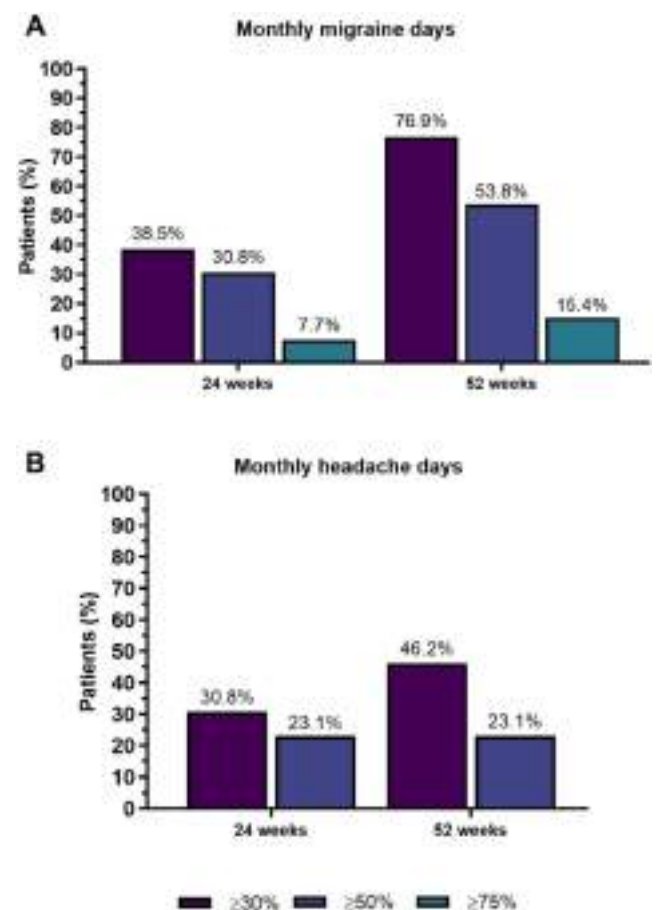


FIGURE 3 Response rates at 24 and 52 weeks.



FIGURE 1 Study design of the TRIPLE study.

Estrategia en Salud 2023 (JR23/00065). He is a junior editor for Cephalalgia. PPR: has received, in the last 3 years, honoraria as a consultant and speaker from AbbVie, Amgen, Dr Reddy's, Eli Lilly, Lundbeck, Medscape, Novartis, Organon, Pfizer and Teva Pharmaceuticals. Her research group has received research grants from AbbVie, AGAUR, EraNet Neuron, FEDER RIS3CAT, Instituto Investigación Carlos III, MICINN, Novartis, and Teva Pharmaceuticals, and has received funding for clinical trials from AbbVie, Amgen, Biohaven, Eli Lilly, Lundbeck, Novartis, Pfizer and Teva Pharmaceuticals. She is the Honorary

Secretary of the International Headache Society, is on the editorial board of *Revista de Neurologia*, is an associate editor for *Cephalalgia*, *Headache*, *Neurologia*, *Frontiers of Neurology*, and is an advisor of the Scientific Committee of the Editorial Board of *The Journal of Headache and Pain*. She is a member of the Clinical Trials Guidelines Committee and Scientific Committee of the International Headache. ET: has received personal fees for lectures/advisory boards: Novartis, Eli Lilly, Abbvie, TEVA, Roche, Lundbeck, Pfizer, Biogen, Organon. Consultant for and owner of stocks and IP in Man & Science. Stocks and IP in Nordic Brain Tech. Stocks in Keimon Medical. Non-personal research grants from several sources, including Norwegian Research Council, KlinBeForsk, EU. Commissioned research (non-personal): Lundbeck. CS: has received honorarium for lectures/advisory boards: Novartis, Abbvie, TEVA, Vigil NeuroScience.

EPO-0091 | CGRP monoclonal antibodies in adolescents with migraine: Real-world persistence and outcomes from the German pain e-Registry

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Background and aims: Preventive treatment options for adolescents with migraine are limited. Conventional oral preventives are often used off-label, poorly tolerated, and frequently discontinued. Real-world data on CGRP monoclonal antibodies (mABs) in this population are scarce. To explore real-world treatment persistence and clinical outcomes of CGRP mABs compared with conventional oral preventives in adolescents with high-burden migraine.

Methods: We analyzed depersonalized data from the German Pain e-Registry. Adolescents (12–17 years) with ≥ 1 documented 6-month episode of high-evidence conventional oral preventives (HECP) and ≥ 1 subsequent CGRP mAB episode were included. The primary endpoint was a composite of 6-month persistence and $\geq 50\%$ reduction in monthly migraine days (MMD).

Results: In 422 adolescents (1,448 HECP; 422 CGRP mAB episodes), 6-month persistence was 30.6% with HECP and 88.2% with CGRP mAB. $A \geq 50\%$ MMD reduction occurred in 25.4% and 70.9%, respectively. The composite endpoint was reached in 23.7% with HECP versus 69.9% with CGRP mAB. Mean MMD decreased from 11.7 to 9.4 with HECP and from 11.6 to 4.4 with CGRP mAB. Improvements in disability and psychosocial outcomes were consistently greater with CGRP mAB.

Conclusion: In this high-burden adolescent cohort, CGRP mAB treatment episodes were associated with markedly higher persistence and broader improvements than prior conventional preventive episodes. Given the non-randomized, sequential design, these findings are hypothesis-generating and support the need for controlled pediatric trials.

Disclosure: M.A. Überall is honorary board member of the German Pain Association and the German Pain League and Director of the Institute of Neurological Sciences (IFNAP) and CEO of O.Meany – Medical Data & Project Management GmbH. The German Pain e-Registry is hosted by O.Meany – MDPM GmbH under scientific governance of IFNAP. No third-party

funding was received. The author declares no other conflicts of interest.

EPO-0092 | Real-world treatment persistence in migraine prevention: A national registry comparison of CGRP monoclonal antibodies and oral preventives

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Background and aims: Oral migraine preventive drugs are frequently discontinued in routine care. Most real-world studies rely on prescription claims and cannot capture clinically validated reasons for treatment termination. In addition, many datasets predate the availability of monoclonal antibodies targeting calcitonin gene-related peptide (CGRP). To compare real-world treatment persistence and documented reasons for discontinuation among patients receiving subcutaneous CGRP monoclonal antibodies (CGRP mAB), oral high-evidence preventive therapies (HEVP), and oral low-evidence preventives (LEVP).

Methods: This observational cohort study analyzed depersonalized routine-care data from the German Pain e-Registry, a nationwide multicenter clinical registry. Patients with physician-confirmed migraine receiving preventive therapy were followed for 182 days. Treatment persistence was defined as continuous use of the index therapy. Discontinuations were classified as due to adverse drug reactions (ADR) or insufficient efficacy. Persistence trajectories were assessed using Kaplan–Meier analyses and pairwise risk comparisons.

Results: A total of 36,465 patients were included (12,347 CGRP mAB; 23,353 HEVP; 765 LEVP). Six-month persistence was markedly higher with CGRP mAB (89.3%) than with HEVP (32.7%) or LEVP (42.6%; $p < 0.001$). ADR-related discontinuation occurred in 6.8% of CGRP mAB patients, compared with 45.1% in HEVP and 35.8% in LEVP. Discontinuation due to insufficient efficacy was 3.6% with CGRP mAB versus 22.2% and 21.6% with HEVP and LEVP. Sex and migraine subtype had no clinically meaningful influence. Within HEVP, tricyclic antidepressants, beta-blockers, and valproic acid showed the lowest persistence.

Conclusion: CGRP monoclonal antibodies demonstrate substantially superior real-world persistence and tolerability compared with conventional oral migraine preventives, highlighting the clinical relevance of mechanism-based prevention.

Disclosure: M.A. Überall is honorary board member of the German Pain Association and the German Pain League and Director of the Institute of Neurological Sciences (IFNAP) and CEO of O.Meany – Medical Data & Project Management GmbH. The German Pain e-Registry is hosted by O.Meany – MDPM GmbH under scientific governance of IFNAP. All other authors declare no competing interests.

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Background and aims: Headache disorders are among the leading causes of disability worldwide, yet undergraduate medical education in headache medicine remains variable and often lacks standardized training. Although the International Classification of Headache Disorders, 3rd edition (ICHD-3), provides a globally accepted framework for headache diagnosis, structured instruction during medical training is inconsistent. We therefore implemented a digital ICHD-3 instructional module and evaluated its impact.

Methods: Medical students from two undergraduate programs participated in a pre-post educational evaluation. Baseline knowledge and confidence were assessed using a pre-intervention survey, followed by a 20-minute structured teaching session focused on use of ICHD-3, including a brief review of SNOOP red-flag features. The post-intervention survey repeated

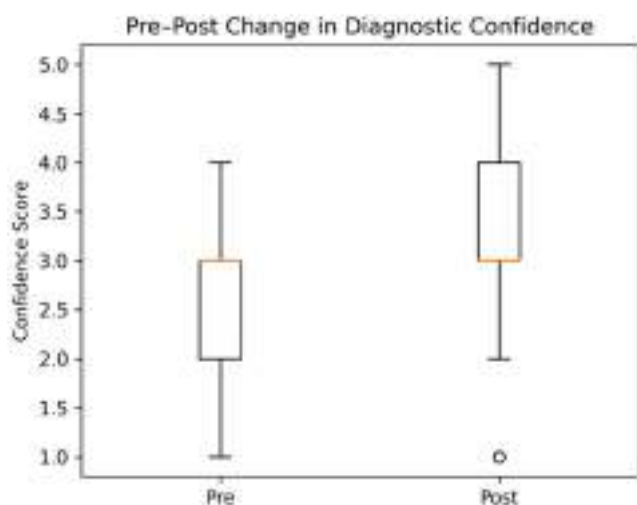


FIGURE 1 Pre-post change in diagnostic confidence scores following the ICHD-3 educational intervention. Post-intervention confidence scores were significantly higher than baseline (Wilcoxon signed-rank test, $p < 0.001$).

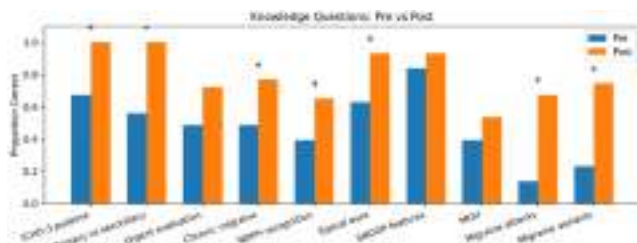


FIGURE 2 Proportion of students correctly answering knowledge questions before and after an ICHD-3-based educational intervention. Questions assessed core diagnostic concepts in headache disorders. Asterisk denotes $p < 0.05$ on McNemar's exact test.

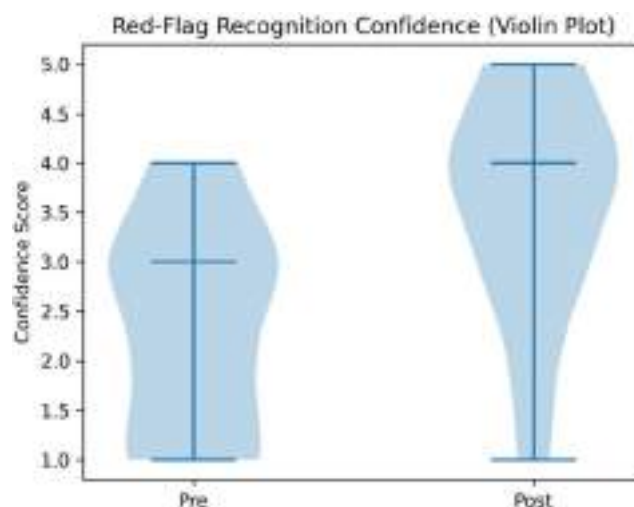


FIGURE 3 Pre-post change in recognition of secondary headache red flags (SNOOP criteria). Post-intervention scores demonstrated a significant improvement compared with baseline (Wilcoxon signed-rank test, $p < 0.001$).

all assessment items. For vignette-based questions, participants were permitted to access the digital ICHD-3 to simulate point-of-care diagnostic support. Pre-post changes were analyzed using the Wilcoxon signed-rank and McNemar's tests.

Results: Forty-three students completed both assessments. Post-intervention confidence increased significantly across all evaluated domains (all $p < 0.001$), including focused history-taking, primary versus secondary headache disorders, SNOOP, ICHD-3 diagnostic criteria, and communication of headache diagnoses. Perceived usefulness of headache classification increased significantly ($p < 0.001$), while interest in a quick diagnostic reference remained high and unchanged ($p = 0.096$). Diagnostic knowledge accuracy improved for key ICHD-3 concepts, including migraine and new daily persistent headache diagnosis, typical aura, and episodic migraine-associated syndromes (all $p \leq 0.019$).

Conclusion: These findings demonstrate that a brief, digitally supported ICHD-3-based intervention can improve diagnostic confidence and accuracy among medical students and support a scalable approach to strengthening structured headache education.

Disclosure: Nothing to disclose.

EPO-0094 | Self-reported redosing and treatment outcomes with oral triptans for acute migraine: Results from the DREAM study

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Background and aims: This nationwide population study evaluated self-reported frequency of redosing after use of oral triptans, treatment effectiveness as assessed by pain relief and return to normal function, and adverse events.

Methods: All Danish residents ≥18 years with a triptan purchase or a migraine diagnosis (ICD-10: G43) between January 2021 – December 2023 were invited to the nationwide DREAM (Danish REgistry Analyses of Migraine) study combining data from national health registers with a survey on acute migraine medication experience.

Results: 41,498 people reported a migraine attack within the past 3 months. Baseline characteristics are presented in Table 1 Among 21,960 individuals using oral triptans as the only or the first medication type to treat their most recent migraine attack (Figure 1) 49% reported redosing with the same or a different medication, most commonly (77%) between 2–24 h. The proportion achieving 2 h pain relief, return to normal function, and sustained pain relief was significantly lower among individuals who redosed between 2–24 h compared with those who did not redose (73% vs 88%, 32% vs 50%, 57% vs 84%, respectively; $p < 0.01$ for all comparisons). Among those redosing a significantly lower proportion experienced no adverse events vs those not redosing (Figure 2) (38% vs 50% $p < 0.01$).

Conclusion: In this nationwide real-world study almost half of individuals taking oral triptans reported redosing, which was associated with poorer pain and functional outcomes and a

TABLE 1 Baseline characteristics

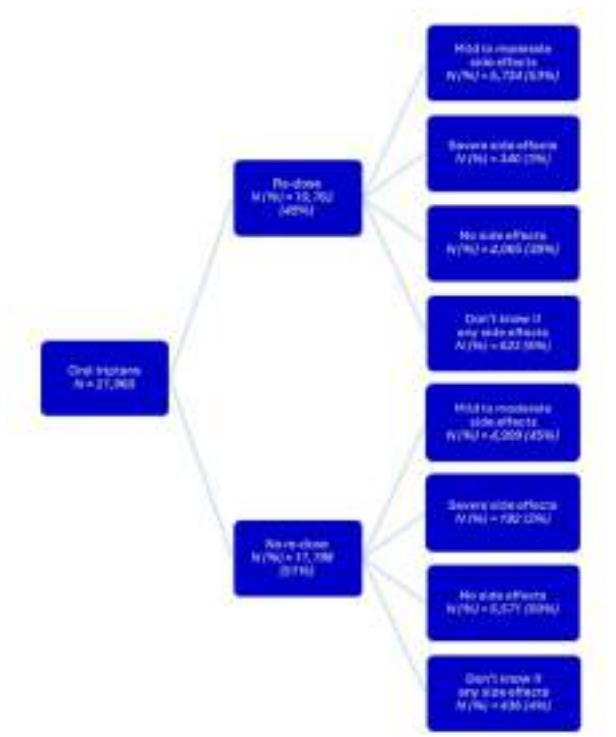
	Migraine population (MF 1) n (%)	Reference population (MF 2) n (%)
N	41,498	134,494
Age		
Age, mean (SD)	48.2 (13)	49.2 (13)
Age, median (Q4)	50 (40, 58)	50 (40, 58)
Sex		
Women	34,285 (84.1%)	134,494 (84.1%)
Men	6,800 (15.9%)	13,800 (15.9%)
Annual income (DKK)		
Income, mean (SD)	43,449 (11,840)	42,170 (12,101)
Income, median (Q4)	47,775 (3,262, 64,586)	46,387 (3,160, 63,813)
Individuals in the workforce	37,426 (90.2%)	112,828 (90.2%)
Income in this reference, mean (SD)	47,240 (11,732)	46,545 (12,126)
Highest attained education		
Primary education	4,566 (11.0%)	12,176 (15.4%)
Upper secondary education	3,189 (7.7%)	9,958 (8.9%)
Vocational education and training	12,627 (30.5%)	39,171 (31.9%)
Short cycle higher education	2,889 (6.9%)	6,842 (5.3%)
Vocational (see below) education/bachelor's programs	10,186 (24.4%)	30,870 (24.0%)
Master's programs	5,707 (13.9%)	15,802 (12.7%)
PhD programs	589 (1.4%)	1,382 (1.1%)
Not stated	335 (0.8%)	1,824 (1.3%)

Note: The reference population consists of individuals who are not identified as being referred to a specialist or having an acute diagnosis. The data are presented as a 1:1 ratio comparing matching on age, sex, region of residence, and age. The annual income is constructed using the Danish tax register data.



Note: The population of 21,960 individuals consists of those who reported using oral triptans for acute treatment of their most recent migraine attack. Individuals were included if they had exclusively used oral triptans (EPO-095) or if it was possible to confirm their oral triptan use as the first medication in a multi-drug treatment regimen (EPO-095). Due to the structure of the questionnaire, the first medication taken was not identified when individuals indicated they had taken their second dose within 2–24 hours. This requirement left 2,522 individuals out of this category, which is why the total number of individuals in the population of 21,960 individuals who used oral triptans for acute treatment of their most recent migraine attack is 21,960. Pain relief indicates pain relief at 2 hours.

FIGURE 1 Redosing, pain relief, and return to normal function after oral triptan use for acute treatment of the most recent migraine attack



Note: The population of 21,960 individuals consists of those who reported using oral triptans for acute treatment of their most recent migraine attack. Individuals were included if they had exclusively used oral triptans (EPO-095) or if it was possible to confirm their oral triptan use as the first medication in a multi-drug treatment regimen (EPO-095). Due to the structure of the questionnaire, the first medication taken was not identified when individuals indicated they had taken their second dose within 2–24 hours. This requirement left 2,522 individuals out of this category, which is why the total number of individuals in the population of 21,960 individuals who used oral triptans for acute treatment of their most recent migraine attack is 21,960. Side effects refer to symptoms, for the 5,724 individuals, they were mild to moderate, or severe.

FIGURE 2 Redosing and side effects of medications for acute treatment of the most recent migraine attack.

higher frequency of adverse events. Need for redosing reflects insufficient effectiveness of the initial dose and highlights an unmet need in acute migraine treatment.

Disclosure: This study was sponsored by Pfizer. Medical writing support was provided by EY Parthenon, a paid vendor to Pfizer.

Infectious diseases 1

EPO-095 | Inflammatory markers in tubercular meningitis and their role in predicting paradoxical reaction

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Background and aims: Tubercular meningitis (TBM) is associated with high morbidity and mortality due to an intense inflammatory response leading to basal exudates, cranial nerve palsies, vasculitic infarcts, and hydrocephalus. Paradoxical reaction (PR) occurs in nearly 30% of patients and is an important cause of clinical deterioration during treatment. Dysregulated inflammatory mediators in serum and cerebrospinal fluid (CSF) have been implicated in PR and adverse outcomes in TBM.

Methods: In this prospective observational study, 151 newly diagnosed TBM patients fulfilling the Marais et al. consensus

diagnostic criteria were enrolled. Baseline clinical, radiological, serum, and CSF inflammatory markers were assessed in treatment-naïve patients and repeated at 3 months or at the time of PR or stroke. Serum markers included hsCRP, ESR, LDH, ferritin, albumin, D-dimer, neutrophil-lymphocyte ratio (NLR), and systemic immune-inflammation index (SII). CSF analysis included ADA, NLR, protein, sugar, and total leukocyte count.

Results: The mean age was 32.7years; headache was the most common symptom (93.4%). Paradoxical reaction developed in

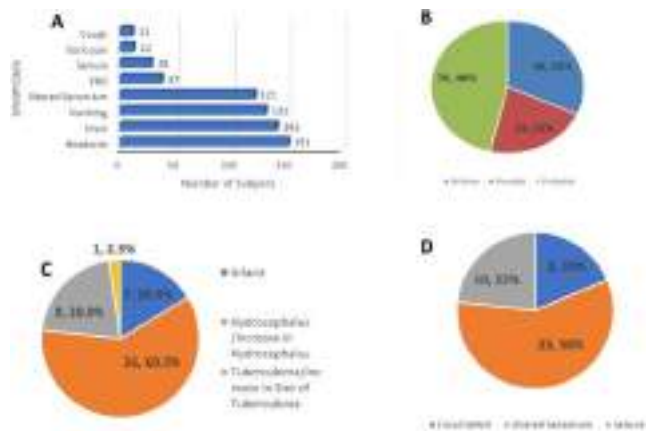


FIGURE 1 (A)- Frequency of Symptoms, (B)- Staging of TBM, (C)- Radiological manifestation of paradoxical reaction, (D)- Clinical Manifestation of Paradoxical reaction.

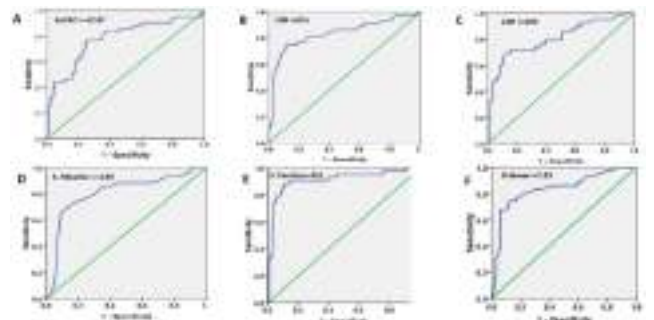


FIGURE 2 The receiver operating characteristic curve showing the discrimination power of (A)- HsCRP, (B)- ESR, (C)- S. LDH, (D)- S. Albumin, (E)- S. Ferritin, (F)- D-dimer between paradoxical reaction and non-paradoxical reaction group.

	Regression coefficient t	Standar rd Error	Signific ant	Odds ratio	95.0% C.I. for OR	
					Lower	Upper
D dimer	.246	.203	.225	1.279	.859	1.905
LDH	-.002	.003	.552	.998	.992	1.004
S ferritin	.004	.001	.001	1.004	1.002	1.007
ESR	.020	.009	.033	1.020	1.002	1.039
HsCRP	.002	.009	.825	1.002	.985	1.019
S. albumin	-.466	.650	.473	.627	.175	2.243
Constant	-2.688	2.503	.283	.068		

TABLE 1 Multivariate logistic regression analysis for predictors of paradoxical reaction in TBM.

43 patients (28.4%), most commonly presenting with altered sensorium. Radiologically, worsening hydrocephalus was the predominant finding. On univariate analysis, serum albumin, D-dimer, LDH, ferritin, ESR, and hsCRP were significantly associated with PR. On multivariate analysis, Serum Ferritin and ESR independently predicted the paradoxical reaction.

Conclusion: Serum inflammatory markers, particularly ferritin and ESR, may serve as useful predictors of paradoxical reaction in TBM. These findings support an immune-mediated mechanism underlying paradoxical worsening and may help identify high-risk patients who could benefit from early anti-inflammatory or immunomodulatory therapy.

Disclosure: Nothing to disclose.

EPO-0096 | An emerging arboviral trigger of Guillain-Barré syndrome: The first documented case linked to Toscana virus

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Background and aims: Guillain-Barré syndrome (GBS) may occur after various infectious triggers, including arboviral infections. Toscana virus (TOSV) is an emerging sand fly-borne phlebovirus endemic in the Mediterranean region, typically associated with neuroinvasive diseases. To date, no cases have established a clear temporal relationship between serologically confirmed acute TOSV infection and the subsequent development of GBS.

Methods: We report the clinical, neurophysiological, and laboratory findings of a patient diagnosed with GBS following acute TOSV infection. The diagnostic work-up included cerebrospinal fluid (CSF) analysis, nerve conduction studies, molecular and serological testing for neurotropic pathogens and assessment of antibodies against gangliosides and sulfatides. A targeted review of the literature was conducted to identify previously reported cases of Guillain-Barré-like syndrome associated with TOSV infection.

Results: CSF findings and electrodiagnostic studies were consistent with the acute inflammatory demyelinating polyneuropathy variant of GBS. Real-time PCR detected TOSV RNA in urine, supporting a recent TOSV infection temporally antecedent to neurological symptom onset. The patient underwent six plasma exchange sessions in addition to supportive care, resulting in progressive neurological improvement.

Conclusion: To our knowledge, this is the first documented case establishing a clear temporal association between TOSV acute infection and GBS onset. This report highlights the importance of considering TOSV as a potential infectious trigger of post-infectious GBS in endemic areas.

Disclosure: Nothing to disclose.

EPO-0098 | Foodborne botulism: Signs beyond descending weakness

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Background and aims: Botulism neurotoxin intoxication is a rare and life-threatening condition that typically causes a descending flaccid paralysis. However, rarely, autonomic dysfunction may be the predominant clinical manifestation and should be considered in the differential diagnosis of pure dysautonomia.

Methods: We describe a case series of three patients with atypical botulism presenting predominantly with dysautonomia and a favourable outcome.

Results: Patient 1 was a 40-year-old man, patient 2 a 35-year-old woman, and patient 3 a 70-year-old man, all of whom sought medical care because of blurred vision and xerostomia. Additional symptoms included gastrointestinal manifestations (abdominal pain, nausea, and vomiting in patient 1 and constipation in all patients), urinary dysfunction (urinary hesitancy in patients 1 and 3), hypotension (patient 3), mild diplopia (patient 3) and bulbar complaints with mild clinical variations among the three cases. Neurological examinations were normal in all patients, except for accommodative paresis. All three patients had consumed cured ham at the same meal. Electromyographic studies didn't reveal neuromuscular junction disfunction. Serum assays were positive for botulinum toxin type B in all patients. In two cases, toxin B-encoding genes were detected in stool samples via PCR. Antitoxin was not administered as all patients were admitted more than two weeks after symptom onset. At a four-month follow-up, all patients showed nearly complete recovery.

Conclusion: Botulism can be a challenging diagnosis when patients do not develop the typical descending weakness or respiratory failure. Careful consideration of the full clinical presentation and epidemiological context is essential for accurate diagnosis, as timely antitoxin administration may be lifesaving.

Disclosure: Nothing to disclose.

EPO-0099 | Toscana virus meningoencephalitis, a case series

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Background and aims: Toscana virus (TOSV) is the neurotropic arbovirus of greatest clinical relevance in the Mediterranean basin and the Middle East. Our objective is to describe a series of TOSV meningoencephalitis cases within our catchment area.

Methods: We describe five cases of TOSV meningoencephalitis identified between 2021 and 2024 at General University

Hospital of Elche and Vega Baja University Hospital, located in the province of Alicante, Spain, in order to describe their clinical and epidemiological characteristics and relevant findings in complementary tests.

Results: Most patients were men in their eighth decade of life. The most frequent comorbidities included diabetes, hypertension, and prostate cancer. On admission, the predominant symptoms were fever or low-grade fever, headache with red-flag features, and confusion. Cerebrospinal fluid analysis most commonly showed lymphocytic pleocytosis and elevated protein levels. Neuroimaging revealed no abnormalities attributable to TOSV infection, and only one patient exhibited epileptiform activity on electroencephalography. Clinical outcomes were favorable, with all patients returning to their baseline status.

Conclusion: TOSV should be included in the differential diagnosis of aseptic meningitis and other central nervous system (CNS) infections in endemic regions. Its incidence is expected to rise in the coming years as increasing global temperatures facilitate the spread of vector-borne diseases into new geographical

TABLE 1 Case characteristics and clinical features (Given the small sample size, results are presented as absolute frequencies).

Characteristics	Frequency
Age - yr (median)	36 -78 (73)
Male sex - n	5/5
Clinical features	Frequency
Headache - n	4/5
Precipitated by Valsalva maneuvers	1/5
Postural aggravation	1/5
Fever - n	5/5
Nausea or vomiting - n	2/5
Meningism - n	2/5
Consciousness impairment - n	4/5
Focal neurological impairment - n	1/5
Seizures - n	1/5
Intensive care unit admission - n	1/5

TABLE 2 Cerebrospinal fluid (CSF) characteristics and complementary test. CRP: reactive C protein; Mon: mononuclear cells; ANA: Antinuclear antibodies; EEG: Electroencephalogram.

	Case 1	Case 2	Case 3	Case 4	Case 5
CRP(ng/dL)	5	5.9	5.1	0.018	0.15
White blood cells/L in plasma	1430	4480	8793	4713	4380
White blood cells/L in CSF	180 (90% Mon)	130 (95% Mon)	36 (99% Mon)	796 (96% Mon)	422 (96% Mon)
CSF/plasma glucose ratio (mg/dL)	77/148 (0.52)	68/120 (0.56)	92/188 (0.48)	70/142 (0.49)	67/137 (0.53)
CSF proteins (mg/dL)	115	88	95	>200	148
Autoimmunity tests	Negative	Negative	ANA positive	ANA positive	Not performed
EEG	Encephalopathy	Not performed	Encephalopathy	Encephalopathy + epileptiform activity	Not performed

areas. Identification and characterization of these cases will enhance our understanding of the disease and help determine its prognosis and management.

Disclosure: Nothing to disclose.

EPO-0100 | Otogenic meningoventriculitis due to *Pseudomonas stutzeri* managed with surgery and TDM-monitored targeted antibiotic therapy: A case report

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Background and aims: Otogenic meningo-ventriculitis is a challenging condition, particularly when caused by uncommon Gram-negative pathogens and complicated by hydrocephalus. We report a rare case of *Pseudomonas stutzeri* meningoventriculitis managed with aggressive surgical strategy and fosfomycin-based combination treatment with serial plasma/CSF fosfomycin monitoring.

Methods: Clinical, microbiological, neuroimaging, and CSF data were collected during admission. Serial CSF analyses were performed from external drains and lumbar punctures. Blood and CSF cultures guided therapy. Fosfomycin concentrations were monitored in plasma and CSF to support dosing optimization.

Results: A 70-year-old woman was admitted with acute hydrocephalus and suspected otogenic meningoventriculitis. Urgent external ventricular drainage was placed. Blood cultures grew *Pseudomonas stutzeri* (wild type; fosfomycin MIC 32 mg/L) and CSF microscopy showed Gram-negative bacilli; CSF culture confirmed *P. stutzeri*. After initial empiric therapy with ceftriaxone plus levofloxacin, antibiotic therapy was escalated to cefepime plus fosfomycin. Neuroimaging supported meningo-ependymal enhancement and acute otomastoiditis; ENT source control included tympanostomy and subsequent mastoid surgery. Despite persistently abnormal CSF cytochemistry, follow-up CSF cultures remained negative. Based on adequate source control, targeted and TDM-guided antibiotic therapy (Figure 1), clinical/radiological improvement and sustained culture negativity, definitive ventriculo-peritoneal shunt was placed on day 30 after admission; intraoperative CSF sampling resulted negative. Antibiotics were discontinued on day 34. At 1-year follow-up, no relapse occurred.



FIGURE 1 Fosfomycin concentrations in plasma and CSF.

Conclusion: *P. stutzeri* is a rare cause of otogenic meningoventriculitis. A combining prompt source control, and fosfomycin-based combination treatment with serial plasma/CSF fosfomycin monitoring may improve the patient's outcome.

Disclosure: Nothing to disclose.

EPO-0101 | Development of a patient reported outcome measure for infectious encephalitis: What matters most to patients

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Background and aims: Research on the long-term outcomes of patients with infectious encephalitis (IE) is limited. Existing evidence from a systematic review indicates that most studies and clinicians use the modified Rankin Scale (mRS), a global disability score developed for stroke which does not capture the cognitive, behavioural, emotional and quality of life (QoL) consequences of IE. The current study aims to develop a disease-specific patient-reported outcome measure (PROM) to address this gap.

Methods: Semi-structured interviews were developed following COSMIN guidance, with international input from clinicians and adults with lived experience of IE. Participants were recruited via Encephalitis International until data saturation was achieved ($n=40$). Transcripts of all interviews were thematically analysed by 2 independent reviewers, to identify concepts for the initial instrument.

TABLE 1 Demographics of the 40 patients who completed a semi-structured interview.

Participants	40
Person affected	37
Caregiver	3
Sex	
Female	21
Male	19
Location	
UK	26
USA	7
Other*	7
Type of encephalitis	
HSV	22
Not specified	12
Tick-borne	3
Other**	3
ITU admission	11
Median age at diagnosis (IQR)	42 (26-54)
Time since diagnosis in months (IQR)	50 (25-192)
Days spent in hospital (IQR)	21 (15-35)

*Australia, Finland, India, Netherlands, Philippines, Spain, Switzerland
** West Nile Virus, Influenza, Mumps

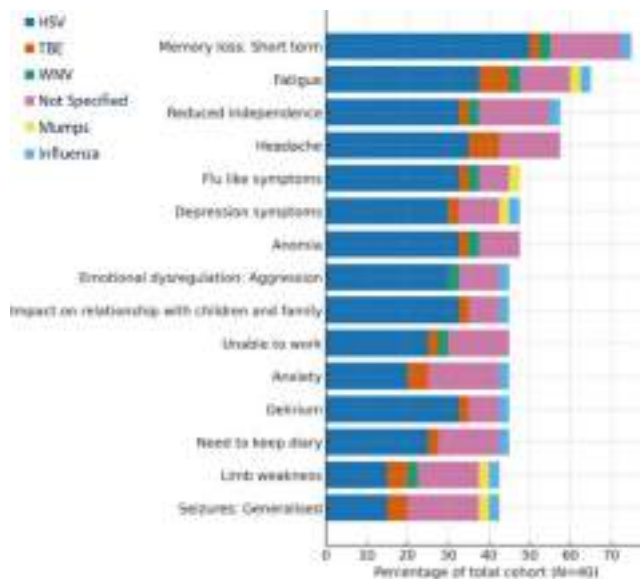


FIGURE 1 A graph of the 20 most prevalent items identified in thematic analysis of semi-structured interviews.

Results: 101 items were initially identified, reduced to 73 items after removing duplicates and items with < 3 responses. Item breakdown by domain included Physical (25), Health related QoL (13), Cognitive (14), Psychological (9), Behavioural (12) and were shared across multiple aetiologies. Participants with a 'good outcome' according to mRS described substantial ongoing symptoms and impact on QoL. 11 participants experienced severe initial disease, and most patients spent an extended duration (21 day mean) in hospital.

Conclusion: This series of semi-structured interviews has generated 73 items, spanning across multiple domains. Common items have been shared across aetiologies, suggesting a generic PROM for IE is justifiable. The next steps in PROM development are to undertake further refinement of items and cognitive debriefing interviews to assess for content validity.

Disclosure: Nothing to disclose.

EPO-0102 | Post-acute COVID-19 effects on standardized cognitive and motor test scores in a longitudinal study of independent elderly subjects

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Background and aims: There is considerable concern about long-term consequences of COVID-19, including cognitive and motor decline. Whether COVID-19 adds burden beyond normal aging remains unclear, particularly in cohorts studying neurological disease. This study examined whether prior COVID-19 illness was associated with differential cognitive or motor decline in older adults over time broadly.

Methods: Subjects were included if they were cognitively normal before the pandemic, completed a COVID questionnaire, and attended subsequent diagnostic conferences. All received serial evaluations with Montreal Cognitive Assessment (MoCA) and Unified Parkinson's Disease Rating Scale (UPDRS). We compared, between those reporting COVID-19 and those denying it, conversion to cognitive impairment or dementia and changes in cognitive and motor scores longitudinally.

Results: 100 participants reported COVID-19 and 71 denied infection. Illness severity was generally mild; none required ventilation. Cognitive and motor performance declined slightly without statistical significance. Conversion occurred in twenty-six percent of COVID-19 participants and thirty-two percent of controls, a nonsignificant difference between groups during follow-up across the study period. Logistic regression indicated that self-reported COVID-19 was not associated with diagnostic conversion, while greater age, male sex, possession of one

TABLE 1 Demographic characteristics of subjects reporting having had COVID-19 or not. Conversion refers to the percentage of subjects that converted from cognitively normal to cognitive impairment or dementia. No significant differences between groups.

COVID-19	Age	Sex	Education Yrs	AgeE+4	Peak PD	Conversion
YES (n = 100)	81.79 (8.67)	34 M (34%)	15.88 (2.43)	23 (23%)	19 (19%)	26 (26%)
NO (n = 71)	82.36 (7.29)	24 M (34%)	15.85 (2.33)	16 (22.5%)	11 (15.5%)	23 (32.4%)

TABLE 2 Comparison of pre- and post-pandemic MoCA and UPDRS motor scores in subjects reporting having had or not having had COVID-19.

COVID-19	MoCA Study Beginning	MoCA Study End	UPDRS Study Beginning	UPDRS Study End
YES	26.56 (2.88) n = 40*	25.96 (2.87) n = 40	2.95 (4.37) n = 40	4.79 (7.63) n = 40
NO	26.37 (2.03) n = 40	24.8 (4.16) n = 40*	2.40 (3.67) n = 42	4.96 (6.27) n = 42

TABLE 3 Logistic regression analysis of relationship of reported COVID-19 on likelihood for conversion from cognitively normal to cognitively impaired or dementia, when also considering multiple covariates.

Variable	Odds Ratio (95% CI)	Probability	Beta Coefficient	Standard Error
Reported COVID-19	0.71 (0.335-1.52)	0.36	-0.338	0.386
Age*	4.67 (1.91-11.46)	0.0007	1.54	0.407
Male Sex*	3.76 (1.30-8.35)	0.0110	1.328	0.404
Education Years	1.42 (0.62-3.25)	0.405	0.352	0.422
AgeE+4*	1.50 (1.32-1.70)	0.0095	1.151	0.444
Predicted PD*	2.863 (1.00-8.20)	0.050	1.052	0.536

or more apolipoprotein E-ε4 alleles, and a diagnosis of probable Parkinson's Disease all conferred a greater likelihood of conversion.

Conclusion: These findings suggest that mild COVID-19 illness is not associated with greater cognitive or motor decline than expected from age-related change alone. Limitations include modest sample size, potential misclassification of COVID-19 status, and reliance on relatively crude screening measures that may miss subtle functional effects and longer-term disease-modifying consequences remain.

Disclosure: Nothing to disclose.

EPO-0103 | Temporal evolution of clinical and cognitive markers following viral encephalitis

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Background and aims: Objectives: To investigate cognitive functioning and clinical markers in patients shortly following viral encephalitis. In addition, the patients' precepted impact of sequelae on their daily functioning.

Methods: Methods: Autoimmune Inflammation as cause of Relapsing Symptoms in patients with Viral Encephalitis (ARISE) was a prospective, observational, single-centre cohort study in Amsterdam UMC, the Netherlands. All patients aged ≥16 years with viral encephalitis and confirmed pathogen were eligible for this study. Patients with prior cognitive impairment, recent neurosurgery or neuro-devices were excluded. Follow-up was at week 1, 4 and 12, and year 1 from the onset of viral encephalitis. Cognitive functioning was assessed each follow-up. Blood samples were drawn at week 1, 4 and 12, and cerebrospinal fluid (CSF) from diagnostic lumbar puncture was collected, to be screened for neuron-specific enolase (NSE), S100b and auto-antibodies.

Results: Results: Eight patients were included between 2020 and 2024, of which 1 of 8 (13%) subjects had died during admission. Outcome at discharge was unfavourable in 7 of 8 (88%) patients. Follow-up data was available for 7 patients. Cognitive functioning showed most pronounced deficits in short-term memory and visuospatial/executive function. Patients reported perceiving to be most limited by physical health and energy. Daily functioning was impaired greatest in their speed, energy and attention. Median values for NSE and S100b did not exceed reference values in CSF or blood samples. No samples showed reactivity for auto-antibodies.

Conclusion: Conclusions: Post-viral encephalitis results primarily in cognitive impairment of short-term memory, speed, and attention, hindering daily functioning one year after onset.

Disclosure: Nothing to disclose.

TABLE 1

Cohort characteristics	ARISE (N = 8)
Sex, Female, n (%)	2 2/8 (25%)
Age, Median [Q1, Q3]	58 [44, 67]
Medical History	
Psychiatric symptoms, n (%)	1 1/8 (13%)
Immunocompromised ^A , n (%)	1 1/8 (13%)
Symptoms at Presentation	
Headache, n (%)	2 2/7 (29%)
Fever, n (%)	6 6/8 (75%)
Neck stiffness, n (%)	0 0/6 (0%)
Seizures preadmission, n (%)	3 3/8 (38%)
Glasgow Coma Scale	
Altered mental status, EMV ≤14, n (%)	2 2/8 (25%)
Coma, EMV ≤8, n (%)	1 1/8 (13%)
Cerebrospinal Fluid (CSF)	
Leucocytes, cells per mm3, Median [Q1, Q3]	54 [35, 185]
CSF to blood glucose ratio, Median [Q1, Q3]	0.55 [0.51, 0.60]
Protein, g/L, Median [Q1, Q3]	0.69 [0.56, 1.13]
Opening pressure, cm H2O, Median [Q1, Q3]	23 [22.0, 29.0]
Causative pathogen	
HSV-1, n (%)	5 5/8 (63%)
HSV-2, n (%)	1 1/8 (13%)
VZV, n (%)	1 1/8 (13%)
HHV - 7, n (%)	1 1/8 (13%)
Treatment	
Received antiviral treatment, n (%)	8 8/8 (100%)
Glasgow Outcome Scale at Discharge	
Favourable outcome, n (%)	4 4/8 (50%)
Unfavourable outcome, n (%)	4 4/8 (50%)
Death, n (%)	1 1/8 (13%)
Location of Discharge	
Home, n (%)	4 4/8 (50%)
Rehabilitation Centre, n (%)	3 3/8 (38%)
Died during Admission, n (%)	1 1/8 (13%)
Admission	
Duration, days, Median [Q1, Q3]	19 [13, 25]

^A Receiving immunosuppressive therapy, underwent splenectomy, documented HIV, diabetes or alcoholism

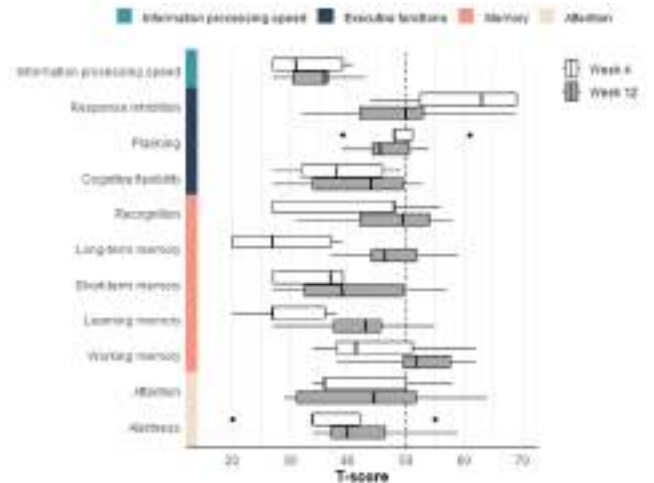


FIGURE 1 COGBAT week 4 and week 12.

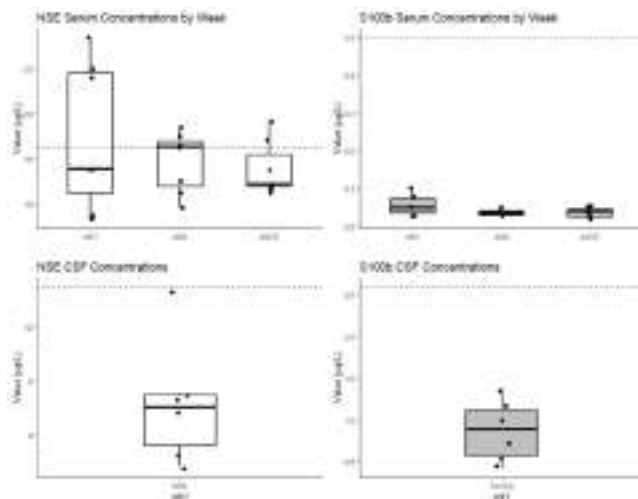


FIGURE 2 NSE and S100b.

EPO-0104 | Inkblot infarcts: A Distinctive MRI pattern of recurrent stroke in varicella zoster virus vasculopathy

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Background and aims: Varicella-zoster virus vasculopathy (VZVV) can manifest with recurrent, antithrombotic-refractory strokes in the absence of rash or prodrome, which presents a diagnostic and therapeutic challenge. This study aims to introduce the novel pattern in MRI brain, which we have named “Inkblot Infarct”, characterised by recurrent, slow-expanding strokes in the same subcortical region, secondary to VZVV.

Methods: We prospectively analysed five adult patients presenting with recurrent ischemic strokes at a tertiary care centre. All patients underwent comprehensive vascular, cardiac, immune, and infectious evaluations, along with serial magnetic resonance imaging. VZV-specific IgG antibody levels in cerebrospinal fluid (CSF) and serum were analysed. Patients were treated with acyclovir, and their clinical and radiological outcomes were assessed.

Results: All five patients demonstrated recurrent infarcts in the subcortical region, showing gradual expansion on serial imaging. None had a preceding rash or fever. Antiplatelets and anticoagulation therapy failed to prevent recurrence. Cerebrospinal fluid analysis revealed elevated anti-varicella zoster virus immunoglobulin G levels in all patients, with raised immunoglobulin G index where available. Following initiation of acyclovir, no further strokes occurred during follow-up.

Conclusion: The “Inkblot Infarct” pattern represents a unique manifestation of VZVV-related strokes, hypothesised to result from persistent viral antigen-mediated vascular remodelling. Atypical presentations, such as absent rashes and delayed symptom progression, emphasise the need for heightened clinical suspicion. Recognition of this pattern may facilitate earlier

TABLE 1 Patient Characteristics, clinical features, treatment and outcomes.

Patient	Age Sex	Medical History	Presented	Acute stroke	First Stroke	Presenting symptoms	Investigation after 1st stroke	Investigation after 2nd stroke	Investigation after 3rd stroke	Investigation after 4th stroke	Investigation after 5th stroke	Outcome
Patient 1	45M	HTN	Yes	Yes	Stroke 1	Exacerbated left hemiparesis due to gradual expansion of the infarcted subcortical region	Final stroke	Acyclovir initiated on 1st day	Subcortical infarct, periventricular	Final stroke	Subcortical infarct	10 days
Patient 2	55F	HTN	Yes	Yes	Stroke 1	Subcortical infarct of internal capsule	Final stroke	Acyclovir initiated on 1st day	Subcortical infarct, periventricular	Final stroke	Subcortical infarct	10 days
Patient 3	65M	HTN	Yes	Yes	Stroke 1	Subcortical infarct of internal capsule	Final stroke	Acyclovir initiated on 1st day	Subcortical infarct, periventricular	Final stroke	Subcortical infarct	10 days
Patient 4	55M	HTN	Yes	Yes	Stroke 1	Subcortical infarct of internal capsule	Final stroke	Acyclovir initiated on 1st day	Subcortical infarct, periventricular	Final stroke	Subcortical infarct	10 days
Patient 5	55M	HTN	Yes	Yes	Stroke 1	Subcortical infarct of internal capsule	Final stroke	Acyclovir initiated on 1st day	Subcortical infarct, periventricular	Final stroke	Subcortical infarct	10 days

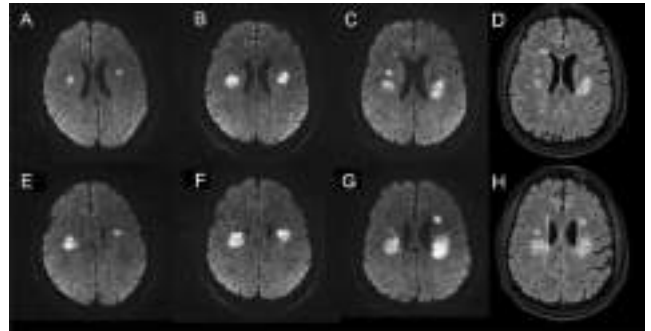


FIGURE 1 MRI Brain diffusion-weighted sequences, axial sections in patient 1 demonstrated restricted diffusion within the bilateral corona radiata and centrum semi-ovale (A, E).

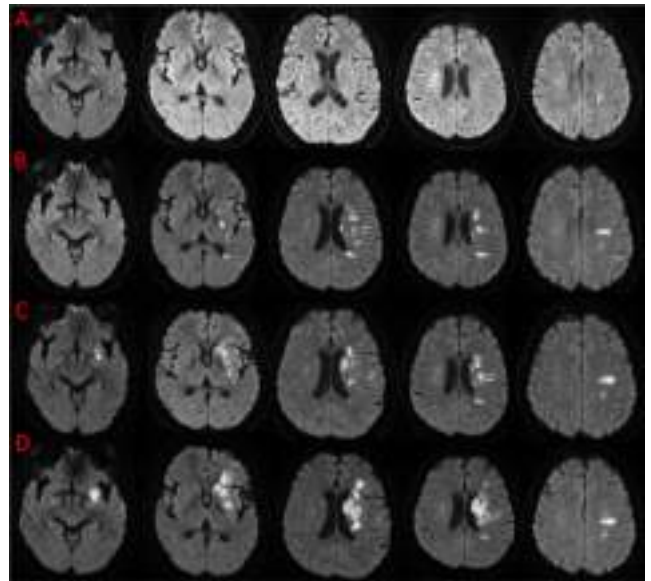


FIGURE 2 Panels A–D show axial diffusion-weighted MRI sequences from patient 2 over time, demonstrating the evolution of ischemic lesions in the brain at 5 anatomical levels (from left to right): the mesencephalic level, basal ganglia level, level above.

diagnosis and targeted antiviral therapy in recurrent cryptogenic stroke.
Disclosure: Nothing to disclose.

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Background and aims: Post-COVID-19 condition (PCC) shows substantial heterogeneity, with primarily neurological symptoms. It is unclear whether this reflects distinct disease entities or a spectrum of a shared condition enriched for neurological manifestations.

Methods: We analyzed data from 509 PCC patients (353 females, 48.15 ± 12.73 years), seen at the RWTH Aachen neurological Post-COVID-19 outpatient clinic from 2021 to 2024. Clinical history, acute and persistent (>12 weeks) symptom profiles, patient-reported outcome measures (PROMs) and cognitive performance were assessed. Multiple correspondence analysis followed by hierarchical clustering was used to derive data-driven phenotypes, which were compared across symptoms, PROMs and organ involvement.

Results: Three clusters emerged that aligned along a pronounced severity and multisystem-complexity continuum. Cognitive complaints were frequent across all clusters, but differed by overall symptom burden, functional impact, and extent of system involvement. Key clinical markers showed stepwise gradients from high-to-low-burden phenotypes, including severe fatigue (FSMC: 61.0% vs 23.8% vs 22.5%), impaired cognition (MoCA: 25.4% vs 11.9% vs 5.6%), depressive symptoms (HADS: 44.1% vs 18.4% vs 9.9%) and arthralgia (96.6% vs 75.5% vs 19.7%). The high-burden phenotype combined prominent neurocognitive symptoms with dyspnea, sensory/chemosensory disturbances, pain and higher psychological burden. Prior vaccination was more frequent in the lower-burden group, while

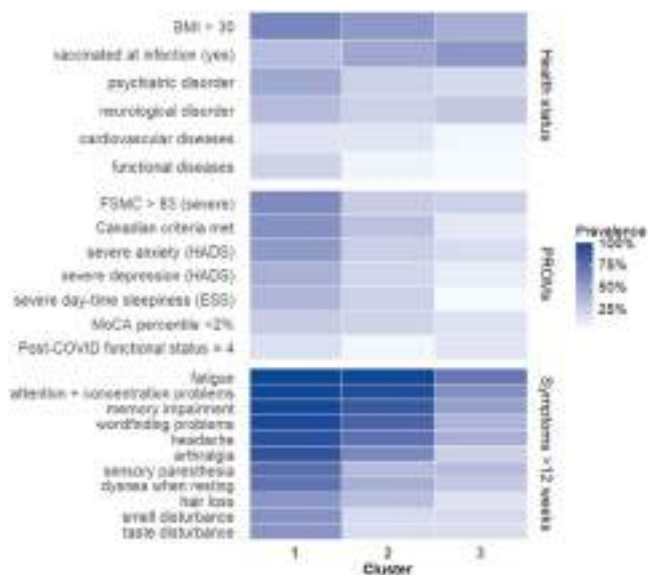


FIGURE 1 Heatmap for health status indicators, patient-reported outcome measures (PROMs) and symptom prevalence per cluster. Illustrated is the respective percentage of patients. Abbreviations: BMI: Body Mass Index; FSMC: Fatigue Scale for Motor and Cog.

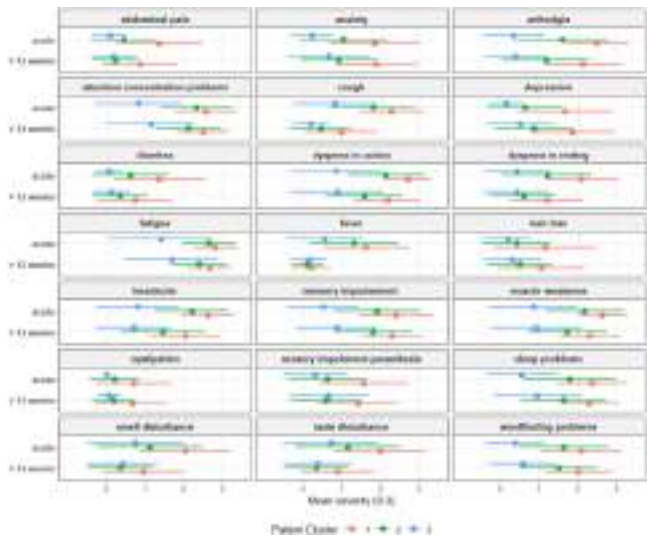


FIGURE 2 Subjective severity rating per cluster for acute and persisting (>12 weeks) symptoms. The mean severity rating and standard deviation is shown for each patient cluster for symptoms in the acute infectious and post-infectious stage after 12 weeks.

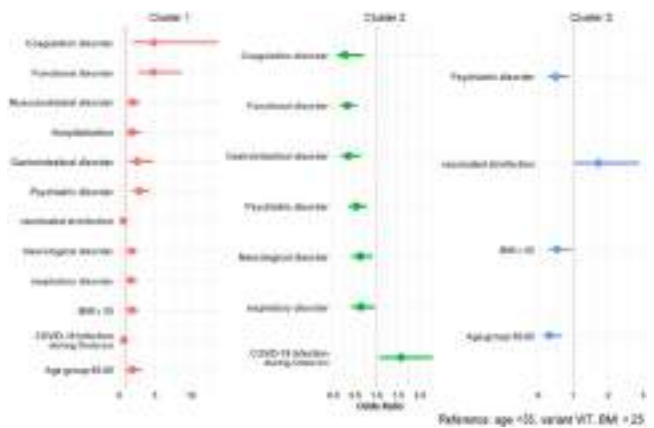


FIGURE 3 Pre-existing morbidities and health status associations with neurological PCC severity. The Odds Ratio is shown for neurological PCC patients to be categorized in a cluster based on their pre-morbid health status in a One-vs-rest approach for each cluster. The reference for age was <35, BMI reference was <25, and COVID-19 infection time-point reference was during wild-type (WT) variant wave. For the remaining factors, absence of the condition was used as reference.

psychiatric comorbidities and higher BMI were enriched towards the high-burden end.

Conclusion: Data-driven phenotyping supports a spectrum model in which neurological symptoms are common throughout. However, severity and disease complexity is driven by non-neurological factors (i.e. vaccination, metabolic health, comorbidities) underscoring the need of a coordinated multidisciplinary management even for patients presenting primarily with neurological symptoms.

Disclosure: Nothing to disclose.

EPO-0106 | Efficacy and tolerability of β -hydroxybutyrate in patients with ALS (KETO-ALS): A randomised, placebo-controlled, double-blind study

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Background and aims: Hypermetabolism and weight loss are prominent features of amyotrophic lateral sclerosis (ALS). Here, we sought to target the energy deficit in ALS by applying oral ketone bodies as high-energetic substrates.

Methods: Patients with ALS were enrolled to this randomised, placebo-controlled, double-blind, monocentre phase 2 trial. Eligible patients had a decline of ALS functional rating scale revised (ALSFRS-R) of at least 0.33 points/month. They were randomly assigned (1:1) to receive β -hydroxybutyrate-ester (β HB, 30 g/day) or placebo. Primary endpoint was change of neurofilament light chain (NfL) per month, measured as slope over 6 months. Multiple linear regressions models were applied to adjust for NfL, ALSFRS-R, age, and slow vital capacity (SVC) at baseline. The trial is registered with clinicaltrials.gov, number NCT04820478.

Results: Between April 5, 2022, and February 12, 2025, 81 patients were randomly assigned to receive β HB ($n=40$) or placebo ($n=41$) and included in the intention-to-treat analysis. NfL slope was -0.5 pg/mL (IQR -3.2 to 2.0 , minimum -11.2 , maximum 10.4) in the β HB group and 0.0 pg/mL (IQR -1.7 to 3.3 , minimum -6.2 , maximum 49.0) in the placebo group ($p=0.19$). In the adjusted analysis, both NfL ($p=0.04$) and BMI slope ($p=0.05$) showed a benefit for the β HB group. β HB was well tolerated.

Conclusion: This study provides preliminary evidence for efficacy and safety of oral ketone bodies in ALS by demonstrating target engagement and a reduction of NfL. Whether these effects can be translated into meaningful clinical effects remains to be clarified by a larger phase 3 trial with extended treatment duration.

Disclosure: Nothing to disclose.

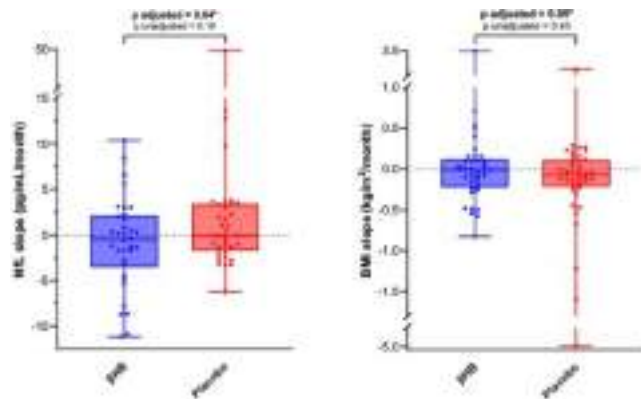


FIGURE 1 NfL and BMI slope.

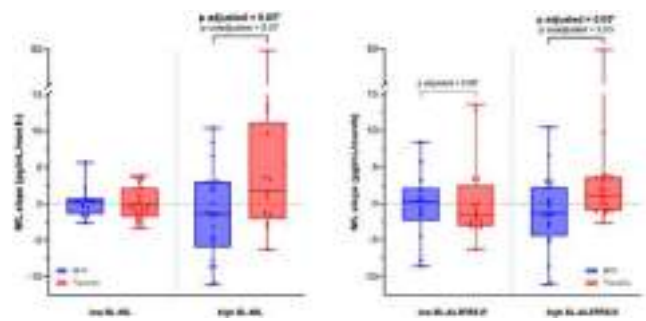


FIGURE 2 NfL subgroups.

	Total	β HB	Placebo	p-value
Number of patients	81	40	41	
Age (years)	62 (50-67)	59 (50-66)	64 (50-67)	0.06
Sex				0.94
Male	39 (48%)	19 (47%)	20 (49%)	
Female	42 (52%)	21 (53%)	21 (51%)	
Weight				0.04
kg	71 (57-78)	70 (57-78)	72 (58-79)	
BMI	24 (20-26)	24 (20-26)	25 (21-27)	
NfL slope (pg/mL/month)	0.0 (-3.2-2.0)	-0.5 (-3.2-2.0)	0.0 (-1.7-3.3)	0.19
ALSFRS-R	33 (27-37)	33 (27-37)	33 (27-38)	0.88
BMI slope (kg/m²/month)	0.0 (-0.1-0.1)	-0.1 (-0.1-0.1)	0.0 (-0.1-0.1)	0.05
SVC (L)	1.2 (0.9-1.5)	1.2 (0.9-1.5)	1.2 (0.9-1.5)	0.98
Weight loss (kg)	1.4 (0.0-2.8)	1.4 (0.0-2.8)	1.4 (0.0-2.8)	0.92
Progression rate (pg/mL/month)	0.0 (-0.1-0.1)	-0.1 (-0.1-0.1)	0.0 (-0.1-0.1)	0.12
Discontinuation (n/total)	2 (2/81)	1 (1/40)	1 (1/41)	0.96

Table 1: Patient Characteristics at Baseline
Data are given as median (interquartile range), n or % (%). Wilcoxon rank-sum test or chi-square test. Bold p-values signify significant differences. β HB: oral β -hydroxybutyrate; NfL: neurofilament light chain level in serum; ALSFRS-R: amyotrophic lateral sclerosis functional rating scale revised; BMI: body mass index; SVC: slow vital capacity; RL: baseline

EPO-0107 | Clinical implications of retinal markers in amyotrophic lateral sclerosis: A systematic review

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Background and aims: Amyotrophic lateral sclerosis (ALS) is a fatal, clinically heterogeneous, multisystem neurodegenerative disease, underscoring the need for biomarkers beyond motor decline. Optical coherence tomography (OCT) enables non-invasive retinal assessment and may identify relevant retinal biomarkers in ALS. Therefore, we systematically synthesised and evaluated the evidence on OCT-derived retinal biomarkers in ALS and their clinical relevance.

Methods: A systematic review was reported in accordance with the PRISMA statement and prospectively registered in PROSPERO (CRD420251242585). We searched MEDLINE, Embase, Scopus, and the Cochrane Library for observational studies reporting OCT-derived retinal biomarkers in adults with ALS, excluding non-eligible designs. The primary outcome was

the association between retinal OCT measures and functional severity. Risk of bias was assessed using QUIPS.

Results: Fifteen studies involving 1,301 patients, published between 2013 and 2025, were included, comprising predominantly cross-sectional designs with three longitudinal studies. Average peripapillary retinal nerve fibre layer (pRNFL) thinning was reported in eight studies, while several others demonstrated thinning confined to specific quadrants. Five studies reported thinning in inner retinal layers or macular thickness. Seven studies reported significant correlations between optical coherence tomography parameters and ALSFRS-R score. All longitudinal studies consistently demonstrated progressive pRNFL thinning over follow-up in ALS patients. One study reported a significant correlation between RNFL thickness and respiratory function (FVC% and FEV1%), while another found no such association.

Conclusion: OCT-derived retinal changes, particularly pRNFL thinning, are reported in ALS and show associations with functional severity, with longitudinal studies suggesting progressive retinal degeneration. Larger, standardised longitudinal studies are needed to define prognostic value and clinical utility.

Disclosure: Nothing to disclose.

EPO-0108 | Cortical motor mapping in ALS: Investigating hand motor representation across disease phenotypes

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Background and aims: Amyotrophic lateral sclerosis (ALS) is clinically heterogeneous, with phenotype-dependent variation in cortical excitability. Reduced short-interval intracortical inhibition (SICI) on transcranial magnetic stimulation (TMS) is a recognised hallmark of ALS, but its role as pathogenic driver versus compensatory plasticity remains unclear. Navigated TMS (nTMS) motor mapping quantifies cortical motor representations and may capture degeneration and plasticity. We examined whether motor map volume differs across phenotypes and relates to SICI, resting motor threshold (RMT), and lower motoneuron (LMN) involvement.

Methods: Twenty ALS patients were consecutively recruited and classified by phenotype. Bilateral nTMS motor mapping of first dorsal interosseous (FDI) was performed. Map volumes were derived by segmentation in 3D Slicer. Threshold-tracking SICI was recorded at the FDI hotspot. LMN involvement was quantified using Neurophysiological Index (NI). A 6-month follow-up is ongoing.

Results: Mean age was 65.8 (± 11.1) years; 17/20 were males. Mean disease duration was 21.2 months (± 10.8) and ALS

Functional Rating Scale-Revised (ALSFRS-R) was 41.1 (± 3.4). Onset was spinal in 13 and bulbar in 7. Phenotypes were classical ($n=9$), bulbar ($n=7$), pyramidal ($n=4$). Map volumes and SICI did not differ by onset site or phenotype. Map volume was inversely associated with RMT ($R^2=0.22$; $p=0.042$). RMT correlated positively with disease duration ($\rho=0.48$; $p=0.039$). Map volume was not associated with SICI, NI, or disease duration.

Conclusion: Cortical motor representation appears unrelated to SICI and LMN measures. The positive correlation between RMT and disease duration suggests a stage-dependent increase in motor threshold that may influence map volume. Follow-up will examine longitudinal changes.

Disclosure: Nothing to disclose.

EPO-0109 | REST inhibition as a therapeutic and prognostic target in amyotrophic lateral sclerosis: From siRNA silencing to serum biomarkers

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Background and aims: The Restrictive Element-1 Silencing Transcription factor (REST) acts as a master repressor of neuronal gene expression. Aberrant REST accumulation has been linked to several neurodegenerative conditions, but its role in amyotrophic lateral sclerosis (ALS) remains unclear.

Methods: REST expression was analyzed in the motor cortex, brainstem, and spinal cord of SOD1-G93A mice at different disease stages. To assess causality, intracerebroventricular injections of a small interfering RNA targeting REST (siREST) were performed weekly for four weeks starting at the presymptomatic stage (day 90). Behavioral, histological, and molecular analyses were conducted to evaluate motor performance, neuronal survival, SOD1 aggregation, and glial activation. In parallel, REST serum levels were quantified by ELISA in ALS patients and healthy controls, and correlated with clinical outcomes.

Results: REST levels were markedly increased in the CNS of SOD1-G93A mice during both early and late symptomatic phases. siREST treatment significantly reduced REST expression, prevented motor neuron loss, decreased misfolded SOD1 aggregates, and attenuated astro- and microgliosis. These effects translated into improved motor performance and prolonged survival (133.2 ± 1.7 vs 124.3 ± 2.1 days, $p < 0.01$). In the clinical cohort, serum REST was significantly higher in ALS patients than in controls ($p = 0.006$). Elevated REST levels independently predicted poorer tracheostomy-free survival (multivariate HR = 13.43, 95% CI: 2.1–86.0, $p = 0.006$).

Conclusion: REST upregulation contributes to ALS pathogenesis by promoting neuronal death and neuroinflammation. Its silencing through siRNA confers neuroprotection and survival

benefit in ALS mice. Elevated serum REST mirrors CNS pathology and predicts disease progression, supporting REST as a novel therapeutic and prognostic target in ALS.

Disclosure: Nothing to disclose.

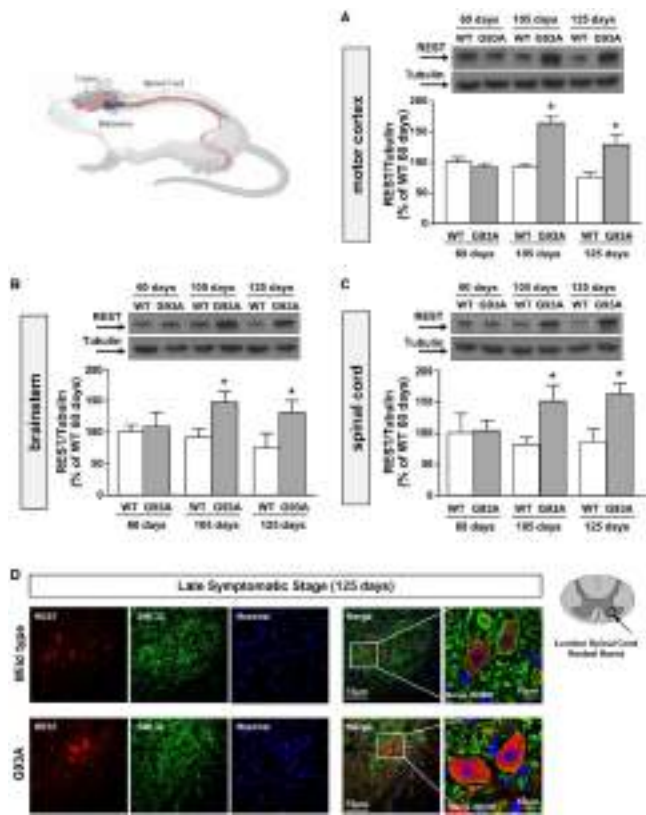


FIGURE 1 Characterization of REST protein expression in motor cortex, brainstem, and spinal cord of G93A mice, compared with WT mice, at early symptomatic phase or late phase of the disease.

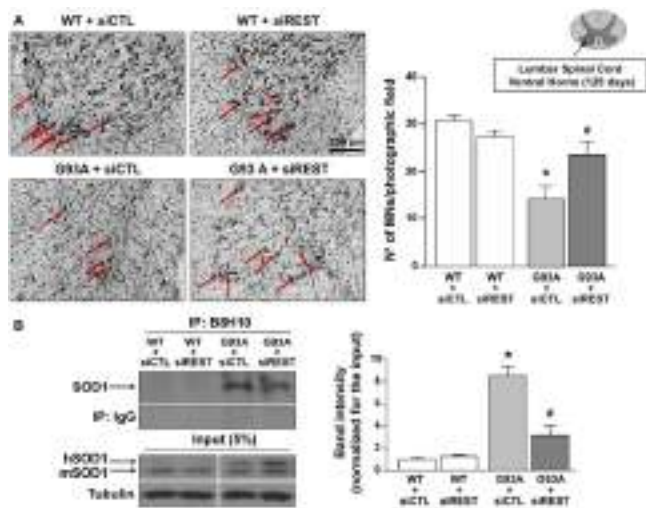


FIGURE 2 Effect of siRNA against REST on MN survival and formation of misfolded SOD1 aggregates in fully symptomatic G93A mice compared with age-matched WT mice.

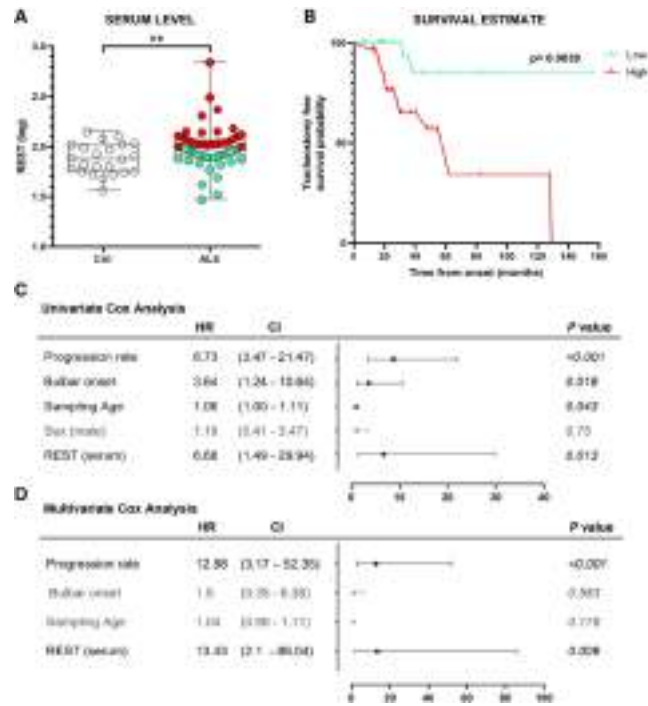


FIGURE 3 Serum levels and prognostic value of REST protein in ALS patients.

EPO-0110 | Fractional anisotropy of the corticospinal tract as a predictor of survival in amyotrophic lateral sclerosis: A DTI study

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Background and aims: Diffusion tensor imaging (DTI) metrics of key white matter (WM) tracts in amyotrophic lateral sclerosis (ALS) are crucial for assessing upper motor neuron involvement and predicting disease progression and survival. This study investigated whether major WM tracts predict survival in ALS and explored differences between spinal- and bulbar-onset cases.

Methods: A total of 411 ALS were recruited at the Turin ALS Centre (2008–2021) and underwent 1.5 Tesla brain MRI with DTI. Fractional anisotropy (FA) was extracted from the bilateral corticospinal tracts (CST; averaged) and the corpus callosum

(CC). Survival was censored in January 2024. Demographic and clinical differences were assessed. Cox proportional hazards models evaluated the association between FA and survival, adjusting for age at MRI, sex, and disease duration, in the full cohort and stratified by onset site.

Results: In the overall cohort, higher CST FA was significantly associated with longer survival (HR=0.0003, 95% CI: 6.20×10^{-6} – 0.0158, $p < 0.001$). When stratified by onset site, CST FA remained a strong predictor of survival in spinal-onset ALS (HR = 5.82×10^{-5} , 95% CI: 4.66×10^{-7} – 0.0073, $p < 0.001$). In bulbar-onset ALS, CST FA showed a trend toward predicting survival but did not reach statistical significance (HR=0.0008, 95% CI: 4.15×10^{-7} – 1.46, $p = 0.063$). FA of the CC was not significantly associated with survival.

Conclusion: CST integrity is a strong predictor of survival in ALS, particularly in spinal-onset disease, supporting CST FA as a prognostic imaging biomarker.

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EPO-0111 | Development and validation of a classification method and personalised prediction models for patients with amyotrophic lateral sclerosis

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Background and aims: The heterogeneity of amyotrophic lateral sclerosis (ALS) in clinical presentations and prognosis poses a significant obstacle to the successful translation of research into therapeutic solutions. A classification method with high stability, excellent clinical applicability and strong generalizability across populations remains to be further investigated. No parametric prognostic model has yet been established for Chinese patients with ALS.

Methods: This retrospective cohort study involved two large cohorts: the Peking University Third Hospital (PUTH) ALS cohort and a Caucasian ALS cohort derived from Pooled Resource Open-Access ALS Clinical Trial (PRO-ACT) database and the Answer ALS database.

Results: 603 patients from PUTH ALS cohort and 3610 patients from Caucasian ALS cohort were enrolled in this study. Statistical analysis suggested that patients could be stratified into three progression groups--high-risk(HR), moderate-risk(MR), and low-risk (LR)--based on diagnostic delay, progression rate, and FVC, namely the DRF classification method. More than 83% of patients remained in the same group 1 year after the initial assessment. The median survival time of PUTH ALS patients with HR progression is 6 months shorter than that of Caucasian ALS patients. PUTH ALS patients with HR progression are more likely to experience group transitions during follow-up compared to Caucasian ALS patients. Our survival models enable the estimation of the survival probability for individual ALS patients.

Conclusion: DRF classification method is an ideal choice for stratification in future multinational clinical trials. The prediction of individual disease progression and prognosis is essential not only for personalized treatment strategies but also for the evaluation of clinical trials outcomes.

Disclosure: Nothing to disclose.

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Background and aims: Disease-modifying therapies (DMTs) have modified the clinical course of spinal muscular atrophy (SMA), improving survival and motor outcomes. Nonetheless, scoliosis remains frequently observed.

Methods: Nine SMA patients receiving DMTs (3 SMA1, 6 SMA3; mean age 17.6 ± 13.1 years) were enrolled at baseline (T0). Scoliosis was assessed using Cobb angle. Clinical history and evaluations were collected including CHOP -INTEND, HFMSE, RULM, MFM-32, forced vital capacity (FVC), SMA Independence Scale-Clinical Report (SMAIS-CR) and SF-36 questionnaire. Associations between Cobb angle and clinical measures were explored using Spearman's rank correlation at baseline and after 1 year follow-up (T1).

Results: At baseline, scoliosis was observed in 78% of patients ($n=7$). SMA 1 patients presented more severe scoliosis (mean Cobb angle at T0: $45.0^\circ \pm 26.0^\circ$, at T1: $48.8^\circ \pm 47.7^\circ$), with one patient undergoing stabilization intervention. SMA 3 patients showed milder curves (mean Cobb angle at T0: $18.5^\circ \pm 4.0^\circ$, at T1: $19.3^\circ \pm 5.1^\circ$). Associations with Cobb angle were found at

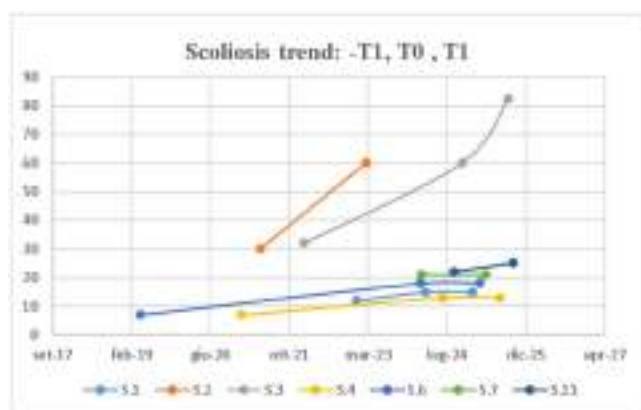


FIGURE 1 Scoliosis progression graph. On the X-axis, it is shown the time-line; on the Y-axis, Cobb angles. For patient S.1, S.3, S.4 and S.6, there are three time-points available (restrospective, baseline and at 1-year follow-up).

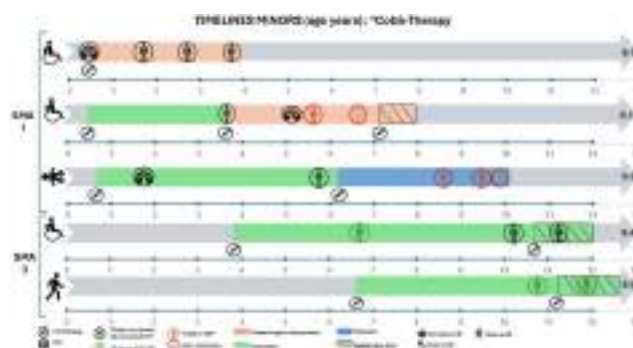


FIGURE 2 Timeline illustrating the clinical history of individual pediatric patients by age (years), including baseline motor function (T0), phenotype, scoliosis, disease-modifying therapies (DMTs), clinical stabilization, and respiratory support (NIV).

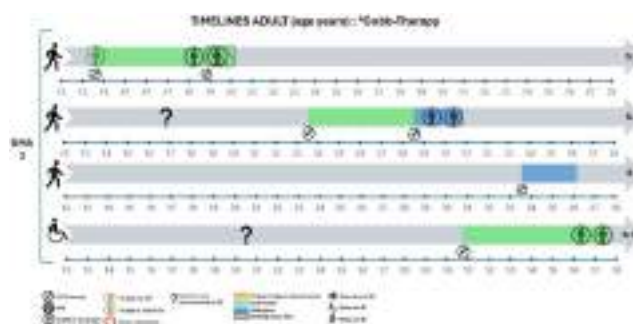


FIGURE 3 Timeline illustrating the clinical history of individual adult patients by age (years), including baseline motor function (T0), phenotype, scoliosis, disease-modifying therapies (DMTs), clinical stabilization, and respiratory support (NIV).

T0 with RULM ($r = -0.832$, $p=0.040$), at T1 with FVC in liters ($r=0.900$, $p=0.037$) and SMAIS-CR ($r = -0.900$, $p=0.037$). These associations should be interpreted cautiously, as increases in absolute FVC may reflect growth or treatment-related effects rather than respiratory function changes per se.

Conclusion: In this small exploratory-cohort, scoliosis was frequent in SMA patients receiving DMTs and appeared to co-occur with motor performance and daily functioning. Larger longitudinal studies are needed to better define scoliosis trajectories and clinical relevance in the DMTs-era.

Disclosure: Nothing to disclose.

EPO-0113 | Multimodal biomarkers for early diagnosis of amyotrophic lateral sclerosis: Integration of transcranial magnetic stimulation and radiomics MRI

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Background and aims: Early diagnosis of amyotrophic lateral sclerosis (ALS) remains challenging due to the limited sensitivity of conventional upper motor neuron markers. Integration of

multimodal biomarkers combining neurophysiological and neuroimaging approaches may improve diagnostic accuracy, particularly in early disease stages.

Methods: In this prospective study, 74 patients with amyotrophic lateral sclerosis and 38 healthy controls were included. ALS patients were stratified into mild and moderate-advanced stages according to ALSFRS-R scores. Bilateral transcranial magnetic stimulation (TMS) was applied to the abductor pollicis brevis and first dorsal interosseous muscles to assess resting motor threshold (RMT), cortical and radicular latencies, central motor conduction time (CMCT), and short-interval intracortical inhibition (SICI). Radiomics features ($n = 555$), including texture and shape parameters, were extracted from the perirolandic cortex, posterior limb of the internal capsule, and bulbus on MRI. Feature selection was performed using the Boruta algorithm, followed by LASSO and Random Forest analyses.

Results: ALS patients demonstrated significant alterations in TMS parameters, including reduced SICI, altered RMT, and prolonged CMCT. Among neurophysiological measures, SICI at 3 ms ISI showed the highest diagnostic performance ($AUC = 0.846$). Radiomics analysis revealed increased entropy and reduced homogeneity in motor-related regions. Multimodal models integrating TMS and radiomics MRI consistently outperformed single-modality approaches and achieved excellent discrimination of early-stage ALS from healthy controls ($AUC = 0.984$).

Conclusion: Multimodal integration of transcranial magnetic stimulation and radiomics MRI improves discrimination of early-stage ALS and supports the potential value of combined functional and structural biomarkers.

Disclosure: Nothing to disclose.

EPO-0114 | **Reduced nuclear TDP-43 and cytoplasmic DLK1 as markers of motor neuron degeneration in amyotrophic lateral sclerosis**

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Background and aims: Loss of upper and lower motor neurons (MNs) is a defining pathological feature underlying the clinical manifestations of amyotrophic lateral sclerosis (ALS). However, the differences in MN loss and TDP-43 pathology between these areas in ALS patients remain unknown.

Methods: This study included seven patients with ALS and three controls from autopsies performed. The cell density and regional density of TDP-43-positive inclusions in four upper MN areas (tongue, arm, trunk, and leg) and their anatomically corresponding lower MN areas were measured. The cell residual rate by comparing the cell density of each case to the average density of the controls was calculated. The numbers of large cells with loss of nuclear TDP-43 and cytoplasmic delta-like 1 homolog (DLK1), a MN-related factor involved in its differentiation, were counted.

Results: The results showed severe MN loss in both upper and lower MN areas; however, TDP-43-positive inclusions differed markedly: they were rare in upper MNs but abundant in lower MN. Although in upper MN areas, TDP-43 density was not

associated with the residual rate of MNs, the density in MNs in lower MN areas was well associated with the cell residual rate. Significantly higher numbers of MNs lacking nuclear TDP-43 and cytoplasmic DLK1 were observed in the upper and lower MN regions in ALS.

Conclusion: These results demonstrated that the frequency and severity of abnormal cytoplasmic TDP-43 aggregation differed significantly between the upper and lower MN regions in ALS patients, while nuclear TDP-43 loss and cytoplasmic DLK1 reduction were consistent in both regions.

Disclosure: Nothing to disclose.

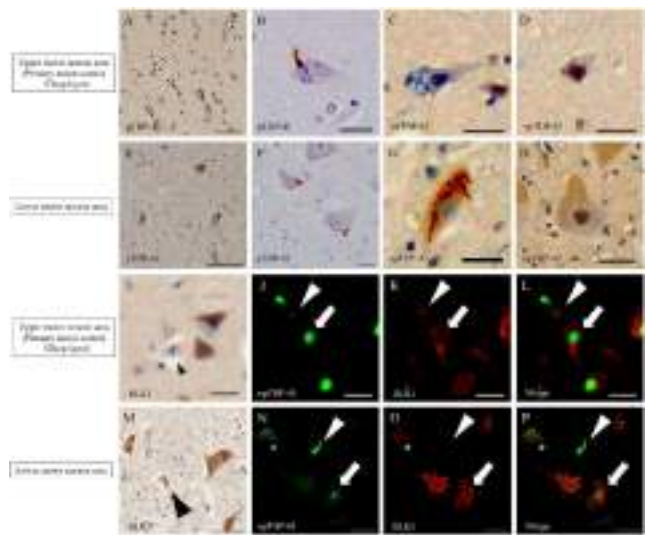


FIGURE 1 Microscopy of TDP-43 and DLK1 immunostaining in upper and lower motor neuron areas in samples from patients with amyotrophic lateral sclerosis.

TABLE 1 Case profile.

Case	Age	Sex	Duration (years)	Onset	Progression	Cause of death	Post-mortem interval (days)	Upper motor neuron				Lower motor neuron			
								Location	Number	Percentage	Residual rate	Location	Number	Percentage	Residual rate
1	65	M	5	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
2	68	M	3	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
3	70	M	2	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
4	72	M	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
5	75	M	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
6	78	M	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
7	80	M	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
8	60	F	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
9	62	F	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
10	64	F	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%

TABLE 2 Prevalence of nuclear/cytoplasmic non-phosphorylated TDP-43 and cytoplasmic DLK1 in motor neurons.

Case	Age	Sex	Duration (years)	Onset	Progression	Cause of death	Post-mortem interval (days)	Upper motor neuron				Lower motor neuron			
								Location	Number	Percentage	Residual rate	Location	Number	Percentage	Residual rate
1	65	M	5	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
2	68	M	3	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
3	70	M	2	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
4	72	M	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
5	75	M	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
6	78	M	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
7	80	M	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
8	60	F	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
9	62	F	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%
10	64	F	1	Hand	Progressive	Respiratory failure	14	Posterior horn	10	100%	100%	Posterior horn	10	100%	100%

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Background and aims: Bulbar dysfunction strongly impacts prognosis in amyotrophic lateral sclerosis (ALS), but early detection remains challenging. Motor evoked potentials (MEPs) are widely used to assess corticospinal tract integrity, yet the clinical value of cranial MEPs is unclear. This study evaluated the diagnostic and prognostic significance of cranial MEPs in a real-world ALS cohort.

Methods: We retrospectively analysed 510 consecutive ALS patients assessed at diagnosis with transcranial magnetic stimulation of three districts: cranial (genioglossus, $n=175$), upper limb (first dorsal interosseous, $n=510$), and lower limb (tibialis anterior or abductor hallucis, $n=479$). Clinical involvement was classified using King's staging and clinical phenotypes. We assessed associations between MEP abnormalities and clinical variables and the impact of cranial MEPs on time to PEG placement.

Results: Cranial MEPs were more frequently abnormal than upper limb (51.4% vs 36.1%, $p<0.001$) and similar to lower limb. They were often abnormal in patients without bulbar symptoms (27.3%) and strongly associated with bulbar involvement ($p<0.001$). Including cranial districts increases the detection of pathological MEPs by 16% across ALS phenotypes, with rates rising along the LMN-UMN spectrum and reaching 100% in UMN-predominant ALS and PLS. The frequency of abnormal cranial MEPs increased across King's stages (34% at stage 1, 47% at stage 2, 64% at stage 3, $p=0.004$). Abnormal cranial MEPs predicted shorter time to PEG (median 10 vs 18 months; $p=0.038$).

Conclusion: Cranial MEPs detect subclinical bulbar involvement and predict earlier bulbar deterioration in ALS. Their integration into routine assessment may improve early stratification and prognostic evaluation in clinical practice.

Disclosure: Nothing to disclose.

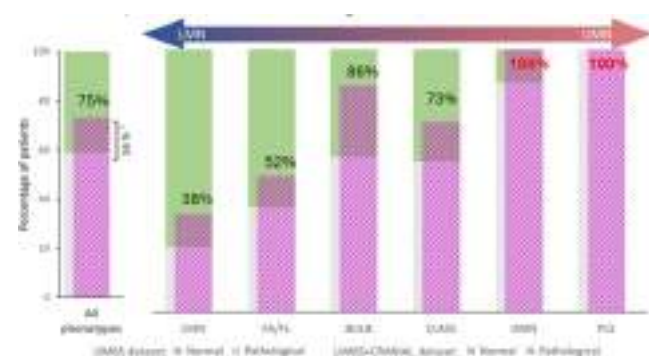


FIGURE 1 Cranial MEPs improve diagnostic sensitivity in ALS and across the phenotypic spectrum.



FIGURE 2 Stage-dependent increase of cranial MEPs involvement: In early disease stages, pathological MEPs predominantly involve the lumbosacral district, whereas cranial involvement increases across King's stages and becomes predominant at stage 3.

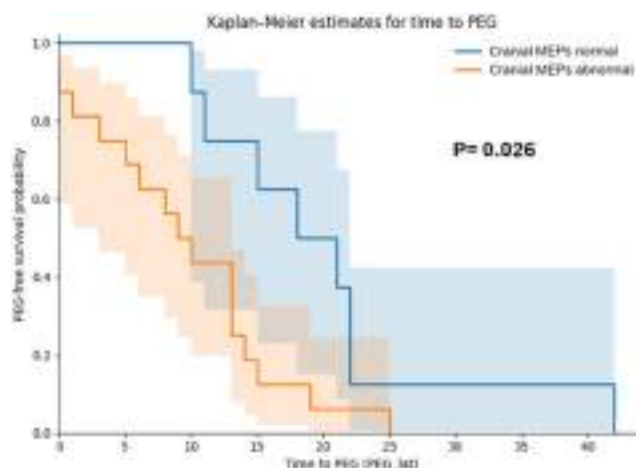


FIGURE 3 Kaplan–Meier analysis showed that patients with abnormal cranial MEPs had a significantly shorter PEG-free survival compared with those with normal cranial MEPs (median 10 vs 18 months; log-rank $p=0.026$; Cox proportional hazards HR=0.37, $p=0.038$).

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Background and aims: Amyotrophic lateral sclerosis (ALS) may be associated with selective vulnerability of specific muscle groups, described as split phenomena. This study aimed to systematically evaluate the split elbow (SE) phenomenon—defined as disproportionate involvement of biceps versus triceps muscles—as a potential clinical and neurophysiological characteristic of ALS.

Methods: This systematic review and meta-analysis followed PRISMA guidelines and was registered in PROSPERO (CRD42024528359). MEDLINE, Scopus, Web of Science, and grey literature were searched up to April 2025 using the terms “split elbow” and “amyotrophic lateral sclerosis.” Eligible English-language studies included randomized, observational, diagnostic accuracy, and case–control designs. Study quality was assessed using the Joanna Briggs Institute critical appraisal tools. Random-effects models were used to pool standardized mean differences of normalized Medical Research Council (MRC) strength scores. The diagnostic performance of the split elbow index based on compound muscle action potentials (SEI-CMAP) was analyzed using a bivariate random-effects model.

Results: Seven studies involving 1,941 patients with ALS (61.8% male) were included. There was no significant difference in strength between elbow extensors and flexors (pooled standardized mean difference [triceps–biceps] -0.17 ; 95% CI -1.03 to 0.69 ; $p=0.63$), with substantial heterogeneity across studies

($I^2=97.1\%$). The SEI-CMAP showed moderate accuracy in distinguishing ALS from controls, with pooled sensitivity of 0.79 , specificity of 0.58 , and an area under the curve of 0.66 .

Conclusion: Current evidence does not support a consistent split elbow pattern in ALS. High methodological heterogeneity and limited sample sizes reduce the clinical relevance of this phenomenon, suggesting limited diagnostic utility in routine practice.

Disclosure: Nothing to disclose.

EPO-0117 | Diagnostic ultrasound pattern of fasciculations in amyotrophic lateral sclerosis

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Background and aims: Muscle ultrasound (MUS) is an effective technique for non invasive fasciculation detection. This study aimed to characterize the fasciculation distribution pattern in amyotrophic lateral sclerosis (ALS) compared to ALS mimicking disorders.

Methods: This prospective study included 182 patients with suspected ALS: 149 ALS patients (median age 57.0 years, ALSFRS R 42) and 33 served as controls (median age 49.0 years), spondylotic myelopathy ($n=17$), radiculopathy ($n=8$), spinal muscular atrophy ($n=5$) and multifocal motor neuropathy ($n=3$). All participants underwent standardized symmetric MUS of 14 muscle groups across bulbar, cervical, thoracic and lumbosacral levels.

Results: Fasciculations were significantly more frequent in the ALS group ($p<0.001$) and were generalized and symmetric with no significant side-to-side differences ($p>0.05$). Detection rates ranged from 59.7% to 94.0%, with the highest frequencies in proximal muscles: m. biceps brachii (91.9%), posterior forearm muscle group (89.0%) and m. quadriceps femoris (92.6%). Controls showed less frequent, distally predominant fasciculations (thenar muscles 30.3–39.4%; posterior calf muscles 45.5–48.5%). The risk of fasciculation was markedly elevated in ALS: proximal muscles had high odds ratios (OR) without side to side differences (m. deltoideus OR 78–100, m. biceps brachii OR 64–89, posterior forearm group OR 113–114, m. quadriceps femoris OR 70–71), while distal muscles had lower ORs (e.g. thenar OR 11–13). A strong association (Cramer's $V=0.60$ – 0.79) linked ALS with proximal fasciculations.

Conclusion: MUS identifies a distinct proximal predominant fasciculation pattern in ALS that robustly differs from mimicking disorders, supporting its value as an objective screening tool for early differential diagnosis.

Disclosure: Nothing to disclose.

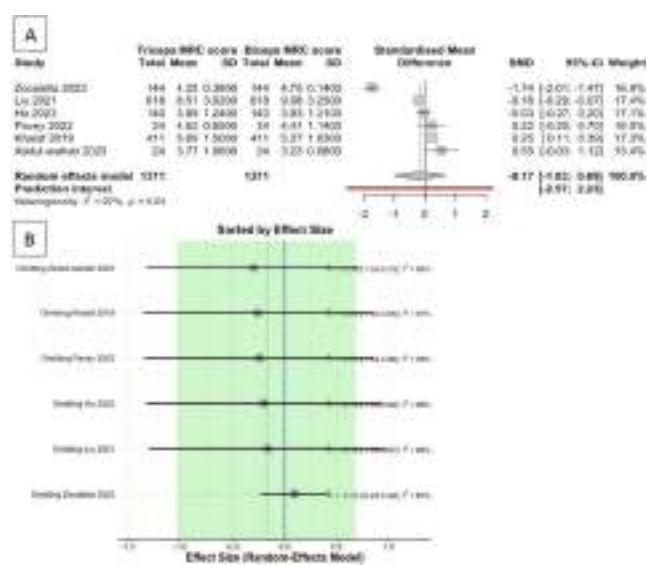


FIGURE 1 (A) Forest plot of the meta-analysis on the standardized mean difference (MRC triceps scores – MRC biceps scores) in the eligible studies. (B) Sensitivity analysis using the leave-one-out method.

Diagnostic accuracy of split elbow index (using CMAP)

Study	TP	TN	FP	FN	Sensitivity	Specificity	AUC
Forrest et al. 2022	14	14	1	0	1.00	1.00	0.99
Waller et al. 2022	14	14	1	0	1.00	1.00	0.99
Li et al. 2021	14	14	1	0	1.00	1.00	0.99
He et al. 2021	14	14	1	0	1.00	1.00	0.99
Porey et al. 2022	14	14	1	0	1.00	1.00	0.99
Kivari et al. 2019	14	14	1	0	1.00	1.00	0.99
Akil et al. 2020	14	14	1	0	1.00	1.00	0.99

FIGURE 2 Diagnostic accuracy of split elbow index (using CMAP).

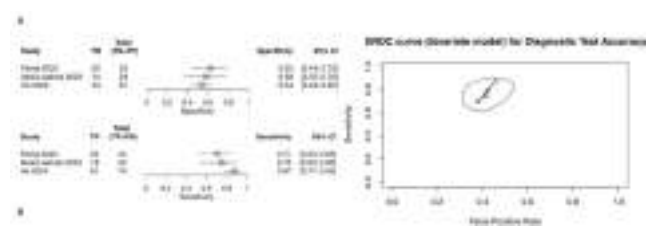


FIGURE 3 Diagnostic accuracy of the split elbow index derived from compound muscle action potentials (SEI-CMAP) for distinguishing amyotrophic lateral sclerosis (ALS) from controls. Forest plots show study-level sensitivity and specificity.

EPO-0118 | Platform-based balance assessment in Parkinson's disease: A summary of tasks and parameters: A systematic review

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Background and aims: Postural Instability (PI) is one of the cardinal motor symptoms of Parkinson's Disease (PD). Force platforms (FP) can be used as an objective balance assessment tool during both the early and advanced phases of PD. This systematic review aims to identify FP-performed tests for PD balance assessment and summarize the most commonly calculated FP parameters.

Methods: We conducted a systematic review of studies published on Scopus, PubMed, and Embase from inception until September 2025. We screened 3204 articles using Rayyan, out of which 164 matched the inclusion criteria of performing FP-based balance assessment of an idiopathic PD (iPD) group. Following eligibility assessment, 113 eligible studies were included. Study quality was assessed using JBI Critical Appraisal Tools.

Results: A total of 3121 iPD patients were assessed. Static balance was mainly assessed by instructing the patients to maintain a feet apart position under the eyes open (EO) or eyes closed (EC) conditions, while standing on the FP or a compliant surface [Figure 1]. Dynamic balance of PD patients was assessed by a fewer number of studies [Figure 2]. The most frequently calculated FP-derived parameters were the Centre of Pressure (CoP) displacement, velocity, path length and the area encompassing all (total area) or 95% of all CoP points (ellipse) [Figure 3].

Conclusion: Force platforms can prove to be a valuable asset, not only for researchers, but also for clinicians. Further research is required to explore the use of FP for detecting subclinical PI in early PD, monitoring disease progression, and assessing the effectiveness of balance rehabilitation.

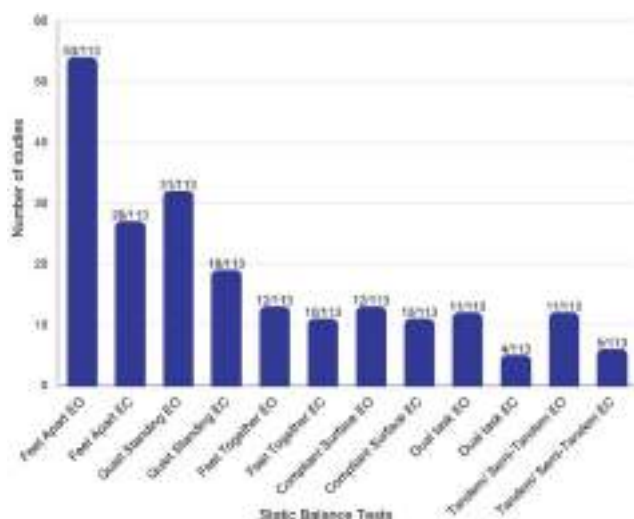


FIGURE 1 Static balance tests.

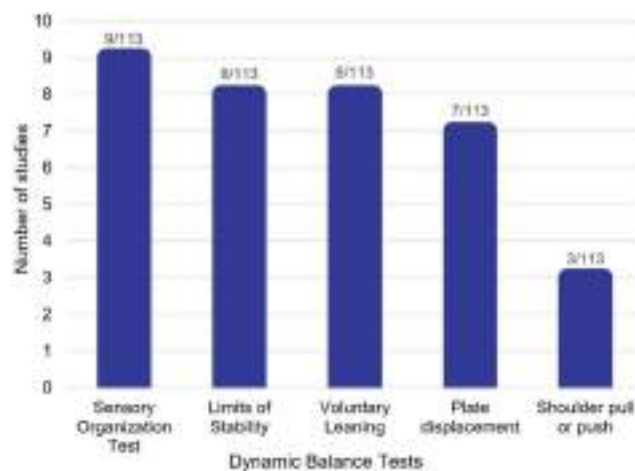


FIGURE 2 Dynamic balance tests.

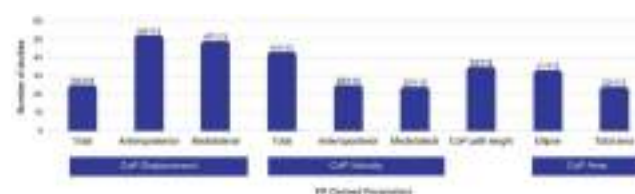


FIGURE 3 FP-derived parameters.

Disclosure: Nothing to disclose.

EPO-0119 | Efficacy of Opicapone in early motor fluctuations in Parkinson's disease: A comparison of early versus later initiation

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Background and aims: Evidence suggests that opicapone use in Parkinson's disease patients with recent-onset wearing-off yields a higher magnitude of effect than that observed in later stages. However, the long-term impact of earlier initiation is not well-studied. This post-hoc analysis assessed long-term outcomes of opicapone early initiation in levodopa-treated Parkinson's patients with early motor fluctuations.

Methods: Data were pooled from randomized, 3.5-month, double-blind, placebo-controlled opicapone trials and their

a) **Body weight and food intake.** The line graph shows body weight (g) on the left y-axis (0-250) and food intake (g chow) on the right y-axis (0-100) over 28 days. Flutamide treatment (25 and 50 mg/kg/day) significantly reduces both body weight and food intake compared to the vehicle group. The bar graph shows the change in body weight (g) and food intake (g chow) for the three groups. Significant differences are indicated by asterisks (*).

Group	Initial Weight (g)	Final Weight (g)	Change in Weight (g)	Initial Food Intake (g)	Final Food Intake (g)	Change in Food Intake (g)
Vehicle	~180	~200	~20	~40	~60	~20
25 mg/kg/day	~180	~190	~10	~30	~40	~10
50 mg/kg/day	~180	~185	~5	~20	~30	~10

b) **Energy expenditure.** The line graph shows energy expenditure (kcal/kg BW) on the left y-axis (0-10) over 28 days. Flutamide treatment (25 and 50 mg/kg/day) significantly reduces energy expenditure compared to the vehicle group. The bar graph shows the change in energy expenditure (kcal/kg BW) for the three groups. Significant differences are indicated by asterisks (*).

Group	Initial EE (kcal/kg BW)	Final EE (kcal/kg BW)	Change in EE (kcal/kg BW)
Vehicle	~4.5	~5.5	~1.0
25 mg/kg/day	~4.5	~4.5	~0.0
50 mg/kg/day	~4.5	~4.5	~0.0

Table 1. Long-term effectiveness of supragingival versus open-toothbrushing methods against <i>Candida albicans</i>	Change from 0th baseline		Change from 1th baseline	
	0th-1st	1st-2nd	0th-1st	1st-2nd
CET score (mm)				
Adjusted mean±SD (Supragingival)	-0.03±0.12	-0.03±0.12	-0.03±0.12	-0.03±0.12
Difference in Total (1st-0th) (%)	-0.20 (95% CI: -0.40)		0.00 (95% CI: 0.00)	
P-value	0.000		0.999	
COT score (mm)				
Adjusted mean±SD (Supragingival)	0.00±0.14	0.00±0.14	0.00±0.14	0.00±0.14
Difference in Total (1st-0th) (%)	0.00 (95% CI: 0.00)		0.00 (95% CI: 0.00)	
P-value	0.999		0.999	
Tooth COT score (mm)				
Adjusted mean±SD (Supragingival)	0.00±0.14	0.00±0.14	0.00±0.14	0.00±0.14
Difference in Total (1st-0th) (%)	0.00 (95% CI: 0.00)		0.00 (95% CI: 0.00)	
P-value	0.999		0.999	
COT index value (percentage of 4th degree cases)				
Adjusted mean±SD (Supragingival)	0.0±0.0	0.0±0.0	0.0±0.0	0.0±0.0
Difference in Total (1st-0th) (%)	0.0 (95% CI: 0.0)		0.0 (95% CI: 0.0)	
P-value	0.999		0.999	

Conclusion: Early opicapone in PD patients with recent-onset wearing-off provided sustained improvements of ~2.5 hours in

Disclosure: Supported by Bial.

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Methods: EPSILON was a randomized, DB, placebo-controlled study followed by a 1-year OLE. Levodopa-treated PD patients without MCs received OPC 50mg or placebo during the DB phase, after which all patients received OPC 50mg. This post-hoc analysis assessed time to MCs, dyskinesia (MDS-UPDRS-IV item 4.1), and OFFs (item 4.3) over 1.5 years in patients initiating

a Progression-free survival (months) for all patients. The y-axis ranges from 0 to 12 months. The x-axis ranges from 0 to 100 months. The 'With anti-VEGF' group (n=100) shows a median PFS of approximately 8 months, while the 'No anti-VEGF' group (n=100) shows a median PFS of approximately 6 months.

b Progression-free survival (months) for patients with MGMT promoter methylation. The y-axis ranges from 0 to 12 months. The x-axis ranges from 0 to 100 months. The 'With anti-VEGF' group (n=50) shows a median PFS of approximately 9 months, while the 'No anti-VEGF' group (n=50) shows a median PFS of approximately 7 months.

c Overall survival (months) for all patients. The y-axis ranges from 0 to 12 months. The x-axis ranges from 0 to 100 months. The 'With anti-VEGF' group (n=100) shows a median OS of approximately 10 months, while the 'No anti-VEGF' group (n=100) shows a median OS of approximately 8 months.

d Overall survival (months) for patients with MGMT promoter methylation. The y-axis ranges from 0 to 12 months. The x-axis ranges from 0 to 100 months. The 'With anti-VEGF' group (n=50) shows a median OS of approximately 11 months, while the 'No anti-VEGF' group (n=50) shows a median OS of approximately 9 months.

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OPC early (OPC-OPC) or after placebo (PLC-OPC), stratified by levodopa dose (<400 vs. ≥400 mg/day). Kaplan–Meier survival analyses with log-rank tests were used.

Results: Higher levodopa doses (≥400 mg/day) showed a trend for increased MCs risk, particularly in patients starting OPC later. Proportions of patients without MCs were 81.9% (<400 mg/day) and 79.0% (≥400 mg/day) in the OPC-OPC group, vs. 73.3% (<400 mg/day) and 66.7% (≥400 mg/day) in the PLC-OPC group ($p=0.04$). Dyskinesia followed a similar trend ($p=0.06$). Proportions of patients without OFFs were slightly higher in the OPC-OPC group but independent of levodopa dose ($p=0.4$); dystonia remained low in all groups (Figure 1a–d).

Conclusion: Most patients remained free of MCs with OPC; however, a trend toward higher MC risk was observed in patients on higher levodopa doses who initiated OPC later. Early OPC introduction may support motor stability, even at higher levodopa doses.

Disclosure: Supported by BIAL.

EPO-0121 | Beta and gamma local field potentials as biomarkers of motor and non-motor outcomes in Parkinson's disease: A systematic review

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Background and aims: Deep brain stimulation (DBS) is an established therapy for idiopathic Parkinson's disease (PD). Sensing-enabled DBS systems facilitate the recording of local field potentials (LFPs), which represent synchronised population-level neuronal activity. Beta-band activity, defined as 13 to 30 hertz, strongly correlates with motor symptoms including bradykinesia and rigidity. Gamma-band oscillations of over 30 hertz have been proposed as potential complementary biomarkers. The objective of this study is to evaluate the clinical relevance of beta- and gamma-band LFP biomarkers derived from postoperative recordings in people living with PD.

Methods: Following PRISMA 2020 guidelines, searches of PubMed, Scopus and ClinicalTrials.gov for human studies published between January 2020 and October 2025 investigating sensing-enabled DBS of the subthalamic nucleus or globus pallidus internus were performed. Studies required quantitative beta and/or gamma analyses linked to validated motor or non-motor outcomes. Twenty-seven studies met the inclusion criteria. Significant methodological heterogeneity precluded quantitative meta-analysis; therefore, the analysis will consist of a systematic narrative review mapping methodological patterns. Risk of bias was assessed using the Newcastle-Ottawa Scale and Cochrane risk of bias tools.

Results: Beta burst-derived metrics were associated with motor impairment, supporting their relevance for adaptive DBS control variables. Gamma-related findings were heterogeneous and

most commonly reported in relation to dyskinesia. Non-motor outcomes were sparsely represented.

Conclusion: This review consolidates evidence on robust beta and gamma local field potential biomarkers. Findings will define methodological priorities and inform multi-feature biomarker models for precise and objective adaptive DBS programming.

Disclosure: Nothing to disclose.

EPO-0122 | Spontaneous blink dynamics reveal distinct subtypes of isolated REM sleep behaviour disorder

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Background and aims: While distinct alterations of blinking and eyelid motor control have been described across different alpha-synucleinopathy phenotypes, eyelid motor behavior remains poorly characterized in isolated REM sleep behavior disorder (iRBD), a recognized prodrome of alpha-synucleinopathies. Given that the kinematics of blinking can index the function of cortical-basal ganglia networks, subtle alterations in blink dynamics may emerge prior to phenoconversion. Here, we investigated whether spontaneous blink subtypes can be identified in iRBD and whether their patterns differ from de novo, drug-naïve PD.

Methods: We analyzed single-camera video recordings of freely spoken monologues from 101 polysomnography-confirmed patients with iRBD and 57 patients with PD. Facial landmark tracking was used to extract nine spontaneous blink parameters, capturing: rate, amplitude, duration, opening and closing velocities, palpebral fissure width, and variability. Unsupervised k-means clustering identified iRBD blink subtypes, which we related to clinical markers and compared to the PD cohort.

Results: Two stable blink clusters were identified, comprising a hyperkinetic subtype (68%) and a hypokinetic subtype (32%) with similar demographics, sex distribution, motor severity, and cognitive performance. Blink rate did not differ between subtypes; however, palpebral fissure width, blink duration, and eyelid opening and closing velocities diverged in opposite directions. Clinically, the hypokinetic subtype was associated with higher autonomic dysfunction and stronger differentiation from the PD cohort.

Conclusion: Distinct spontaneous blink subtypes may reflect divergent underlying pathophysiology, potentially specific to synucleinopathy phenotype. Establishing discriminating blink characteristics may aid in identifying meaningful prodromal subgroups, supporting an earlier intervention time, and informing future neuroprotective or disease-modifying interventions.

Disclosure: This study received support from the Ministry of Health of the Czech Republic, grant no. NU23J-04-00042 and the Grant Agency of Charles University, project no. 604425.

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Background and aims: Continuous dopaminergic infusion therapies are used in advanced Parkinson's disease (PD) to improve motor fluctuations; however, studies in patients treated with levodopa/carbidopa intestinal gel (LCIG) have reported peripheral neuropathies, including early small-fiber involvement [1,2]. We aimed at evaluating peripheral nerve involvement during continuous subcutaneous foslevodopa infusion (SC foslevodopa) using integrated electrophysiological and morphological evaluations.

Methods: In this prospective observational study, we enrolled PD patients initiating SC foslevodopa who underwent baseline and 6-month follow-up assessments. Large-fiber function was assessed by standard motor and sensory nerve conduction studies. Small-fiber involvement was evaluated by validated questionnaires and distal leg skin biopsy with quantification of intraepidermal nerve fiber density (IENFD).

Results: 21 patients completed baseline and follow-up nerve conduction studies: peripheral neuropathy was present in eight patients, including four sensory and four sensorimotor axonal neuropathies, at baseline. No new cases or progression of neuropathy were observed at follow-up. Skin biopsy data at baseline and 6 months were available in 21 patients. Asymptomatic pathological IENFD was detected in 2 patients at baseline, remaining stable over time, while two additional patients developed pathological IENFD at follow-up.

Conclusion: In this cohort SC foslevodopa was not associated with incident or progressive large-fiber peripheral neuropathy over medium-term follow-up while skin biopsy revealed small-fiber involvement in a subset of patients. Whether this reflects dissociated fiber involvement or disease-related small-fiber denervation remains to be elucidated. Combined electrophysiological and morphological monitoring may improve the detection of early peripheral nerve involvement during infusion therapies.

Disclosure: Nothing to disclose.

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Background and aims: This study evaluated the safety and efficacy of staged-bilateral magnetic resonance-guided focused ultrasound (MRgFUS) Pallidothalamic-tractotomy (PTT) in patients with Parkinson's disease (PD) experiencing motor complications. MRgFUS is an incisionless technique that enables targeting of the Pallidothalamic-tract within the subthalamic region, offering a novel alternative for PD management.

Methods: In this prospective, open-label, single-arm, multicenter phase III trial (NCT04728295), 84 participants (≥30 years) were enrolled across nine international centers. Eligible subjects underwent first-side MRgFUS PTT followed, when appropriate, by staged-bilateral treatment after a minimum 6-month interval. The primary endpoint was change in MDS-UPDRS Part III OFF bilateral upper and lower extremities (ULE) 3 months after 2nd side treatment. Secondary measures included MDS-UPDRS Parts III total OFF and total IV scores, with safety assessed through treatment-emergent adverse events. Nonparametric tests were used for statistical analysis.

Results: Fifty-four subjects received unilateral MRgFUS PTT, and 40 underwent staged-bilateral treatment. Following second-side procedures, Part III OFF (ULE) improved by 32.1% [median 33 (IQR 28–40) to 21 (16–27); $p < 0.0001$] (Figure 1), total Part III OFF improved by 34% [median 50 (44–56) to 32 (26–40); $p < 0.0001$] (Figure 2), and Part IV improved by 67% [median 12 (9–13) to 4 (0–5); $p < 0.0001$] (Figure 3). Benefits persisted through 12 months. After unilateral treatment, 30 events occurred in 21 subjects (87% mild; non-severe), 60% resolving within 6 months; after bilateral, 43 events in 22 subjects (70% mild; 2% severe), 40% resolving within 12 months. The most frequent events were imbalance, dysarthria, and fatigue.

Conclusion: MRgFUS PTT significantly improved motor function and complications with a favorable safety profile, supporting its role as an incisionless therapy for advanced PD. The recent FDA approval enables real-world evaluation and larger confirmatory studies. These results may also support additional regulatory approvals; the full manuscript is under review.

Disclosure: PW, LZ, WCC, VB, MP and MK declare no conflicts of interest. MM has received contracts or grants from the International Parkinson and Movement Disorders Society (MDS Podcast), and Michael J Fox Foundation (Grant ID: 16451); honoraria for lectures, presentations, speaker's bureaus, or educational events from Bial, Insightec, International Parkinson and Movement Disorders Society, Sociedad Española de Neurología, and Zambon. Stock: Biogen. AD has received honoraria and travel funds from Insightec and honoraria from Acadia pharmaceuticals. HME has received grants for Insightec Haifa. HS has received clinical trial support from Bial, Biogen, Bluerock, Bukwang, Cerevance, Genentech, Insightec, MeiraGTX, Neuroderm, Novo Nordisk, Preval, Sun Pharma, and UCB. She has served as a consultant for Neuroderm and Novo Nordisk and has been a speaker for Insightec. PSF has received research funding from the FUS Foundation and Insightec. MdA is a consultant for Abbott, Boston Scientific, Insightec, and Medtronic; and has received honoraria as a speaker from Palex and reimbursement of travel expenses to attend scientific conferences from Boston Scientific, Medtronic, and Palex. LHG-Q has received honoraria from Boston Scientific, Insightec, Medtronic, Rebrain, Siemens and for lectures, travel, and accommodation to attend scientific meetings. He had also received honoraria as a consultant from Insightec and Siemens. He is a member of the medical advisory board of Rebrain. MCR-O has received financial support to attend scientific meetings, honoraria for lectures, advisory boards and research from Boston Scientific, Insightec, Medtronic and Palex. She has also received grants for research projects from different calls of the Spanish and Navarrian Government and from Siemens. SS has received research grant funding from Abbott, Boston Scientific, and Medtronic, NIH R01 grant funding and ad hoc paid proctorship from Insightec. PG has received honoraria for medical advisory boards for Petal Surgical, Profound Medical, and SonALAsense NP has received honoraria as a consultant for AbbVie Pharmaceuticals, Amneal Pharmaceuticals, and Supernus. AM has received grant support from Insightec. JO has received honoraria for lecturing in scientific meetings from Insightec Ltd. He has received consulting honoraria from Biogen for participating at Adverse Events Advisory Board 2023–2025, and consulting honoraria from Bayern and Roche for attending one Advisory Board for each. He also holds several non-paid, non-profit research grants from the Spanish Ministry of Education and Science, European Funding Agencies, Focused Ultrasound Foundation and ASAP coalition.

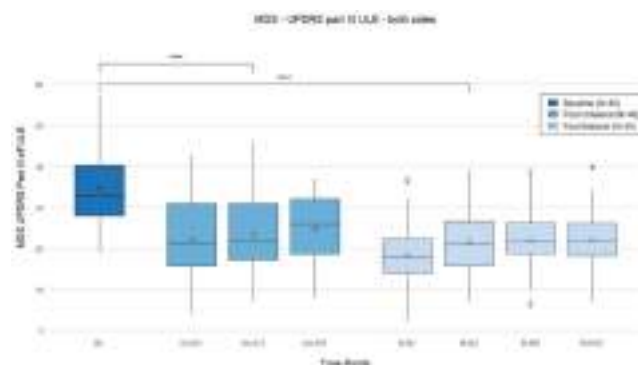


FIGURE 1 Change from baseline in the MDS-UPDRS Part III OFF ULE score [median (IQR)]. BL: baseline; Uni = unilateral; Bi = bilateral; M = month; ****= $p < 0.0001$.

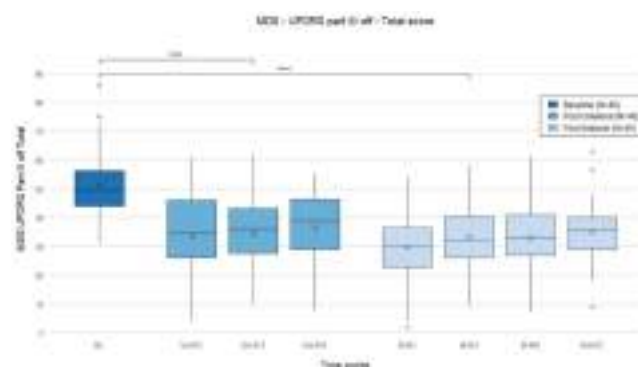


FIGURE 2 Change from baseline in the MDS-UPDRS Part III total score [median (IQR)]. BL: baseline; Uni = unilateral; Bi = bilateral; M = month; ****= $p < 0.0001$.

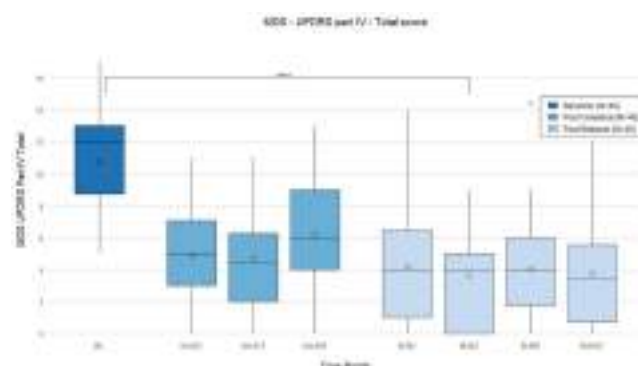


FIGURE 3 Change from baseline in the MDS-UPDRS Part IV score [median (IQR)]. BL: baseline; Uni = unilateral; Bi = bilateral; M = month; ****= $p < 0.0001$.

EPO-0125 | Mapping the availability of palliative care for parkinsonian syndromes in African countries: A scoping review

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Background and aims: Neurodegenerative disorders (NDDs) are progressive conditions leading to severe disability. They are

affecting patients and caregivers. Neuro-palliative care (NPC) aims to improve the quality of life of patients by addressing the multidimensional burdens of NDD, and reducing the burden on caregivers. Globally and especially in African countries, access to NPC in NDD is scarce both institutionally and professionally. This study aims to evaluate the availability and integration of NPC in the management of parkinsonian syndromes (PS) across African countries.

Methods: We conducted a scoping review following the Arksey and O'Malley framework, with Levac and JBI enhancements, to evaluate the availability and integration of palliative care in the management of parkinsonian syndromes across African countries. PubMed, Scopus, Web of Science, and Google Scholar were searched. Observational studies and grey literature were included. Two reviewers independently screened studies and extracted predefined data on degree of integration and reported barriers, with disagreements resolved by a third reviewer.

Results: Substantial gaps in the availability and implementation of NPC for PS across Africa were identified. NPC is rarely incorporated into NDD care pathways and remains poorly represented in national neurological policies. Services are limited, unevenly distributed, and primarily focused on oncology. Financial constraints, inadequate infrastructure, and high out-of-pocket costs further restrict access. Cultural beliefs, low awareness, and limited training in NPC significantly contribute to delayed referral and underutilization of palliative services.

Conclusion: NPC for PS in Africa remains critically insufficient. Increasing awareness and strengthening policies in sustainable palliative care models are essential to improve quality of life for affected individuals and their caregivers.

Disclosure: Nothing to disclose.

EPO-0126 | Safety and tolerability of opicapone in elderly Parkinson's disease patients: A single-center, real-life retrospective study

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Background and aims: Opicapone is a Catechol-O-Methyltransferase inhibitor, used in patients with Parkinson's Disease (PwPD) experiencing motor fluctuations¹. Registrative trials of Opicapone demonstrated its effectiveness and its favorable profile of safety and tolerability. However, these trials primarily enrolled non-elderly patients, who represent a higher percentage of PwPD^{2,3}. Our aim was to assess safety and tolerability of Opicapone in elderly patients in a real-life clinical setting.

Methods: We retrospectively collected data from PwPD treated with Opicapone at our outpatient clinic. Patients were stratified according to age at drug initiation (60–75 vs ≥75 years). Comorbidities, time between fluctuation-onset and drug start, clinical assessments, including Hoehn and Yahr (H&Y) stage, MDS-UPDRS-III, LEDD, concomitant therapy and adverse events, were evaluated at baseline (T0) and at follow-up (T1; treatment discontinuation or last available visit).

Results: No statistically significant differences were observed between the two age groups regarding disease duration, H&Y stage, or total LEDD at either baseline (T0) or follow-up (T1). Adverse events were more frequent in patients aged ≥75 years (50% vs 19%, $p=0.02$), without significant difference in discontinuation rates. Subgroup analysis revealed that patients who discontinued Opicapone had higher disease severity (H&Y-stage, $p=0.01$; UPDRS-III at T0 and T1, $p=0.02$ and 0.01 , respectively), a lower increase of LEDD at T1 ($p=0.001$) and were more frequently female ($p=0.031$). A tendency towards significance was found for differences in age at visit ($p=0.005$).

Conclusion: These preliminary findings suggest that safety and tolerability were higher in patients with less advanced age and disease severity, with possible gender-related differences. Larger, multicenter prospective studies are warranted.

Disclosure: Nothing to disclose.

EPO-0127 | Family resilience, rights utilizations, dyadic relationship and caregiver care-efficacy in families of persons with Parkinson's disease

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Background and aims: Parkinson's disease (PD) is a chronic, progressive neurodegenerative disorder that severely affects daily functioning. Globally, an estimated 12–13 million people are currently living with PD, with numbers expected to double by 2050. Consistent with Aging in Place policies, most PDs remain at home, placing increasing physical and emotional demands on family members. Little is known about the factors that help family caregivers cope effectively with these ongoing challenges.

Methods: The study included 168 family caregivers of community-dwelling individuals with PD in Israel. Data were collected online (January–June 2025), and multiple regression analyses examined associations among caregiving efficacy, family resilience, rights utilization, and dyadic relationship.

Results: Caregivers were mostly women (80%), partnered (95%), and retired (58%), with a mean age of 66.7 ± 10.9 years. Care recipients with PD were predominantly men (84%) with a mean age of 72.6 ± 9.7 years. On average, caregivers provided 9.7 ± 7.0 hours of care per day. Caregiving care-efficacy was associated with family resilience ($r=0.68$, $p<0.001$), dyadic relationship ($r=0.35$, $p<0.001$), and emotional rights utilization ($r=0.22$, $p=0.014$). Multivariate analysis showed that caregiver care-efficacy was significantly associated with family resilience ($\beta=0.569$, $p<0.001$). Emotional rights utilization significantly moderated the relationship between family resilience and care-efficacy ($B=1.99$, $p=0.009$), while dyadic relationship did not show a moderating effect. Female caregivers reported higher care-efficacy ($\beta=.154$, $p=0.044$). The overall model explained 50.9% of the variance ($R^2=0.509$, $p<0.001$).

Conclusion: Within the Aging in Place framework, enhancing family resilience, emotional rights utilization, and Caregiver care-efficacy is essential to support family members in providing effective and sustainable home care for individuals with PD.

Disclosure: This research was partially supported by a scholarship grant from the CUENCA Institute.

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Background and aims: Omaveloxolone is the first approved treatment for Friedreich Ataxia (FA), an autosomal recessive neurodegenerative disorder caused by a trinucleotide GAA expansion in FXN gene encoding frataxin. In FA, reduced frataxin levels and suppression of the Nrf2 pathway lead to oxidative stress, mitochondrial dysfunction, and cellular damage. Omaveloxolone, a synthetic oleanane triterpenoid, pharmacologically activates Nrf2 and enhances antioxidant effects (1). In a phase II trial, Omaveloxolone (150 mg/die) significantly improved neurological function compared to placebo (2). This retrospective study aimed to evaluate the efficacy and safety of Omaveloxolone in FRDA patients treated in clinical practice.

Methods: Patients underwent baseline and follow-up visits after 3 and 6 months. Neurological assessments included modified FA rating scale (mFARS) and FA activities of daily living (FA-ADL) scores. Serum laboratory analyses at baseline and months 1, 2, 3, and 6 included liver enzymes and lipid profile. Clinical scale change in time, and the interaction of factors and time were assessed with a linear mixed model for repeated measures.

Results: Fifty-seven patients (29 female, 28 male; mean age 41.3 ± 13.6 years; disease duration 22.1 ± 10.6 years) were enrolled. Baseline mFARS was 58.6 ± 16.9 , decreasing to 57.5 ± 16.7 after 3 months; 13 patients reached 6 months (59.7 ± 10.6 ; $p = 0.186$). FA-ADL slightly declined from 16.2 ± 7.4 to 15.8 ± 6.8 ($p = 0.547$). Higher baseline mFARS and FA-ADL scores predicted greater improvement ($p < 0.001$). AST/ALT increased by month 3, then decreased ($p < 0.001$). Eleven patients temporarily discontinued therapy; one permanently due to gastrointestinal side effects.

Conclusion: Omaveloxolone was safe and well tolerated, with mFARS and FA-ADL improvements more evident in patients with advanced disease and higher baseline scores.

Disclosure: Nothing to disclose.

EPO-0129 | Using of remote smartphone language screening can predict clinical markers in Huntington disease

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Background and aims: Screening symptoms of Huntington's disease (HD) remains limited to infrequent, clinic-based evaluations. We evaluated whether fully automated linguistic analysis of speech tasks recorded via smartphone can remotely capture core clinical markers of HD severity.

Methods: In this cross-sectional multicenter study across Czech and German sites, 53 participants including 30 HD (ranging from 0 to 3 according to Integrated Staging System) and 23 healthy controls completed smartphone-based speech assessment, including spontaneous monologue and fairy tale retelling for 7

consequent days. Recordings were automatically transcribed and analyzed using natural language processing to derive 3 lexical and 3 syntactic features. Predictive models for clinical outcomes based on Unified Huntington's Disease Rating Scale were built using multivariate linear regression with cross-validation.

Results: Language features predicted HD severity across multiple domains with high accuracy, explaining up to 47% of the variance in cognitive performance, 63% in motor impairment, and 59% in functional capacity. Retelling required only 3 days of recordings to reach 90% of maximum predictive accuracy, while monologue required 6 days. Compared to controls, HD participants showed reduced vocabulary range and increase phrase repetition in both tasks ($p < 0.05$), with additional monologue-specific deficits in sentence length and syntactic complexity ($p < 0.05$).

Conclusion: Fully automated analysis of smartphone-based speech assessment can remotely quantify cognitive, motor, and functional impairment in HD, offering a scalable, low-burden digital biomarker for clinical trials and decentralized monitoring.

Disclosure: This project was supported by Czech Ministry of Health, project number NW25-04-00052.

MS and Related Disorders 1

EPO-0130 | Shedding light on kappa free light chain index in multiple sclerosis: Experience from a Portuguese tertiary centre

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Background and aims: In the latest McDonald criteria update, Kappa Free Light Chain (k-FLC) index was considered a proper test for the diagnosis of multiple sclerosis (MS) and could be used instead of oligoclonal bands (OCBs) when using a cut-off point of 6.1. This study aims to describe the applications of this biomarker in MS diagnosis and to address potential correlations with disease characteristics.

Methods: We included all patients from our center who had an established diagnosis and underwent k-FLC index quantification from November 2019 to June 2025. Sensitivity and specificity of k-FLC index and OCBs were calculated. Relations with clinical, imaging and laboratory variables were addressed.

Results: Of the 500 patients eligible for analysis, 145 had MS, 10 CIS, 8 NMOSD, 7 MOGAD, 22 SNC infections and 308 other diagnoses. Using the cut-off point of 6.1, k-FLC index had higher sensitivity than OCB (86.45% vs 77.42%) but lower specificity (88.41% vs 92.46%). κ-FLC and OCBs concordance rate was 90.8%. In the univariate analysis, k-FLC index was significantly higher in patients with high MRI lesion load (Median $46.20 \pm \text{IQR } 79.08$ vs 29.82 ± 77.95 ; $p = 0.015$), with active lesions (53.50 ± 68.68 vs 31.55 ± 74.00 ; $p = 0.018$), with infratentorial lesions (44.27 ± 72.52 vs 28.62 ± 80.52 ; $p = 0.045$) and with spinal cord lesions (54.69 ± 96.65 ; $p = 0.020$ vs 32.03 ± 53.65).

Conclusion: k-FLC index may not only be used as an OCBs substitute for MS diagnosis but also as a biomarker for prognosis.
Disclosure: The authors have nothing to declare.

EPO-0131 | Dynamics of ratios T1/T2 for evaluation of multiple sclerosis (MS) activity

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Background and aims: T1/T2 ratio is a one of the markers, which is related to microstructural integrity for the cases of MS lesions and white matter in general. Lower T1/T2 ratios are named as associated with clinical disability as well as differential impact of neuroinflammation. Note, that T1 and T2 magnetic resonance images (MRI) are divided as a result there in no necessity to convert them into the similar brightness basis.

Methods: MR-data collected for 15 patients with RRMS (2023 – 2025) and 9 – with SPMS (2023 – 2025). T2FLAIR images (256×256×272) are recorded to be reference during the some consequential years as well as T1. T1 images are co-registered and re-sliced with reference (SPM12) to be located in the similar spatial basis (Figure 1). Then, T1[year 1]/T2[year 1], T1[year N]/T2[year 1] are slice-by-slice calculated to be in the same spatial basis. Consequently, temporal differences (calculated for each pair of years) are analyzed. This allows to check differences in T1/T2 ratios qualitatively and quantitatively.

Results: This approach allows to analyze both increasing of MR-density, which corresponds to growth of T1/T2 ratio (fig. 2), and decreasing T1/T2 ratio (fig. 3) Moreover, in accordance with publications, they can be qualified as signs of partial remyelination and neuroinflammation correspondingly.

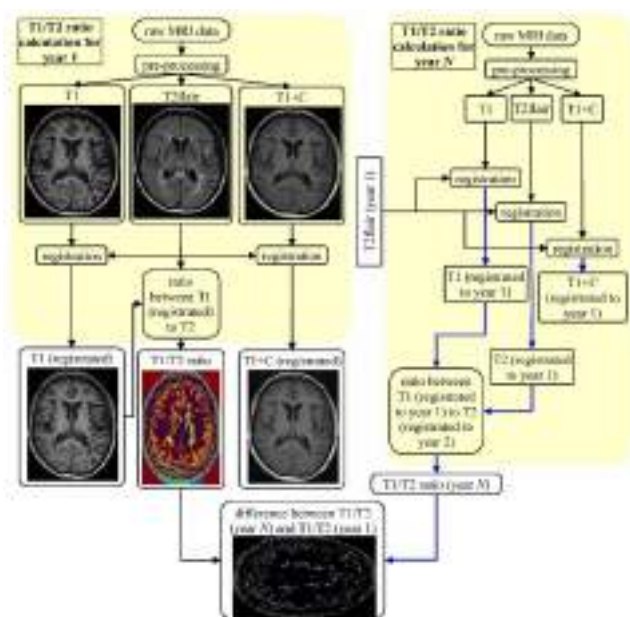


FIGURE 1 Method of T1/T2 ratio MRI calculation.

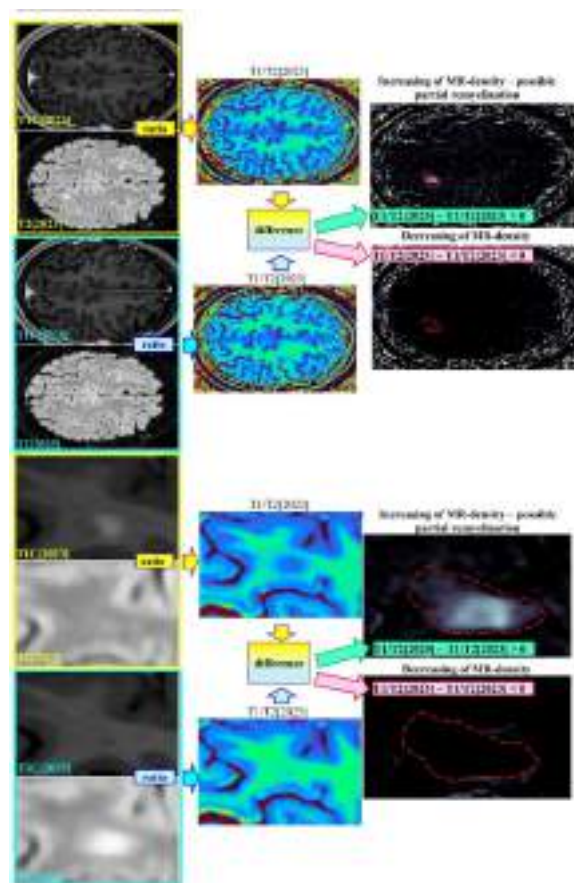


FIGURE 2 The analyze of MR-density with growth of T1/T2 ratio.

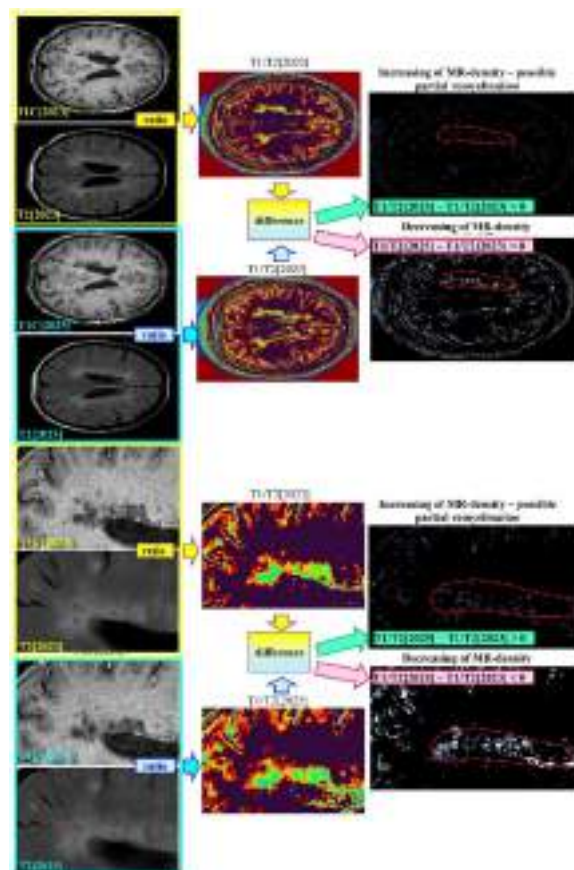


FIGURE 3 The analyze of MR-density with decreasing of T1/T2 ratio

Conclusion: Increasing of T1/T2 ratio near lesions correspond to clinically confirmed remission in RRMS, decreasing – with progression is SPMS, that correlates well with increasing of neurological symptoms.

Disclosure: Nothing to disclose.

EPO-0132 | Dynamics of statistical properties of tract density as additional confirmation of fulfillment of NEDA for MS patients

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Background and aims: The concept of NEDA (no evidence of disease activity) is widely used to describe multiple sclerosis (MS) patients under treatment. Radiological sustainability is one of the most important factors for this criterion. Due to a set of factors residual white matter microstructural changes can be missed on the magnetic resonance imaging (MRI). This research is dedicated to demonstrate that usage of statistical properties of tract density allows to confirm of fulfillment of NEDA for MS patients.

Methods: MR-data collected for 15 patients with relapsing remitting (RR) MS (2023 – 2025), MRI is performed with GE SIGNA Pioneer (3T, 25 directions of b-value 1000 s/mm²). Tractography is realized by DSI Studio with RK4 method, 10⁸ tracts are calculated. For each slice, calculation of histogram gradients and both entropy and sum of TD are performed (Figure 1)

Results: This approach allows to directly compare histogram of gradients spatial distribution and of statistical properties of TD (entropy and sum). Additionally, histogram difference can be visualized. In the case under study, RRMS with confirmed NEDA is characterized by sustainable histogram gradients (Fig. 2, 3). E.g. average entropy has changed negligibly on ~2.9% (from 0.69 to 0.71 [bit]), as well as sum – on 0.8% (from 2.29 to 2.27

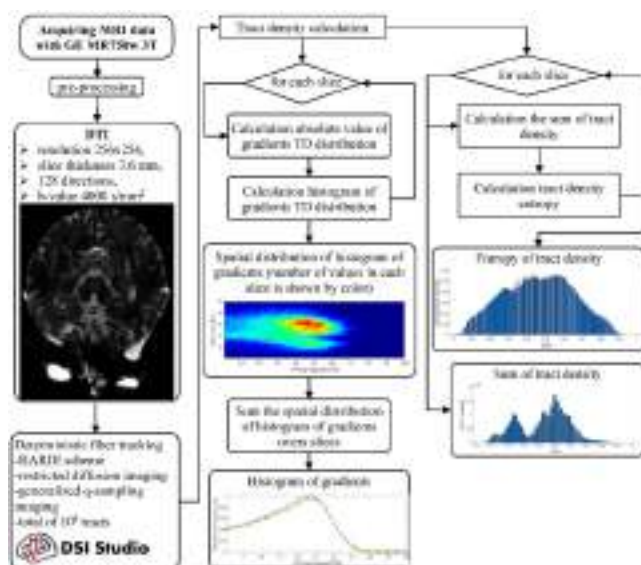


FIGURE 1 Calculation method of histogram gradients, entropy and sum of TD.

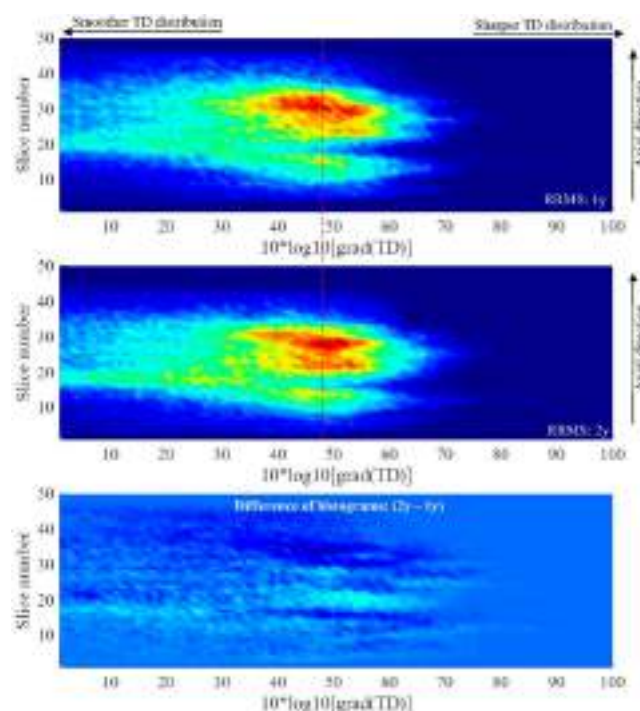


FIGURE 2 Histogram gradients in 1 and 2y.

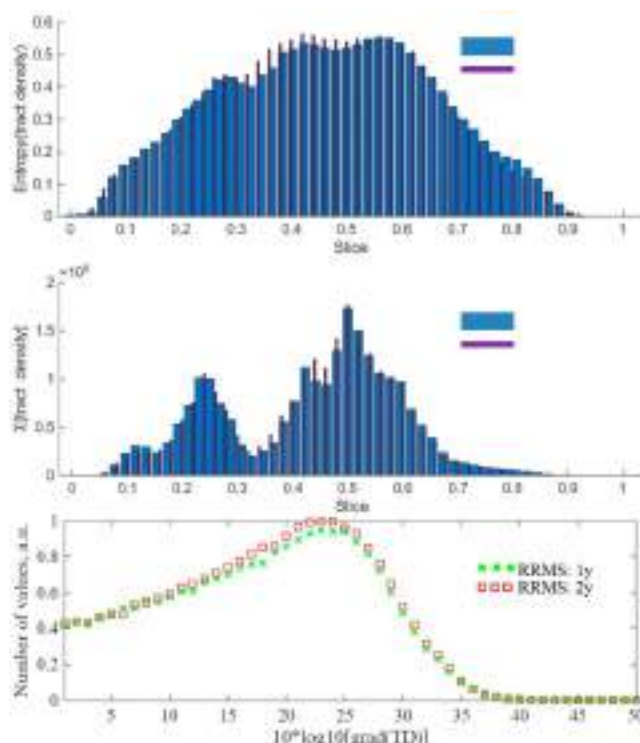


FIGURE 3 Histograms of tract density entropy

[10⁹ tracts/voxel]) (fig. 3). Clinically all patients were stable and EDSS score didn't change.

Conclusion: It is shown that RRMS with confirmed NEDA also demonstrate sustainability of histogram of gradients spatial distribution, histogram difference, average entropy and sum of TD. This can be additional factor to confirm the fulfillment of NEDA

Disclosure: Nothing to disclose.

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Background and aims: Tolebrutinib is an oral, brain-penetrant and bioactive Bruton's tyrosine kinase inhibitor that reduced the risk of disability accumulation by 31% versus placebo in non-relapsing secondary progressive multiple sclerosis (nrSPMS) and by 29% versus teriflunomide in relapsing multiple sclerosis (RMS) in phase 3 trials. This post-hoc analysis assessed the relationship between on-trial lesion formation and risk of disability accumulation in the phase 3 tolebrutinib trials in nrSPMS and RMS.

Methods: HERCULES (NCT04411641), GEMINI 1 (NCT04410978) and GEMINI 2 (NCT04410991) were phase 3, double-blind trials of tolebrutinib 60 mg once-daily taken with food. In HERCULES, participants with nrSPMS were randomised 2:1 to tolebrutinib or placebo. In GEMINI, participants with RMS were randomised 1:1 to tolebrutinib or teriflunomide (14 mg). We analysed the effect of tolebrutinib on time to onset of 6-month confirmed disability accumulation (CDA) by the presence/absence of on-trial new gadolinium-enhancing (Gd+) lesions and new/enlarging T2 lesions.

Results: In HERCULES, the formation of Gd+ or new/enlarging T2 lesions was associated with an increased risk of 6-month CDA in the placebo group, which was mitigated with tolebrutinib, particularly for participants with new/enlarging T2 lesions (15% risk reduction in those without vs 45% in those with on-trial lesions). In GEMINI, the risk of 6-month CDA was similar irrespective of lesion formation in the teriflunomide group, and all analyses favoured tolebrutinib.

Conclusion: This post-hoc analysis suggests that tolebrutinib decreases the risk of disability accumulation in participants with nrSPMS and RMS in both those who develop focal lesions while receiving treatment and those who do not.

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Siemens, TG Therapeutics); advisory committees (AB Science, Biogen, Immunic, Novartis, Sanofi); clinical trial contract and research grant funding (Biogen, Novartis, and Sanofi); HW: Honoraria (AbbVie, Alexion, Argenx, Biogen, BMS, F Hoffmann-La Roche, Janssen, Merck, Neurodiem, Novartis, Roche, Sanofi, Teva, WebMD); consulting (AbbVie, Actelion, Argenx, Biogen, BMS, EMD Serono, Fondazione Cariplo, Gossamer Bio, Idorsia, Immunic, Immunovant, Janssen, Lundbeck, Merck, NexGen, Novartis, PSI, Roche, Sanofi, Swiss Multiple Sclerosis Society, UCB, Worldwide Clinical Trials); research funding (Alexion, Amicus, Argenx, Biogen, CSL, Deutsche Forschungsgesellschaft, Deutsche Myasthenie Gesellschaft, F Hoffmann-La Roche, German Ministry for Education & Research, Merck, Novartis, Roche, Sanofi, UCB); AB-O: Grant support to the University of Pennsylvania (Biogen Idec, EMD Serono, Novartis, Roche Genentech); speaking and/or consulting (Accure, Atara Biotherapeutics, Biogen, Bristol Myers Squibb, EMD Serono, GlaxoSmithKline, Gossamer, Janssen, MedImmune, Novartis, Roche Genentech, Sanofi); PV: Honoraria or consulting (AB Science, Ad Scientiam, Biogen, Celgene-BMS, Imcyse, Merck, Novartis, Roche, Sanofi); research support (Biogen, F. Hoffmann-La Roche, Merck, Novartis, Sanofi); BACC: Consulting (Alexion, Atara, Autobahn, Avotres, Biogen, Boston Pharma, EMD Serono, Gossamer Bio, Horizon, Immunic, Neuron23, Novartis, Sandoz, Sanofi, Siemens, TG Therapeutics, Therini); research (Genentech); SS, YL, WSV, TJT, EW: Employees of Sanofi (may hold shares and/or stock options in the company); JO: Consulting and/or speaking (Amgen, Biogen, Eli Lilly and Company, EMD Serono, Novartis, Roche, Sanofi); research (Biogen, Roche).

EPO-0134 | Blood protein profiles are associated with brain atrophy in multiple sclerosis

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Background and aims: Brain atrophy is a key determinant of long-term disability in multiple sclerosis (MS). While MRI-based volumetry provides prognostic information, its limited scalability makes blood-based biomarkers an attractive tool for monitoring neurodegeneration. The MS Disease Activity (MSDA) panel profiles 18 proteins reflecting key pathways, including neuroinflammation, immunomodulation, myelin biology, and neuro-axonal integrity. In this study, we investigate whether serum MSDA scores are predictive of subsequent brain atrophy.

Methods: In this retrospective study, 140 MS patients with paired serum samples and longitudinal 3T MRI (median follow-up 2.1 years, IQR: 1.2–3.2) were analyzed. Patients were stratified into high and low annualized brain volume change groups, matched for age, sex, treatment status, and disease duration ($n=70$; 35 per group). Protein levels were measured on the Olink™ platform. Group differences were assessed using

non-parametric tests and regression models adjusted for age and sex, with Bonferroni correction for multiple testing.

Results: Patients in the high-atrophy group showed greater loss of whole brain, white matter, deep gray matter, and thalamic volumes. All proteomic pathway scores, except for neuroinflammation, were significantly elevated in the high-atrophy group compared low atrophy group. At the individual protein level, serum neurofilament light chain (NfL) was increased and CXCL9 decreased in high-atrophy group. Higher NfL levels predicted greater brain volume loss, and combining with CXCL9 levels improved distinction between high- and low-atrophy patients.

Conclusion: These results demonstrate that distinct serum proteomic profiles are associated with accelerated brain atrophy in MS and support using multi-analyte blood biomarkers alongside MRI for early risk stratification and monitoring of neurodegeneration.

Disclosure: C. Tafrali has received travel funding and speaker honoraria from Merck and Novartis. E. Hofer has nothing to disclose. M. Martinez-Serrat has received travel funding and speaker honoraria from Novartis. MT. Haindl has nothing to disclose. T. Kaiser has nothing to disclose. R. Demjaha has received travel funding from Janssen, Novartis and Sanofi. S. Hechenberger has received speaking honoraria from Roche, Bristol-Myers Squibb, and Merck. B. Helmlinger has received speaking honoraria from Roche, Sanofi, and Bristol-Myers Squibb, and travel funding from Janssen. P. Opriessnig has nothing to disclose. D. Pinter is a member of the advisory board for “Cognition and MS” for Novartis and has received speaking honoraria from Biogen, Novartis, MedAhead and Bristol-Myers Squibb. B. Heschl has nothing to disclose. A. Damulina has participated in meetings sponsored by, received speaker honoraria or travel funding from Sanofi-Aventis, Novartis and Janssen. G. Datta is an employee of Octave Bioscience D. Brazel is an employee of Octave Bioscience F. Qureshi is an employee of Octave Bioscience S. Ropele has nothing to disclose. C. Enzinger has received funding for traveling and speaker honoraria from Biogen Idec, Bayer Schering Pharma, Merck Serono, Novartis, Genzyme and Teva Pharmaceutical Industries Ltd./Sanofi-Aventis, Shire; received research support from Merck Serono, Biogen Idec, and Teva Pharmaceutical Industries Ltd./Sanofi-Aventis; and serves on scientific advisory boards for Bayer Schering Pharma, Biogen Idec, Merck Serono, Novartis, Genzyme, Roche, and Teva Pharmaceutical Industries Ltd./Sanofi-Aventis. M. Khalil has received travel funding and speaker honoraria from Bayer, Biogen, Novartis, Merck, Sanofi and Teva and serves on scientific advisory boards for Biogen, Bristol-Myers Squibb, Gilead, Merck, Novartis, Alexion, Amgen and Roche. He received research grants from Biogen, Novartis and Teva.

EPO-0135 | Comparative efficacy and safety of disease-modifying therapies in pediatric relapsing-remitting multiple sclerosis: A network meta-analysis

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Background and aims: Pediatric multiple sclerosis (MS) is a rare disease but increasingly reported, with higher inflammatory activity and relapses than adult-onset MS. Available treatments include interferon beta-1a, glatiramer acetate (GA), fingolimod, dimethyl fumarate (DMF), and teriflunomide, but comprehensive comparisons of disease-modifying therapies (DMTs) remain limited.

Methods: PubMed, Cochrane CENTRAL, Web of Science, and Scopus were searched for studies published up to December 2025. Outcomes assessed the efficacy of DMTs in pediatric MS, including annualized relapse rate (ARR), MRI lesion activity,



FIGURE 1 Graphical abstract.

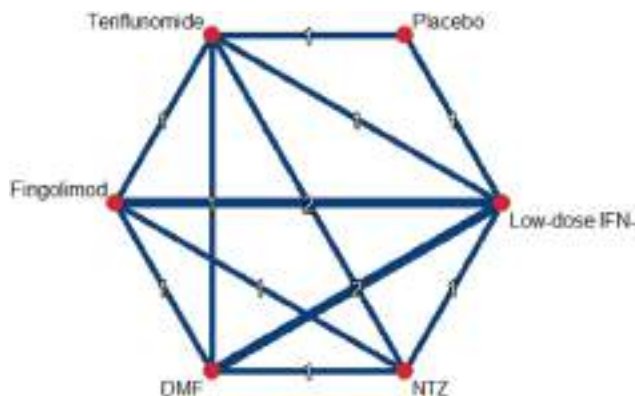


FIGURE 2 Net graph.

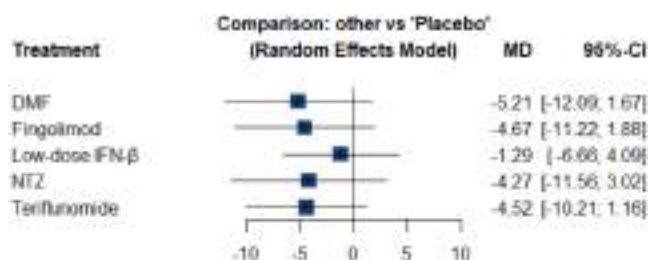


FIGURE 3 Forest plot ARR.

safety, free relapse rate, and disability progression. All Analyses were conducted using R v4.3.1.

Results: Fifteen studies, including over 11,000 pediatric and young adult MS patients, were analyzed. Rituximab produced the largest ARR reduction (-1.12), followed by GA (-1.02) and natalizumab (-1.01), while ocrelizumab, DMF, and fingolimod also showed significant ARR reductions; interferon beta and teriflunomide were non-significant. At 24 months, the absence of gadolinium-enhancing lesions was most frequent with rituximab (89%), followed by natalizumab (84%) and ocrelizumab (81%). Fingolimod showed higher relapse-free rates than low-dose interferon beta (86% vs 39%) and ocrelizumab (77% vs 52%), and lower disability progression (4.7% vs 14.8%). Teriflunomide reduced disability progression versus placebo (11.9% vs 17.5%). Serious adverse events were more frequent with fingolimod than with low-dose interferon beta (17% vs 7%) but less frequent with DMF (23% vs 29%).

Conclusion: Rituximab was the most effective therapy for reducing relapse rates in pediatric and young MS patients, with additional MRI, relapse-free, and disability benefits observed across agents and variable safety profiles.

Disclosure: The authors declare no conflicts of interest.

EPO-0136 | Reproductive-life planning, pregnancy, and peri- and post-partum management in multiple sclerosis: International expert consensus

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Background and aims: A modified Delphi consensus program was conducted to generate evidence-based, practical clinical recommendations (CRs) for healthcare professionals (HCPs) to address gaps in the literature and guidelines relating to reproductive-life planning, pregnancy, and peri- and post-partum management for people with multiple sclerosis (PwMS).

Methods: A multidisciplinary steering committee (SC) of 14 international multiple sclerosis (MS) experts identified 14 key clinical questions across five themes. A total of 14 CRs were drafted, informed by evidence from a systematic literature review and expert insights. The SC and an extended faculty comprising 23 HCPs from 12 countries voted to ascertain expert agreement (≥75% scoring 7–9).

Results: Consensus was reached on all 14 CRs after two rounds of voting, with 13 achieving 90–100% agreement. CRs are provided across five themes: 1) General reproductive-life planning – timing for discussions, key counseling points, and challenges when adjusting treatment; 2) Trying to conceive – factors influencing fertility and MS treatment considerations; 3) Fertility treatments – when to consider them and best practices; 4) Pregnancy – pre-conception and early-pregnancy care, relapse risk, and pregnancy-related complications; and 5) Peri- and post-partum – labor and delivery, relapse risk, MS treatment considerations, and breastfeeding. An overarching CR provides expert guidance on comprehensive reproductive care and effective interdisciplinary coordination.

Conclusion: These recommendations underscore the importance of patient-centered reproductive-life planning for PwMS, providing expert guidance that supports timely counseling, safe management, and informed patient-driven clinical decisions across all reproductive stages.

Disclosure: This work was supported by Merck (CrossRef Funder ID: 10.13039/100009945), who provided funding for the project.

EPO-0137 | Rethinking annual MRI monitoring in ocrelizumab-treated multiple sclerosis patients: A real-world number needed to scan analysis

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Background and aims: Ocrelizumab is a widely used high-efficacy therapy for multiple sclerosis (MS). Although current guidelines recommend annual MRI-monitoring, the low rate of radiological disease activity during long-term ocrelizumab therapy suggests that this recommendation should be re-evaluated.

Methods: We included ocrelizumab-treated MS patients, both relapsing- and primary progressive-, from the Amsterdam MS Cohort with at least two brain MRI-scans performed during ≥3 months of treatment. Radiological activity and significant radiological activity were defined as either ≥1 or ≥2 CELs on a MRI-scan performed ≥3 months after ocrelizumab initiation or any or ≥3 new/enlarging T2 lesions on a subsequent MRI-scan compared with a scan performed at least ≥3 months after ocrelizumab initiation. The number needed to scan (NNS) was calculated as the inverse of the predicted probability of disease activity.

Results: Among 1,639 MRI-scans in 342 patients, (significant) radiological disease activity was detected in 27 scans in 23 patients (1.8% of scans; 6.8% of patients) over a median treatment duration of 49 months. All three patients in whom MRI findings prompted a treatment switch had clinical symptoms preceding the MRI scan. NNS increased with treatment duration. No new pharmacovigilance findings were identified.

Conclusion: Routine annual MRI-monitoring in asymptomatic patients never led to treatments changes in clinical practice. Increasing NNS over time, especially after two years of treatment, indicates that MRI intervals can be safely extended in stable patients; potentially doubling the interval from year 2 onwards. Radiological monitoring remains essential during early treatment and in clinically symptomatic patients to identify cases requiring treatment re-evaluation.

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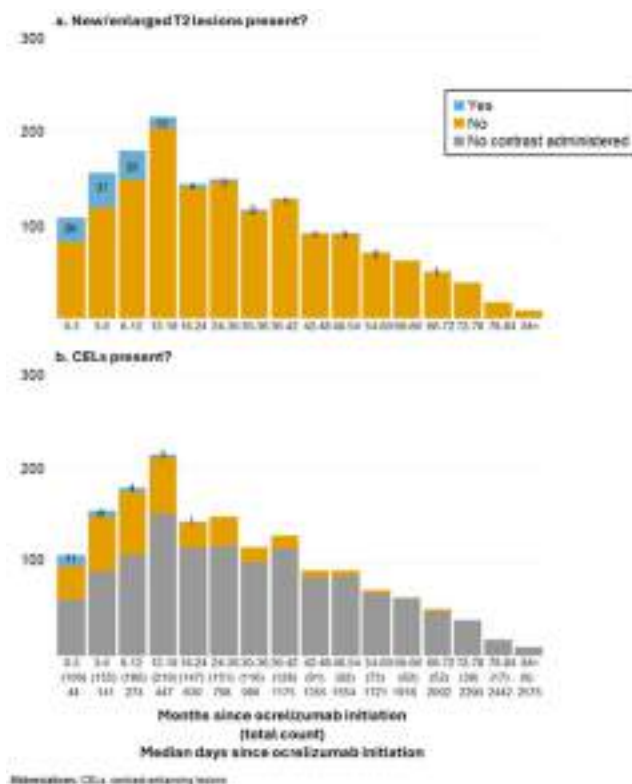


FIGURE 1 MRI scans showing new/enlarged T2 lesions or CELs during ocrelizumab treatment.

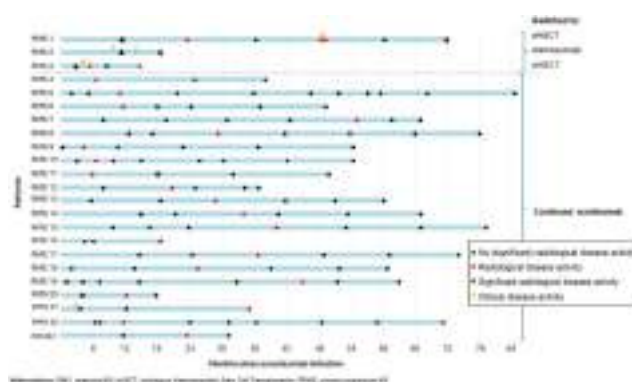


FIGURE 2 Individual patients with (significant) radiological disease activity during ocrelizumab treatment.

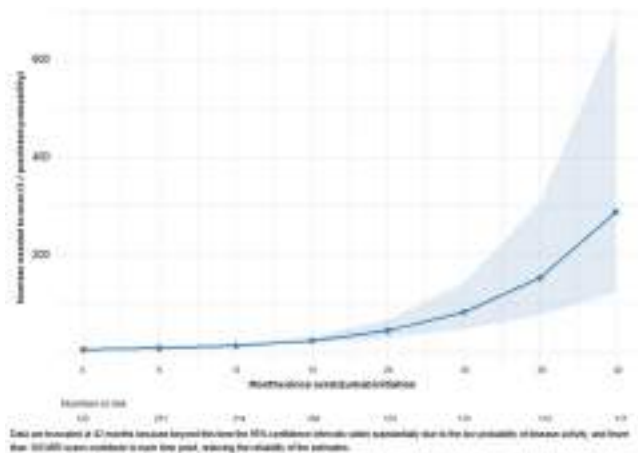


FIGURE 3 Number needed to scan per treatment duration in months.

Novartis and Sanofi/Genzyme (payments to institution); adjudication committee of MS clinical trials of Immunic (payments to institution). FB: is part of the steering committee or is a data safety monitoring board member for Biogen, Merck, Eisai, and Prothena; is an advisory board member for Combinostics and Scottish Brain Sciences; is a consultant for Roche, Celltrion, Rewind Therapeutics, Merck, and Bracco; has research agreements with ADDI, Merck, Biogen, GE Healthcare, and Roche; and is cofounder and shareholder of Queen Square Analytics LTD. ES: has received speaker fees from Merck and Novartis.

EPO-0138 | Relapse activity after ponesimod discontinuation in the OPTIMUM long-term extension study

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Background and aims: OPTIMUM was a 2-year phase 3 trial comparing ponesimod versus teriflunomide in relapsing multiple sclerosis. In the OPTIMUM Long-Term Extension (LTE), all participants received ponesimod 20 mg for up to 5 additional years. In the core study analysis there were no signals of rebound or reactivation after discontinuation. We assessed whether stopping ponesimod after longer exposure was followed by clinical reactivation.

Methods: This post-hoc analysis included participants who discontinued ponesimod in the OPTIMUM-LTE, which were followed per protocol for 30 to 44 days. Confirmed relapses were counted and annualized relapse rate (ARR) was estimated for the 6 months before discontinuation (on-treatment) and the available post-discontinuation period.

Results: Among 877 participants enrolled in the OPTIMUM long-term extension, 551 discontinued ponesimod and were included (core ponesimod: *n* = 265; core teriflunomide: *n* = 286). Median post-discontinuation follow-up was 38 days (IQR 31 – 48 days). There were 58 relapses during the on-treatment reference period versus 7 relapses post-discontinuation, corresponding to ARR 0.21 versus 0.11, respectively (RR 0.51; 95% CI [0.23 - 1.12]) (Table 1). No post-discontinuation relapse was associated with an Expanded Disability Status Scale (EDSS) increase ≥2 points (Figure 1). Subgroup analyses showed that 423 patients did not receive subsequent therapy within 30 days after discontinuation and had an ARR of 0.12 during this period, compared with 0.18 during the on-treatment period.

Conclusion: In this analysis, ARR and relapse EDSS did not suggest a clinically meaningful reactivation or rebound after ponesimod discontinuation within the available follow-up. Real-world data with longer follow-up are needed to confirm these findings.

Disclosure: L.K. declares no personal compensation. Over the past 3 years, the University Hospital Basel / Foundation Clinical Neuroimmunology and Neuroscience Basel received payments for steering committees, advisory boards, DSMB participation, consultancy, and educational activities from Autolus, Bayer, Biogen, BMS, Celltrion, Clene Nanomedicine, EMD Serono, Er-KIM, Galapagos, Genentech, Idorsia, Immunic, Janssen,

TABLE 1 Annualized Relapse Rate (ARR) estimates before and after ponesimod discontinuation among patients that permanently discontinued ponesimod at the end of study; Negative binomial model was applied; RR: Rate Ratio, CL: Confidence Level.

Period	ARR	Before discontinuation	After discontinuation
Extension Period	Mean ARR estimate	0.210	0.107
	95% CI	(0.161, 0.276)	(0.061, 0.226)
	Treatment Effect (RR)	0.508	
	95% CI	(0.238, 1.123)	
	Number of subjects	551	551
	Number of relapses	58	7
	Total time (years)	276	66

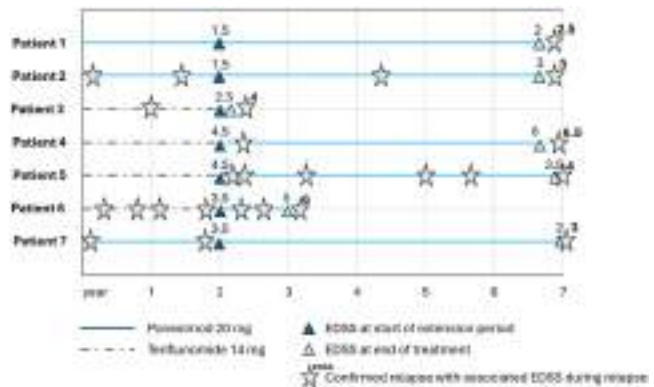


FIGURE 1 Representative visualization of the evolution of EDSS and relapses of the 7 patients which presented relapses after discontinuation of ponesimod. EDSS: Expanded Disability Status Scale

Laboratoires Juvise, Kiniksa, Merck Healthcare, MSD, Minoryx, Neurostatus UHB, Novartis, Roche, Sanofi, Shionogi, Wellmera and Zai Lab, as well as research support from Innosuisse, Novartis and Roche. X.M. has received speaker honoraria and travel expenses, and has served on steering committees or advisory boards for Abbvie, Actelion, Alexion, Biogen, BMS/Celgene, EMD Serono, Genzyme, Roche, Immunic, Janssen, Medday, Merck, Mylan, Nervgen, Novartis, Sandoz, Sanofi-Genzyme, Teva, TG Therapeutics, Excemed, MSIF and NMSS. C.P. has served on advisory boards for Novartis, Merck, Biogen, Sanofi-Genzyme, Roche, Janssen and Alexion; received travel and speaker honoraria from Merck, Serono, Biogen, Sanofi-Genzyme, Roche, Almirall, Janssen, Alexion and Novartis; and research support from Merck, Biogen, Novartis and Almirall. R.H. has received personal fees from Novartis, Sanofi, Merck, Biogen, Teva, Janssen/J&J and Roche. E.K.H. has received grants or contracts from the Czech Ministry of Education (Cooperatio LF1 and EXCELES projects). She has received payments or honoraria for lectures, presentations, speaker bureaus, writing, or educational events from Actelion/Janssen, Biogen, Celgene/BMS, Merck, Novartis, Roche, Sanofi and Teva, and has participated in DSMBs or advisory boards for Actelion/Janssen, Biogen, Celgene/BMS, Merck, Novartis, Roche and Sanofi. R.F. has served as consultant or advisory board member for Arrowhead, Astoria Biologica, Biogen, BMS, Cognito, Eli Lilly, Roche, Galvani, Immunic, Inmune Bio, Innocare, Kiniksa, NGM Bio, Novartis, Pfizer, Population Council, Sanofi, Siemens, TG Therapeutics, Tr1X, Ventus and Viracta. His institution has received clinical trial contracts from Sanofi and Synaptogenix. He has participated in DSMBs for AB Science and has been employed by Janssen Pharmaceuticals. M.S.F. has received research or educational grants from Sanofi-Genzyme Canada; consulting fees or honoraria from Alexion/AstraZeneca, BiogenIdec, EMD Serono/Merck Serono, Find Therapeutics, Roche, Novartis, Quanterix, Sanofi-Genzyme and Teva Canada. He serves on advisory boards or boards of directors for Alexion/AZ, Atara, Bayer, Celestra, EMD/Merck, Find Therapeutics, Roche, Actelion/Janssen, Novartis, Sanofi-Genzyme and Setpoint Medical, and has participated in speaker bureaus for EMD Serono and Sanofi-Genzyme. M.H., S.G., A.A. and A.S. are employees of Juvise Pharmaceuticals.

EPO-0139 | Automated analysis of natural speech reveals emotional alterations in multiple sclerosis

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Background and aims: Speech conveys emotional states that standard clinical assessments often miss. In multiple sclerosis (MS), affective disturbances are common yet difficult to quantify objectively. This study investigated whether automated language-based emotion analysis can identify affective alterations in MS.

Methods: We analyzed speech from 240 native Czech speakers, including 120 patients with MS and 120 age-, sex-, and education-matched healthy controls. Participants completed two speech tasks: a spontaneous monologue and a story-telling narration. Speech recordings were automatically transcribed using an automatic speech recognition system. Emotional content was quantified using a fine-tuned multilingual BERT (mBERT) model adapted to recognize six emotional states: joy, surprise, love, anger, fear, and sadness.

Results: In spontaneous monologues, patients with MS demonstrated significantly higher levels of anger ($p=0.034$) and lower levels of joy ($p=0.003$) compared to controls (Figure 1). During the story-telling task, MS patients showed reduced joy ($p=0.024$) relative to controls. Anger was the dominant emotion in the MS group, accounting for 31% of the discourse, followed by joy (24%) and sadness (22%). In contrast, healthy controls exhibited joy as the predominant emotion (30%), followed by anger (29%) and sadness (21%). Surprise emotion was not analyzed because it was not detected in any sentence in 82% of participants.

Conclusion: Language-based emotion analysis revealed distinct affective alterations in individuals with MS, most notably reduced joy and increased anger. Fine-tuned large language model enables a fully automated and objective assessment of these changes and may complement and extend conventional clinical evaluations in MS.

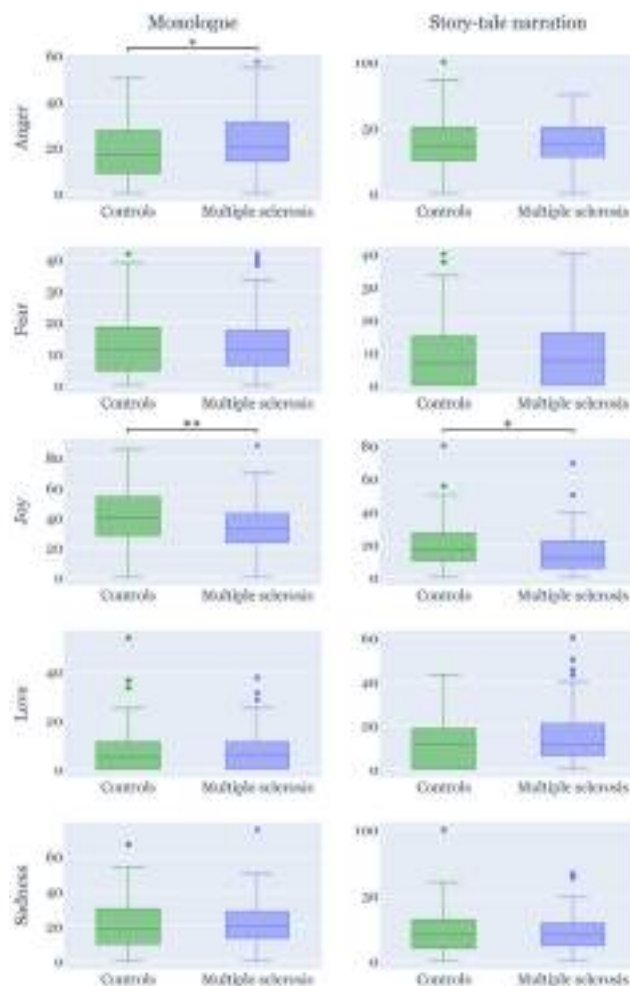


FIGURE 1 Between-group differences in individual emotional features. Asterisks indicate statistically significant differences based on independent-samples *t*-tests (* $p < 0.05$, ** $p < 0.01$).

Disclosure: This project was supported by the ERC–CZ program from the Ministry of Education Youth and Sports (project no. LL2504).

EPO-0140 | Diffusion tensor image analysis along the perivascular space in multiple sclerosis: Microstructural influences & considerations for glymphatic function

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Background and aims: The glymphatic system facilitates waste clearance through perivascular pathways and has been implicated in multiple sclerosis(MS) pathophysiology. Diffusion tensor image analysis along the perivascular space(DTI-ALPS) has been proposed as a noninvasive marker of glymphatic function; however, its specificity in demyelinating disease remains uncertain. This study examined DTI-ALPS in MS and its associations with structural MRI markers and indicators of disease progression.

Methods: Twenty-six MS patients and 28 healthy controls (Table 1) underwent diffusion-weighted imaging. Bilateral DTI-ALPS indices were calculated for all participants. Structural MRI measures included lesion-load(LL), choroid-plexus volume(ChPV), and lateral-ventricle volume(LVV). Clinical measures included Expanded Disability Status Scale (EDSS) and disease duration. Group comparisons were performed (Table 2), and within the MS group, correlations between DTI-ALPS and imaging and clinical measures were assessed.

TABLE 1 Demographics of study participants. Abbreviations: PwMS = Patient with Multiple Sclerosis; HC = Healthy Control; SD = Standard Deviation; IQR = Interquartile Range; EDSS = Expanded Disability Status Scale; *p*-values calculated using *Chi-Squared Te.

	PwMS (n = 26)	HC (n = 28)	P-values
Sex, female, n (%)	18 (73.1)	20 (71.4)	0.89 ⁺
Age in years, mean (SD)	33.9 (9.6)	32.3 (5.5)	0.45 ⁺
Disease duration in years, median (IQR)	4.5 (3.6)	-	-
EDSS, median (IQR)	1.5 (1.0)	-	-

TABLE 2 MR Imaging Characteristics describing mean normalized volumetry results. Abbreviations: PwMS = Patient with Multiple Sclerosis; HC = Healthy Control; SD = Standard Deviation. *p*-values calculated using Independent Samples T-Test.

	PwMS (n = 26)	HC (n = 28)	P-values
T2 lesion volume, mL (SD)	7.79 (16.72)	0.07 (0.10)	<0.001
Lesion load, % (SD)	0.001 (0.001)	0.000 (0.000)	0.00
Total lateral ventricle volume, % (SD)	1.33 (0.55)	1.03 (0.54)	0.03
Choroid plexus volume, % (SD)	0.13 (0.04)	0.10 (0.02)	0.004

Results: DTI-ALPS indices did not differ significantly between MS patients and healthy controls (*p*-FDR > 0.4). Within the MS group, DTI-ALPS was not significantly associated with structural or clinical markers after correction for multiple comparisons (*p*-FDR > 0.76). Importantly, component analysis in patients showed that ALPS indices were strongly associated with diffusion perpendicular to the perivascular axis (the denominator of the ALPS ratio; *p*-FDR < 0.001). In contrast, correlations with diffusion along the perivascular direction (the numerator) were not significant (*p*-FDR > 0.1).

Conclusion: These findings suggest that DTI-ALPS in MS may be influenced by white matter microstructural alterations rather than reflecting perivascular glymphatic function. Caution is therefore warranted when interpreting DTI-ALPS as a marker of glymphatic function, especially in demyelinating disease, and complementary approaches may be required to assess glymphatic pathway involvement.

Disclosure: The authors report no conflicts of interest related to this work.

Muscle and Neuromuscular Junction Disorder 1

EPO-0141 | Steroids tapering among patients with generalized myasthenia gravis (gMG) receiving efgartigimod: Interim results from the STRIVE-gMG chart review

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Background and aims: Long-term steroids use among patients with gMG is associated with substantial complications. Efgartigimod, approved in the United States (US) in 2021 for AChR antibody-positive gMG, has demonstrated clinical benefits that may facilitate steroids reduction. This study evaluated real-world patterns of steroids tapering following efgartigimod initiation in the US.

Methods: STRIVE-gMG is a retrospective chart review of patients with gMG who received efgartigimod (≥2 cycles) and concurrent steroids at efgartigimod initiation. Data are collected from neurologists in a specialty panel and from participating medical centers. This interim analysis summarizes initial data from the neurologist panel.

Results: As of November 10, 2025, 41 patient charts from 15 neurologists were analyzed. All patients were AChR antibody-positive. At efgartigimod initiation, patients had a mean age of 48.9 years, mean disease duration of 3.7 years, and mean duration on current steroids of 1.1 years. Most (87.8%) had MGFA class II–III disease. Following efgartigimod initiation, all patients tapered steroids, with mean prednisone-equivalent dose decreasing from 30.4 mg/day at efgartigimod initiation to 8.6,

TABLE 1 Baseline characteristics of patients.

	Patients (N=41)
Age at efgartigimod initiation (years), mean (SD)	48.3 (11.6)
Years of gMG before efgartigimod initiation, mean (SD)	3.7 (4.2)
Sex, n (%)	
Male	18 (43.9)
Female	23 (56.1)
Race, n (%)	
White or Caucasian	28 (68.3)
Black or African American	7 (17.1)
Asian/Other	6 (14.6)
Insurance type ^a , n (%)	
Commercial	35 (85.4)
Medicare	3 (7.3)
Medicaid	3 (7.3)
Comorbidity ^b , n (%)	
Hypertension	17 (41.5)
Hyperlipidemia	13 (31.7)
Anxiety	11 (26.8)
Depression	10 (24.4)
Fatigue	9 (22.0)
Chronic pain	6 (14.6)
Autoimmune diseases	5 (12.2)
Cardiovascular diseases	5 (12.2)
MGFA classification at efgartigimod initiation, n (%)	
0 (remission)	1 (2.4)
1 (ocular only)	2 (4.9)
2A (mild generalized, predominantly limb/axial muscle weakness)	7 (17.1)
2B (mild generalized, predominantly bulbar muscle weakness)	1 (2.4)
3A (moderate generalized, predominantly limb/axial muscle weakness)	14 (34.1)
3B (moderate generalized, predominantly bulbar muscle weakness)	2 (4.9)
4A (severe generalized, predominantly limb/axial muscle weakness)	1 (2.4)
4B (severe generalized, predominantly bulbar muscle weakness)	0 (0.0)
5 (tracheal requires ventilatory support)	1 (2.4)
Treatment received in the 3-year period prior to efgartigimod initiation ^c , n (%)	
Steroids	41 (100%)
ACNE inhibitors	14 (34.1)
NS5Ts	7 (17.1)
Biologics ^{d,e}	0 (0.0)
IVIGs/SCIGs	5 (12.2)
Plasma exchange	1 (2.4)

Abbreviations: ACNE, acetylcholinesterase; MGFA, Myasthenia Gravis Foundation of America; gMG, generalized Myasthenia Gravis; NS5T, non-steroidal immunosuppressant therapies; IVIG, intravenous immunoglobulin; SCIG, subcutaneous immunoglobulin; SD, standard deviation.

Notes:

^a Multiple responses were allowed; therefore, totals may exceed 100%.

^{d,e} Biologics include separate Fc receptor (FcR) inhibitors, complement inhibitors, and B-cell therapies.

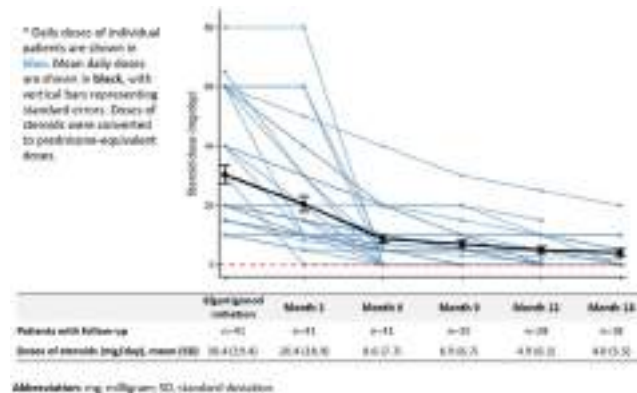


FIGURE 1 Steroids tapering while on efgartigimod.

4.9, and 4.0 mg/day at months 6, 12, and 18, respectively; 68.3% discontinued steroids entirely, primarily due to stable disease control. At the end of follow-up, 92.7% remained on efgartigimod, with a median treatment duration of 17.1 months. All patients showed improvements in functional limitations and muscle weakness.

Conclusion: In this interim analysis, efgartigimod was associated with profound reductions in steroids dose while maintaining clinical improvement among patients with gMG. Future studies are needed to examine the impacts of steroids reduction on comorbidities and healthcare utilization.

TABLE 2 Efgartigimod treatment response and steroids tapering while on efgartigimod.

	Patients (N=41)
Improvement in functional status, n (%)	
No functional limitations at any time during treatment	7 (17.1%)
Improvement, but some functional limitations remained	34 (82.9%)
Improvement in muscle weakness, n (%)	
No weakness or only some weakness during treatment	32 (78.0%)
Improvement, but moderate to severe muscle weakness remained	9 (22.0%)
Patients tapered steroids while on efgartigimod, n (%)	41 (100.0%)
Steroids tapering amount while on efgartigimod ^a	
Mean change in daily dose (mg/day), mean (SD)	27.4 (13.2)
With ≥ 50% reduction in daily dose, n (%)	40 (97.6%)
Last dose of steroids while on efgartigimod ^b , n (%)	
>30 mg/day	1 (2.4%)
>5 to ≤30 mg/day	4 (9.8%)
>0 to ≤5 mg/day	8 (19.5%)
0 (i.e., discontinued)	28 (68.3%)
Reasons for steroids discontinuation ^c , n (%)	N = 28
Able to maintain disease control without steroids	25 (89.3%)
Transitioned to other therapies	4 (14.3%)
Tolerability issues	2 (7.1%)
Patient preference	2 (7.1%)

Abbreviations: mg, milligram; SD, standard deviation.

Notes:

^a Based on the last available dose of steroids while on efgartigimod in medical charts; some patients were still undergoing tapering.

^b Multiple responses were allowed; therefore, totals may exceed 100%.

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Background and aims: Efgartigimod is a human immunoglobulin G1 (IgG1) Fc fragment that blocks the neonatal Fc receptor. ADAPT-SC+ (NCT04818671) was an open-label extension study evaluating long-term safety and efficacy of subcutaneous (SC) efgartigimod PH20 (coformulated with recombinant human hyaluronidase PH20) in participants with generalized myasthenia gravis (gMG).

Methods: Efgartigimod PH20 SC 1000 mg was administered in cycles of 4 once-weekly injections. Subsequent cycles were

initiated based on clinical evaluation (Year 1, ≥ 4 weeks between cycles; Year 2 and onward, ≥ 1 week between cycles following protocol amendment). Efficacy was assessed using Myasthenia Gravis Activities of Daily Living (MG-ADL). Quality of life (QoL) impact was assessed during Year 1 using Myasthenia Gravis Quality of Life 15-Item Questionnaire, Revised (MG-QoL15r).

Results: During ADAPT-SC+, 180 participants received ≥ 1 efgartigimod PH20 SC injection (mean [SD] study duration, 2.6 [0.9] years; 459.4 participant years of follow-up). Adverse events were predominantly mild/moderate (Table 1). After Year 1, efgartigimod remained well tolerated in both participants who averaged <4 weeks and ≥ 4 weeks between treatment cycles. Among anti-acetylcholine receptor antibody-positive (AChR-Ab+) participants, mean MG-ADL improvements were sustained from Week 1 through Week 163 (Figure 1). Most AChR-Ab+ participants (59.2%) achieved minimal symptom expression (MSE; MG-ADL score, 0–1) at least once, and 88.1% of those with MSE at least once sustained MSE during ≥ 4 weeks. Mean MG-QoL15r improvements were observed by Week 1 and sustained through Week 55 (Figure 2).

Conclusion: Long-term efgartigimod PH20 SC treatment was well tolerated and led to sustained efficacy and QoL improvements during ADAPT-SC+.

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TABLE 1 Summary of TEAEs: Overall Population.

	Efgartigimod PH20 SC (N=180; PYFU=459.4)	
	ER ^a	n (%)
Any TEAE	7.2	169 (93.9)
Any TEAE grade ≥ 3	0.3	62 (34.4)
Any serious TEAE	0.2	55 (30.6)
Fatal event ^b	<0.1	5 (2.8)
Discontinued study treatment due to TEAEs ^c	<0.1	8 (4.4)
Any injection site reaction ^d	2.6	63 (46.1)
Most commonly observed TEAEs ^e		
COVID-19	0.1	53 (29.4)
Headache	0.4	46 (25.7)
Nasopharyngitis	0.1	38 (20.0)
Diarrhea	0.1	32 (17.8)
Upper respiratory tract infection	0.2	32 (17.8)
Myasthenia gravis worsening	0.1	25 (13.9)
Back pain	0.1	22 (12.2)
Anemia	0.1	19 (10.6)

^aEvent rate (ER) was calculated as number of events per participant year of follow-up (PYFU). ^bFatal events (metastatic renal cell cancer, cardiac arrest, respiratory failure [$n=2$], and COVID-19/respiratory failure) were not related to efgartigimod PH20 SC treatment, as determined by investigators. ^cTreatment discontinuations were due to metastatic renal cell cancer (Cycle 1, fatal), cardiac arrest (Cycle 2, fatal), pulmonary mass (Cycle 3), COVID-19/respiratory failure (Cycle 3, fatal), and MG crisis (Cycle 1), thymoma (Cycle 4), metastatic renal cancer (Cycle 21 [Amended Cycle 6]), and Bouveret's disease (Cycle 16 [Amended Cycle 5]). Among injection site reactions, the most frequently reported were arthralgia (57.8%), edema (20.0%), and swelling (7.8%). ^dMost frequent TEAEs occurring in $>10\%$ of participants in the overall population: ER, event rate; MG, myasthenia gravis; PYFU, participant year(s) of follow-up; SC, subcutaneous; TEAE, treatment-emergent adverse event.

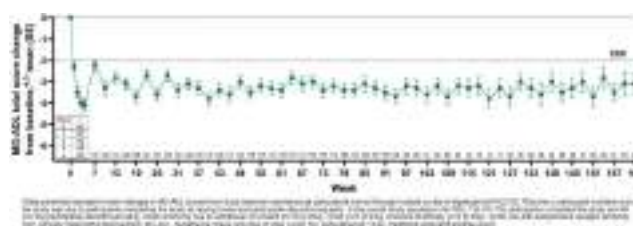


FIGURE 1 Mean Change from Baseline in MG-ADL Total Score Over Time: AChR-Ab+ Population.

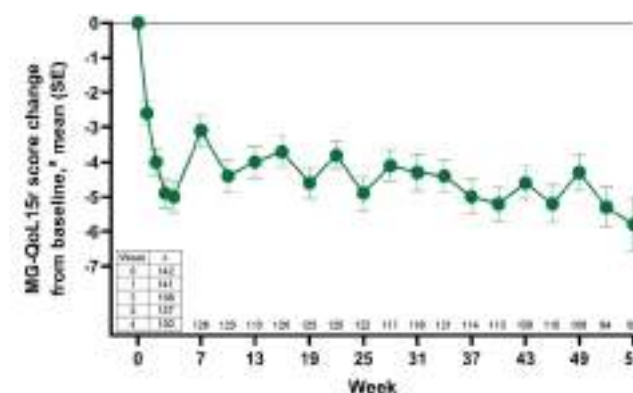


FIGURE 2 Mean Change from Baseline in MG-QoL15r Score Over Time: AChR-Ab+ Population.

safety of efgartigimod in gMG, ITP, and CIDP, administered via varying dosing regimens.

Methods: Data were pooled per disease from global clinical trials investigating varying efgartigimod dosing regimens and administration routes (intravenous 10 mg/kg; subcutaneous 1000 mg PH20 [coformulated with recombinant human hyaluronidase PH20]). Event rates (ER) were reported per participant year of follow-up (PYFU).

Results: The analysis includes data from at least 874 patients representing ~1350 PYFU. Treatment-emergent adverse events (TEAE) were mostly mild-to-moderate in severity. During controlled trials, severe infection ERs were comparable between placebo- and efgartigimod-treated participants. Reported TEAE-related treatment discontinuation rates were consistently low. Rates of discontinuation and severe and serious TEAEs did not increase with additional treatment cycles (gMG) or prolonged exposure to weekly administration (ITP, CIDP). Further analyses of participant population subgroups are ongoing. No albumin reduction or increases in low-density lipoprotein cholesterol levels were reported in efgartigimod-treated participants. No vaccinations, laboratory value monitoring, or pretreatment medications are required to receive efgartigimod. Efgartigimod was consistently well tolerated across trials in autoimmune diseases, including those with subcutaneous administration of efgartigimod.

Conclusion: Efgartigimod was well tolerated and demonstrated a similar safety profile regardless of disease state, dosing regimen, and length of exposure.

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EPO-0145 | Italian real-life experience with Zilucoplan for generalized myasthenia gravis: One year of ZILU25 study

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Background and aims: Generalized Myasthenia gravis (gMG) is a rare chronic autoimmune disease affecting the postsynaptic membrane of the neuromuscular junction. New treatment options, such as the C5 inhibitors, are growing with promising results. In this study we investigated the real-world use of Zilucoplan for AChR-positive gMG in Italy.

Methods: Zilucoplan was self-administered by daily subcutaneous injections. Efficacy was assessed by Myasthenia Gravis Foundation of America Post-intervention status (MGFA-PIS), Myasthenia Gravis Activity of Daily Living (MG-ADL), Myasthenia Gravis Composite (MGC), Myasthenia Gravis quantitative (QMG) scales and 15-items Myasthenia Gravis Quality of Life (MG-QOL-15) questionnaire at baseline and after 1, 4, 12, 24 and 48 weeks.

Results: 54 patients (37 females, aged 57.3 years) were included in the follow-up analysis. Clinical improvements observed in the initial study were sustained over time, with stable or further reduced MG-ADL, QMG, MGC and MG-QOL-15r scored over weeks. Moreover, it was also observed a continued steroid-sparing effect and a reduction of hospitalization due to MG worsening. Treatment adherence remained high, and no new safety report emerged during the extended follow-up.

Conclusion: Zilucoplan has proven to be well-tolerated and effective in most patients with AChR-positive gMG. A clinical meaningful effect was reported since the first week and was sustained during the following weeks. Further studies are needed to confirm these preliminary real-life data.

Disclosure: Nothing to disclose.

EPO-0146 | Quality of gravis patients achieving sustained versus transient clinical outcomes in the phase 3 vivacity-MG3 trial

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Background and aims: Minimal clinically important improvement in the Myasthenia-Gravis-Activities-of-Daily-Living (MG-ADL) score (MCI; ≥ 2 -point improvement) or a composite response (CR) of ≥ 2 -point improvement in MG-ADL and ≥ 3 -point improvement in Quantitative-Myasthenia-Gravis scores are plausible treatment goals in generalized myasthenia gravis (gMG). This Vivacity-MG3 posthoc analysis evaluated health-related quality of life (HRQoL) in patients achieving sustained versus transient MCI or CR.

Methods: Nipocalimab- and placebo-treated participants were stratified by MCI or CR status over the 24-week double-blind phase: never achieved MCI/CR (No-MCI/CR), achieved MCI/CR sustained ≥ 8 weeks (sustained-MCI/CR), or achieved MCI/CR ≥ 1 time but not sustained for ≥ 8 weeks (transient-MCI/CR). Mean changes from baseline (CFB) to week 24 (W24) in MGQoL-15r and Neuro-QoL Fatigue scores were compared across groups using ANCOVA models.

Results: The MCI analysis included 129 patients (91 sustained-MCI, 29 transient-MCI, 9 no-MCI) and the CR analysis included 125 patients (50 sustained-CR, 37 transient-CR, 38 no-CR). Sustained- versus transient-MCI patients demonstrated greater improvements in MGQoL-15r (least-square-mean CFB, -6.2 versus -2.0 , $p < 0.001$). Additionally, the sustained-MCI group had greater improvements in Neuro-QoL Fatigue (-19.5) versus transient-MCI (-7.5 , $p < 0.001$) and No-MCI (-8.5 , $p = 0.04$). Similarly, sustained-CR patients demonstrated greater improvements in MGQoL-15r (-9.6) versus transient-CR (-5.4 , $p < 0.001$) and No-CR (-3.6 , $p < 0.001$) and those with sustained-CR had greater improvements in Neuro-QoL Fatigue (-29.2) versus transient-CR (-16.4 , $p < 0.001$) and No-CR (-12.2 , $p < 0.001$).

Conclusion: Patients achieving sustained versus transient MCI or CR demonstrated greater improvements in HRQoL and fatigue, suggesting 8-week sustained outcomes may be more

impactful treatment goals than short-term transient response for gMG patients receiving advanced treatments.

Disclosure: Funding This study was funded by Johnson & Johnson. Author Disclosures JV has received consultant fees for serving on advisory boards for Alexion Pharmaceuticals Inc, argenx BV, Dianthus Therapeutics, Horizon Therapeutics (now Amgen Inc.), Johnson & Johnson, Regeneron, Roche, and UCB Pharma SA. CA has received funding for travel, meeting attendance, and advisory board participation from Alexion, Momenta, Sanofi, argenx, UCB, and Johnson & Johnson. KGG has served as a paid consultant for Alexion Pharmaceuticals, argenx, and UCB; and she has served on the speakers' bureau for Alexion Pharmaceuticals. KG, IT, SP, and NC are employees of and may hold stock in Johnson & Johnson.

EPO-0147 | Myasthenia gravis Inebilizumab trial: 26-week randomised-placebo controlled and 26-week open-label results in anti-muscle-specific kinase subgroup

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Background and aims: Generalised myasthenia gravis (gMG) is caused by anti-muscle-specific kinase (MuSK+) and anti-acetylcholine receptor (AChR+) antibodies targeting corresponding proteins in the synapse. The placebo (PBO)-controlled MG Inebilizumab (INEB) Trial (MINT) assessed INEB, a monoclonal antibody targeting CD19+ B cells, in patients with MuSK+ or AChR+ gMG. INEB vs PBO yielded significantly greater improvements in MG Activities of Daily Living (MG-ADL) and Quantitative MG (QMG) scores at week 26 in MINT. Here we report key outcomes for the MuSK+ subpopulation from the first 26 weeks of the open-label period (OLP).

Methods: Participants with MuSK+ gMG in MINT (NCT04524273) were randomised 1:1 to 300 mg intravenous INEB or PBO for 26 weeks. Participants underwent a protocol-specified steroid taper (≤ 5 mg/day by week 24). After the randomised, controlled period (RCP), participants could receive INEB in the OLP. This pre-specified, interim, descriptive analysis included participants who received any dose of INEB during MINT. No statistical testing was done.

Results: Overall, 48 participants with MuSK+ were enrolled and randomised: 46 entered the OLP and 44 completed 26 weeks. Baseline MG-ADL and QMG scores were similar between groups, and mean scores decreased from baseline to week 26 during the RCP (INEB -4.1 , -5.0 ; PBO -3.1 , -4.2 , respectively). During the initial 26-week OLP (week 52), the mean MG-ADL and QMG scores continued to decrease from baseline (INEB/INEB: -5.6 , -6.1 ; PBO/INEB: -5.1 , -7.4 , respectively; Figures 1–2; Table 1).

Conclusion: In this descriptive analysis, INEB demonstrated ongoing improvement in MG-ADL and QMG scores compared to baseline irrespective of initial randomisation.

Disclosure: JV has been a consultant for and/or received speaker honoraria from Alexion Pharmaceuticals, Amgen Inc, argenx, Dianthus Therapeutics, Johnson & Johnson, NMD Pharma, Novartis, Regeneron Pharmaceuticals, Roche, Toleranzia AB, and UCB. MIL received funding from the National Health Service (MRDS and NSCG for NMOSD) and the University of Oxford. She was awarded research grants from the UK association for patients with myasthenia (myaware) and the University of Oxford. She received speaker honoraria/travel grants from the Guthy-Jackson Charitable Foundation and UCB. She served on scientific/educational advisory boards for argenx, UCB, and Amgen Inc. JFH received research funding (paid to his institution) from Ad Scientiam, Alexion AstraZeneca Rare Disease, argenx, Cartesian Therapeutics, the Centers for Disease Control and Prevention, EMD Serono, Merck, the Muscular Dystrophy Association, Myasthenia Gravis Foundation of America, the National Institutes of Health, NMD Pharma, and UCB; honoraria/consulting fees from AcademicCME, Alexion AstraZeneca Rare Disease, Amgen Inc, Biohaven, CheckRare CME, CorEvitas, Curie.Bio, EMD Serono, Hansa Biopharma, Lundbeck, Merck, Novartis, PeerView CME, Physicians' Education Resource CME,

TABLE 1 Change from BL in MG-ADL and QMG Scores.

	RCP Week 26	OLP Week 26 (Week 52)
	Mean Change From Baseline (SE)	
MG-ADL Score		
PBO/INEB	-3.1 (0.7)	-5.1 (0.8)
INEB/INEB	-4.1 (0.7)	-5.6 (0.7)
QMG Score		
PBO/INEB	-4.2 (1.1)	-7.4 (1.1)
INEB/INEB	-5.0 (1.0)	-6.1 (1.1)

This is a descriptive analysis. All statistical hypothesis testing was conducted for the primary analysis after the 26-week RCP.

INEB, inebilizumab; MG-ADL, Myasthenia Gravis Activities of Daily Living; OLP, open-label period; PBO, placebo; QMG, Quantitative Myasthenia Gravis; RCP, randomised, controlled period; SE, standard error.

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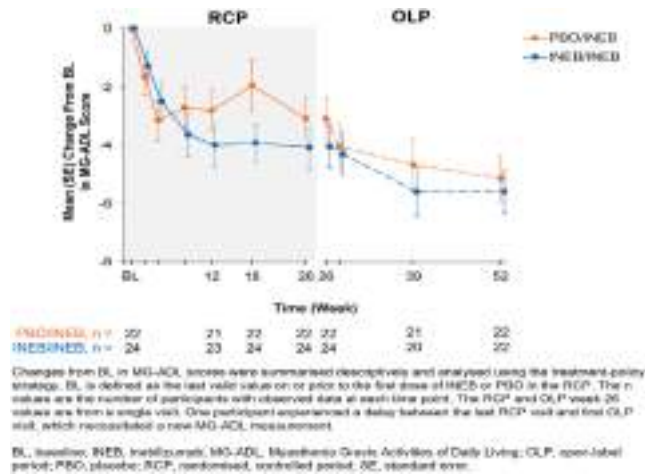


FIGURE 1 Change from BL in MG-ADL Score.

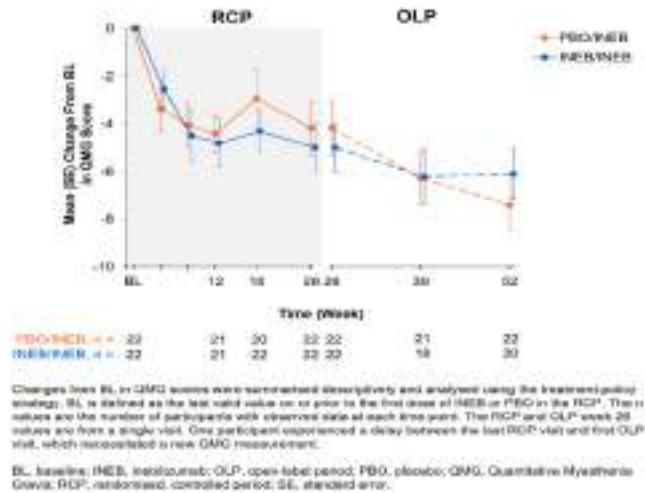


FIGURE 2 Change from BL in QMG Score.

EPO-0148 | Multiple responder analyses with claspribart, an active C1s inhibitor, in patients with generalized myasthenia gravis

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Background and aims: The classical complement pathway plays a significant role in acetylcholine receptor-positive (AChR+) generalized myasthenia gravis (gMG) pathology. Claseprubart is an investigational, clinical-stage, potent monoclonal antibody engineered to selectively target the classical complement pathway, by selectively inhibiting activated C1s protein. The clinical benefit of claseprubart in gMG may be best supported by improvement across multiple validated scales to capture multi-dimensional improvement beyond single-scale analyses.

Methods: AChR+ gMG patients treated with 300 mg claseprubart subcutaneously once every 2 weeks in the MaGic study (NCT06282159) – a global Phase 2, randomized, double-blind, placebo-controlled trial – were analyzed for responses based on multiple assessments compared to placebo. *p*-values are one-sided, with nominal significance assessed at an alpha of 0.1. A responder was defined as achieving an improvement in MG-ADL ≥ 3 and QMG ≥ 4 . Endpoints include proportion of dual MG-ADL and QMG responders at Week 13, time to response, and proportion of responders sustaining response for at least 6 weeks.

Results: 63% of claseprubart patients had dual response improvement in MG-ADL ≥ 3 and QMG ≥ 4 , versus 14% for placebo (OR 29.00; *p* = 0.0006). Dual responders were seen as early as Week 1. The median time to response was Week 3. All claseprubart patients with dual response at Week 13 had maintained response for at least 6 weeks.

Conclusion: Claseprubart-treated patients were more likely to achieve multi-dimensional response than placebo. Responses emerged early and were sustained, confirming rapid, robust, and durable benefit from claseprubart across functional and quantitative measures from both patient and clinician perspectives.

Disclosure: MD grant or honoraria from Abcuro, Alexion/AstraZeneca, Alnylam, Amicus, Annexon, ArgenX, Astellas, AstraZeneca, Biosensics, Bristol-Myers Squibb, Cabaletta, Candid, Catalyst, Covance, CSL-Behring, Creyon, Dianthus, EMD Serono/Merck, GlaxoSmithKline, Genentech, Grifols, Horizon/Immunovant, Janssen, Kriya, Medlink, Nkarta, Novartis, Nuvig, Mitsubishi Tanabe, Octapharma, Orphazyme, Priovant, Sanofi Genzyme, Shire Takeda, TACT/Treat NMD, UCB Biopharma, and Vertex Pharmaceuticals. FS nothing to declare. MA employee of Dianthus Therapeutics CB employee of Dianthus Therapeutics US employee of Dianthus Therapeutics LH employee of Dianthus Therapeutics ML employee of Dianthus Therapeutics MS nothing to declare.

EPO-0149 | Treatment response in anti-HMGCR and anti-SRP immune-mediated necrotizing myopathy: Impact of demographic and racioethnic factors

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Background and aims: Immune-mediated necrotizing myopathy (IMNM) associated with anti-HMGCR and anti-SRP autoantibodies is a rare autoimmune neuromuscular condition characterized by significant phenotypic heterogeneity. Despite varying clinical presentations, the impact of demographic and racioethnic factors on treatment response remains poorly characterized.

Methods: This multi-center retrospective study analyzed 56 patients with IMNM (48 anti-HMGCR, 8 anti-SRP). We assessed demographics, racioethnicity, number of immune therapies, and therapy escalations (defined as addition of therapy or increase in dose/frequency). Statistical analysis included Mann-Whitney U, Kruskal-Wallis, and Fisher's Exact tests. Significance was set at *p* < 0.05.

Results: The cohort (mean age 59.8 years, 54.6% female, 82.1% statin-exposed) predominantly included anti-HMGCR patients (85.7%). Anti-HMGCR patients were significantly older (mean 62.3 vs. 44.8 years, *p* = 0.003) with higher statin exposure (91.7% vs. 25.0%, *p* < 0.001) than anti-SRP patients. Mean time to diagnosis (12.3 months) was similar between groups. Peak CK levels varied significantly across racioethnic groups in the overall cohort, with the highest levels observed in Black (11,978 U/L) and Asian (10,332 U/L) patients compared to Hispanic (9,156 U/L) and White (6,751 U/L) patients. Younger patients (<59.8 years) required significantly more immune therapies (mean 3.4 vs. 2.8, *p* = 0.044) and treatment escalations (2.8 vs. 1.2, *p* = 0.025). Hispanic patients required significantly higher treatment escalations, nearly three times more than White patients (mean 2.9 vs. 0.9, *p* = 0.026). Subgroup analysis confirmed similar significant trends within the anti-HMGCR cohort.

Conclusion: Demographic and racioethnic factors significantly influence IMNM disease severity and treatment intensity. This study underscores the need for larger studies to validate these findings and guide personalized therapeutic strategies.

Disclosure: Nothing to disclose.

EPO-0150 | Efficacy of long-term zilucoplan treatment in subgroups of patients with generalised myasthenia gravis: A 120-week analysis of RAISE-XT

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Background and aims: Clinically meaningful improvements in myasthenia gravis (MG)-specific outcomes were observed with zilucoplan treatment up to Week 120 in patients with

generalised MG (gMG) in the Phase 3, ongoing, open-label extension study, RAISE-XT (NCT04225871). Here, we evaluate the efficacy of long-term zilucoplan treatment in patients with gMG, stratified by specific disease characteristics.

Methods: Adults with anti-acetylcholine receptor antibody-positive gMG who completed a qualifying double-blind, placebo-controlled study (NCT03315130 or NCT04115293 [RAISE]) could enter RAISE-XT and self-administer once-daily subcutaneous zilucoplan 0.3 mg/kg injections. Change from baseline (CFB) to Week 120 in MG-specific outcome measure scores was assessed (data cut-off: 11 November 2023). Subgroups were prespecified according to the following baseline characteristics: age <65 or ≥65 years, MG Activities of Daily Living (MG-ADL) score ≤9 or ≥10, and disease duration <5.2 or ≥5.2 years.

Results: Overall, 200 patients enrolled. At Week 120, mean (standard error) CFB in MG-ADL scores across all subgroups were: age: <65 years, $n=64$: -7.3 (0.5) and ≥65 years, $n=22$: -6.7 (0.8); MG-ADL: ≤9, $n=68$: -7.0 (0.4) and ≥10, $n=18$: -7.6 (1.4); and disease duration: <5.2 years, $n=42$: -6.7 (0.7) and ≥5.2 years, $n=44$: -7.5 (0.6). Improvements were also observed at Week 120 for Quantitative MG, MG Quality of Life 15-item revised, and Quality of Life in Neurological Disorders Short Form Fatigue scores. Treatment-emergent adverse events occurred in 97.0% ($n/N=194/200$) of patients; most were mild or moderate and unrelated to the study drug.

Conclusion: Long-term zilucoplan treatment demonstrated consistent improvements in MG-specific outcomes up to Week 120, irrespective of disease severity, duration or patient age.

Disclosure: This study was funded by UCB. Natasa Savic, Babak Borojerdi and Paul Mahoney are employees and shareholders of UCB. Full disclosure of all industry relationships will be made during congress presentation if accepted.

EPO-0151 | Health-related quality of life in patients with generalised myasthenia gravis receiving gefurulumab in the PREVAIL trial

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Background and aims: Gefurulumab is a novel dual-binding nanobody complement component 5 inhibitor. Efficacy and safety of gefurulumab in adults with anti-acetylcholine receptor antibody-positive (AChR-Ab+) generalised myasthenia gravis (gMG) were demonstrated in PREVAIL, a phase 3, randomised, placebo-controlled trial (NCT05556096). To provide a holistic view of the treatment effect, we present health-related (HR) quality of life (QoL) outcomes.

Methods: Patients with AChR-Ab+ gMG were randomised to receive weekly subcutaneous self-injection of gefurulumab or placebo. Exploratory endpoints assessed HRQoL changes from baseline through wk26 (randomised period completion) using: MG-QOL 15-Item Questionnaire-Revised (MG-QOL15r); QoL in

Neurological Disorders (Neuro-QoL) Fatigue Scale; EuroQol-5 Dimensions-5-Level (EQ-5D-5L) Visual Analogue Scale (VAS) and Health State Index (HSI).

Results: Overall, 260 patients (female, 60.4%; mean ± SD age, 52.8 ± 15.7 yrs; mean ± SD baseline MG-ADL total score, 9.0 ± 2.2) were randomised to gefurulumab ($n=131$) or placebo ($n=129$). Significantly greater improvements were observed with gefurulumab compared with placebo, starting at wk4 in MG-QOL15r and Neuro-QoL Fatigue and wk8 in EQ-5D-5L. These improvements were sustained throughout the randomised period. At wk26, the least squares mean (95% CI) changes from baseline among patients with available data receiving gefurulumab ($n=123$) vs placebo ($n=117$) were -4.9 ($-6.0, -3.8$) vs -2.3 ($-3.2, -1.4$) ($p=0.0005$) in MG-QOL15r; -4.8 ($-6.2, -3.4$) vs -2.5 ($-3.8, -1.1$) ($p=0.0164$) in Neuro-QoL Fatigue; 9.2 (6.2, 12.1) vs 4.6 (1.6, 7.6) ($p=0.0349$) in EQ-5D-5L VAS; 0.12 (0.076, 0.155) vs 0.04 ($-0.004, 0.079$) ($p=0.0088$) in EQ-5D-5L HSI.

Conclusion: In PREVAIL, patients with AChR-Ab+ gMG receiving gefurulumab had significant improvements in HRQoL compared with those receiving placebo, demonstrated by MG-specific and general HRQoL measures.

Disclosure: TR reports grants from the German Ministry of Education, Science, Research, and Technology; grants and personal fees from Alexion, AstraZeneca Rare Disease, argenx, Johnson & Johnson, Roche, Sanofi, and UCB; personal fees from Amgen, Merck, Novartis, and Roche outside of the submitted work. FS has received public speaking honoraria from Alexion, argenx, Biogen, Genpharm, Medpharma, Medison Pharma, Mylan, Neopharm Israel, Novartis, Roche, Sanofi, UCB, and Zai Lab; compensation for advisory boards from Alexion, Almirall, Amgen, argenx, AstraZeneca, Avexis, Biogen, Dianthus, Johnson & Johnson, Lexeo, Novartis GmbH, Reata, Sandoz, UCB, and Zai Lab; consultation fees from Alexion, argenx, Dianthus, EPG Communication Holdings Ltd, Johnson & Johnson, Medscape, PeerView, Vitaccess; clinical trial support from Almirall; funding from Agenzia Italiana del Farmaco (AIFA), Alexion, Associazione Italiana per la lotta alle Sindromi Atassiche (AISA), Friedreich Ataxia Research Alliance (FARA), Roche SpA, Tristan Allamby Research Fund (TARFfa); and was a clinical trial principal investigator from Alexion, argenx, Dianthus, Immunovant, Leditant, Lexeo, Novartis, Prilenia, RemeGen, and Sanofi. TV has served on advisory board and/or as consultant for Alexion, AstraZeneca Rare Disease, argenx, Amgen, Dianthus, Johnson & Johnson, ImmunAbs, NMD Pharma, and VOR; served as a clinical trial principal investigator for Alexion, AstraZeneca Rare Disease, Amgen, argenx, Cartesian, COUR, Dianthus, EMD Seono, ImmunAbs, Immunovant, Johnson & Johnson, NMD Pharma, Regeneron, VOR, and UCB; and participated in CME events sponsored by AcademicCME, CMEO, PER, and MedLive. SR, AMR, and SP are employees of Alexion, AstraZeneca Rare Disease, and hold stock or stock options in AstraZeneca. AAL has served as a consultant for argenx, Cartesian, and Johnson & Johnson.

EPO-0153 | Compound POMGNT1 variants associated with muscle-eye-brain disease: Necessity of comprehensive genetic testing

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Background and aims: Congenital muscular dystrophies (CMD) are a heterogeneous group of rare autosomal recessive myopathies. Subtypes like dystroglycanopathies, collagen VI-related dystrophies, and dystroglycan–sarcoglycan complex disorders pose significant diagnostic challenges due to overlapping ocular and neurological involvement.

Methods: We report a 32-year-old male patient presenting in a comatose and febrile state following status epilepticus. Neurological examination revealed spastic paraparesis with bilateral clonus and knee contractures, right-arm hypotonia, and horizontal nystagmus. His history included global neurodevelopmental delay with subsequent loss of ambulation and speech, drug-resistant epilepsy, and ophthalmological abnormalities including bilateral optic atrophy, severe refractive error, and cataract.

Results: Brain imaging demonstrated ventriculomegaly, lissencephaly and brainstem and cerebellum hypoplasia (Figure 1). Electroencephalography confirmed refractory epileptic encephalopathy, which ultimately responded to a four-drug antiepileptic regimen (valproate sodium, brivaracetam, zonisamide, lacosamide). Muscle biopsy revealed myopathic changes with reduced α -dystroglycan, laminin $\alpha 2$, and laminin $\beta 2$ expression on immunohistochemistry. Next-Generation-Sequencing identified compound heterozygosity in POMGNT1, comprising a nonsense variant (c.1282C>T, p.Gln428*) and a missense variant (c.793C>T, p.Arg265Cys) (Figure 2). The missense variant is predicted to preserve partial enzymatic activity and likely contributes to the patient's phenotype.

Conclusion: Combined genetic and histopathological results were consistent with Muscle-Eye-Brain disease (MEB), a dystroglycanopathy within the CMD spectrum. Over one-third of MEB patients have epilepsy, approximately 20% of which suffer from refractory seizures. This case illustrates how compound heterozygosity with residual POMGNT1 activity permits prolonged survival, while predisposing to severe adult-onset neurological



FIGURE 1 T1-weighted MRI imaging of the brain showing brainstem and cerebellar hypoplasia, ventricular enlargement and lissencephaly predominantly involving the frontal lobes (red arrows).

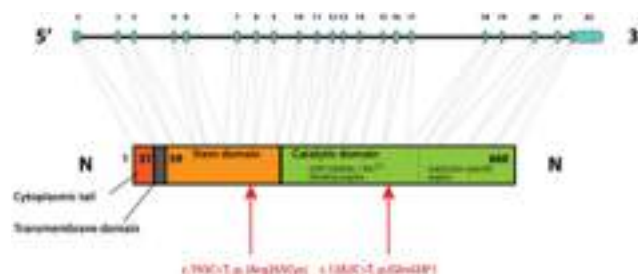


FIGURE 2 POMGNT1 gene and the corresponding protein with its main domains; the variants found in the present case are shown with red arrows.

complications, necessitating extensive genetic testing to resolve complex neuromuscular phenotypes.

Disclosure: The authors report no conflicts of interest or external funding.

EPO-0154 | Diagnostic clues: A multidisciplinary review of 600 cases with suspected inherited white matter disorder

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Background and aims: Adult patients with suspected inherited white matter disorders (IWMDs) present diagnostic challenges due to phenotypic overlap with acquired inflammatory, vascular, and toxic leukoencephalopathies. Delayed or incorrect classification may result in inappropriate treatment, missed therapeutic windows, and prolonged diagnostic odysseys. Multidisciplinary team (MDT) evaluation integrating clinical features, neuroimaging, cerebrospinal fluid (CSF) analysis, genetics, and longitudinal disease evolution is increasingly recognised as central to accurate diagnosis; however, real-world data quantifying which features most reliably contribute to diagnostic confidence remain limited.

Methods: We performed a retrospective review of approximately 600 consecutive cases discussed at a tertiary Inherited White Matter Disorders MDT. Clinical, radiological, laboratory, and genetic data were extracted from our IWMD database supplemented with up to date electronic health records. Variables included presenting phenotype, age at onset, family history, MRI pattern and distribution, CSF findings, genetic testing results, and use of ancillary investigations (e.g. brain biopsy). Cases were classified as inherited, acquired, in work-up, or unresolved following MDT consensus.

Results: Preliminary analysis demonstrates that no single clinical, radiological, or laboratory feature reliably distinguishes inherited from acquired white matter disease in isolation. Diagnostic confidence most commonly arose from the convergence of multiple domains, particularly characteristic MRI patterns, absence of inflammatory CSF markers, multisystem features, and longitudinal disease evolution. Brain biopsy was infrequently contributory and reserved for diagnostically ambiguous cases.

Conclusion: These findings highlight the value of integrated MDT assessment in complex adult white matter disorders and may inform more efficient diagnostic pathways, reducing unnecessary invasive investigations and supporting earlier recognition of genetic disease.

Disclosure: Nothing to disclose.

EPO-0155 | Miglustat in Alzheimer's disease associated with heterozygous NPC1 mutation: Exploratory case series and preliminary findings

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Background and aims: Several studies have previously demonstrated an increased risk of dementia and brain amyloid deposition in individuals with heterozygous NPC1 mutations. Moreover, in a recent study, we identified the first family with autosomal dominant late-onset Alzheimer's disease (AD) caused by a heterozygous NPC1 mutation. Unfortunately, there are currently no effective treatments available for this condition. Miglustat, which impacts the metabolism of oxysterols, has been shown to exert an anti-amyloidogenic effect in a human cellular model of AD.

Methods: In our exploratory uncontrolled study, three patients from the previously published family were orally treated with miglustat for 12 months. They underwent monthly clinical evaluations and routine blood tests. Additionally, neuropsychological evaluations, brain amyloid-PET imaging, and biochemical analyses on plasma and CSF were performed.

Results: All three patients achieved clinical stability, showed a sustained reduction in serum oxysterol levels, and experienced a marked decrease in brain amyloid burden.

Conclusion: Based on our preliminary observations and hypothesis-generating findings, along with the growing evidence suggesting AD as a lipid disorder, miglustat should be further tested in a larger cohort of heterozygous NPC1 mutated patients and probably evaluated as a potential disease-modifying treatment for AD.

Disclosure: H.Z. has served on scientific advisory boards and/or as a consultant for Abbvie, Acumen, Alector, Alzinova, ALZPath, Amylyx, Annexon, Apellis, Artery Therapeutics, AZTherapies, Cognito Therapeutics, CogRx, Denali, Eisai,

LabCorp, Merry Life, Nervgen, Novo Nordisk, Optoceutics, Passage Bio, Pinteon Therapeutics, Prothena, Red Abbey Labs, reMYND, Roche, Samumed, Siemens Healthineers, Triplet Therapeutics, and Wave, has given lectures sponsored by Alzecure, BioArctic, Biogen, Cellectricon, Fujirebio, Lilly, Novo Nordisk, Roche, and WebMD, and is a co-founder of Brain Biomarker Solutions in Gothenburg AB (BBS), which is a part of the GU Ventures Incubator Program (outside submitted work). None of the other authors declare conflicts of interest.

EPO-0156 | Predicting neurodevelopmental outcome in KCNQ2-related disorders

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Background and aims: KCNQ2-related disorders (RD) are a major cause of neonatal-onset epilepsy, presenting a spectrum from self-limited (familial) neonatal epilepsy (SeL(F)NE) to severe developmental and epileptic encephalopathy (DEE). Haploinsufficient variants typically cause milder SeL(F)NE, whereas dominant-negative variants often lead to DEE. Yet, clinical outcomes are variable, neonatal seizures occur across phenotypes, and functional data are often unavailable, complicating early prognostic assessment.

Methods: We performed a multicentric retrospective cohort study including patients with a (likely) pathogenic KCNQ2 variant and ≥ 3 years follow-up. Mosaic variants were excluded, yielding 235 patients. Ten neonatal predictors were selected based on statistical significance ($p \leq 0.01$), clinical relevance, and data availability. The cohort was randomly split into a training set (70%) and an independent validation set (30%). Three random forest models were trained using these predictors to forecast neurodevelopmental delay, age at first words, and age at walking.

Results: Six clinical predictors were retained: neonatal hypotonia, family history of neonatal seizures, EEG features, age at seizure onset, seizure type, and seizure frequency at onset. Four genetic predictors included exon location, truncating or de novo variant, and localization within KCNQ2-DEE hotspot regions. Models predicted neurodevelopmental outcome (normal, mild, moderate to profound intellectual disability), age of language acquisition (< 16 months, ≥ 16 months, never), and age of walking (< 18 months, ≥ 18 months, never) with validation accuracies of 74%, 80%, and 81%, respectively.

Conclusion: These prediction models offer a practical tool for early, individualized prognostic counselling in KCNQ2-RD, supporting patient stratification for early interventions, including sodium channel blockers, and guiding enrolment in precision medicine trials.

Disclosure: Nothing to disclose.

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Background and aims: Deep intronic GAA repeat expansions in intron 1 of the FGF14 gene have recently been identified as a frequent cause of late-onset cerebellar ataxia (LOCA), known as GAA-FGF14-related ataxia (SCA27B). While SCA27B is increasingly recognized worldwide, data from South America remain scarce and limited to isolated reports.

Methods: We report the clinical, neuropsychiatric, neuroimaging, and genetic findings of a patient evaluated for progressive cerebellar ataxia. Molecular analysis of FGF14 was performed using PCR-based techniques to identify GAA repeat expansions.

Results: We report a 62-year-old man with Spanish and Italian ancestry, carrying a pathogenic FGF14 GAA repeat expansion of 331 repeats. Originally referred due to paroxysmal ataxia and dysphonia, in the context of a long-standing neuropsychiatric history and prolonged alcohol and substance abuse. Neurological examination revealed progressive cerebellar ataxia, multidirectional nystagmus, dysphonia, dysphagia, and autonomic dysfunction, including bladder urgency and orthostatic hypotension. Family history showed a maternal diagnosis of amyotrophic lateral sclerosis. Early clinical features and personal history led to an initial diagnosis of alcohol-related cerebellar degeneration. Brain MRI showed nonspecific cerebellar atrophy. Subsequent progression to a pancerebellar ataxia-plus syndrome and a marked symptomatic response to 4-aminopyridine prompted genetic testing, confirming SCA27B.

Conclusion: This case represents, to our knowledge, the first genetically confirmed report of SCA27B in Argentina, expanding the phenotypic spectrum of SCA27B by highlighting concomitant neuropsychiatric manifestations and vulnerability to alcohol use as early disease features. In regions where alcohol-related cerebellar ataxia is frequently diagnosed, careful phenotypic assessment may facilitate recognition of treatable late-onset ataxias such as SCA27B

Disclosure: Nothing to disclose.

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Background and aims: Genome-wide association studies (GWAS) have substantially contributed to elucidating the genetic architecture of Parkinson's disease (PD). The present study investigated the burden of susceptibility variants associated with sporadic PD in the Italian population, with the goal of identifying specific genetic susceptibility profiles and candidate biomarkers.

Methods: A total of 180 Single Nucleotide Polymorphisms (SNPs) were analysed in 730 Italian patients with sporadic PD using OpenArray technology. Variants were selected from GWAS, meta-analyses, public databases and PD-related biological pathways. Case-control association analyses with multiple testing correction and odds ratio estimation were integrated with machine-learning and bioinformatic analyses.

Results: The study identified 72 SNPs significantly associated with PD susceptibility. Among the tested machine learning models, the neural network showed the best performance and identified five variants (rs2012070, ATP6V0A1; rs1800795, IL6; rs329652, MIR4697; rs2641116, PARK7; rs9275326, HLA-DRB6/HLA-DQA1) as the most predictive. eQTL analysis revealed predominant regulatory effects of six PD-associated variants in the basal ganglia, cerebellum, cerebral cortex, hippocampus and hypothalamus regions of the brain. Pathway enrichment analysis indicated that genes harbouring the associated variants are involved in PD-relevant biological processes, including immune and inflammatory response, neurodevelopment, synaptic function, protein degradation, cellular metabolism, membrane organization and intracellular trafficking, as well as regulation of neuronal survival and cell death.

Conclusion: These findings delineate a distinct genetic susceptibility profile for PD in the Italian population and provide a rationale for developing a population-tailored genetic card as a tool to support risk stratification and identify individuals who may benefit from closer monitoring.

Disclosure: Nothing to disclose.

EPO-0159 | Landscape of dystrophin variants in Hungary

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Background and aims: Dystrophinopathies are one of the most important causes of muscular dystrophies in boys. With the advent of targeted therapies, it became extremely important to know the exact molecular background of each patient.

Methods: We collected molecular results of patients across the country. We collected date of birth, date of diagnosis, and the genetic results from all patients. Cases, first tested by multiplex PCR, were retested with MLPA. For a subset of patients (cared at the Semmelweis University), we collected phenotypic data.

Results: Between 1992 and 2025, 558 DMD molecular tests were performed. Formerly, only copy number variant (CNV) analysis was easily available, but since 2020 single nucleotide variant (SNV) analysis is also more accessible. 169 in-frame CNV (30.3%), 306 out-of-frame CNVs (54.8%), and 55 SNVs (9.9%) were detected. Among the out-of-frame CNVs, 115 different events occurred: in 9.3% duplication, and in 90.7% deletion. Most common events were del45-50 (8% of cases), del45 (6.5%), and del48-50 (6.5%). The number of theoretically amenable patients for exon skipping therapy (exon skipping 45/51/53) would be 122 (39.9%). We have available phenotype data from 109 patients in the whole cohort, with 43 Becker and 66 Duchenne phenotypes.

Conclusion: Routine availability of CNV and SNV testing, including NGS, allows reliable incidence estimates and reveals an increasing number of rare damaging variants and clinically affected females. Twelve common variants accounted for half of the cases, and a substantial proportion of patients are theoretically eligible for targeted therapies.

Disclosure: First author received support for congress attendance from the Medis, Medical Marketing Company.

EPO-0160 | Hereditary spastic paraparesis type 18: Shedding light upon a novel mutation in the ERLIN2 gene

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Background and aims: Hereditary spastic paraparesis (HSP) type 18 is caused by mutations in the ERLIN2 gene, which encodes a protein involved in the endoplasmic reticulum-associated degradation pathway. Autosomal recessive mutations are involved in complicated HSP, while autosomal dominant mutations present as pure HSP. We describe a case of a new mutation in the ERLIN2 gene.

Methods: A 60-year-old woman who developed progressive gait disturbance since the age of 16. At 50 years old, she also presented weakness in the lower limbs. Her maternal grandfather, a maternal great-aunt, and two second-degree cousins had gait abnormalities. However, her non-consanguineous parents, two siblings, and one daughter had no neurological symptoms. On examination, muscle strength 4/5 in lower limbs with spasticity, hyperreflexia, Babinski sign, and bilateral clonus were observed. Modified Ashworth Scale: 3, and Timed 25-Foot Walk Test (T25FW): 24 s.

Results: Brain and spinal MRI: normal. Whole exome sequencing (WES) identified the index case as a heterozygous carrier of the c.205G > C (p.D69H) mutation in the ERLIN2 gene. Previous treatments with oral baclofen, tizanidine, and botulinum toxin showed minimal response. Extended-release fampridine (10 mg b.i.d.) was administered as a therapeutic alternative.

Conclusion: The proband case confirms the genotype-phenotype correlation between monoallelic variants of the ERLIN2 gene and pure HSP. This finding suggests a potential dominant negative effect of the erlin2 protein on oligomerization, as the cause of a milder phenotype. Furthermore, this case introduces a new variant in the ERLIN2 gene, contributing to a better understanding of the genetic factors underlying HSP type 18.

Disclosure: Nothing to disclose.

EPO-0161 | Abstract withdrawn

EPO-0162 | Investigating natural history and genotype influence in ALS2-related infantile-onset ascending hereditary spastic paraplegia

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Background and aims: Infantile-onset ascending hereditary spastic paraplegia (IAHSP) is a rare motor neuron disorder caused by biallelic ALS2 mutations. Despite a distinctive phenotype, quantitative data on disease progression and genotype-phenotype correlations remain limited, hindering clinical trial design.

Methods: We conducted a natural history study of 83 individuals with biallelic-ALS2 mutations, combining published cases with patients enrolled in a multicenter cohort. Collected variables included psychomotor development, age at onset of lower-limb, upper-limb and bulbar involvement, loss of independent ambulation, additional neurological features. Based on predicted residual protein function, patients were classified into three genotype groups: biallelic-loss-of-function{00},

mixed{01}, non-loss-of-function{11}. Disease progression was analyzed using Kaplan–Meier curves and Cox proportional hazards models. Functional progression was assessed with the SPATAX-disability score, allowing longitudinal modeling and preliminary sample size estimation. Unsupervised clustering was applied to identify phenotypic subgroups.

Results: Age at symptom onset was consistent across genotypes (median 1.4–1.6 years), identifying early lower-limb involvement as a genotype-independent hallmark of IAHS. In contrast, progression rates differed by genotype: {00}-patients showed faster and more homogeneous progression, {01}-patients an early but more gradual course, and {11}-patients the slowest progression. Longitudinal SPATAX analyses indicated a mean progression rate ~0.5 points/year. Based on this estimate, preliminary modeling suggested that approximately 10 patients may be sufficient to detect a 50% reduction in progression in an interventional trial. Unsupervised clustering identified three preliminary phenotypic groups that did not map one-to-one onto genotype classes, suggesting additional sources of clinical heterogeneity currently under further investigation.

Conclusion: These data support time-based endpoints and data-driven stratification for IAHS trials.

Disclosure: Nothing to disclose.

EPO-0163 | Applicability of PPCS as diagnostic biomarker for Niemann Pick type C2 patients

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Background and aims: N-palmitoyl-O-phosphocholineserine (PPCS) formerly known as Lyso-SM-509 has been shown to be a diagnostic marker for Niemann Pick type C1 (NPC1) with high sensitivity and specificity (Griese 2015). It has been reported to correlate in NPC1 patients with the annual severity increment score (Sidhu R 2020) and with disease severity (clinical presentation) and progression (measured in dried blood spot - DBS - samples) (Guatibonza 2023). So far, this biomarker has only been investigated for NPC1 patients. For NPC2 patients however, biomarker data is not yet publicly available since pathogenic NPC2 variants are accountable for only 5 percent of all NPC cases which corresponds to an incidence rate of only 1 of 2,858,998 (Wassif 2016).

Methods: With this work, we aim to establish a PPCS reference range for NPC2 patients based on data that has been acquired in our laboratories over 10 years from DBS samples of 33 patients.

Results: All patients were characterized genetically with the predominant variants being p.Cys47Ter, p.Cys99Arg, and p.Ser63fs. The PPCS blood levels of all NPC2 patients were above the diagnostic cut-off and hence above the 97th percentile of values measured in blood of healthy individuals. Furthermore, clinical data was dissected using HPO terms, and genotype-phenotype analysis was performed.

Conclusion: This work should serve as evidence for the applicability of PPCS as diagnostic biomarker for NPC2 and should be the starting point for investigation of its use as monitoring biomarker in treatment efficacy studies.

Disclosure: Steffen Fischer, Luba Pardo, Aida Bertoli-Avella and Peter Bauer are employees of Centogene GmbH.

EPO-0164 | Genetic, biochemical and clinical properties of GLA p.Asp313Tyr variant in a large global cohort – No indication of Fabry disease

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Background and aims: Fabry disease (FD) is a multisystemic disorder caused by pathogenic variants in the GLA gene. Premature stroke and neuropathic pain are typical for neurological involvement. The disease-causing properties of the GLA variant c.937G > T (p.Asp313Tyr), reclassified from “Variant of uncertain significance” to “Benign” by our lab, are still debated. Recently, we demonstrated no specific phenotype for this variant in a large German cohort. To confirm the data globally we extended our evaluation.

Methods: Leveraging Centogene’s Biodatabank we analyzed all samples (n = 154,447) molecularly tested for Fabry disease during 2015 and 2020 from 72 countries for GLA genotypes, clinical presentations, and biochemical phenotypes.

Results: The allele frequency of GLA variant p.Asp313Tyr in this cohort was increased compared to gnomAD v4.1.0 data resulting in an odds ratio of 3.77 (95% CI: 3.61–3.94; p < 0.0001). Again, there

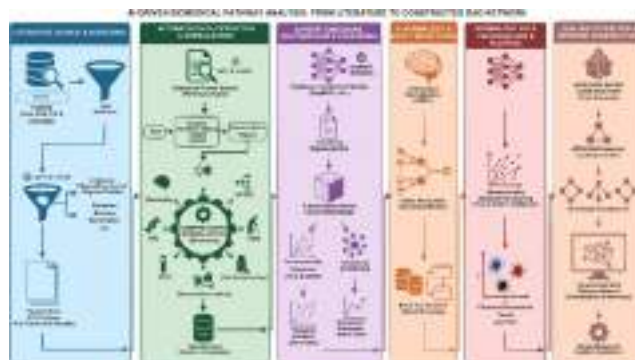


FIGURE 1

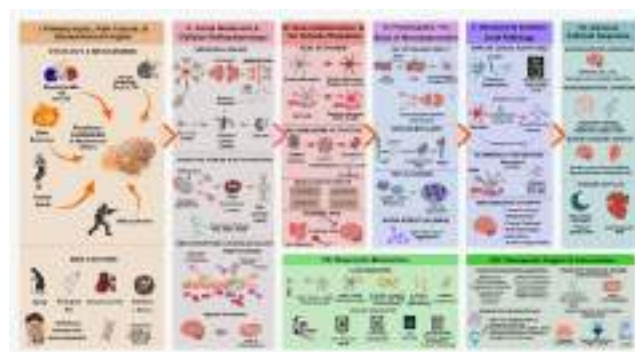


FIGURE 2 Integrated evidence.

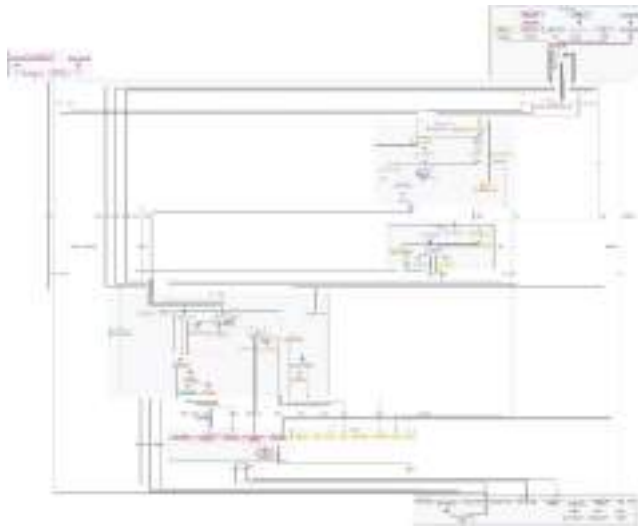


FIGURE 3 Clinical evidence.

was no specific clinical nor biochemical phenotype compared to GLA reference individuals, supporting a diagnosis of FD.

Conclusion: The GLA variant p.Asp313Tyr is classified as a benign variant and does not cause FD. Neither clinical symptoms nor biochemical characteristics evidenced a specific effect in our cohort. The fact that the allele frequency here is higher than expected from general population reference data and publications describing a higher prevalence of GLA p.Asp313Tyr in patients with peripheral nervous system involvement or cerebrovascular disease, demand further clarification of a potential role as risk factor for a microangiopathic/neurological dysfunction, possibly comparable to the role of GBA1 variants in Parkinson's disease, must be elucidated. Therefore, properly designed case-control studies are needed.

Disclosure: No support has been received to conduct the study. T.B, C.B, E.Z, S.S, A.K, J.P.B. and P.B. are employees of Centogene GmbH.

Ageing and dementia 2

EPO-0165 | Mapping causal pathways from traumatic brain injury to dementia a systematic synthesis with computational inference

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Background and aims: Traumatic brain injury (TBI) represents a critical global health burden, and serves as an environmental risk factor for neurodegenerative diseases, and increasing dementia risk. However, the mechanistic heterogeneity linking acute injury to chronic neurodegeneration remains poorly integrated across the vast clinical and preclinical literature.

Methods: 1,347 abstracts screened using a validated prompt engineering system, extracting structured biomedical triples from 823 relevant studies. Semantic path analysis was performed

using SapBERT embeddings, dimensionality reduction (PCA-UMAP), and hierarchical clustering (HDBSCAN) to construct directed acyclic graphs (DAGs) of causal evidence.

Results: Our analysis reconstructed the causal map of the post-TBI events leading to dementia, delineating distinct phases of pathogenesis. Primary axonal degeneration is driven by SARM1-mediated NAD⁺ depletion and calpain-dependent spectrin proteolysis, distinct from classical apoptosis. This transitions into a chronic neuroinflammatory phase characterized by HMGB1-TLR4 signaling, NF-κB activation, and the assembly of the NLRP3 inflammasome, which drives gasdermin-D mediated pyroptosis. We identified critical failures in clearance mechanisms, specifically AQP4 mislocalization leading to glymphatic dysfunction, which exacerbates the accumulation of hyperphosphorylated cis-tau, amyloid-beta, and TDP-43. Furthermore, our synthesis highlights the integrated stress response (ISR) and mitochondrial fission (Drp1) as central metabolic drivers of synaptic failure. The constructed causal maps identify specific therapeutic targets, including SARM1 inhibitors, NLRP3 antagonists, and senolytics, and validate fluid biomarkers such as NfL and GFAP for prognostic stratification.

Conclusion: This computational synthesis bridges the gap between molecular reductionism and clinical phenomenology, offering a unified framework for precision intervention in TBI-associated dementia.

Disclosure: Nothing to disclose.

EPO-0166 | Regional amyloid-PET dynamics in patients treated with anti-Aβ monoclonal antibody therapy

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Background and aims: Anti-amyloid-β monoclonal antibodies (AAA) have demonstrated substantial reductions in cerebral amyloid burden in early symptomatic Alzheimer's disease (AD). However, interpretation of amyloid PET changes largely relies on global metrics that may mask regional heterogeneity of amyloid clearance.

Methods: We analyzed real-world amyloid PET data from 19 patients treated with Lecanemab or Donanemab who completed six months of therapy. Global Centiloid values and regional standardized uptake value ratios were quantified at baseline and follow-up using [18F]flutemetamol or [18F]florbetaben. Regional amyloid changes were examined according to treatment, clinical diagnosis, and clinical disease stage using ANCOVA models adjusted for tracer and AAA.

Results: Amyloid reduction showed pronounced regional variability, with the largest decreases in anterior cingulate, caudate and precuneus, and smaller changes in mesial temporal and occipital regions. Patients with early-onset AD showed higher baseline amyloid burden than those with late-onset disease but similar longitudinal reductions. Despite comparable baseline amyloid burden, patients with mild cognitive impairment demonstrated greater cortical amyloid reduction than patients with mild dementia. Among patients reaching global amyloid positivity thresholds, residual regional uptake—most consistently in the occipital cortex—was frequently observed.

Conclusion: Early amyloid clearance following AAA therapy is regionally heterogeneous and incompletely captured by global amyloid PET measures.

Disclosure: A Ghirelli, EG Spinelli, AM Samanes Gajate, G Rugarli, S Pisano, V Castelnovo, E Sibilla, A Gilioli, C Tripodi F Freri, A Panzacchi, and G Pepe have nothing to disclose. Giordano Cecchetti received speaker honoraria from Neopharmed Gentili. Elisa Canu receives research support from the Italian Ministry of Health. A. Chiti reports consulting or advisory role for Blue Earth Diagnostics, Telix Pharmaceuticals, InnovaRadi Therapeutic, and General Electric Healthcare; and Speaker's Bureaus for Bracco Diagnostics, General Electric Healthcare, Novartis, Telix Pharmaceuticals, and United Imaging; he is Editor in Chief of The EANM Journal. M Filippi is Editor-in-Chief of the Journal of Neurology, Associate Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmed Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. F Agosta is Associate Editor of NeuroImage: Clinical and the European Journal of Neurology; has received speaker honoraria from Biogen Idec, Bristol Myers Squibb, Eisai, Eli Lilly, GE Healthcare, Neuraxpharm, and Roche; and receives or has received research supports from the Italian Ministry of Health, the Italian Ministry of University and Research, AriSLA (Fondazione Italiana di Ricerca per la SLA), the European Research Council (ERC), the EU Joint Programme – Neurodegenerative Disease Research (JPND), and Foundation Research on Alzheimer Disease (France).

EPO-0167 | Family history and associated risk factors in patients with dementia with Lewy bodies

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Background and aims: Dementia with Lewy bodies (DLB) is one of the most common neurodegenerative diseases. Its aetiology and possible risk factors remain largely unclear. Evidence on whether general family history of dementia confers higher risk of DLB remains scarce. We aim to assess whether family history of dementia is associated with increased risk of DLB. We explore inheritance patterns (maternal, paternal, and siblings), and possible link to consanguinity.

Methods: We conducted a tertiary hospital-based cohort study with 1836 participants, including 152 patients with DLB, 843 patients with Alzheimer's disease (AD) and 841 sex- and age-matched cognitively healthy controls over 75 years old. Participants were questioned regarding family history of dementia (maternal, paternal, siblings). Parental birthplace and history of consanguinity was also registered. Binary logistic regressions and comparative analyses were performed between DLB, AD, and controls.

Results: In the total 1836 participants, 152 had DLB, with mean current age 80.2 years (SD: 5.9), mean onset age 74.3 years (SD: 6.2), and 62.1% were women. We found that having a sister with dementia was associated with higher odds of DLB compared with controls (OR 51.33, 95% CI=20.02–131.65) and AD (OR 2.61, 95% CI=1.66–4.10). Higher odds of DLB were also associated with maternal dementia history (OR 1.89, 95% CI=1.16–3.08) versus controls.

Conclusion: This study provides new evidence that maternal and sibling family history of dementia may be associated with an increased risk of DLB, refining our understanding of a possible heritable architecture, highlighting the importance of detailed family history in clinical assessment and risk stratification.

Disclosure: Nothing to disclose.

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Background and aims: The Boston Criteria v2.0 integrates clinical-radiological features to diagnose Cerebral Amyloid Angiopathy (CAA), with deep hemorrhagic manifestations, the so-called mixed small vessel disease (SVD), precluding a CAA diagnosis, given the high likelihood of underlying hypertensive SVD. This study aims to evaluate whether the A β 42/40 ratio outperforms isolated biomarkers (A β 42, A β 40) as a proxy of neuropathology in a heterogeneous SVD cohort.

Methods: We analyzed a cohort of patients with probable CAA (Boston Criteria v2.0) or mixed SVD with available CSF biomarkers (A β 42, A β 40). We quantified MRI markers of SVD, including cerebral microbleeds (CMB), cortical superficial siderosis (cSS), enlarged perivascular spaces (EPVS), and the CAA-SVD score. Associations between MRI scales and Z-score normalized CSF biomarkers were evaluated using logistic regression models.

Results: We included 79 patients (58 probable CAA, 21 mixed SVD). A β 40 showed a significant association with cSS (OR = 0.54; p = 0.005), A β 42 with cSS (OR = 0.40; p < 0.001), CAA-SVD score (OR = 0.55; p = 0.003), and deep EPVS (OR = 2.45; p < 0.001), while the A β 42/40 ratio was associated with cSS (OR = 0.54; p = 0.010), CAA-SVD score (OR = 0.63; p = 0.021), deep EPVS (OR = 2.42; p < 0.001), and deep CMBs (OR = 3.29; p < 0.001). The only CSF biomarker that showed a bimodal distribution was the A β 42/40 ratio.

Conclusion: In our cohort, lower levels of A β 42 and A β 40 are closely linked to specific CAA features. However, the A β 42/40 ratio gains more reliability in relation to deep hemorrhagic markers. This suggests, given also the binomial distribution, that the A β 42/40 ratio may better differentiate the underlying SVD compared to isolated CSF biomarkers. This should be confirmed in further studies, verifying also the diagnostic accuracy of a biology-integrated approach compared to the current radiological classification.

Disclosure: Nothing to disclose.

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Background and aims: Epilepsy is common in neurodegenerative diseases, particularly Alzheimer's disease (AD). However, the impact of amyloid- β on epilepsy prevalence in clinical populations remains uncertain, as does the association between epilepsy and specific cognitive profiles or brain atrophy. This study aimed to compare cognitive characteristics, AD cerebrospinal fluid (CSF) biomarkers, and brain morphometry between patients with neurodegenerative diseases with and without epilepsy. Additionally, we examined whether epilepsy prevalence differs between patients with AD (as confirmed by CSF biomarkers) and patients with non-Alzheimer's neurodegenerative diseases (Non-AD).

Methods: In a cross-sectional study, we retrospectively analyzed well-characterized patients in a University Memory Clinic cohort (Leenaards Memory Center; Lausanne University Hospital, Switzerland). CSF biomarkers, global cognitive functioning, dominant impaired cognitive domain, brain morphometry, epilepsy diagnosis and EEG report were assessed. Patients were classified using the ATN system to differentiate AD from other neurodegenerative conditions (Non-AD).

Results: In a Memory Clinic cohort (n = 479, age: 69.1 $\pm$ 9.2, 44% female), no significant differences were found in either cognitive impairment, brain morphometry, CSF amyloid- β and phosphorylated Tau levels or ATN classification between patients with or without epilepsy. In addition, epilepsy prevalence was comparable between AD and Non-AD groups (5.2% and 5.4% respectively, p = 0.965).

Conclusion: Epilepsy was not associated with a distinct cognitive, morphometric, AD CSF biomarker or ATN profile. Importantly, Alzheimer's disease did not correlate with an increased prevalence of epilepsy. These findings point toward mechanisms beyond amyloid- β - potentially shared across neurodegenerative diseases - as contributors to epileptogenesis, emphasizing the need for further targeted research.

Disclosure: Gilles Allali was supported by a grant from the Swiss National Science Foundation (grant number # 214855), from the Leenaards Foundation, the Solis Foundation, the Empiris Foundation, the Marina Cuennet-Mauvernay Foundation, the Faculty of Biology and Medicine of the University of Lausanne.

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Background and aims: Limbic-predominant age-related TDP-43 encephalopathy (LATE) is a recently recognised neurodegenerative disease with clinical characteristics, cognitive deficits, and structural brain changes similar to those seen in Alzheimer's disease (AD). The diagnosis of LATE is challenging due to the absence of validated specific biomarkers. This study evaluated diagnostic and clinical usefulness of non-invasive biomarkers in a memory clinic cohort, exploring the potential of these biomarkers as tools for the early diagnosis of LATE and for differentiating it from AD.

Methods: 83 participants from the Czech Brain Aging Study, including individuals with amnesic mild cognitive impairment (aMCI) and biomarker-confirmed AD ($n=27$), individuals with aMCI meeting criteria for probable LATE ($n=33$), and cognitively normal older adults (CN, $n=23$), underwent cognitive assessment, structural brain MRI, blood-based biomarker assessment, and genetic testing. Additionally, the aMCI participants underwent amyloid PET imaging and cerebrospinal fluid biomarker assessment.

Results: Participants with LATE aMCI outperformed AD aMCI and were similar to CN on visuospatial tests (Figure 1), exhibited greater reductions in entorhinal cortical thickness and a lower ratio of entorhinal-to-parahippocampal cortical thickness compared to AD aMCI and CN (Figure 2), and had lower plasma glial fibrillary acidic protein levels and a lower frequency of the APOE4 allele than AD aMCI, and similar to CN (Figure 3). A combination of these markers differentiated between LATE and AD aMCI with 86% sensitivity and 74% specificity.

Conclusion: A combination of specific non-invasive biomarkers may distinguish early LATE from early AD, providing moderate-to-high sensitivity and specificity. This makes them valuable tools for differential diagnosis in clinical settings.

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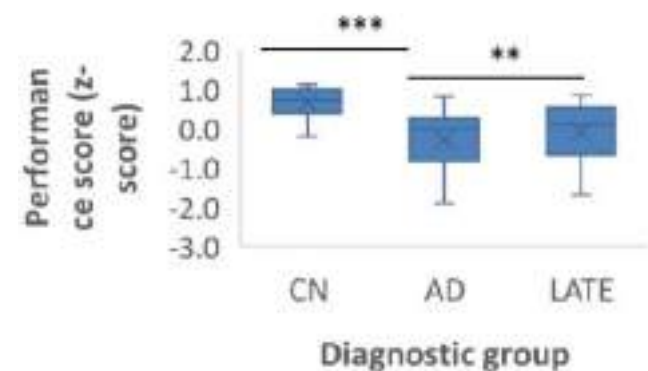


FIGURE 1 Between-group differences in visuospatial function.

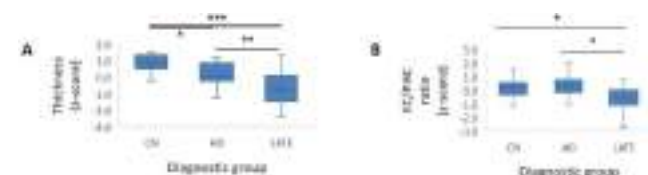


FIGURE 2 Between-group differences in A) entorhinal cortical thickness and B) entorhinal-to-parahippocampal cortical thickness ratio.

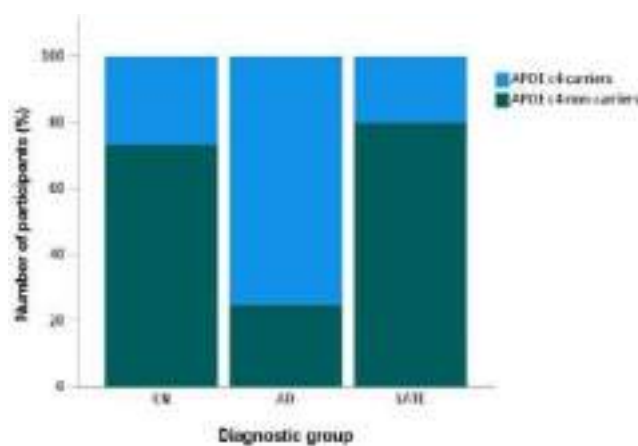


FIGURE 3 Between-group differences in the distribution of the APOE4 allele.

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EPO-0171 | GALECTIN-3 as an emerging biomarker of neuroinflammation in Alzheimer's disease and frontotemporal dementia

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Background and aims: Frontotemporal dementia (FTD) and Alzheimer's disease (AD) are the most common causes of dementia and neuroinflammation plays an important role in their pathogenesis. Elevated levels of Galectin-3 (a galactoside-binding lectin expressed by activated microglia, Gal-3), were reported elevated in the cerebrospinal fluid (CSF) of AD and FTD patients. This study aims to evaluate CSF Gal-3 levels in AD, FTD and patients without diagnosed neurodegenerative disorders (NND), and to investigate its relationship with established biomarkers of neurodegeneration.

Methods: CSF samples were collected from 118 patients recruited at Turin University Dementia Center: 51 with AD, 40 with FTD and 27 classified as NND. Gal-3 concentrations were measured using an Enzyme-Linked Immunosorbent Assay (ELISA) kit (Abcam, Cambridge, UK) while CSF Amyloid-beta-42 (A β 42), Amyloid-Beta-42/40 ratio (A β 40/42), total-tau (t-Tau), phosphorylated-tau-at-threonine-181 (p-Tau181), and neurofilament-light-chain (NFL) using a Lumipulse platform (Fujirebio, Ghent, Belgium).

Results: Mean Gal-3 levels were higher in the FTD group (562.1 ± 226.1 pg/mL) and in the AD group (473.1 ± 128.3 pg/mL) compared to the NND group (256.7 ± 164.6 pg/mL; $p < 0.001$). Gal-3 levels did not differ between the FTD and AD groups ($p = 0.064$). In the FTD subgroup Gal-3 showed a significant

correlation with t-Tau ($r=+0.33$, $p=0.045$), while in the overall population Gal-3 correlated with NfL ($r=+0.28$, $p=0.039$).

Conclusion: CSF Gal-3 levels were elevated in patients with AD and FTD, supporting the contribution of neuroinflammation to neurodegeneration and reinforcing Gal-3 as a potential biomarker. The correlation between Gal-3 and t-Tau further suggests a relationship between microglial activation and tau-related pathology.

Disclosure: Nothing to disclose.

EPO-0172 | Gut-brain communication via extracellular vesicles enables amyloid- β transport to the central nervous system

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Background and aims: Alzheimer's disease (AD) is defined by progressive neurodegeneration and cerebral deposition of amyloid- β (A β). While central amyloid pathology has been extensively studied, emerging evidence suggests that peripheral tissues may contribute to disease initiation and progression. However, the cellular pathways enabling amyloid proteins to traverse the gut-brain axis remain poorly defined.

Methods: Gut-to-brain transport was examined using complementary in vitro and in vivo models. A co-culture was established to model directional transport. Epithelial barrier integrity was confirmed using TEER measurements, Dextran-FITC permeability assays, and ZO-1 immunostaining. EV involvement in A β transport was assessed by tracking EV-associated A β transfer. Neuronal oxidative stress and cytotoxicity were quantified following exposure to free or EV-associated A β . In vivo validation was performed in zebrafish larvae.

Results: Intestinal barriers remained structurally and functionally intact across in vitro models. A β was preferentially packaged into EVs released by intestinal epithelial cells and subsequently internalized by neurons. Neurons exposed directly to free A β exhibited significantly higher oxidative stress and cytotoxicity compared to neurons receiving EV-associated A β through epithelial-neuronal interfaces. In vivo imaging demonstrated that A β delivered into the zebrafish gut accumulated in the brain.

Conclusion: These findings establish extracellular vesicles as critical mediators of amyloid- β transport from the gut to the brain via transcellular pathways, independent of intestinal barrier disruption.

Disclosure: Nothing to disclose.

EPO-0173 | Neurological perspectives in solid organ transplantation: Advancing ethical and equitable care for people with cognitive impairment

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Background and aims: Cognitive impairment (CI) affects a significant proportion of liver (up to 80%), kidney (up to 55%), lung (45%), and heart transplant candidates (25–74%). As organ failure increasingly affects older adults, the number of solid organ transplant candidates with CI is expected to increase. However, the lack of standardized cognitive assessments leads to variability across centers, potentially resulting in inequities in transplant accessibility and outcomes. Incorporating CI assessments into pre-transplant evaluations is therefore crucial to better predict post-transplant outcomes and to support ethically sound decision-making.

Methods: We reviewed the available literature on the debated issue of organ allocation for patients with CI, synthesizing arguments both in favor of and against transplantation, as well as proposed approaches to address these challenges.

Results: It has been recommended to establish independent regional boards to address allocation challenges and reduce the risk of discrimination against patients with CI. These boards should include transplant and non-transplant professionals (e.g. social workers, psychologists, bioethicists) and other relevant stakeholders (e.g. patients and family members) to enhance transparency and consensus. Integrating ethical assessments and review into pre-transplant evaluations may further support multidisciplinary discussions.

Conclusion: We propose that transplant eligibility for individuals with CI would be approached as a specialized clinical evaluation, conducted early in the transplant process by experts in cognitive disorders to assess factors relevant to perioperative and long-term outcomes, including severity, etiology, and prognosis of the CI; this clinical assessment should be systematically integrated with structured ethical evaluation to support fair and transparent decision-making.

Disclosure: The authors have nothing to disclose.

EPO-0174 | Plasma metabolic profiles predict future dementia and dementia subtypes: A prospective analysis of 274,160 participants

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Background and aims: Blood-based biomarkers for dementia are gaining attention due to their non-invasive nature and feasibility in regular healthcare settings. Here, we explored the associations between 249 metabolites with all-cause dementia (ACD), Alzheimer's disease (AD), and vascular dementia (VaD) and assessed their predictive potential.

Methods: This study included 274,160 participants from the UK Biobank. Cox proportional hazard models were employed to investigate longitudinal associations between metabolites and dementia. The importance of these metabolites was quantified using machine learning algorithms, and a metabolic risk score (MetRS) was subsequently developed for each dementia type. We further investigated how MetRS stratified the risk of dementia onset and assessed its predictive performance, both alone and in combination with demographic and cognitive predictors.

Results: During a median follow-up of 14.01 years, 5,274 participants developed dementia. Of the 249 metabolites examined, 143 were significantly associated with incident ACD, 130 with

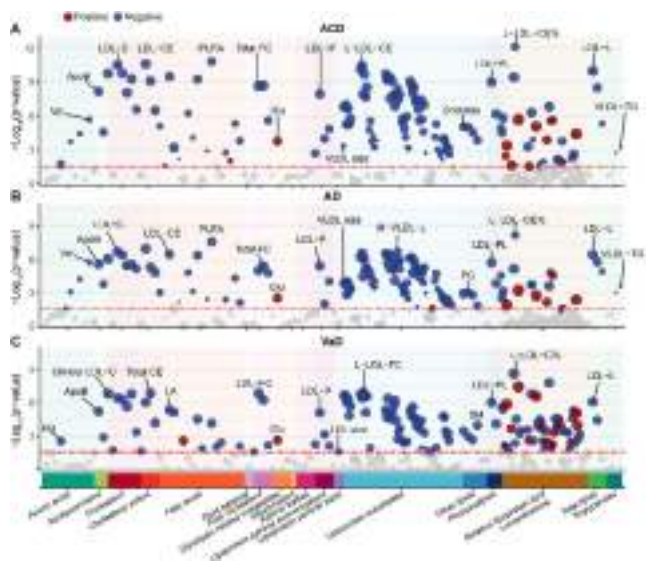


FIGURE 1 Dementia-associated metabolites in the association analysis.

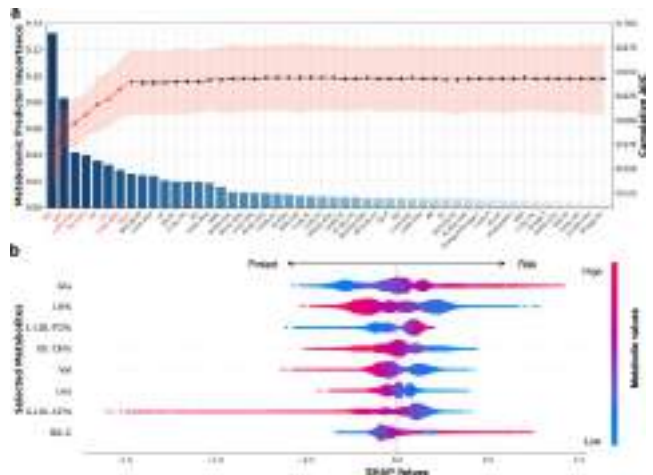


FIGURE 2 Metabolites importance ranking and SHAP visualization of modeling based on incident ACD populations.

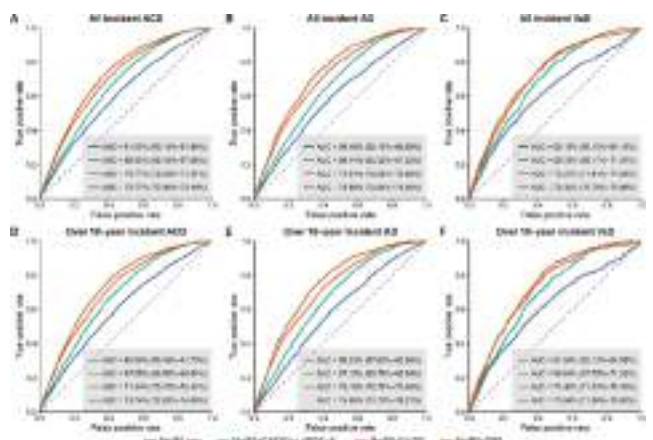


FIGURE 3 Prediction of incident ACD, AD, and VaD.

AD, and 140 with VaD. Among metabolites significantly associated with dementia, lipoprotein lipid concentrations, linoleic acid, sphingomyelin, glucose, and branched-chain amino acids ranked top in importance. Individuals within the top tertile of MetRS faced a significantly greater risk of developing dementia than those in the lowest tertile. When MetRS was combined with demographic and cognitive predictors, the model yielded the area under the receiver operating characteristic curve (AUC) values of 0.857 for ACD, 0.861 for AD, and 0.873 for VaD.

Conclusion: We conducted the largest metabolome investigation of dementia to date, for the first time revealed the metabolite importance ranking, and highlighted the contribution of plasma metabolites for dementia prediction.

Disclosure: Nothing to disclose.

EPO-0175 | Serum clinical laboratory tests and risk of incident dementia: A prospective cohort study of 407,190 individuals

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Background and aims: Prevention of dementia is a public health priority, and the identification of potential biomarkers may provide benefits for early detection and prevention.

Methods: This study investigates the association of common serum laboratory tests with the risk of incident dementia. Among 407 190 participants from the UK Biobank (median follow-up of 9.19 years), we investigated the linear and nonlinear effects of 30 laboratory measures on the risk of all-cause dementia using Cox models and restricted cubic spline models.

Results: We found that dementia incidence was associated with low vitamin D concentration (hazard ratio 0.994, 95% confidence interval 0.993–0.996), indicators of endocrine disorders: IGF-1 level (*P* for non-linearity=1.1E-05), testosterone level (*p* for non-linearity=0.006); high sex-hormone-binding globulin level

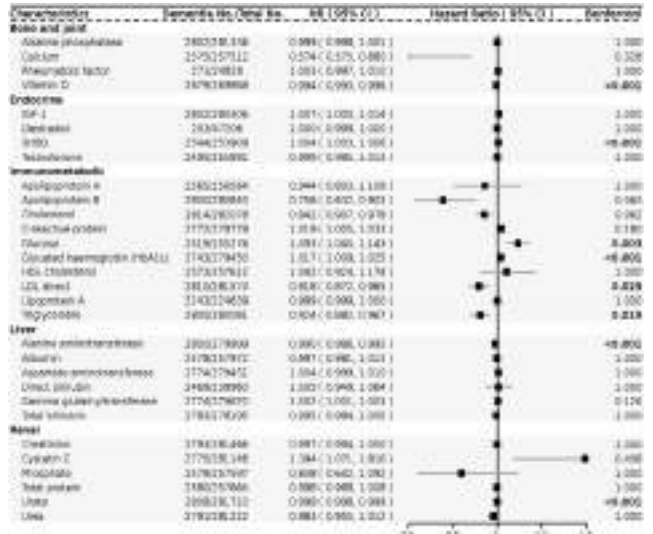


FIGURE 1 Linear associations between serum laboratory tests and risk of incident dementia.

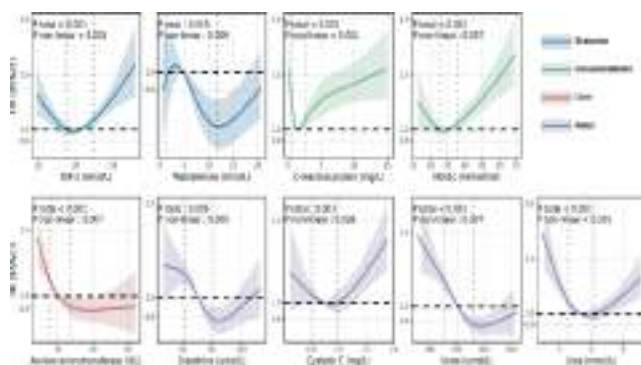


FIGURE 2 Nonlinear associations between serum laboratory tests and risk of incident dementia.

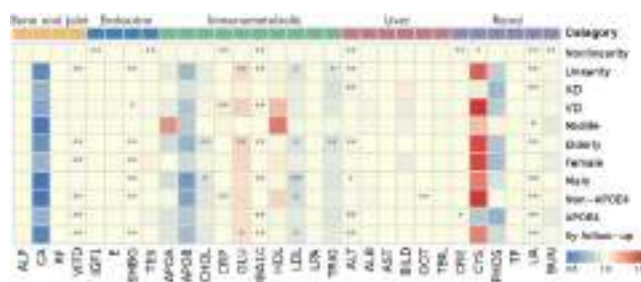


FIGURE 3 Heatmap to overview the associations of serum laboratory tests with dementia risks.

(HR 1.004, 95% CI 1.003–1.006); reduced liver function: lower alanine aminotransferase (HR 0.990, 95% CI 0.986–0.995); renal dysfunction: cystatin C level (p for non-linearity = 0.028); oxidative stress: lower urate level (HR 0.998, 95% CI 0.998–0.999); lipids dysregulation: lower LDL (HR 0.918, 95% CI 0.872–0.965) and triglycerides (HR 0.924, 95% CI 0.882–0.967) concentrations; insulin resistance: high glucose (HR 1.093, 95% CI 1.045–1.143) and HbA1c (HR 1.017, 95% CI 1.009–1.025) levels; immune dysbiosis: C–reactive protein (p for non-linearity = 5.5E-09).

Conclusion: In conclusion, markers of vitamin D deficiency, GH-IGF-1 axis disorders, bioactive sex hormone deficiency, reduced liver function, renal abnormalities, oxidation, insulin resistance, immune dysbiosis, and lipids dysregulation were associated with incident dementia. Our results support a contributory role of systemic disorders and diverse biological processes to onset of dementia.

Disclosure: Nothing to disclose.

Neurotoxicology/Occupational Neurology

EPO-0176 | Cannabis-induced PRES with acute cerebral infarctions involving the bilateral posterior territories and left thalamus

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Background and aims: Posterior Reversible Encephalopathy Syndrome (PRES) is an uncommon but clinically significant condition characterized by headache, visual disturbances, seizures,

altered mental status, and posterior white-matter changes. Cerebral infarction occurs in a minority of cases. Although many triggers have been described, cannabis-associated PRES remains rare.

Methods: Case report.

Results: A 36 year old patient was admitted after mixed alcohol and cannabis intoxication. On admission, he reported several days of headache, vertigo, and visual perceptual disturbances. Blood pressure was 161/91 mmHg. Due to markedly elevated urinary tetrahydrocannabinol levels and recent heavy cannabis use, symptoms were initially attributed to intoxication. Because neurological symptoms persisted, MRI was performed and revealed subacute infarctions in the bilateral posterior territories and the left thalamus, with PRES-compatible changes and minor bilateral haemorrhagic transformation. Transcranial and extracranial Doppler studies showed normal flow without vasospasm or stenosis. During monitored care, mildly elevated blood pressure stabilized under calcium channel blocker therapy. A follow up CT after three days confirmed stable haemorrhagic findings, allowing initiation of aspirin for secondary stroke prevention. With ongoing visuospatial rehabilitation, the patient showed gradual but incomplete improvement. Visual agnosia partially resolved, but deficits in colour discrimination and prosopagnosia persisted. He was discharged with a structured outpatient neurorehabilitation program.

Conclusion: This case illustrates an atypical form of PRES with concurrent posterior infarctions, reflecting severe autoregulatory dysfunction. Early recognition and blood pressure control supported partial recovery. The case highlights the importance of considering PRES in patients presenting with neuropsychiatric symptoms after substance use, where initial impressions may be misleading.

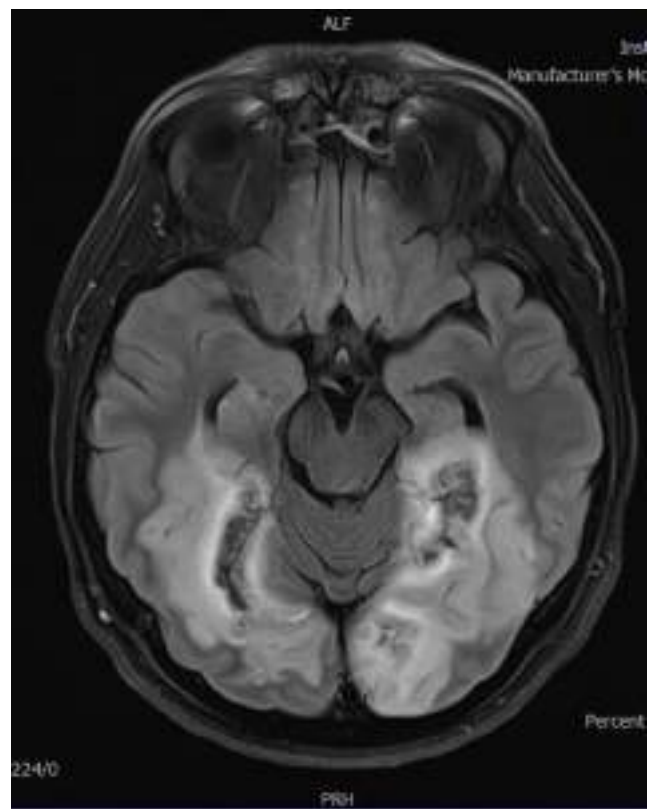


FIGURE 1 MRI FLAIR.

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Background and aims: Hyperacute hour-lasting confusional states frequently suggest exposure to intoxicating substances, but associations may be hard to establish outside the typical contexts.

Methods: We discuss a case of a healthy 69-year-old Portuguese female patient.

Results: She was brought to the emergency department after sudden confusion. Hypertension, sinus tachycardia and flushed skin were noted. Temperature was normal and she did not have headache or vomiting. Neurologic and mental exams were remarkable for disorientation, periods of delusional incoherent and disconnected ideas in between regular thought processing, slurred speech and extremity/truncal ataxia, with no other focal deficits. Laboratory tests were normal including: no detectable alcohol, amphetamines, cocaine, cannabinoids, opioid and barbiturates. Brain CT/MRI were unremarkable. She was kept under surveillance and all symptoms subsided within 12 hours. She recalled only parts of the episode, but remembered having eaten locally bought sprouts that day. Her son whom she had had lunch with described a dry mouth feeling afterwards. She was discharged upon clinical stability and photographed the greens, where different-looking fragments with bristly stems could be seen among the other tuberous roots and leaves.

Conclusion: Fragments of *Ecballium elaterium* (squirting cucumber), a flowering plant found in some parts of southern

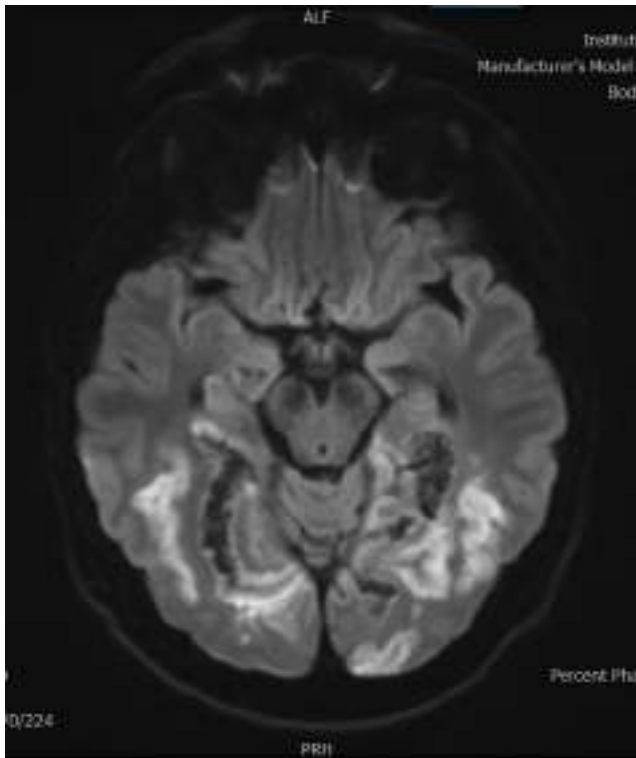


FIGURE 2 MRI DWI.

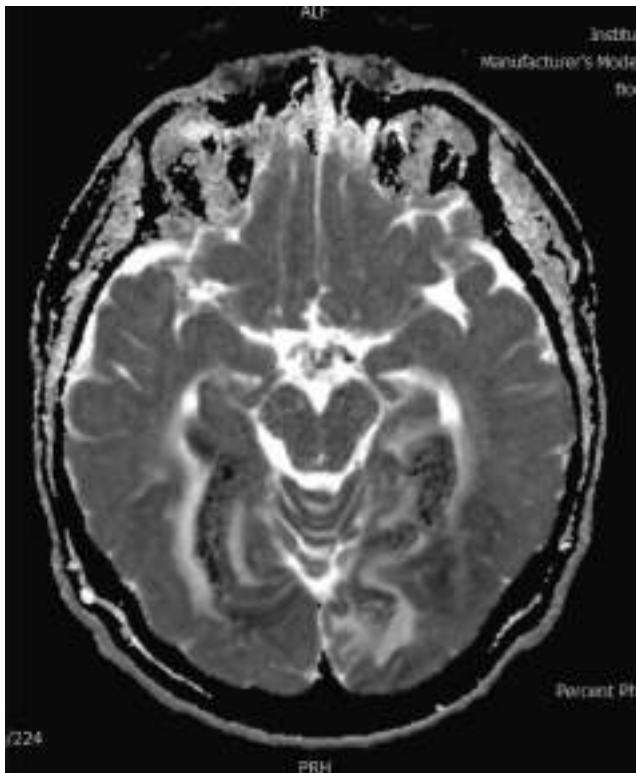


FIGURE 3 MRI ADC.

Disclosure: Nothing to disclose.

EPO-0177 | Abstract withdrawn



FIGURE 1 Patient's own vegetable mix (the dark leaves are the sprouts, and the different-looking tuberous root fragments are the lighter green, bulkier ones).



FIGURE 2 Closer look at the fragments (1).



FIGURE 3 Closer look at the fragments (2).

Europe, including Portugal, and northwestern Africa, were probably inadvertently consumed by our patient. It is allergenic and a strong purgative but, unlike plants of the Solanaceae family, has not been implicated in anticholinergic or central nervous system toxicity. We emphasize reviewing the proliferation of these species in some rural areas for both health and cultivation control.

Disclosure: Nothing to disclose.

EPO-0179 | Neurological spectrum of recreational nitrous oxide use: A case series including monozygotic twins

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Background and aims: Nitrous oxide (N₂O) is a gas widely used in medical and dental practice that has gained increasing popularity as a recreational substance due to its euphoric effects, earning the nickname “laughing gas.” Its abuse can induce a functional vitamin B12 deficiency through oxidation of the cobalt ion, reducing myelin synthesis and leading to neurological manifestations. The aim of this study is to describe the clinical presentations associated with recreational N₂O use.

Methods: Case series of four patients with neurological impairment following prolonged N₂O consumption.

Results: Four male patients aged 18 to 28 years were included, with an average N₂O use of four months, presenting with muscle weakness and/or sensory symptoms. Neurological outcomes varied: one patient was asymptomatic, one presented with axonal polyneuropathy, another with cervical subacute combined degeneration (SCD), and another with both conditions. The series included monozygotic twins with identical exposure: one developed combined SCD and motor axonal polyneuropathy with elevated homocysteine and reduced B12 levels, whereas the twin who intermittently self-supplemented with B-group vitamins showed only mild hypoesthesia and had normal MRI,

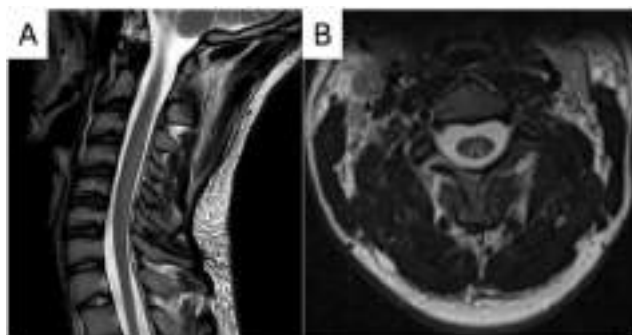


FIGURE 1 Cervical MRI in SCD. (A) Sagittal T2-weighted of Patient 1: longitudinal hyperintense signal in posterior columns. (B) Axial T2-weighted of Patient 4: bilateral and symmetric posterior column hyperintensity forming the “inverted V sign”.

electromyography (EMG), nerve conduction studies (NCS) and B12 values. Clinical improvement was observed following N2O cessation and vitamin supplementation.

Conclusion: Inhalation of N2O may cause a spectrum of neurological injury ranging from axonal polyneuropathy to myelopathy. Normal B12 levels do not exclude neurotoxicity, limiting their usefulness as a sole diagnostic marker. Vitamin

supplementation may delay symptom onset, but cessation of N2O use is essential to halt disease progression.

Disclosure: Nothing to declare.

EPO-0180 | Curcumin exhibits comparable effects to rapamycin in reducing neuronal soma size and intracellular calcium expression in SH-SY5Y cells

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Background and aims: The mTORC2 complex is vital for neuronal function and structure. There are no specific inhibitors for mTORC2; however, chronic high-dose rapamycin and curcumin may have indirect effects. This study compares their impact on mTORC2 inhibition in neuronal cells, focusing on calcium levels and soma size.

Methods: This in vitro study involved SH-SY5Y neuroblastoma cell cultures divided into four groups: a control, one treated with 100 nM rapamycin, and two treated with curcumin at 5 µM and 10 µM for 48 hours to target mTORC2. Soma size was measured in µm², and intracellular calcium levels were labeled with Fluo-4 and quantified with confocal laser scanning microscopy (CLSM). Statistical analysis assessed data differences.

Results: Soma sizes were measured as: control (7892.07 µm²), 5 µM curcumin (6101.25 µm²), 10 µM curcumin (3413.44 µm²), and rapamycin (3341.95 µm²). Intracellular calcium intensities were: control (29.35 nPhoton), rapamycin (7.69 nPhoton), 10 µM curcumin (3.80 nPhoton), and 5 µM curcumin (2.19 nPhoton). Significant differences ($p < 0.001$) were found between all treatment groups and the control for both parameters. The 5 µM curcumin group differed significantly from rapamycin ($p < 0.001$), but there was no significant difference between the 10 µM curcumin and rapamycin groups ($p = 0.84$). The 5 µM curcumin had the lowest calcium intensity, indicating a non-linear dose-dependent effect

Conclusion: Rapamycin is comparable to 10 µM curcumin in inhibiting mTORC2, which reduces neuronal soma size and calcium levels. This suggests that curcumin may naturally modulate the mTOR pathway, highlighting its potential for treating neurological disorders related to mTOR.

Disclosure: Nothing to disclose.

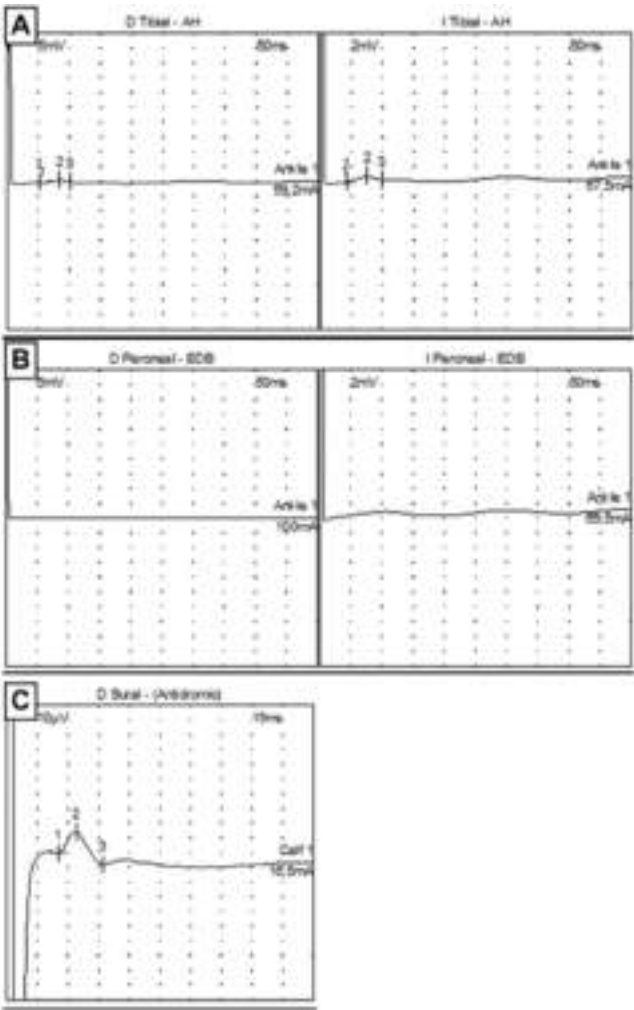


FIGURE 2 NCS of Patient 1 showing a pure motor axonal polyneuropathy of the lower limbs. (A) Reduced compound muscle action potential amplitude in tibial nerves. (B) Absent peroneal nerves responses. (C) Preserved sural nerve sensory response.

TABLE 1 Main clinical characteristics of patients.

	PATIENT 1 (Sister of patient 2)	PATIENT 2 (Sister of patient 1)	PATIENT 3	PATIENT 4
Age	38 years	38 years	20 years	38 years
Onset	3 months	3 months	3 months	3 months
First symptoms	1 month before	1 month before	1 month before	1 month before
Neurological signs	1 day	1 day	1 day	1 day
Presenting complaint	Upper and lower limb weakness Muscle cramps Cognitive decline	Upper limb weakness perforating Tongue numbness GPIIb-IIIa inhibitor	Lower limb weakness Upper limb numbness Cognitive decline	Perforating weakness Upper limb numbness Muscle cramps
Physical examination	Proximal weakness in upper limbs weakness in lower limbs dysarthria Cognitive decline	Weakness	Weakness	Upper limb numbness Weakness in lower limbs dysarthria Cognitive decline
Vitamin B12 levels (pg/mL)	100-150 pg/mL	200-300 pg/mL	100-150 pg/mL (before treatment)	100-150 pg/mL
Homocysteine (µmol/L)	15-20 µmol/L	15-20 µmol/L	15-20 µmol/L	15-20 µmol/L
Genetics	MTOR mutation	MTOR mutation	MTOR mutation	MTOR mutation
EMG/NCV	Upper limb motor axonal polyneuropathy in lower limbs	Upper limb polyneuropathy	Upper limb motor axonal polyneuropathy in lower limbs	Upper limb motor axonal polyneuropathy in lower limbs
EMG to lower limbs	Weakness in lower limbs with normal amplitude in the left side	Normal	Weakness in lower limbs with normal amplitude in the left side	Normal
Follow-up	Stable condition with improving gait	Improvement	Improvement	Improvement with difficulties in hands and lower limbs. Upper limb numbness

EPO-0181 | Boldine and the nervous system: A case of reversible myoneuropathy

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Background and aims: Phytotherapeutic preparations are widely used and often perceived as safe; however, many contain bioactive compounds with potential neuromuscular toxicity that remains poorly characterized. Boldine is an aporphine alkaloid extracted from *Peumus boldus* and commonly used for its presumed hepatoprotective properties. We report a case of reversible myoneuropathy associated with rhabdomyolysis, likely related to boldine consumption.

Methods: A comprehensive clinical, laboratory, neurophysiological, and radiological evaluation was performed, including brain, spinal cord, and muscle magnetic resonance imaging (MRI), lumbar puncture, neurophysiological studies, serial measurements of creatine kinase (CK) and serum neurofilament light chain (sNfL).

Results: A 55-year-old man with alcoholic cirrhosis presented with a three-week history of bilateral proximal lower limb weakness and ascending paresthesias. Neurological examination revealed weakness of hip and knee movements, thigh hypoesthesia, stocking-type sensory loss in the feet, and absent lower limb reflexes. Symptom onset temporally coincided with the initiation of boldine for liver function improvement. Acute inflammatory polyradiculoneuropathy was initially suspected, and intravenous immunoglobulins were administered; however, this diagnosis was excluded due to normal electrophysiological findings and the absence of albuminocytological dissociation. Laboratory tests showed elevated CK levels, peaking at 4,176 U/L. Whole-body MRI demonstrated focal muscle edema involving the right scalene, pectoral, vastus medialis, and left rectus femoris muscles, consistent with rhabdomyolysis. sNfL were elevated.

Conclusion: The clinical and laboratory course was favorable after discontinuation of boldine, with near-complete recovery within one month, normalization of biomarkers, and electrophysiological improvement. The temporal relationship with

boldine exposure, absence of alternative etiologies, and reversibility support a probable toxic myoneuropathic mechanism.

Disclosure: Nothing to disclose.

EPO-0182 | Wernicke encephalopathy: Learning from experience

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Background and aims: Wernicke encephalopathy (WE) is a neurological emergency caused by thiamine deficiency, frequently associated with heavy alcohol drinking, malnutrition or consumptive diseases. Not all cases manifest with the complete classical triad (i.e, ophthalmoplegia, gait ataxia and encephalopathy), leading to underdiagnosis.

Methods: We conducted a descriptive study using the electronic medical record database of Colentina Clinical Hospital, Bucharest, Romania. We included data from patients admitted between January 2019 and December 2025 with a diagnosis of WE (ICD-10 code E51.2). For statistical analysis we used Microsoft Excel and IBM SPSS.

Results: Datasets from 41 patients (90% male, 10% female) complied with the inclusion criteria. The median age was 66 years. Alcohol use disorder was the main identified risk factor for WE in 95% of cases. Gait ataxia was the most frequent sign upon evaluation. Only 21% of patients exhibited the full classical triad. All patients received high doses of intravenous thiamine for several days (mean total dose 5634 mg +/-3607 mg). Brain MRI was performed in 12 patients (29%), of whom only 2 had typical signs of WE. Clinical improvement at discharge was observed in 82% of patients, only a third achieving previous neurological status. We found no significant correlation between the degree of clinical improvement and initial clinical presentation or dose of thiamine administered.

Conclusion: Our findings are in line with the published literature. We reinforce the importance of maintaining a high index of clinical suspicion and prompt initiation of parenteral thiamine treatment, particularly in patients with known risk factors for WE.

Disclosure: Nothing to disclose.

EPO-0183 | Chronic noise exposure induces cis-P-tau accumulation, an early Alzheimer's disease-related pathological marker, and cognitive deficits in rats

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Background and aims: Chronic noise exposure is a stressor linked to central nervous system dysfunction and cognitive decline, including Alzheimer's disease (AD). Hyperphosphorylation of tau protein, particularly Thr231, is a hallmark of AD. Cis

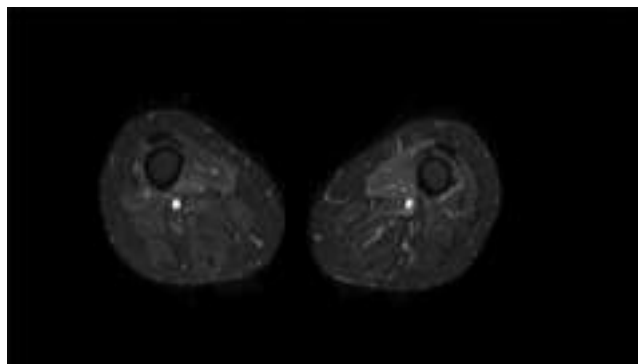


FIGURE 1 Edema of the left quadriceps.

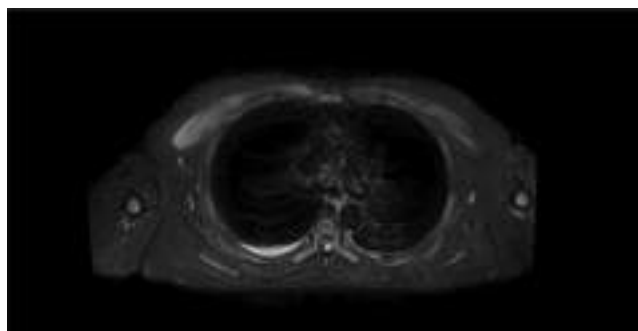


FIGURE 2 Edema of the right pectoral muscle.

p-tau is highly neurotoxic and an early driver of tauopathy, causing microtubule disruption and neurodegeneration, a process termed Cistauosis.

Methods: Rats were exposed to 95 dB SPL white noise for 4 hours daily over 15 days. Cognitive and anxiety-related behaviors were assessed using the elevated plus maze and Y-maze. Western blotting and immunohistochemistry were performed to evaluate tau pathology.

Results: Noise-exposed rats exhibited increased anxiety-like behaviors and memory deficits. Molecular analyses demonstrated that chronic noise significantly induced cis p-tau accumulation and Cistauosis in multiple brain regions.

Conclusion: Chronic noise exposure promotes Cistauosis, linking environmental stress to neurodegeneration and AD development. These findings highlight the potential role of noise-induced tau pathology in the early stages of Alzheimer's disease.

Disclosure: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

EPO-0184 | Neurological manifestations related to toxic exposure in children: A clinical study

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Background and aims: Neurological manifestations due to toxic exposure in children represent a significant cause of pediatric morbidity. Their clinical heterogeneity often complicates diagnosis and delays appropriate management. This study aimed to describe the epidemiological characteristics, clinical presentation, management, and outcomes of toxic-induced neurological manifestations in a pediatric population.

Methods: We conducted a retrospective study including patients aged under 18 years, hospitalized between 2017 and 2025 in the Pediatric Neurology Department of the National Institute of Neurology in Tunis, for neurological symptoms attributed to toxic exposure. Demographic, clinical, paraclinical, therapeutic, and outcome data were collected and analyzed.

Results: Eighteen patients were included, with a median age of 6 years. Toxic exposure was drug-related in 13 cases, including chemotherapy (5 cases), antiepileptic drugs (4 cases), haloperidol (1 case), and flunarizine (1 case). One case each was attributed to carbon monoxide and N-hexane exposure. The circumstances were iatrogenic in nine cases, accidental in six cases, and intentional in five cases. Five patients presented with an encephalitic-like syndrome. Peripheral neuropathy, movement disorders, and ataxia were each observed in four patients. The outcome was favorable in 13 cases, while two patients required admission to the intensive care unit.

Conclusion: In our series, neurological manifestations related to toxic exposure in children were predominantly iatrogenic. Early recognition and prompt discontinuation of the offending agent are crucial for a favorable prognosis. These conditions should be systematically considered in the differential diagnosis of acute or subacute neurological presentations in children.

Disclosure: The authors declare no conflicts of interest.

Cerebrovascular Diseases 2

EPO-0185 | Asymptomatic hemorrhagic transformation after mechanical thrombectomy is not benign: Early and long-term outcomes

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Background and aims: The prognostic relevance of asymptomatic hemorrhagic transformation (aHT) after mechanical thrombectomy (MT) for acute ischemic stroke (AIS) remains uncertain and is often considered clinically benign. We evaluated the impact of aHT on early and long-term outcomes after MT.

Methods: We retrospectively analyzed consecutive patients with AIS treated with MT at a comprehensive stroke center. A follow-up CT scan was performed after 24 hours and hemorrhagic transformation was assessed using Heidelberg Bleeding Classification. Patients were followed up for 12 months. Associations between aHT, functional outcomes, and mortality were analyzed using multivariable regression models.

Results: Among 363 patients, 237 (65.3%) had no HT, 84 (23.1%) developed aHT, and 35 (9.6%) developed symptomatic HT. Patients with aHT had worse functional outcomes than those without HT. Functional independence (mRS 0–2) at 90 days was achieved in 45.1% of patients without HT (23.8% with aHT). After adjustment, aHT was independently associated with reduced odds of functional independence at 90 days (OR 0.29, 95% CI 0.15–0.57), a shift toward worse functional outcome at 12 months (common OR 2.11, 95% CI 1.30–3.41), and increased mortality during follow-up (HR 1.64, 95% CI 1.07–2.52). Patients developing aHT more frequently presented with higher baseline NIHSS, while no single baseline clinical or treatment-related variable emerged as a strong independent predictor of aHT.

Conclusion: aHT after MT is associated with worse early and long-term functional outcomes and increased mortality. Further validation of our findings in large cohort studies of MT-treated patients is warranted.

Disclosure: Nothing to disclose.

EPO-0186 | Epidemiological trends in intracerebral haemorrhage: A decadal analysis of aetiology, prior blood pressure control, and direct oral anticoagulant use

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Background and aims: The incidence of intracerebral haemorrhage (ICH) in Hong Kong remains high compared to developed countries globally. As the most severe form of stroke, where treatment offers only modest benefit, ICH prevention is crucial to reducing disease burden. We aim to study changes in ICH epidemiology, particularly focusing on pre-ICH blood pressure (BP) control, direct oral anticoagulants (DOAC) use, and

TABLE 1 Baseline demographic and clinical characteristics of ICH patients over 3 temporal groups.

	All (n=1044)	2014–2016 (n=379)	2017–2019 (n=333)	2020–2022 (n=332)	P-value
Age, yr	67.9 (15.5)	66.7 (16.1)	69.9 (15.2)	67.7 (15.1)	0.529
Male gender, no. (%)	599 (57.4%)	223 (58.8%)	193 (58.1%)	183 (55.0%)	0.794
Aetiology of ICH, no. (%)					0.108
Hypertension	760 (72.8%)	293 (77.3%)	242 (72.4%)	221 (66.6%)	
Cerebral amyloid angiopathy	243 (23.3%)	71 (18.7%)	81 (24.3%)	91 (27.4%)	
Undiagnosed	36 (3.4%)	13 (3.4%)	8 (2.4%)	5 (1.5%)	
On anticoagulation, no. (%)	186 (17.8%)	77 (20.3%)	38 (11.4%)	41 (12.4%)	0.023
Warfarin	38 (3.6%)	21 (5.5%)	36 (10.8%)	13 (3.9%)	
DOAC	49 (4.7%)	6 (1.6%)	2 (0.6%)	28 (8.5%)	0.001
None	100 (9.5%)	5 (1.3%)	0 (0.0%)	1 (0.3%)	
Hypertension (HTN) controlled status					0.011
Undiagnosed/Uncontrolled HTN	871 (84.4%)	281 (74.1%)	268 (80.2%)	287 (86.2%)	
Controlled HTN	174 (16.6%)	61 (16.1%)	64 (19.3%)	45 (13.5%)	
No HTN / uncertain status of HTN	259 (24.8%)	79 (20.8%)	99 (29.6%)	81 (24.3%)	
Previous history of:					
Ischemic	126 (12.1%)	79 (20.8%)	79 (23.7%)	78 (23.6%)	0.086
Ischemic stroke	106 (10.1%)	61 (16.1%)	61 (18.3%)	64 (19.3%)	
Transient ischemic attack	20 (1.9%)	6 (1.6%)	18 (5.4%)	14 (4.2%)	0.004
Ischemic stroke	145 (13.9%)	85 (22.4%)	97 (29.1%)	92 (27.8%)	0.017
Ischemic heart disease	136 (13.1%)	44 (11.6%)	23 (7.0%)	37 (11.2%)	0.206
Peripheral vascular disease	6 (0.6%)	1 (0.3%)	1 (0.3%)	4 (1.2%)	0.238
Atrial fibrillation	118 (11.4%)	21 (5.5%)	31 (9.3%)	61 (18.4%)	0.008

1. Hypertensive ICH defined by ICH site and/or vessel, including thalamus, basal ganglia, brainstem, cerebellum and cerebellum.
2. Cerebral amyloid angiopathy defined by presence with imaging, and/or clinical findings, following modified Boston criteria.
3. Uncontrolled hypertension defined by recorded blood pressure consistently greater or above 140/90 mmHg (supine/standing).

the cause of ICH. Our findings will help inform public health strategies to prevent ICH.

Methods: We conducted a single-centre observational study of spontaneous ICH patients admitted from 2014 to 2022. Demographic and clinical data were collected, including pre-ICH BP control, anticoagulant use and ICH aetiology using the modified Boston criteria. Temporal shifts in the aetiology of ICH and BP control were analysed.

Results: A total of 1,044 ICH patients were categorised into three temporal groups: 2014–16, 2017–19, and 2020–22. There was a numerical increase in the proportion of ICH attributed to cerebral amyloid angiopathy (CAA) from 19.8% (75/379; 2014–16) to 25.8% (86/333; 2020–22) ($p=0.110$). The rate of undiagnosed/uncontrolled hypertension decreased from 67.3% (255/379; 2014–16) to 62.2% (207/333; 2020–22) ($p=0.023$) but remained high. The prevalence of anticoagulant-associated ICH roughly doubled from 2014–16 to 2020–22 (7.1% vs 13.0%, $p=0.028$), driven by DOAC-ICH. Among DOAC-ICH, 54.3% had uncontrolled/ undiagnosed hypertension and 30.6% had CAA.

Conclusion: Pre-ICH BP control had slightly improved over the decade, but the rate of undiagnosed/uncontrolled hypertension remains unacceptably high. Strengthening hypertension control in both the general population and, as importantly, DOAC users, is essential to reduce ICH burden.

Disclosure: Nothing to disclose.

EPO-0187 | Comparative cardiovascular and bleeding outcomes with PPIs versus H2-receptor blockers: A nationwide cohort study in Korea

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Background and aims: Among stroke survivors on anti-thrombotic therapy, gastroprotection is common, yet the cardiovascular safety of proton pump inhibitors (PPIs) relative to H2-receptor blockers (H2Bs) remains debated.

Methods: We performed a retrospective cohort study using linked Korean National Health Insurance claims and national stroke registry data. Adults with imaging-confirmed index ischemic stroke (2016–2020) who received antithrombotics and

initiated either a PPI or an H2B within 30 days after the index event were included. The primary outcome was major adverse cardiovascular events (MACE)—composite of myocardial infarction, recurrent stroke, or all-cause death. Secondary outcomes were gastrointestinal (GI) bleeding, intracranial hemorrhage, and other bleeding. Propensity-score matching balanced baseline covariates, and Cox models estimated hazard ratios (HRs).

Results: From 850,053 screened records, 4,569 eligible patients were identified and grouped as PPI ($n=2,515$) and H2B ($n=2,054$). After propensity-score matching, there was no significant difference in MACE between PPI and H2B users across crude, PS-matched, and fully adjusted analyses. In contrast, GI bleeding was significantly lower with PPIs than with H2Bs, while intracranial hemorrhage and other bleeding showed no excess risk with PPIs. Findings were consistent across sensitivity analyses.

Conclusion: In ischemic stroke patients treated with anti-thrombotics, PPIs did not increase MACE risk compared with H2Bs and were associated with lower GI-bleeding risk. These results support the cardiovascular safety of PPIs and suggest they may be the preferred gastroprotective option in this setting, particularly for patients at heightened risk of GI bleeding.

Disclosure: Nothing to disclose.

EPO-0188 | Impact of tenecteplase implementation in a remote region without on-site thrombectomy

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Background and aims: Intravenous thrombolysis (IVT) remains the main treatment for acute ischemic stroke (AIS) in hospitals without endovascular treatment (EVT). Tenecteplase may simplify workflow and reduce treatment delays compared with alteplase. We aimed to evaluate the impact of switching from alteplase to tenecteplase on workflow metrics, safety, and early clinical outcomes in an insular hospital.

Methods: An observational study including consecutive patients with AIS treated with IVT at a hospital without on-site EVT. Two periods were compared: 2024 (alteplase) and 2025 (tenecteplase). The primary outcome was DTN time. Secondary outcomes included the proportion treated <60 minutes, intracranial hemorrhage, in-hospital mortality, and functional outcomes at discharge.

Results: 14 patients in the alteplase group and 7 in the tenecteplase group. Mean age was 69 and 62 years, respectively ($p=0.18$), and mean baseline NIHSS was 12.5 vs 9 ($p=0.17$). Median DTN was significantly shorter with tenecteplase (45 vs 71 minutes), representing a reduction of 26 minutes ($p<0.01$). The proportion treated within 60 minutes increased from 35.7% to 71.4% ($p=0.04$). Intracranial hemorrhage rates were similar (21.4% vs 14.3%; $p=1$). One in-hospital death occurred in the alteplase group. NIHSS at discharge did not differ between groups (mean 5.4 vs 4.1; $p=0.29$). Functional independence at discharge (mRS 0–2) was observed in 61.5% of the alteplase group and 57.1% of the tenecteplase group.

Conclusion: In remote regions without onsite EVT, implementation of tenecteplase for IVT was associated with a significant improvement in workflow efficiency, particularly door-to-needle time, without compromising safety.

Disclosure: Nothing to disclose.

EPO-0189 | External validation of ASPECTS–NIHSS clinical-imaging equations for predicting 90-day outcome after endovascular thrombectomy in acute ischemic stroke

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Background and aims: Despite the widespread adoption of endovascular thrombectomy (EVT) in acute ischemic stroke (AIS) patients with large vessel occlusions, rapid bedside tools for prognostication are limited. Novel simple clinical-imaging equations based on Alberta Stroke Program Early CT Score (ASPECTS) and National Institutes of Health Stroke Scale (NIHSS) were recently developed (Nie et al, 2025) to predict post-EVT outcomes. We aimed to externally validate the equations and the published logistic model for predicting 90-day functional independence in an independent cohort.

Methods: Consecutive AIS patients undergoing anterior circulation EVT within 24 hours at the University Hospital Krakow (January–December 2023) with available NIHSS, ASPECTS and 90-day modified Rankin Scale (mRS) were included. Three predictive approaches were validated: (1) clinical–imaging equation (CIE=ASPECTS –0.5×NIHSS), (2) age-adjusted-CIE (AdjCIE = ASPECTS–0.5×NIHSS–0.2×Age), and (3) published logistic model probability (p_FI). Primary outcome was functional independence (mRS 0–2). Discrimination was assessed using area

TABLE 1 Baseline characteristics of the study cohort stratified by modified Rankin Score.

Variable	Total Cohort (n=149)	mRS 0–2 (n=101)	mRS 3–6 (n=48)
Demographics			
Age, median (IQR)	72 (15)	69 (15)	76.5 (13.75)
Female sex, n (%)	90 (60.4)	59 (58.4)	31 (64.6)
Previous mRS ≥ 2, n (%)	19 (12.8)	7 (6.9)	12 (25)
Baseline NIHSS, median (IQR)	15 (9)	13 (10)	18 (6.75)
Vascular risk factors			
Diabetes, n (%)	40 (26.8)	21 (20.8)	19 (39.6)
Hypertension, n (%)	114 (76.5)	75 (74.3)	39 (81.3)
Atrial fibrillation, n (%)	51 (34.2)	37 (36.6)	14 (29.2)
Clinical characteristics			
Baseline ASPECTS, median (IQR)	8 (3)	9 (3)	8 (3)
Oedema site, n (%)			
– A1	1 (0.7)	0 (0)	1 (2.1)
– M1	38 (65.8)	63 (62.4)	35 (72.9)
– M2	43 (28.9)	32 (31.7)	11 (22.9)
– M5	7 (4.7)	6 (5.9)	1 (2.1)
Treatment			
IV thrombolysis, n (%)	24 (49.7)	52 (53.5)	22 (45.8)
Procedural variables			
Door-to-EVT time, min, median (IQR)	304 (163.5)	312 (164)	298.5 (163.5)

*mRS- modified Rankin Score; NIHSS- National Institutes of Health Stroke Scale; ASPECTS- Alberta Stroke Program Early CT Score; EVT- endovascular thrombectomy.

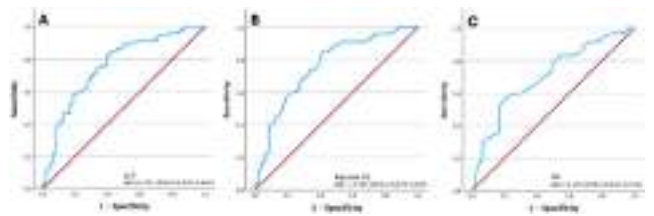


FIGURE 1 ROC curves of (A) the published logistic regression model probability of functional independence (p_FI), (B) age-adjusted clinical–imaging equation (AdjCIE) and (C) clinical–imaging equation (CIE) for 90-day functional independence.

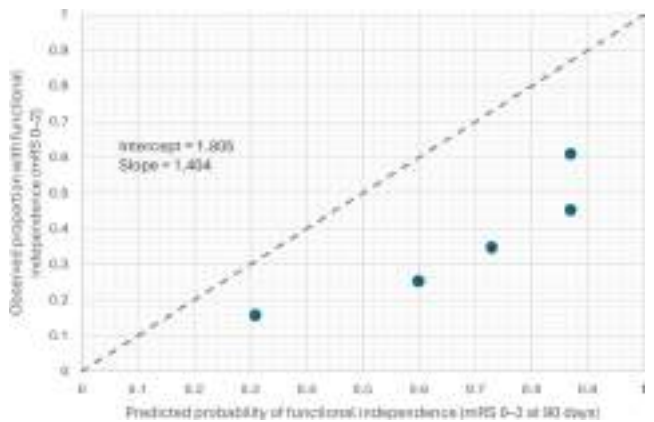


FIGURE 2 Calibration plot for the published logistic model predicting functional independence after 90 days. Predicted probabilities grouped into quintiles of predicted risk in the validation cohort (n=149). The dashed line is perfect calibration.

under the ROC curve (AUC), calibration using intercept and slope and, overall accuracy using Brier score.

Results: 149 of 361 patients were included (mean age 72, 60.4% female, median NIHSS 15 [9]) and 67.8% achieved functional independence. Discriminative ability was good for p_FI (AUC 0.761; 95% CI 0.676–0.846) and AdjCIE (AUC 0.763; 95% CI 0.679–0.847), and moderate for CIE (AUC 0.705; 95% CI 0.616–0.793). Calibration of p_FI showed systematic underprediction (intercept 1.805, slope 1.404) and Brier score was 0.281.

Conclusion: Proposed clinical-imaging equations show good discrimination for 90-day functional independence in our cohort but systematic underprediction in calibration, indicating the possible requirement for regional recalibration before clinical application.

Disclosure: Nothing to disclose.

EPO-0190 | Cancer-related stroke in esus patients: A Tunisian cross-sectional study

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Background and aims: Malignancy is identified in ~10% of patients with embolic stroke of undetermined source (ESUS).

Ischemic stroke (IS) may precede or follow cancer diagnosis, yet no consensus exists on systematic malignancy screening in stroke patients. This study aimed to evaluate the clinical, biological, and radiological features of cancer-related stroke (CRS) in a Tunisian cohort.

Methods: We conducted a 4-year cross-sectional study including patients with acute IS confirmed by CT or MRI and admitted to the Neurovascular Unit of Sahloul University Hospital, Tunisia. CRS was defined as ESUS with a negative etiological workup (brain MRI, CT angiography, 48-hour Holter monitoring, transthoracic and transesophageal echocardiography) in patients with active cancer diagnosed before or during stroke evaluation.

Results: Twenty-four patients were included (median age 66.2 ± 9.1 years; male-to-female ratio 3.8). Cancer was revealed during stroke workup in 16 patients (66.7%). Common malignancies were lung (41.7%), pancreatic (20.8%), colon (12.5%), and prostate (8.3%). Median NIHSS was 6. Five patients had prior venous thrombosis or pulmonary embolism, suggestive of Trousseau syndrome. Imaging showed multiple infarcts in ≥ 2 vascular territories in 50% and the Three Territory Sign in 37.5%. Median D-dimer was $4.0 \mu\text{g/mL}$ [IQR 1.93–13.1], and mean fibrinogen was $3.18 \pm 1.89 \text{ g/L}$. Eight patients (33.3%) died shortly after stroke; eleven received curative anticoagulation.

Conclusion: Elevated D-dimer levels and embolic infarct patterns, particularly the three territory Sign, are key markers of CRS in ESUS patients. D-dimer alone lacks specificity, but combined markers improve risk stratification and enable earlier detection in high-risk patients.

Disclosure: Nothing to disclose.

EPO-0191 | Serum brain-derived tau in moyamoya angiopathy: A case-control study of associations with cognitive impairment

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Background and aims: Brain-derived tau (BD-tau) is a sensitive blood-based biomarker of neuronal injury in acute ischemic stroke; but its relevance in conditions characterized by chronic cerebral hypoperfusion remains unclear. Moyamoya angiopathy (MMA) is a progressive steno-occlusive disorder causing regional cerebral hypoperfusion, impaired vasomotor reserve, and heterogenous clinical phenotypes, including cognitive impairment, complicating risk stratification. Biomarkers reflecting hypoperfusion-related neuronal injury may complement imaging and clinical assessment in neurosurgical decision-making. We investigated serum BD-tau levels in MMA

patients and healthy controls and explored relations with cognitive performance.

Methods: This cross-sectional, register-based case-control study included MMA patients without recent stroke ($n=37$) and healthy controls ($n=14$). Serum total BD-tau levels were compared using Mann-Whitney U test. MMA patients underwent comprehensive neuropsychological assessment across nine cognitive domains. A Global Cognitive Impairment Index (GCII) was calculated as the proportion of domains with at least one test score ≥ 1 standard deviation below normative means. Associations between BD-tau and GCII were assessed using Spearman's rank correlation.

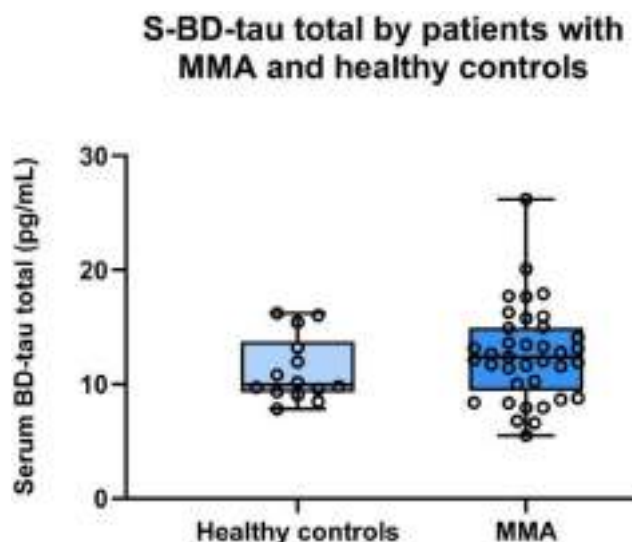


FIGURE 1 Box-plot comparing BD-tau total levels between MMA-patients ($n=37$) and healthy controls ($n=14$). Difference was non-significant ($p=0.263$, Mann-whitney U test). S-BD-tau: serum brain-derived tau total. MMA: moyamoya angiopathy.

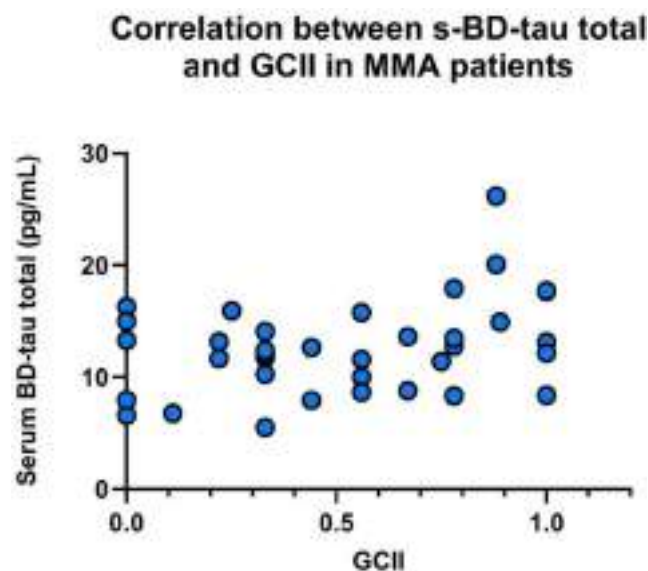


FIGURE 2 Scatterplot demonstrating no statistical correlation between GCII and BD-tau levels in MMA patients ($n=37$, Spearman $\rho=0.311$ $p=0.061$). S-BD-tau: serum brain-derived tau. MMA: moyamoya angiopathy. GCII: Global Cognitive Impairment Index.

Results: Median serum BD-tau levels were 12.38 [9.40–14.96] pg/mL in the MMA group and 9.97 [9.27–13.77] pg/mL in the control group, without significant between-group difference ($p=0.263$). Within the MMA group, BD-tau levels showed no significant correlation with GCII ($p=0.061$).

Conclusion: Serum BD-tau levels were not elevated in MMA patients and were not associated with global impairment. Despite small sample size, these findings suggest limited general utility of BD-tau in MMA, though its potential in selected subgroups warrants further investigation in larger cohorts.

Disclosure: Anne Hege Aamodt has received personal fees for lectures/advisory boards; Novartis, Abbvis, TEVA, Roche, Lundbeck, Pfizer, Boehringer Ingelheim, non-personal research grants from Norwegian National Association for Public Health, South-Eastern Norway regional Health Authority, Odd Fellow, National Program for Clinical Treatment Research in the Specialist Health Service (KLINBEFORSK), EU, Boehringer Ingelheim, Medtronic, BMS. Bente Halvorsen is an SAB member in CircM, Linköping, Sweden and is an evaluator in MH panel, Swedish Research council. The remaining authors have nothing to disclose.

EPO-0192 | Determinants of functional outcome in patients with intracranial atherosclerotic stenosis: Analysis of the bernese intracranial stenosis study

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Background and aims: Functional outcome after intracranial atherosclerotic stenosis (ICAS) is highly variable, yet the prognostic determinants of outcome in these patients are still not well defined. Ultra-high-field 7-Tesla MRI enables detailed evaluation of ICAS.

Methods: We analyzed 131 ICAS patients (of which $n=58$ symptomatic) under best medical management from the prospective Bernese Intracranial Stenosis Study, dichotomized by 1-year functional outcome (favorable: mRS 0–1, $n=58$; unfavorable: mRS 2–6, $n=73$). Group comparisons were performed for demographics, vascular risk factors, laboratory values, and 3/7T MR imaging markers at baseline and follow-up.

Results: At baseline, the unfavorable vs. favorable outcome group had a significantly higher prevalence of symptomatic ICAS (52.1% vs. 34.5%, $p=0.044$), diabetes mellitus (39.7% vs. 20.7%, $p<0.02$), physical inactivity (30.1% vs. 5.2%, $p<0.001$), peripheral artery disease (15.1% vs. 3.4%, $p=0.027$), higher median baseline NIHSS (1 vs. 0, $p=0.012$), median baseline BDI

(7 vs. 3, $p<0.001$), alongside lower median baseline MoCA (26 vs. 27, $p=0.001$) and median baseline EuroQoL VAS (70 vs. 83, $p<0.001$). At 1-year follow-up, the unfavorable outcome group had significantly worse median NIHSS (2 vs. 1, $p<0.001$), BDI-II (6 vs. 3, $p=0.005$), MoCA (26 vs. 28, $p<0.001$), and EuroQoL VAS (75 vs. 87, $p<0.001$) in univariable analysis. In multivariable analysis, symptomatic ICAS (OR 2.74, 95% CI 1.11–6.80, $p=0.029$) remained to be the only independent predictor of unfavorable outcome.

Conclusion: In summary, symptomatic ICAS was independently associated with 2.74-fold increased odds of an unfavorable functional outcome. This highlights the importance of early identification and close monitoring of symptomatic ICAS patients to optimize functional recovery.

Disclosure: Nothing to disclose.

EPO-0193 | Tenecteplase in the extended treatment window for acute ischemic stroke: Evidence from randomized trials

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Background and aims: The role of intravenous tenecteplase (TNK) for acute ischemic stroke treated between 4.5 and 24 hours remains debated, particularly across healthcare settings with variable access to endovascular thrombectomy (EVT).

Methods: Randomized controlled trials comparing TNK (0.25 mg/kg) with placebo or standard care in adults treated 4.5–24 hours after stroke onset were systematically reviewed. The primary outcome was excellent functional recovery (mRS 0–1) at 90 days. Secondary outcomes included recanalization, neurological improvement, and safety endpoints. Random-effects meta-analyses were performed overall and stratified by EVT availability. PROSPERO (CRD420251274200)

Results: Four multicenter RCTs ($n=1,278$) were included. TNK significantly increased the likelihood of excellent functional outcome and vessel recanalization. Overall differences in good functional outcome and early neurological improvement were not significant. In settings without EVT access, TNK improved multiple functional and early recovery outcomes, whereas benefits in EVT-capable settings were limited to recanalization. Rates of symptomatic intracerebral hemorrhage and 90-day mortality were similar between groups.

Conclusion: TNK improves excellent functional recovery and recanalization in the 4.5–24-hour window, particularly where EVT is unavailable, supporting its role as a pragmatic reperfusion option in resource-limited contexts.

Disclosure: Nothing to disclose.

EPO-0194 | Greater occipital nerve block as a treatment for non-traumatic subarachnoid haemorrhage related headache: Systematic review of the available evidence

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Background and aims: Non-traumatic subarachnoid haemorrhage (SAH) is characterized by severe headache refractory to conventional analgesia, leading to frequent use of opioids. Greater occipital nerve blocks (GONB) are commonly used in migraine and other headache disorders with great success. The goal of this study was to review available evidence on the use of GONB in the treatment of SAH-related headache.

Methods: Systematic review of available evidence following the PRISMA-2020 guidelines from inception to 01/2026 with a narrative synthesis of the results. We included studies with adult patients with non-traumatic SAH-related headache, which used GONB (with/without other nerve blocks) as adjunctive

analgesia and monitored headache intensity with validated scores (VAS, NPRS [both 0–10]). The search was performed in electronic databases (PubMed, Embase, Cochrane CENTRAL, ClinicalTrials.gov) using controlled terms related to SAH, headache and GONB. Bias risk was assessed using validated tools (JBI/ROBINS-I).

Results: 48 studies were identified, of which three met inclusion criteria (Figure 1). Two were case series ($n=2$, $n=10$) and one was a non-randomized observational study with historical controls ($n=10$). All studies reported a decrease in headache intensity scores after the procedure (Δ NPRS 2.67–6, timeframe 1 h–1 week), and a reduced need for rescue analgesia. No significant adverse events were reported. All studies had a moderate-to-high bias risk due to lack of proper controls and small sample sizes (Table 1).

Conclusion: Current evidence on GONB for treating non-traumatic SAH-related headache is limited due to small-sample non-controlled studies, but consistently suggests potential analgesic benefits. Randomized controlled data are needed to further examine its efficacy.

Disclosure: The authors declare no conflicts of interest.

EPO-0195 | Predicting neurological functional improvement using machine learning in acute minor stroke: A Vietnamese cohort study

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Background and aims: Although acute minor stroke is often considered benign, many patients develop persistent functional limitations. Early prediction of neurological functional improvement, assessed by the 90-day modified Rankin Scale (mRS), may support treatment decisions and rehabilitation planning. This study aimed to develop machine learning models to predict 90-day mRS improvement in patients with acute minor stroke using newly collected data from Vietnam.

Methods: We analyzed data from 736 patients with acute minor stroke admitted to Bach Mai Hospital, Vietnam, between 2023 and 2024. The dataset was split into a training set (70%, $n=515$) and a test set (30%, $n=221$). Predictor selection was performed in the training set using LASSO regression with 5-fold cross-validation. Four binary classifiers - Extra Trees, Random Forest, Extreme Gradient Boosting, and Support Vector Machine with a Radial Basis Function kernel - were trained using the selected predictors. Hyperparameters were optimized using GridSearch cross-validation, and performance was evaluated in the test set.

Results: Fourteen predictors were identified. All models achieved an area under the receiver operating characteristic curve above 0.75. The Extra Trees model showed the best performance, with an Area Under the Receiver Operating Characteristic Curve (AUC) of 0.780 (95% CI, 0.704–0.854), Precision-Recall AUC of 0.890, accuracy of 0.810, and F1-score of 0.878.

Conclusion: Machine learning models may help predict 90-day functional improvement in patients with acute minor stroke

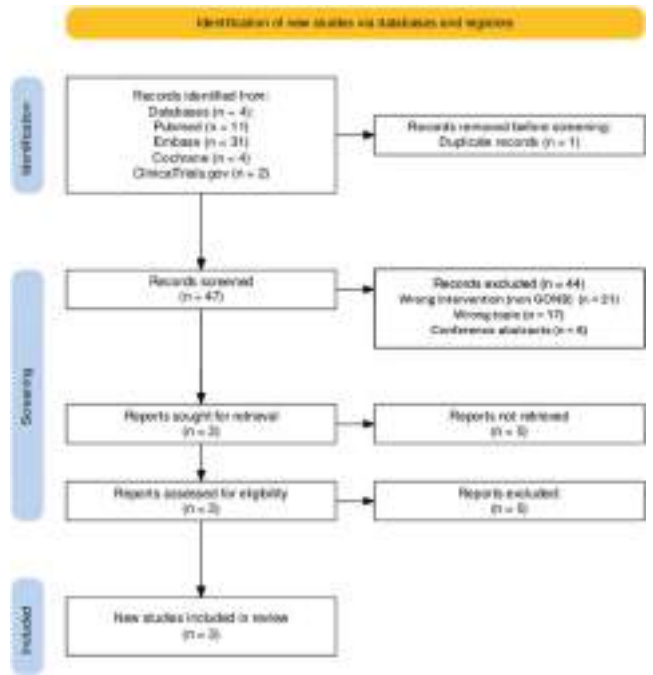


FIGURE 1 PRISMA-2020 flow diagram.

TABLE 1 Risk of bias assessment Abbreviations: JBI = Joanna Briggs Institute; ROBINS-I = Risk of Bias in Non-randomized Studies – of Interventions.

Study	Design	Assessment tool	Bias risk
Dogan et al., 2020	Case report (n=2)	JBI	High
Venkatesulu et al., 2019	Case series (n=10)	JBI	High
Rajagopalan et al., 2023	Non-randomized observational trial with historical cohort (n=10)	ROBINS-I	Moderate

using real-world data from Vietnam. Further validation in larger and more diverse cohorts is required.
Disclosure: Nothing to disclose.

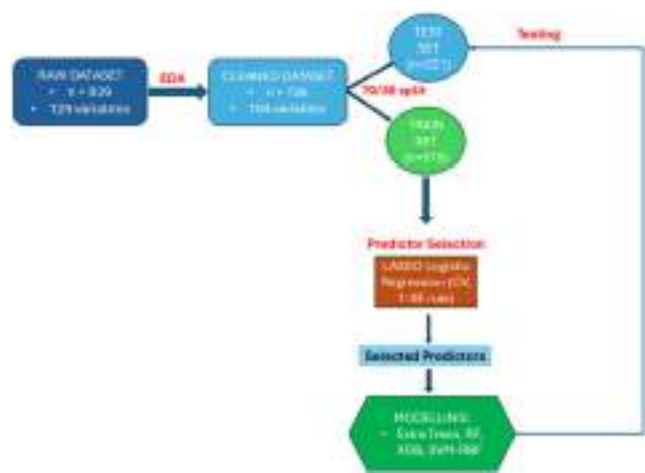


FIGURE 1 The overall workflow diagram illustrating the main sequential steps

Model	AUROC (95% CI)	AUPRC	Accuracy	F1-score
Extra Trees	0.780 (0.704–0.854)	0.890	0.810	0.878
RF	0.766 (0.689–0.842)	0.886	0.824	0.894
XGB	0.758 (0.678–0.838)	0.878	0.789	0.848
SVM_RBF	0.758 (0.683–0.831)	0.888	0.778	0.870

AUROC = Area Under the Receiver Operating Characteristic Curve; AUPRC = Area Under the Precision-Recall Curve; RF = Random Forest; SVM = a Support Vector Machine; SVM_RBF = Support Vector Machine with Radial Basis Function kernel

FIGURE 2 Performance of the four models

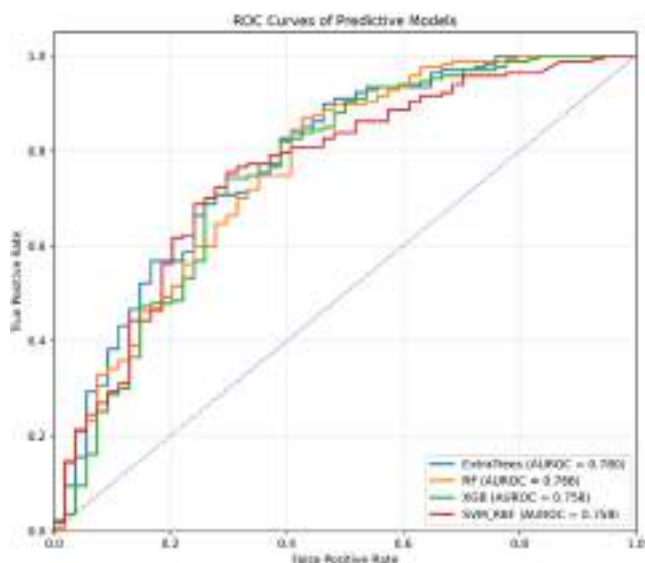


FIGURE 3 ROC curves of the four models.

EPO-0196 | ATAD3A stabilizes Drp1 via lysosome-dependent regulation to sustain pathological mitochondrial fission after cerebral ischemia/reperfusion

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Background and aims: Excessive mitochondrial fission is a key driver of neuronal death after cerebral ischemia/reperfusion (I/R), yet the mechanisms sustaining this process beyond the acute phase remain unclear. Dynamin-related protein 1 (Drp1) is central to mitochondrial fission, but how its pathological activation is maintained following I/R is poorly understood. This study investigated whether ATPase family AAA domain-containing protein 3A (ATAD3A) regulates mitochondrial fission and neuronal death through control of dynamin-related protein 1 stability.

Methods: Mouse middle cerebral artery occlusion/reperfusion (MCAO/R) and neuronal oxygen–glucose deprivation/reoxygenation (OGD/R) models were established. ATPase family AAA domain-containing protein 3A was suppressed genetically in vivo and in vitro. Protein interaction, degradation pathways, mitochondrial dynamics, and neuronal apoptosis were analyzed using biochemical, imaging, and functional approaches.

Results: Cerebral I/R induced sustained upregulation of ATAD3A in neurons. ATAD3A interacted with Drp1 and inhibited its lysosome-dependent degradation, thereby preserving Drp1 protein stability. Persistent stabilization of Drp1 promoted excessive mitochondrial fission and disruption of mitochondrial dynamics, leading to increased neuronal apoptosis. Suppression of ATAD3A accelerated Drp1 degradation, attenuated pathological mitochondrial fission, and reduced neuronal apoptosis after ischemia/reperfusion.

Conclusion: ATAD3A sustains pathological mitochondrial fission after cerebral I/R by preventing lysosome-dependent degradation of Drp1. This proteostatic mechanism represents a key driver of persistent mitochondrial dynamics disruption and neuronal death following ischemic injury.

Disclosure: Nothing to disclose.

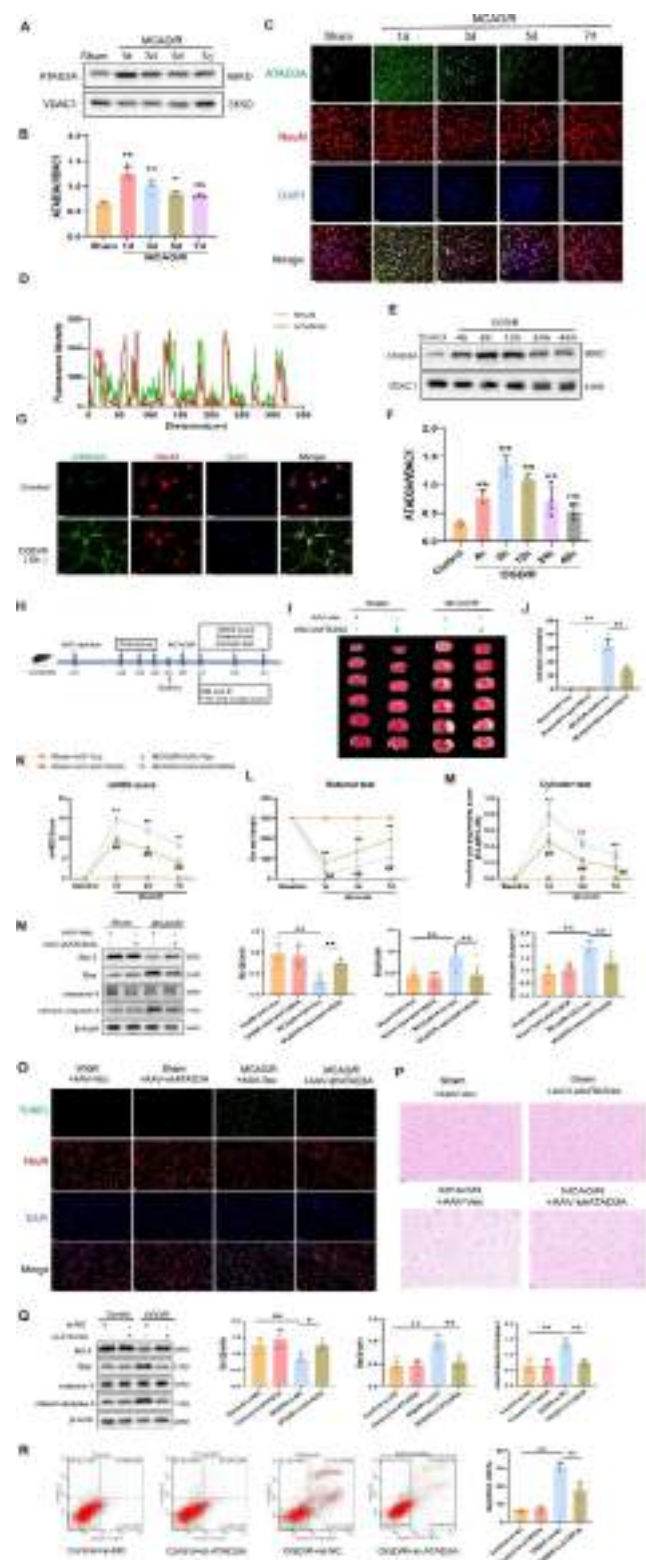


FIGURE 1 ATAD3A is upregulated in neurons after and knockdown of ATAD3A improves neurological function and ameliorates neuronal apoptosis following cerebral ischemia/reperfusion injury.

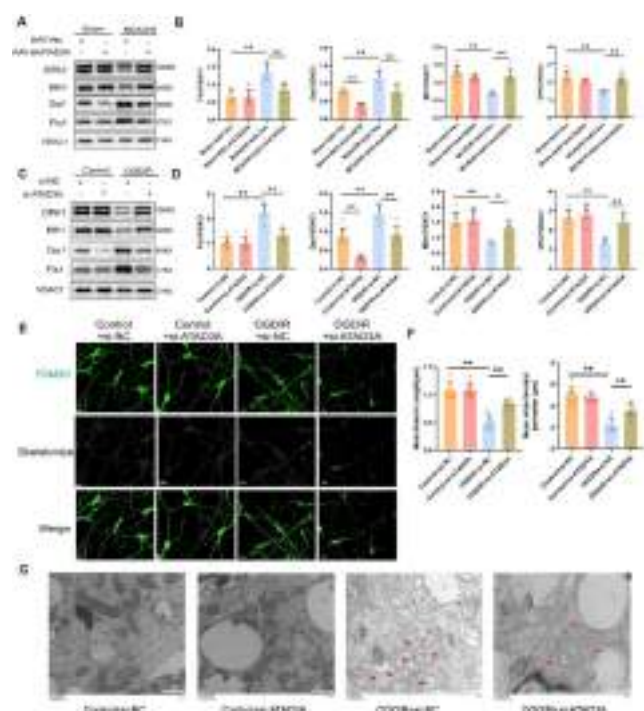


FIGURE 2 ATAD3A promotes mitochondrial fission and suppresses mitochondrial fusion.

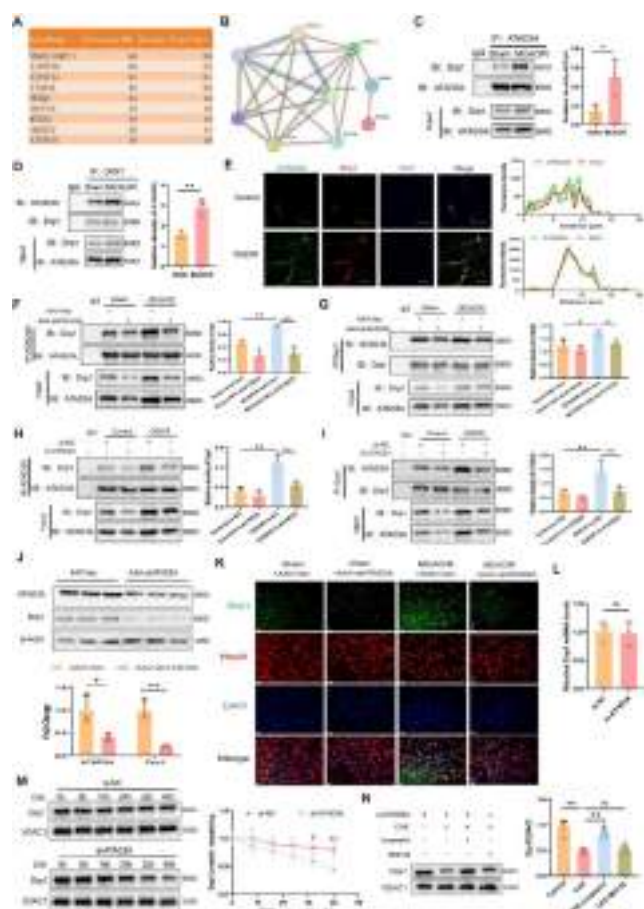


FIGURE 3 ATAD3A interacts with and stabilizes Drp1 by inhibition of lysosome-mediated degradation.

EPO-0197 | Recovery of post-stroke dysphagia: A 16-month follow-up study in 455 post stroke patients using the Functional Oral Intake Scale (FOIS)

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Background and aims: Tracking swallowing function with validated tools is essential for understanding recovery patterns and guiding care. To evaluate the progression and predictors of dysphagia recovery in stroke patients over 16 months using the Functional Oral Intake Scale (FOIS).

Methods: This prospective cohort study included 455 patients with ischemic or hemorrhagic stroke and clinically confirmed dysphagia, admitted between June 2023 and January 2025. Swallowing ability was assessed at baseline, 1, 3, 6, 12, and 16 months post-stroke using the FOIS score. Recovery was defined as reaching FOIS level ≥ 6 . Multivariate logistic regression identified factors associated with persistent dysphagia (FOIS ≤ 5) at 16 months.

Results: At baseline, all patients scored between FOIS 1 and 3. At 3 months, 54.1% had reached FOIS ≥ 4 . By 6 months, 68.3% had achieved FOIS ≥ 6 , and by 16 months, 82.2% of patients reached FOIS levels 6–7, indicating functional oral intake. Persistent dysphagia (FOIS ≤ 5) at 16 months was independently associated with brainstem stroke (OR 3.5, $p < 0.001$), higher NIHSS scores on admission ($p = 0.003$), and age > 75 ($p = 0.01$). A total of 28 patients (6.2%) remained dependent on partial or full enteral feeding at the end of the follow-up.

Conclusion: The majority of patients with post-stroke dysphagia demonstrate significant improvement on the FOIS within the first 6 months, with gradual gains continuing through 16 months. Severe stroke, brainstem involvement, and advanced age are predictors of poor recovery.

Disclosure: Nothing to disclose.

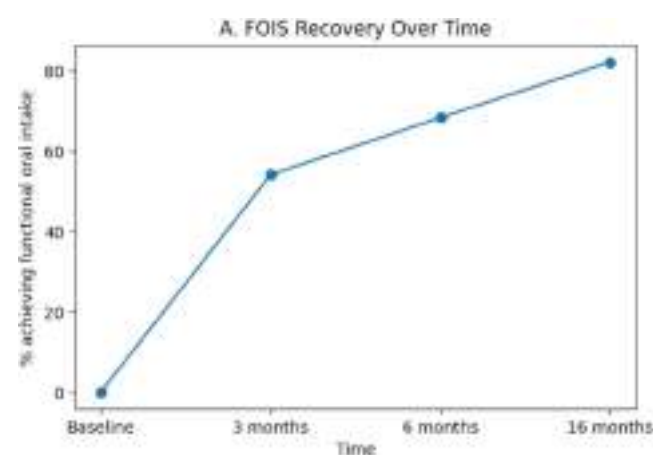


FIGURE 1 FOIS in months.

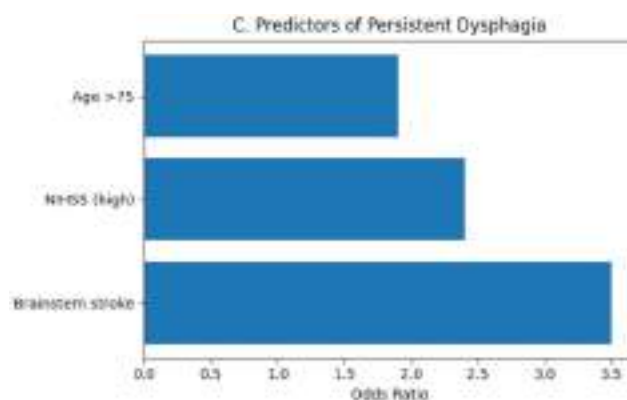


FIGURE 2 Predictors of persistent dysphagia.

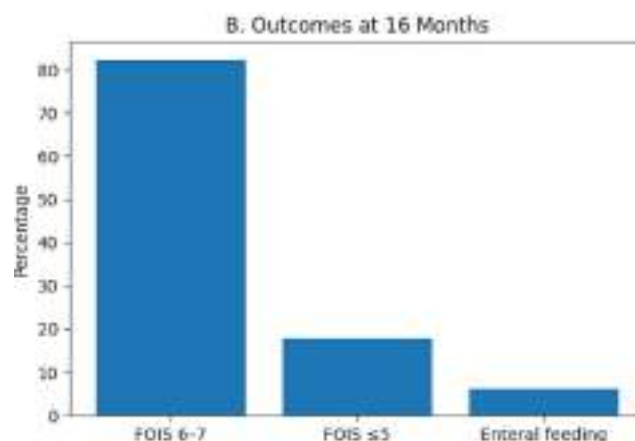


FIGURE 3 Outcomes over time.

EPO-0198 | Explainable machine learning-based prediction of poor functional outcome after ischemic stroke using routine clinical data

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Background and aims: Early prediction of poor functional outcome after ischemic stroke remains challenging. Conventional prognostic models show limited performance in heterogeneous real-world populations. We aimed to evaluate whether a machine learning model using routinely available clinical variables improves prediction of poor functional outcome compared with logistic regression.

Methods: We retrospectively analyzed 642 ischemic stroke patients with available baseline clinical, laboratory, electrocardiographic, and imaging data. Poor functional outcome was defined as modified Rankin Scale score greater than 3 at follow-up. Data were split into training and test sets. A Random Forest classifier was developed and compared with logistic regression. Model performance was evaluated using receiver operating characteristic area under the curve. Model interpretability was assessed using SHapley Additive exPlanations.

Results: The Random Forest model showed good discriminative performance with a receiver operating characteristic area under the curve of 0.87 on the test set, outperforming logistic

regression. Sensitivity and specificity varied according to probability thresholds. SHapley Additive exPlanations identified stroke severity, mortality, functional status, and selected laboratory markers as key contributors to model predictions.

Conclusion: Explainable machine learning using routine clinical data improved prediction of poor functional outcome after ischemic stroke. Explainable models may support individualized risk stratification in clinical practice.

Disclosure: Nothing to disclose.

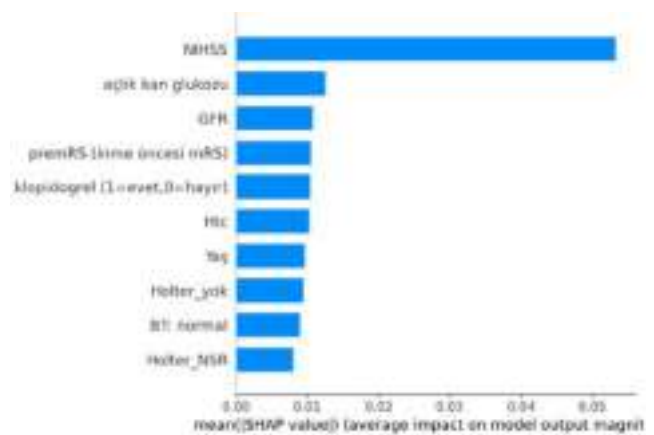


FIGURE 1 Mean absolute SHAP values showing the relative contribution of each variable to poor outcome prediction. NIHSS had the strongest impact, followed by glucose, renal function, premorbid disability, and age.

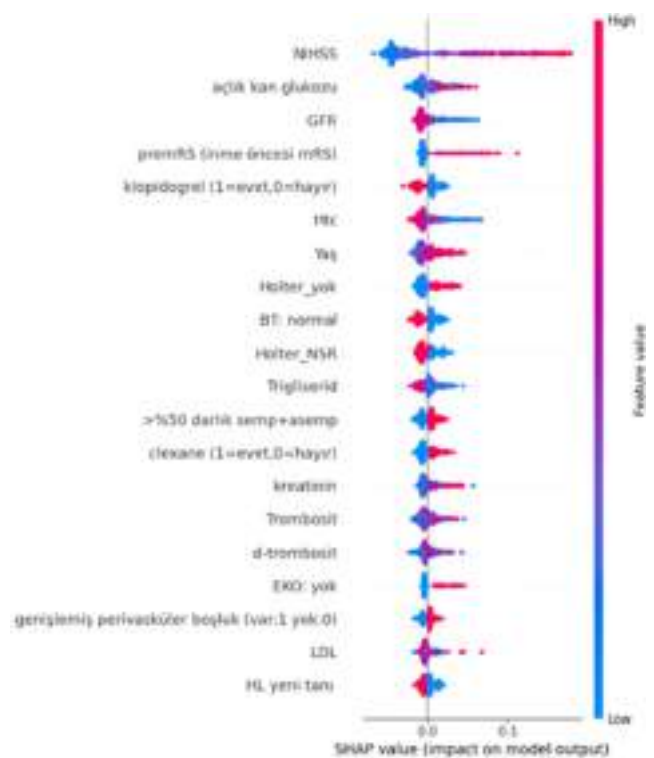


FIGURE 2 SHAP summary plot illustrating direction and magnitude of feature effects. Higher NIHSS and glucose values increased poor outcome risk, while better renal function shifted predictions toward favorable outcome.

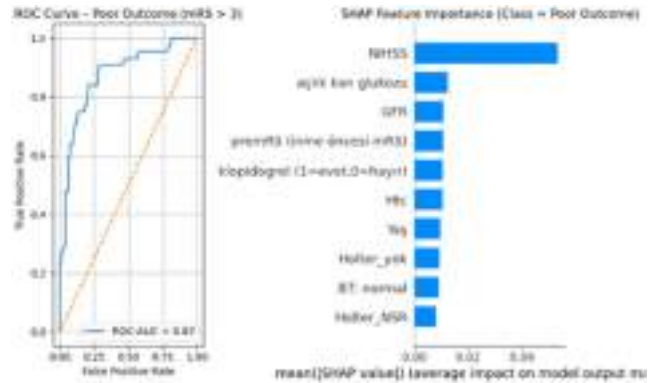


FIGURE 3 Receiver operating characteristic curve demonstrating strong discrimination for poor outcome (area under the curve 0.87). SHAP importance confirms NIHSS as the dominant predictor.

EPO-0199 | The impact of advanced practice nurse-led follow-up model on medication adherence for secondary prevention in patients with acute ischaemic stroke

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Background and aims: This study explores the impact of a follow-up model led by advanced practice nurses on medication adherence for secondary prevention in patients with acute ischaemic stroke, aiming to provide a reference for long-term medication management in this patient group.

Methods: A follow-up programme led by advanced practice nurses was developed, encompassing both in-hospital and post-discharge management. In-hospital management involved advanced practice nurses providing structured health education and guidance during the patient's hospital stay. Upon discharge, patients were given personalised medication management plans. Post-discharge management included follow-ups conducted by advanced practice nurses at 1 month, 3 months, 6 months, and 1 year after discharge via telephone, WeChat, and online platforms. During these follow-ups, individualised education and guidance were provided, and assistance with outpatient appointment bookings was offered when necessary.

Results: A total of 190 patients were included. After one year of intervention, the adherence rates to antithrombotic medication at 3 months, 6 months, and 1 year post-discharge were 94.7% (180/190), 94.2% (179/190), and 95.3% (181/190), respectively; adherence rates to statins were 95.3% (181/190), 92.1% (175/190), and 89.5% (170/190), respectively; adherence rates to hypoglycaemic drugs were 100% (52/52), 96.2% (50/52), and 92.3% (48/52), respectively; adherence rates to antihypertensive drugs were 96.5% (110/114), 99.1% (113/114), and 96.5% (110/114), respectively.

Conclusion: The advanced practice nurse-led follow-up model has a positive impact on medication adherence for secondary prevention in patients with acute ischaemic stroke and merits further promotion and application.

Disclosure: Nothing to disclose.

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Background and aims: Venous outflow (VO) is essential for maintaining cerebral hemodynamics. Impaired venous outflow (VO) has been associated with poor outcomes and hemorrhagic transformation in ischemic stroke; however, its prevalence and determinants in acute intracerebral hemorrhage (ICH) remain understudied.

Methods: We retrospectively included patients admitted to Charité University Hospital, Berlin (January 2019-June 2025) with supratentorial ICH and available baseline CT angiography. VO assessment was performed according to the validated Comprehensive Venous Outflow (CVO) score of the ipsilateral hemisphere. The association between clinical and radiological variables and the CVO score was assessed using a multivariable Bayesian beta-binomial model to account for overdispersion in the bounded count outcome. The variables included in the model were age, systolic blood pressure, ICH volume, intraventricular hemorrhage, and edema volume.

Results: A total of 533 patients were included in this analysis. Baseline characteristics revealed a mean age of 73 years (SD 13.8) and mean ICH volume of 41.41 ml (SD 42.9). A total of 300 (56.3%) patients had deep ICH. The median CVO was 8 (IQR 5–8), with a score <6 in 149 (27.9%) patients, indicating significant impairment. ICH volume (aOR for 1 point reduction for 1 SD increase, 1.84, 95%CrI 1.34–2.49), IVH volume (aOR 1.41, 1.27–1.57), and edema volume (aOR 1.29, 0.95–1.77) along with deep location (aOR 1.54, 1.16–2.06) were associated with a decrease in the CVO score.

Conclusion: VO impairment is common in patients with ICH and is associated with ICH features such as ICH volume, edema, and location. Further research is warranted to evaluate its clinical significance.

Disclosure: F. Mazzacane was supported by EAN Research Fellowship grant. A. Dell'Orco: Nothing to disclose. B. Del Bello: Nothing to disclose. G. Busto: Nothing to disclose. E. Fainardi: Nothing to disclose. M. Wattjes: Nothing to disclose. A. Morotti: Nothing to disclose. J. Nawabi: Nothing to disclose.

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Background and aims: Atrial fibrillation (AF) is a major cause of ischemic stroke and is associated with higher rates of recurrence and worse outcomes. Oral anticoagulation is essential for secondary prevention; however, the optimal timing of initiation following acute ischemic stroke remains uncertain due to concerns regarding hemorrhagic complications. This study aims to evaluate the safety and efficacy of early compared with delayed initiation of oral anticoagulant therapy in patients with AF-related ischemic stroke.

Methods: A systematic review and meta-analysis were conducted following PRISMA guidelines. Randomized controlled trials and observational studies involving adult patients with ischemic stroke and non-valvular AF were included. Studies comparing early and delayed initiation of oral anticoagulants were analyzed. Primary outcomes included recurrent ischemic events or transient ischemic attacks, intracranial hemorrhage, major bleeding, and all-cause mortality. Random-effects models were used for pooled analyses, with sensitivity analyses performed to assess result robustness.

Results: Seventeen studies met inclusion criteria, with thirteen eligible for quantitative synthesis, comprising more than 32,000 patients. Early initiation of oral anticoagulation was not

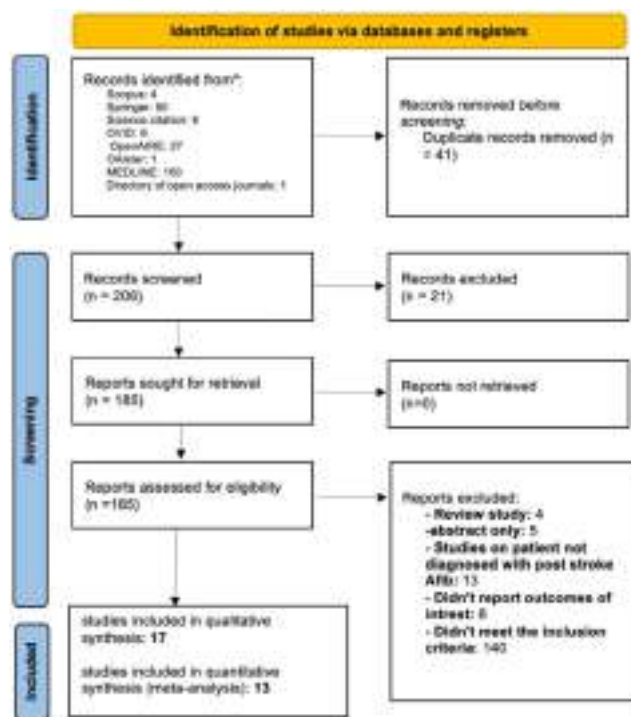


FIGURE 1 PRISMA flow diagram illustrating the study selection process for included randomized controlled trials and observational studies.

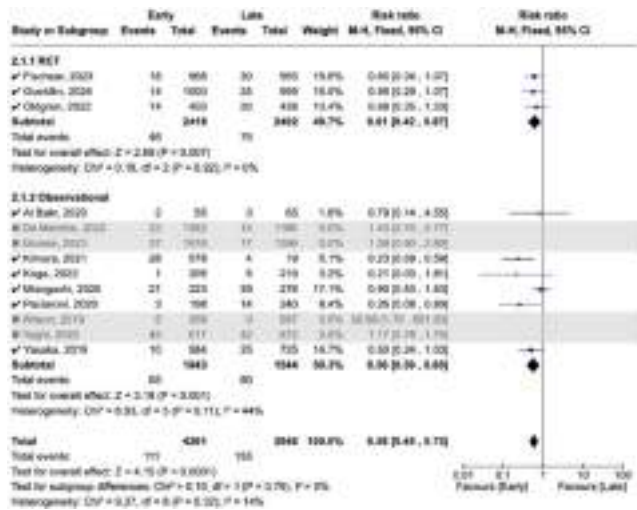


FIGURE 2 Forest plot comparing early versus delayed initiation of oral anticoagulation for ischemic events or transient ischemic attack in patients with atrial fibrillation related ischemic stroke.

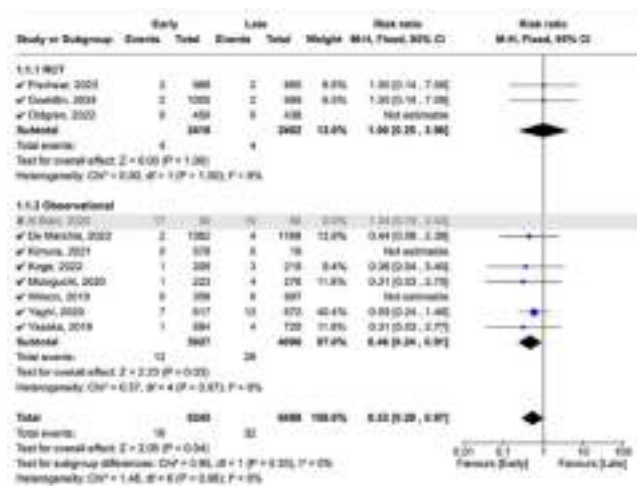


FIGURE 3 Forest plot showing the risk of intracranial hemorrhage associated with early versus delayed oral anticoagulation initiation after atrial fibrillation related ischemic stroke.

associated with increased all-cause mortality or major bleeding compared with delayed initiation. Sensitivity analyses demonstrated a lower risk of recurrent ischemic events without an increased risk of intracranial hemorrhage among patients receiving early therapy.

Conclusion: Early initiation of oral anticoagulation following AF-related ischemic stroke appears safe and may offer superior protection against recurrent ischemic events. These findings support early, individualized, imaging-guided anticoagulation strategies and may inform future clinical guideline updates.

Disclosure: Nothing to disclose.

EPO-0202 | Endovascular treatment for anterior circulation strokes beyond 6 hours: Better late than never

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Background and aims: Endovascular treatment beyond 6 hours has been shown to improve outcomes in selected stroke patients, usually based on advanced imaging. This study aimed to evaluate the outcomes of patients undergoing thrombectomy beyond 6 hours without the use of advanced imaging.

Methods: We retrospectively included patients with anterior circulation strokes who underwent thrombectomy without prior thrombolysis and divided them into late window (LW) and non-late window (NLW) groups. Baseline characteristics included age, sex, National Institutes of Health Stroke Scale (NIHSS), and Alberta Stroke Program Early CT Score (ASPECTS). Outcomes were assessed using parenchymal hemorrhagic transformation (PHT), ASPECTS at 24 hours, and modified Rankin Scale (mRS) at discharge. In the LW group, associations between age, baseline ASPECTS, number of thrombectomy passes and outcomes were analyzed.

Results: A total of 117 patients were included (47 LW, 70 NLW), with no significant baseline differences between groups. No statistically significant differences were observed in PHT rates ($p=0.3649$) or mean ASPECTS at 24 hours ($p=0.12356$). Mean mRS at discharge was $3.4043 (\pm 1.6896)$ in the LW group and $3.9857 (\pm 1.9523)$ in the NLW group ($p=0.05118$). In the LW group, mRS at discharge was not associated with age but showed significant correlations with baseline ASPECTS ($r = -0.30732$, $p=0.03562$) and number of thrombectomy passes ($r=0.41476$, $p=0.00375$).

Conclusion: Thrombectomy beyond 6 hours without advanced imaging resulted in outcomes comparable to those achieved within the conventional time window. Higher baseline ASPECTS and fewer thrombectomy passes were associated with better functional outcomes.

Disclosure: Nothing to disclose.

EPO-0203 | Cardiac ultrasound in stroke by neurologists: A comparative study of diagnostic accuracy

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Background and aims: Cardioembolic stroke is one of the most prevalent causes of ischemic stroke, making it crucial to identify cardiac embolic sources accurately. Transthoracic echocardiography (TTE) is widely used for this purpose, but logistical barriers often delay its use when performed outside neurology departments, potentially, reducing the rate of cardiac imaging in stroke patients. Thus, the objective of the present study was to evaluate the diagnostic accuracy of neurologist-performed TTEs

following specialised training, using cardiologist readings as the reference standard.

Methods: Neurologists were trained using a simplified 10-point echocardiographic protocol developed by the ESNCH Working Group. A total of 74 TTEs performed by neurologists in ischemic stroke patients were compared by cardiologists. Diagnostic sensitivity, specificity, interobserver agreement (Cohen's kappa), accuracy and predictive values were calculated.

Results: Perfect interobserver agreement was found for pericardial fluid ($k=1.0$; accuracy 100%) and left atrial (LA) enlargement ($k=0.832$; sensitivity 90.5%, specificity 93.9%). Moderate agreement was observed for significant valvular dysfunction ($k=0.572$) and left ventricular (LV) function impairment ($k=0.489$). Notably, the negative predictive value (NPV) was high ($\geq 95\%$) for seven out of nine parameters, suggesting Neurologist-performed TTE is a reliable tool for ruling out major structural pathologies.

Conclusion: Trained neurologists can accurately identify significant cardiac abnormalities on TTE relevant to stroke care, with strong specificity across parameters. Neurologist-performed TTE may serve as a reliable diagnostic component in acute stroke settings, improving access and efficiency in interdisciplinary stroke care.

Disclosure: Nothing to disclose.

EPO-0204 | Adjunctive hypothermia after endovascular reperfusion therapy for acute ischemic stroke: Evidence from a meta-analysis

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Background and aims: Endovascular thrombectomy (EVT) is the standard of care for acute ischemic stroke due to large vessel occlusion; however, reperfusion injury limits neurological recovery. Selective or mild therapeutic hypothermia has emerged as a potential neuroprotective adjunct to EVT, aiming to mitigate ischemia-reperfusion injury while preserving procedural safety.

Methods: We conducted a systematic review and meta-analysis of clinical studies evaluating therapeutic hypothermia combined with EVT in patients with acute ischemic stroke. PubMed, Embase, Scopus, and Web of Science were searched from inception to December 2025. Primary outcomes were favorable functional outcome (modified Rankin Scale [mRS] 0–2 at 90 days). Secondary outcomes included excellent functional outcome (mRS 0–1), mortality, and symptomatic intracranial hemorrhage (sICH).

Results: Seven studies comprising 603 patients were included, of whom 294 received therapeutic hypothermia combined with EVT and 309 underwent thrombectomy alone. The addition of hypothermia was associated with a significantly higher rate of favorable functional outcome at 90 days (mRS 0–2: 59.2% vs 49.2%; RR=1.23, 95% CI 1.06–1.42). Patients treated with hypothermia also demonstrated a greater likelihood of excellent functional recovery (mRS 0–1: 48.6% vs 33.1%; RR=1.45, 95% CI 1.12–1.88). There were no significant differences between groups in mortality (RR=0.97, 95% CI 0.75–1.26) or symptomatic intracranial hemorrhage (RR=0.78, 95% CI 0.50–1.21). Statistical heterogeneity was low to moderate across outcomes.

Conclusion: Therapeutic hypothermia combined with EVT is associated with improved functional outcomes without increased mortality or hemorrhagic risk. Larger randomized trials are needed to confirm efficacy and optimize cooling strategies.

Disclosure: Nothing to disclose.

EPO-0205 | MicroRNA-based point-of-care potential for stroke subtype differentiation

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Background and aims: Timely differentiation between acute ischemic stroke and intracerebral hemorrhage is essential for safe treatment. However, neuroimaging may delay care in resource-limited settings. Circulating microRNAs (miRNAs) are stable and linked to cerebrovascular injury, making them promising diagnostic markers.

Methods: Adults presenting within six hours of stroke onset were enrolled before neuroimaging, with STARD guidelines as reference. The diagnosis was confirmed with standard imaging. Blood samples were collected at presentation and analyzed via next-generation sequencing to identify miRNA, then validated by qRT-PCR. Participants included patients with acute ischemic stroke ($n=15$), intracerebral hemorrhage ($n=15$), and healthy controls ($n=30$). miRNA cycle thresholds were normalized. Diagnostic performance was evaluated using nonparametric analysis, ROC curves, PCA, and machine-learning classifiers. Pathway enrichment supported biological plausibility.

Results: Distinct circulating miRNA expression patterns were observed across stroke subtypes and controls. miR-21 and miR-146a were significantly downregulated, whereas miR-155 was upregulated in stroke cohorts relative to healthy individuals. Diagnostic performance analyses demonstrated robust discriminatory capacity, with miR-21 achieving the highest classification accuracy (AUC=0.80). PCA revealed clear separation between ischemic, hemorrhagic, and control groups. Among machine-learning approaches, Random Forest models achieved superior predictive performance, particularly in differentiating intracerebral hemorrhage from controls (AUC=0.79). Pathway analyses linked these miRNAs to inflammatory signaling, immune modulation, and cell survival mechanisms relevant to acute cerebrovascular injury.

Conclusion: Circulating miRNA signatures enable early discrimination between ischemic and hemorrhagic stroke before imaging confirmation. These findings support further validation of miRNA-based assays as adjunctive diagnostic tools to accelerate stroke triage and precision care.

Disclosure: Nothing to Disclose.

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Background and aims: Intracranial atherosclerotic stenosis (ICAS) and chronic kidney disease (CKD) are both significant contributors to stroke risk and poor outcomes.

Methods: In this retrospective observational cohort study, 488 first-ever ischemic stroke patients from the Bernese Stroke Registry (April 2021 – March 2023) were analyzed. Patients were stratified into four groups based on ICAS presence and eGFR: no ICAS/eGFR ≥ 60 ($n = 226$), no ICAS/eGFR < 60 ($n = 73$), ICAS/eGFR ≥ 60 ($n = 107$), and ICAS/eGFR < 60 ($n = 82$). Data on demographics, vascular risk factors, stroke aetiology, ICAS location, acute treatment, and 3-month outcomes (mRS) were analyzed.

Results: Patients with ICAS and eGFR < 60 were the oldest (median 83.9 years, range 55.4–99.5) and had significantly worse renal function (median creatinine 117 $\mu\text{mol/L}$, median eGFR 44 ml/min/1.73 m^2) compared to other groups ($p < 0.001$). Arterial hypertension was significantly more prevalent in the ICAS/eGFR < 60 group (85.4%) than in the no ICAS/eGFR ≥ 60 group (63.1%) ($p < 0.001$). Diabetes mellitus prevalence was highest in the ICAS/eGFR < 60 group (37.8%, $p = 0.009$). According to TOAST classification, large artery atherosclerosis was the dominant stroke mechanism in ICAS groups (44.9% in ICAS/eGFR ≥ 60 vs. 7.5% in no ICAS/eGFR ≥ 60 , $p < 0.001$). At 3-month follow-up, patients with ICAS/eGFR < 60 had the poorest functional outcomes, with only 58.2% achieving a favourable mRS (0–2) compared to 81.3% in the no ICAS/eGFR ≥ 60 group ($p < 0.001$).

Conclusion: The co-existence of ICAS and impaired renal function identifies a high-risk stroke group characterized by a greater burden of vascular risk factors, large artery atherosclerosis as stroke aetiology, and worse short-term functional recovery. These findings underscore the importance of integrated vascular risk assessment and tailored management strategies in this vulnerable patient group.

Disclosure: Nothing to disclose.

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Background and aims: Hematoma growth occurs in approximately one-third of patients with intracerebral hemorrhage (ICH) and is associated with a high rate of morbidity and mortality. However, radiological hematoma growth does not always coincide with neurological deterioration. This study aimed to differentiate symptomatic and asymptomatic hematoma growth and examine their respective clinical implications using data from the Antihypertensive Treatment of Acute Cerebral Hemorrhage 2 (ATACH-2) trial.

Methods: Among 917 ICH patients (568 male patients) from ATACH-2 with complete clinical and imaging data, hematoma growth was defined as a $> 33\%$ relative or $> 6 \text{ mL}$ absolute increase in volume on 24-hour computed tomography. Symptomatic hematoma growth was defined by concurrent neurological deterioration (≥ 1 -point NIHSS increase), while asymptomatic growth occurred without NIHSS worsening. Associations between hematoma growth types and 90-day functional outcomes (modified Rankin Scale [mRS]) were evaluated using multivariable logistic regression.

Results: Symptomatic hematoma growth occurred in 5.9% and asymptomatic growth in 16.7% of patients. Both were independently associated with poor functional outcomes (mRS 4–6), with the strongest effect observed for symptomatic growth (adjusted OR 6.891; 95% CI 3.411–13.921). Mortality was highest among symptomatic patients (25.9%), compared with asymptomatic (10.5%) and no-growth groups (3.7%). Larger baseline hematoma volume, ultra-early hematoma growth, and intraventricular hemorrhage growth were significantly more frequent in symptomatic hematoma growth.

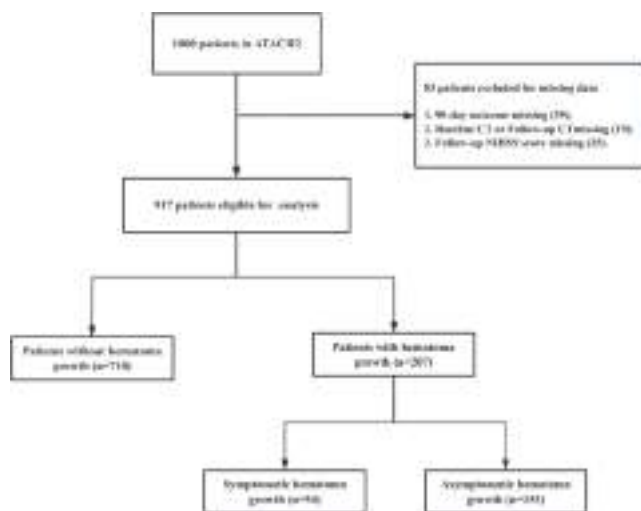


FIGURE 1 Flow diagram of the study participants.

TABLE 1 Relationship between different types of hematoma growth and prognosis.

Variable	Subtype	Adjusted odds ratio	95% confidence interval	P value
90-day major disability (mRS 3-5)	Asymptomatic	1.307	0.764-1.987	0.341
	Symptomatic	1.844	1.073-3.527	0.029
90-day unfavorable outcome (mRS 3-6)	Asymptomatic	1.444	0.977-2.135	0.063
	Symptomatic	10.659	4.032-28.041	<0.001
90-day poor outcome (mRS 4-6)	Asymptomatic	2.114	1.402-3.188	<0.001
	Symptomatic	6.891	3.411-13.921	<0.001
90-day mortality (mRS 5)	Asymptomatic	2.810	1.402-5.632	0.004
	Symptomatic	7.840	3.443-17.848	<0.001

mRS: modified Rankin Scale.

Conclusion: Asymptomatic hematoma growth is not uncommon and predicts poor outcomes after ICH, although symptomatic growth carries a substantially higher risk of death and disability. Early identification through integrated clinical-radiological assessment and vigilant monitoring may improve prognostication and facilitate timely intervention.

Disclosure: JNG has received consulting from AstraZeneca, CSL Behring, Octapharma, Takeda, and Cayuga.

EPO-0208 | Morbidity and mortality of ruptured cerebral aneurysms in patients aged over 80 years: A systematic review and meta-analysis

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Background and aims: Aneurysmal subarachnoid hemorrhage (aSAH) in patients over 80 years is associated with high mortality and morbidity, yet evidence guiding management in this population is limited. As life expectancy increases,

clarifying outcomes of different treatment strategies in very elderly patients has become increasingly important.

Methods: PubMed, Embase, Scopus, and Web of Science were systematically searched till October 2025 in accordance with PRISMA guidelines. Studies reporting outcomes of ruptured intracranial aneurysms in patients aged ≥ 80 years were included. Primary outcomes were favorable functional outcome and mortality and complications of ruptured cerebral aneurysms. Clinical severity was assessed using Hunt & Hess, or WFNS grading systems. Random-effects meta-analyses were performed, and study quality was evaluated using the ROBINS-I tool.

Results: 26 retrospective studies including 14,469 patients were analyzed; approximately two-thirds were female, and the mean age was 84.3 years. Surgical clipping was performed in 4,863

TABLE 1 Favorable Outcome in SAH patients ≥ 80 years old.
Meta-Analysis: Favorable Outcome in SAH Patients ≥ 80 Years

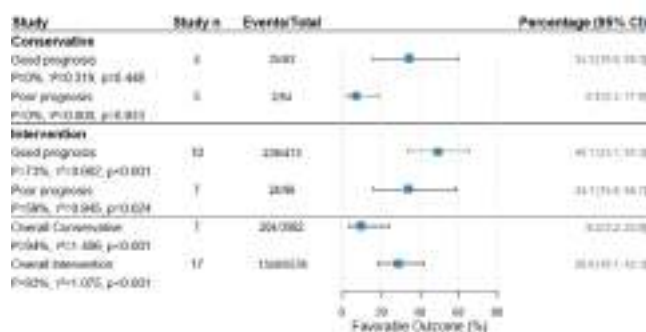


TABLE 2 Mortality in SAH patients ≥ 80 years old.

Meta-Analysis: Mortality in SAH Patients ≥ 80 Years

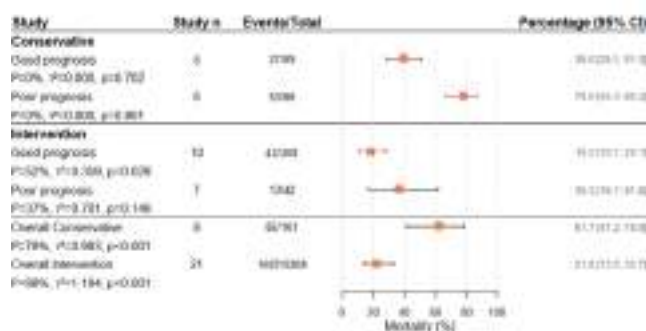
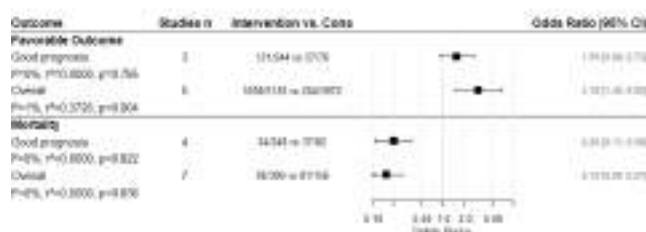


TABLE 3 Odds Ratios between Intervention and Conservative management in SAH patient ≥ 80 years.

Meta-Analysis: Odds Ratios (Intervention vs. Conservative) in SAH Patients ≥ 80 Years



patients, endovascular treatment in 5,444, and conservative management in 4,135. Among 1,252 patients with reported admission grade, 577 had poor-grade SAH. Interventional treatment was associated with a higher pooled rate of favorable outcomes than conservative management (28.6% vs 9.2%) and significantly improved odds of favorable outcome (OR 3.18) and reduced mortality (OR 0.16). Benefits were greatest in patients with good initial clinical grades. The pooled complication rate was 49%, with cerebral vasospasm and hydrocephalus each affecting about one-quarter of patients.

Conclusion: Despite high overall mortality, selected very elderly patients, particularly those with good clinical grades, may benefit from interventional treatment. Treatment decisions should be individualized rather than based on age alone.

Disclosure: Behnam Rezai Jahromi and Mika Niemelä have patents pending on aneurysm imaging.

Neuropathies 2

EPO-0209 | Management of disabling spasticity with functional neurosurgery (selective microsurgical neurotomy)

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Background and aims: Spasticity is one of the most common sequelae of central neurological disorders. It is defined as a motor disorder characterized by a velocity-dependent increase in the tonic stretch reflex associated with an exaggeration of deep tendon reflexes. When disabling spasticity is focused on one or a few muscle groups, selective peripheral neurotomy may be considered.

Methods: Before undergoing these surgical programs, patients undergo a complete preoperative assessment using a block test of the targeted nerves using local anesthetics. After an approach tailored to the targeted nerve, the first step is microsurgical dissection of the nerve branches and the fascicles of each branch. The study of motor responses allows for the differentiation of motor fascicles from sensory fascicles, and then hyperexcited fascicles from others. After this identification, the fascicles are partially transected according to the preoperative plan.

Results: In our series, patients who underwent selective neurotomy of the upper and lower limbs experienced significant suppression or reduction of spasticity and a reduction or disappearance of spasticity-induced pain. Functional improvement was significant (upper limb flexibility, hand and finger opening, limb reuse, improved gait and foot stability during stance and walking), with improvements in body image, comfort when dressing, and shoe fitting.

Conclusion: In the management of spasticity, selective microsurgical neurotomy has predictable effects based on preoperative testing. By eliminating excessive spasticity, correcting abnormal postures, and reducing associated pain, neurotomy allows for the resumption of active and effective rehabilitation

and, in some cases, the reappearance or improvement of voluntary and useful motor skills.

Disclosure: Selective Microsurgical Neurotomy is a modular and adaptable technique for each patient. It allows for the long-term reduction of focal spasticity at the cost of low morbidity.

EPO-0210 | Silent dysphagia in CANVAS: Videofluoroscopic abnormalities despite low EAT-10

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Background and aims: Dysphagia has been reported within the clinical spectrum of cerebellar ataxia, neuropathy and vestibular areflexia syndrome (CANVAS), but it is often unrecognized and not systematically assessed. The objective of this study was to characterize dysphagia in genetically confirmed CANVAS and evaluate concordance between symptom-based screening and instrumental assessment.

Methods: We conducted a retrospective case series of 12 adult patients with CANVAS. All patients underwent dysphagia screening using the Eating Assessment Tool-10 (EAT-10) and videofluoroscopic swallowing study (VFSS). VFSS reports were reviewed for swallowing safety (laryngeal penetration, aspiration, cough reflex) and efficiency (pharyngeal residue), with analysis according to bolus consistency.

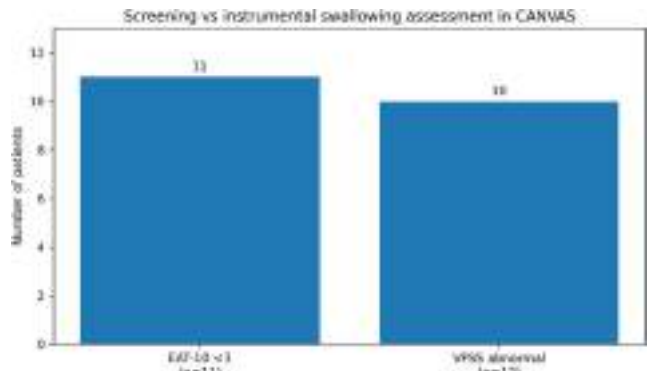


FIGURE 1 Screening versus instrumental swallowing assessment in CANVAS.

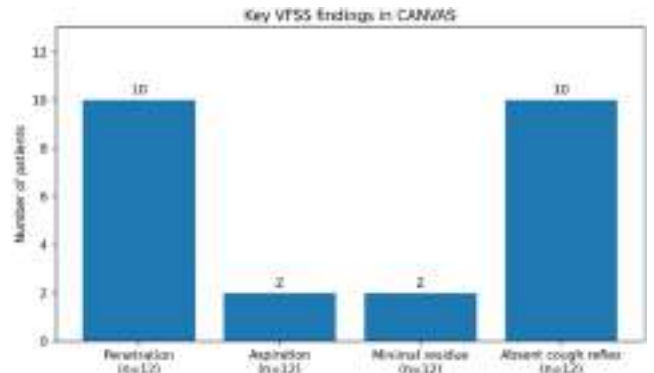


FIGURE 2 Key videofluoroscopic swallowing findings in CANVAS.

Results: The cohort comprised 12 patients (8 women), with a mean age of 66.5 years. Chronic cough was present in most patients (10/12). All patients exhibited sensory axonal polyneuropathy and bilateral vestibular hypofunction, consistent with the CANVAS phenotype. Cerebellar involvement, defined by neurological examination and/or brain MRI findings, was identified in 2/12 patients. EAT-10 scores were below the pathological cut-off (<3) in 11/12 patients, while one patient had advanced dysphagia requiring percutaneous endoscopic gastrostomy. In contrast, VFSS demonstrated objective abnormalities in 10/12 patients (83.3%) (Figure 1) Laryngeal penetration was predominantly observed with thin liquids. All patients with abnormal VFSS lacked an effective cough reflex (10/10, 100%). Aspiration occurred in 2/12 patients (16.7%). Minimal pharyngeal residue was reported in 2/12 patients (16.7%) (Figure 2).

Conclusion: In RFC1-CANVAS, dysphagia appears to follow a silent progression, with normal EAT-10 scores preceding objective videofluoroscopic abnormalities and aspiration risk.

Disclosure: Nothing to disclose.

EPO-0211 | Clinical characteristics and therapeutic response in patients with autoimmune nodopathies: A multi-center study from the US

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Background and aims: Autoimmune nodopathies (AINs) with circulating IgG4 autoantibodies targeting nodes of Ranvier antigens are rare. About 5–10% of patients with electrodiagnostic features of a demyelinating neuropathy have autoimmune nodopathies. AINs usually have distinct clinical features and often don't respond to intravenous immunoglobulin (IVIg), but improve with B-cell-depleting therapy. Most of the current literature on AIN came from Europe and Japan, and data from the US is limited.

Methods: This multi-center study, including over 12 centers in the US, is actively capturing the clinical features and therapeutic responses of patients with AINs.

Results: To date, we have included 33 patients, 25 (76%) with anti-neurofascin 155 (NF155) and six (18%) with anti-Contactin IgG4 autoantibodies, and two patients positive for both autoantibodies. The average age of disease onset was 50.3 ± 19.6 years, and 18 (54.5%) patients were male. About 55% of patients had both distal and proximal weakness, and 36% had predominant distal weakness. Distal loss of vibration sensation and ataxia were common. Areflexia was observed in >80% patients. Over 90% of this cohort received IVIg, but only 33% had partial benefit. Steroids were used in 50% of cases, neonatal Fc receptor

(FcRn) inhibitors in 13% of cases, and PLEX in 20% of cases. Over 70% of patients were treated with rituximab, and most showed objective benefit on clinical examination.

Conclusion: This important collaborative effort will provide new insights into the clinical characteristics of AIN and critical information on management strategies.

Disclosure: CK, MW, WK, DL, AD, have nothing to disclose. KG reports consulting or Adjudication committee for the following: Sanofi, Immunovant, Takeda, Argenx, CSL Behring, Grifols, Annexon, Dianthus C.Karam reports consultant and/or advisory board Accelleron: Alpine, Alexion, Alnylam, Amgen, Amicus, Annexon, Applied Therapeutics, Argenx, Astra Zeneca, Catalyst, Corino, Biogen, CSL Behring, Genentech, Immunovant, Intellia, Ionis, J&J, Medison, Neuroderm, Novo Nordisk, Nuvig, Octapharma, Pfizer, Sanofi, UCB, Takeda, Vertex, and Zai Lab. AM reports to be on the scientific advisory board for Candid Therapeutics MMD received consultancy fees, payment or honoraria for lectures, presentations, manuscript writing or educational events, or participated on a Data Safety Monitoring Board or Advisory Board for AAN, AANEM, Abcuro, Amicus, Annexon, ArgenX, Astellas, Astra Zeneca, Cabaletta Bio, Candid Therapeutics, Catalyst, Covance/Labcorp/Fortrea, CSL-Behring, Creyon Bio, Inc, Dianthus, EMD Serono/Merck, Horizon, Ig Society, Inc, Janssen, Kriya Therapeutics, Inc, Medlink, Nuvig, Octapharma, Sanofi Genzyme, Shire Takeda, and TACT/Treat NMD, UCB and Vertex. MMD has received grants or from Abcuro, Alexion/ AstraZeneca, Alnylam Pharmaceuticals, Amicus, ArgenX, Biosensics, Bristol-Myers Squibb, Cabaletta Bio, Catalyst, CSL-Behring, EMD Serono, FDA/OOPD, GlaxoSmithKline, Genentech, Grifols, Horizon Immunovant, Mitsubishi Tanabe Pharma, MDA, NIH, Nkarta, Novartis, Octapharma, Orphazyme, Priovant, Ra Pharma/UCB Biopharma, Sanofi Genzyme, Sarepta Therapeutics, Shire Takeda, Spark Therapeutics, and The Myositis Association. MMD received royalty for manuscript writing from Wolters Kluwer Health/UpToDate and from Elsevier and for discovery relating to health care from Zevra Therapeutics. NC reports to serve as a consultant for argenx, sanofi, amicus, and arcclx. JA has received honoraria/consultation fee or being in the advisory boards of advisory board: Akcea therapeutics, Amgen, Argenx, Alexion, Alnylam, Astra Zeneca, Annexon, BioCryst, CSL Behring, Grifols, Immunovant, Immunopharma, ImmunoAbs, Octapharma, Dianthus, Sanofi, Johnson & Johnson, Pfizer, Takeda. BR reports serving as a consultant/advisor for Alexion (now part of AstraZeneca), Takeda, Sanofi, and argenx. Additionally, BR has received research support from the Martin Shubik Fund for IBM at Yale University, NIH, Abcuro Pharmaceuticals, argenx, Immunovant, and Takeda. BR has small stocks in Cabaletta Bio, CAVA, and Pfizer.

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Background and aims: Chronic inflammatory demyelinating polyneuropathy (CIDP) is a heterogeneous immune-mediated neuropathy requiring long-term immunomodulatory treatment. Although different effective therapies are available, real-world data on treatment-sequencing, dosing patterns, and escalation strategies remain limited.

Methods: This multicenter retrospective observational study included patients with CIDP fulfilling the 2021 EAN/PNS diagnostic criteria treated at specialized German centers (Neuritis Network). Individual treatment sequences and dosages across consecutive therapy lines were reconstructed. Disability and functional impairment were assessed using the Inflammatory Neuropathy Cause and Treatment (INCAT) score and the Inflammatory Rasch-built Overall Disability Scale (RODS).

Results: 511 patients were included (median age at diagnosis 59 years, 29% female) with 79% diagnosed with typical CIDP and a median follow-up of 80 months. Initial treatment consisted mostly of intravenous immunoglobulins (IVIg, 47%) or intravenous glucocorticoids (46%). While most patients starting on IVIg remained on immunoglobulin therapy, the majority of those initially treated with glucocorticoids later switched to IVIg. Over the disease course, IVIg was used in 90% of patients, intravenous glucocorticoids in 56%, subcutaneous immunoglobulins (SCIg) in 17%, and oral glucocorticoids in 8%. At data cut-off, 62% of patients were treated with IVIg and 17% with SCIg. IVIg dosing was highly variable, ranging from 0.12 to 2.9 g/kg body weight per four weeks. Escalation therapies were applied in 24% of patients, most commonly rituximab.

Conclusion: These findings highlight immunoglobulin therapy as the cornerstone of CIDP management in real-world practice, while heterogeneous dosing patterns and frequent escalation underscore ongoing disease activity and the need for more targeted therapeutic options.

Disclosure: The study received no external commercial or institutional funding. FFK received honoraria for lectures and travel compensation from Alexion, Argenx, Merck, Novartis, and Takeda. He received research support as fellow of the German Research Foundation (DFG)– and Hannover Medical School (MHH)-funded Clinician Scientist Program (PRACTIS) at MHH and from Merck, Siemens and Erwin-Röver-Foundation. KK received honoraria for lectures and travel compensation from Argenx and AstraZeneca. ALF holds shares in Novartis, Novo Nordisk, Coloplast; received consulting fees by Takeda and travel expenses reimbursement by CSL Behring. FS received travel/accommodation/meeting expenses from Alexion Pharmaceuticals and argenx and received speaking honoraria and honoraria for attendance at

advisory boards from Alexion Pharmaceuticals, argnx and UCB pharma. She receives financial research support (paid to her institution) from Alexion Pharmaceuticals and argenx. She serves as a member of the medical advisory of the German Myasthenia Gravis Society e.V. KFJ received research support from Else Kröner Fresenius Foundation and travel compensation and congress fee from Neuraxpharm, Merck and Novartis. SG reports research support from Alnylam Pharmaceuticals, CSL Behring, Else Kröner Fresenius Foundation, Deutsche Forschungsgemeinschaft and Hannover Biomedical Research School (HBRS) and consulting and/or speaker honoraria from Alexion, Alnylam Pharmaceuticals, AstraZeneca, CSL Behring, GSK, Pfizer, Merck and Takeda Pharmaceuticals. TG reports no conflict of interest. RK declares stock ownership from Novo Nordisk; research funding from LFB Group France and Ruhr-University Bochum; travel funding from Grifols, Takeda, and Alnylam; and speaker honoraria from Argenx. JM declares stock ownership from Amgen, Bayer, and Sanofi; travel grants from Alnylam, Biogen Idec, Novartis, Teva, Eisai, Neuraxpharm, Bristol Myers Squibb, and Kyverna; consulting fees from Novartis and Alnylam; and research funding from Klaus Tschira Foundation, Ruhr-University Bochum (FoRUM program), Deutsche Multiple Sklerose Gesellschaft, Hertie Foundation, Novartis, and Kyverna. AG reports research support from CSL Behring, Canon; honoraria for lectures, travel support for meeting attendance, and/or consultancy fees from Alexion, Alnylam, Amgen, argenx, CSL Behring, Grifols, Johnson and Johnson, Roche, Sanofi, Takeda and UCB. KP received research funding from Biogen, CSL Behring, Grünenthal, Argenx, Ruhr University Bochum and speaker honoraria and travel support from Grifols, Argenx, Novartis and Sanofi. TS reports research support from Alnylam, CSL Behring, Merck, Novartis, Siemens; honoraria for lectures, travel support for meeting attendance, and/or consultancy fees from Alexion, Alnylam, argenx, Bayer, Biogen, Bristol Myers Squibb, Centogene, CSL Behring, Grifols, Hexal AG, Horizon, Janssen, Merck, Novartis, Pfizer, Purpose Pharma, Roche, Sanofi, Siemens, SOBI, Teva, Viatriis.

EPO-0213 | Therapeutic effects of repeated immunoadsorption in CIDP: A long-term, prospective, observational study

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Background and aims: Corticosteroids, intravenous immunoglobulins (IVIGs), and plasma exchange (PLEX) constitute the main therapeutic options in CIDP, but immunoadsorption (IA) represents a noteworthy alternative to PLEX due to its excellent tolerability. In this study, we sought to evaluate the short- and long-term therapeutic effects of IA in CIDP compared to PLEX and preceding therapies (IVIGs and corticosteroids).

Methods: Between 02/12/2013 and 03/03/2025, we prospectively evaluated the course of disease of patients with CIDP who received at least one cycle of IA or PLEX. One cycle of IA or PLEX consisted of 5 treatments on 5 consecutive days. As the primary outcome parameter, we used a combined CIDP score of 3 validated scales comprising disability (Inflammatory Neuropathy Cause and Treatment (INCAT) score), motor score (Medical Research Council, MRC), and vibration sensitivity (tuning fork test).

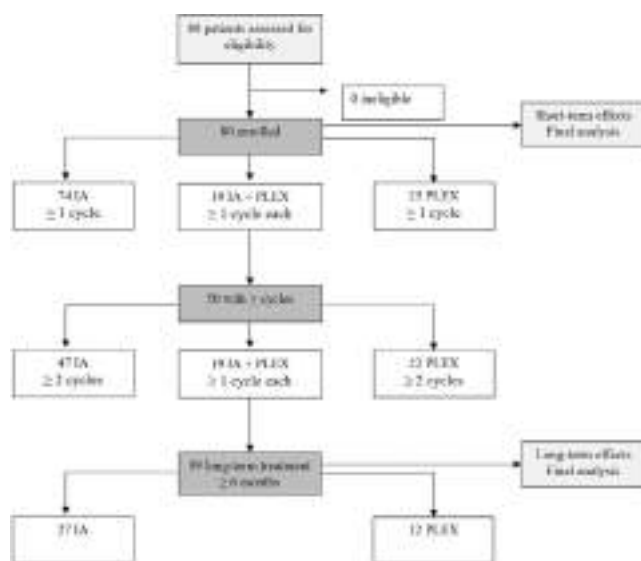


FIGURE 1 Study flow diagram.

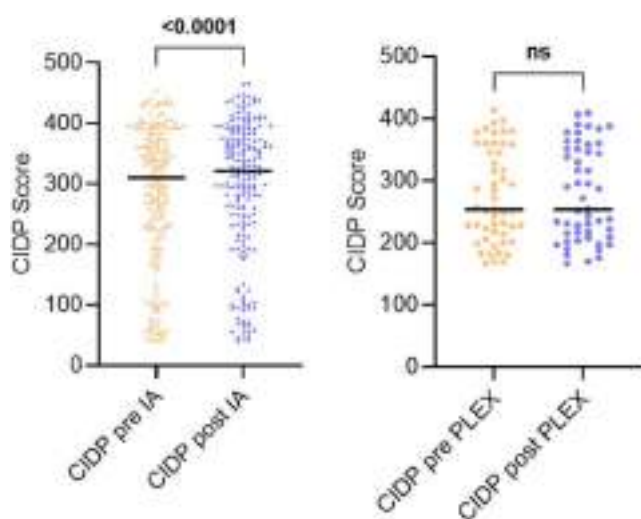


FIGURE 2 Short-term effects.

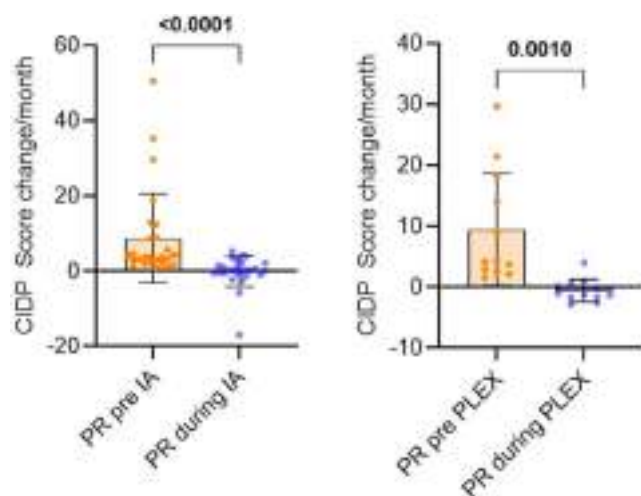


FIGURE 3 Long-term effects.

Results: In total, 80 patients were included. We observed improvements of CIDP scores post-treatment in the IA group (median improvement from 310 (224–374) to 321 (234–373) points; $p < 0.0001$), but not in the PLEX group (median 254 (214–358) vs. 254 (209–351), $p = 0.12$). Long-term progression rates compared to preceding corticosteroid and IVIG therapies decreased from 3.8 (2.2–9.1) to 0.2 (–0.5–2.2) points/month in the IA group, and from 4.2 (2.6–17.2) to –1.1 (–1.6–0.5) points/month in the PLEX group, corresponding to a clinical stabilization of disease progression.

Conclusion: Our results suggest that repeated IA might constitute promising therapeutic options in patients with CIDP who do not sufficiently respond to corticosteroids and IVIGs, stabilizing the disease in the majority of patients.

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EPO-0214 | Paediatric genetic neuropathies: Characterisation and molecular diagnosis in Central Portugal

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Background and aims: Paediatric genetic neuropathies are a group of heterogeneous neuromuscular diseases that are often diagnosed late. This study aims to characterise patients with genetic neuropathy and evaluate the genetic tests that enabled diagnosis.

Methods: A retrospective study of cases since 2000 was conducted, focusing on those with genetic confirmation, with descriptive analyses of demographic, clinical data and diagnostic methods.

Results: We obtained a sample of 68 patients (53.5% male) with suspected genetic neuropathy, in which we identified pathogenic or likely pathogenic variants in 43 (median age of 20.9 years (IQR 16.0–29.5)). The median age at symptom onset was 4.5 years (1.7–8.0), and the median diagnostic delay was 6.7 years

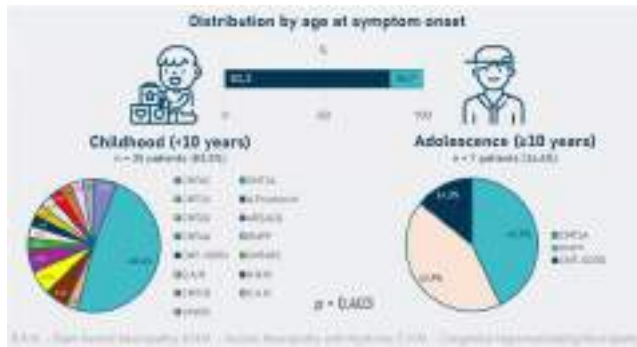


FIGURE 1 Distribution of diagnoses by age at symptom onset.

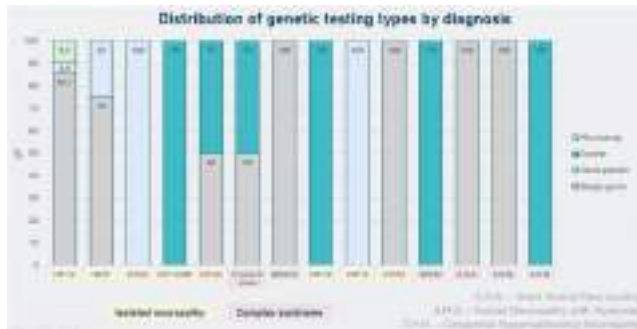


FIGURE 2 Distribution of genetic testing types by diagnosis.



FIGURE 3 Cumulative incidence of diagnosis: Pre- vs. Post-2015 first visit cohorts.

(IQR 2.3–11.8). A family history was present in 50% and consanguinity in 11.6%. Isolated neuropathies accounted for 81.4%, while 18.6% were associated with complex syndromes. Fifteen diagnoses were identified, most commonly CMT1A (48.8%), followed by HNPP (9.3%), and CMT4C, CMT-SORD, CMT2A, Friedreich's ataxia and ARSACS (4.7% each); remaining diagnoses were single cases. Electrophysiology showed demyelinating neuropathy in 58.1%, axonal in 27.9% and intermediate in 2.3%, with sensory-motor involvement in 76.7%. Genetic diagnosis was achieved by single-gene testing in 65.1%, exome sequencing in 16.3%, gene panels in 11.6% and microarray in 4.7%. Prior genetic testing had been performed in 37.2%. Seven (10.8%) variants of uncertain significance were identified, for which we cannot reclassify their pathogenicity.

Conclusion: Paediatric genetic neuropathies show marked heterogeneity and prolonged diagnostic delay. While targeted testing remains valuable in selected scenarios, advanced sequencing techniques are essential in complex cases, supporting individualised diagnostic strategies.

Disclosure: Nothing to disclose.

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Background and aims: Intravenous immunoglobulin (IVIg) is recommended as a first-line therapy for chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) and multifocal motor neuropathy (MMN) according to EAN/PNS and EFNS/PNS guidelines. However, long-term real-world data on IVIg treatment and treatment decision-making are limited and have thus been investigated during this study.

Methods: This non-interventional, multicenter, longitudinal study evaluated treatment patterns in 84 patients (65 typical CIDP, 9 CIDP variants, 10 MMN) treated with IVIg (Gamunex® 10%) across 1,192 visits over up to 18 months. At each infusion cycle, physicians documented criteria for individual treatment decision making with regard of IVIg therapy.

Results: Patient and treatment characteristics are shown in Table 1. Treatment was continued without modification in 1,112 (93.3%) visits of which patients were evaluated clinically stable in 1,051 visits, supported by stable INCAT scores over time (Figure 1). Adaptations for medical reasons occurred in 22/84 (26.2%) patients, which were more frequent in patients with shorter time since diagnosis (≤ 1 year: adaptations in 36.4% of patients; > 1 year: 22.6%) and in patients with CIDP variants (adaptations in 55.6%) compared to typical CIDP (21.5%). Adverse drug reactions occurred in 8/1,176 Gamunex® 10% infusions (0.7%), were mild or moderate, and did not lead to any discontinuation.

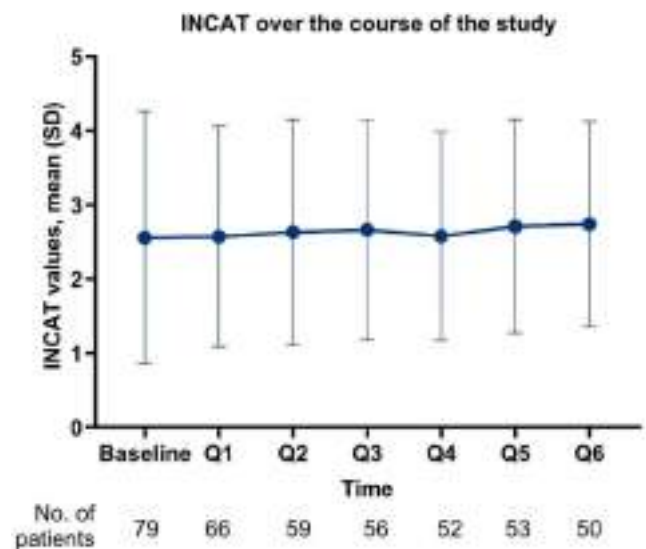


FIGURE 1 INCAT measures remained stable over the course of the study (18 months).

TABLE 1 Patient demographics and treatment characteristics.

Demographics	
Patients, n	84
Typical CIDP	65
Variant CIDP	6
MMN	13
Time since diagnosis, median (range), y	7.0 (0 – 28)
Time in study, median (range), weeks	74.5 (3 – 78)
Visits per patient, median (range), n	16 (2 – 31)
Total documented visits, n	1,192
Previous immunoglobulin, n (%)	79 (94.0)
Time since therapy start with Gamunex® 10%, median (range), y	2.3 (0 – 22.0)
Treatment characteristics	
Loading dose (IVG naïve), mean ± SD, g/kg BW	2.0 ± 0.0
Maintenance dose, median (range), g/kg BW	1.0 (0.2 – 2.0)
Number of infusion days per cycle, mean ± SD	1.5 ± 0.7
Infusion cycle intervals, median (range), days	28 (10 – 126)
Visits with stable dosing and intervals, n/N (%)	1,112/1,192 (93.3)
[Physician's] decision due to stability of the patient, n/N (%)	1,051/1,112 (94.5)

Abbreviations: CIDP, chronic inflammation demyelinating polyradiculoneuropathy; MMN, multifocal motor neuropathy; IVG, intravenous immunoglobulin; BW, body weight; SD, standard deviation.

Conclusion: Therapeutic decisions were individualized, and therapy was adjusted during the observation period in a significant proportion of patients. Most patients remained clinically stable over 78 weeks. IVIg 10% demonstrated a favorable safety profile in real-world practice.

Disclosure: The study was supported by Grifols, manufacturer of Gamunex® 10%. SP: Employee at Grifols FK: Honoraria for lecturing and advisory activities from Abbvie, Stadapharm, Esteve, CSL Behring, Takeda, Zambon, Octapharma, and Argenx. MM: Served in advisory boards for Abbvie, Bial, Desitin, Zambon, UCB, Ipsen and Merz and received honoraria from Abbvie, Bial, UCB, Zambon, Merz, Ipsen, Sanofi-Genzyme, Novartis, Merz and Takeda KP: Research funding from Biogen, CSL Behring, Grünenthal, GBS/CIDP Foundation International, LFB Group France and Ruhr University Bochum (FORUM) and has received speaker honoraria from ARGENTX, CSL Behring, Pfizer, Grifols. AG: Received travel reimbursement and lecture fee by Grifols, CSL Behring, Argenx, Alexion, Alnylam, Akcea, AstraZeneca, Takeda, Pfizer, Johnson and Johnson and Amgen, not directly related to the research topic FH: Payment or honoraria for lectures, presentations, speakers bureaus, manuscript writing or educational events: Alexion, Bayer, Biogen, BMS, Grifols, Janssen, Merck, Novartis, Roche, Sanofi, TEVA. Support for attending meetings and/or travel: Merck, TEVA, Janssen MS: Served on the scientific advisory boards and/or received speaker honoraria, travel funding or honoraria for medical writing from Argenx; Bayer; Biogen Idec; Biotest; CSL Behring; Genzyme; Grifols; Immunovant; Kedrion; Merck; Novartis; Octapharma; PPTA; Roche; Sanofi-Aventis; TEVA; UCB.

EPO-0216 | Neuropathic pain in RFC1-related CANVAS: A systematic review and meta-analysis

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Background and aims: Cerebellar ataxia, neuropathy and vestibular areflexia syndrome (CANVAS), caused by biallelic intronic repeat expansions in RFC1, is a multisystem neurological disorder in which sensory neuronopathy is a core feature. Although neuropathic pain is increasingly reported in clinical cohorts and narrative reviews of RFC1-related disease, its prevalence has not been systematically evaluated. We conducted a systematic review and meta-analysis to determine the prevalence of neuropathic pain in RFC1-related CANVAS.

Methods: A systematic literature search was performed on 13/01/26 using the terms (pain OR painful OR neuropathy OR ganglionopathy OR neuronopathy) AND (CANVAS OR RFC1). The search identified 243 records. After title and abstract screening, 146 articles underwent full-text review. Eligible studies included genetically confirmed CANVAS reporting the presence neuropathic pain. Study selection followed PRISMA guidelines. Data were extracted independently by two reviewers. Statistically pooled proportion calculations were conducted in R language using the default settings of the “meta” package and the “metaprop” function with a random effects model, with heterogeneity assessed using the I² statistic.

Results: The meta-analysis included 433 patients with CANVAS. The pooled prevalence of sensory neuronopathy was 94.7% (95% CI 85.9%–98.2%). The pooled prevalence of neuropathic pain was 46.6% (95% CI 35.6%–57.9%). The heterogeneity was moderate (I² = 73%).

Conclusion: Neuropathic pain is common as it affects a substantial proportion of patients with RFC1-related CANVAS. This systematic review and meta-analysis provides the first pooled prevalence estimate of neuropathic pain in this disorder, highlighting the need for routine pain assessment and increased clinical awareness.

Disclosure: Nothing to disclose.

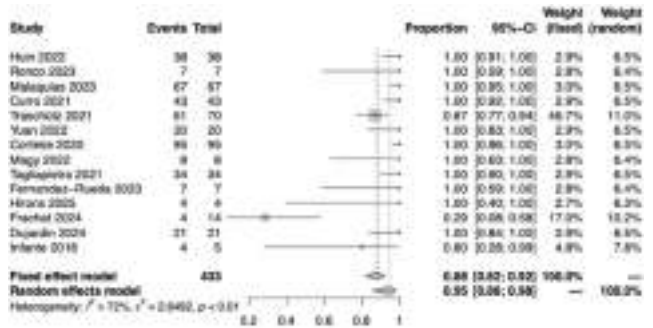


FIGURE 1 Forest plot showing the pooled prevalence of sensory neuronopathy in patients with CANVAS across 14 studies. The pooled prevalence of sensory neuronopathy was 94.7% (95% CI 85.9%–98.2%).

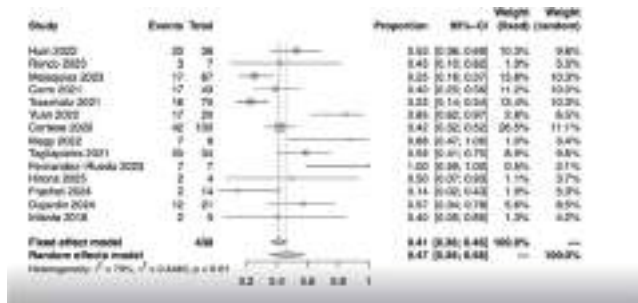


FIGURE 2 Forest plot showing the pooled prevalence of pain in patients with CANVAS across 14 studies. The pooled prevalence of neuropathic pain was 46.6% (95% CI 35.6%–57.9%). The heterogeneity was moderate ($I^2 = 73\%$).

EPO-0217 | Electrical stimulation of spinal cord alleviates pain and restores sensory and autonomic functions in painful diabetic peripheral neuropathy

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Background and aims: Effective management of pain and other neurological symptoms remains challenging in painful Diabetic Peripheral Neuropathy (pDPN). We investigated the clinical and physiological effects of Spinal Cord Stimulation (SCS) in pDPN. **Methods:** Twelve patients with “stocking-and-glove” pDPN were implanted with epidural electrodes at thoracic (T10-T12) level. Assessments were performed pre- and post-SCS implantation (3-, 6- and 12-months) and included questionnaires related to pain, neuropathic and autonomic symptoms and neurological tests examining large- and small-fibers such as sensory and motor nerves conduction studies (NCS), sensorimotor physical exam, limbs skin conductance and thermography. **Results:** Baseline assessments confirmed a severe neuropathy profile denoted by impaired pin sensation test and abundant NCS abnormalities: 40/96 of sensory and 15/72 of motor nerves with absent response. After implant, patients reported significant relief in pain (VRS from 8.2-to-2.0) neuropathic (NPSI from 50-to-15) and autonomic symptoms (BASQ from 28-to-14). We observed a progressive recovery in nerve function at both large- and

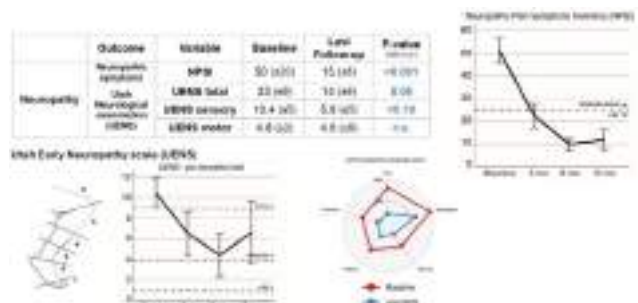


FIGURE 2 Neuropathic symptoms and clinical examination.

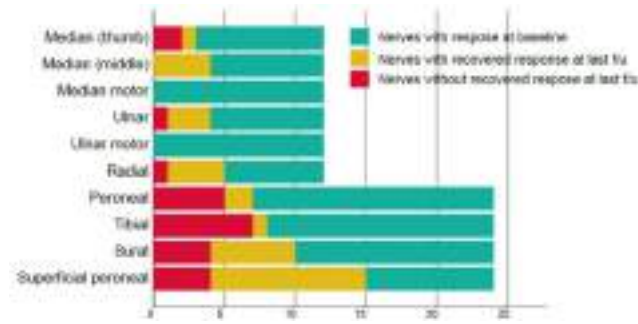


FIGURE 3 Nerve conduction-response recovery studies.

small-fibers. Specifically, to last visit, 29/40 (72%) of sensory and 3/15 of motor responses became detectable (Δ SNAP-2 μ V). Also, in responding nerves, there were noticeable conduction improvements (e.g. $\sim$ Δ SNCV at sural (+2.7 m/s), sup. peroneal (+4.1 m/s) and radial (+5.5 m/s)). This was congruent with improvements in large-fiber-related symptoms (e.g. dysesthesia from 7.2-to-2.5). **Conclusion:** Our results indicate that SCS is able to not only effectively treat pain and other neurological symptoms but also to produce a quantifiable gain in nerve function in pDPN. This suggests that SCS might cause changes in excitability as well as, potentially, some degree of regeneration of nerve fibers. **Disclosure:** This study was sponsored by Boston Scientific.

EPO-0218 | Empasiprubart versus placebo (EMNERGIZE) or immunoglobulin (EMVIGORATE) in chronic inflammatory demyelinating polyradiculoneuropathy: Study designs

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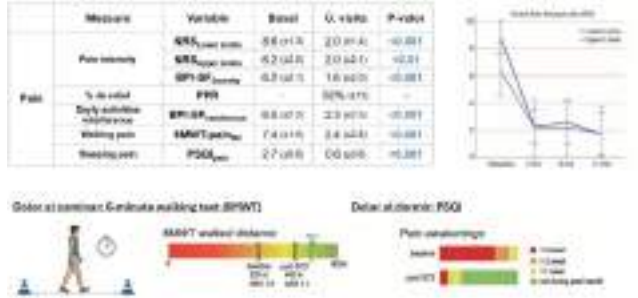


FIGURE 1 Pain results.

Background and aims: Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an immune-mediated neuropathy involving complement-driven macrophage-mediated demyelination. Empasiprubart binds complement C2, blocking activation of classical and lectin complement pathways. Two phase 3, randomised, double-blinded studies in participants with CIDP will compare intravenous (IV) empasiprubart versus placebo (EMNERGIZE, NCT07091630) and IV empasiprubart versus IV immunoglobulin (IVIg) (EMVIGORATE, NCT06920004).

Methods: EMNERGIZE (placebo-controlled) will randomise ~160 adults with CIDP 2:1 to receive either empasiprubart or placebo on Days 1 and 8 then once every 4 weeks in a 24-week double-blind treatment period (part A) (Figure 1). EMVIGORATE (double-dummy) will randomise ~218 adults with CIDP 1:1 on stable maintenance IVIg to receive either empasiprubart or continue the stable IVIg dose in a 24-week double-blind treatment period (part A) (Figure 2). Both studies are followed by a 4-week double-blind rollover, 23-month open-label period (part B) and 15-month safety follow-up.

Results: The primary endpoint for both studies is a reduction of ≥ 1 point versus baseline in adjusted Inflammatory Neuropathy Cause and Treatment (aINCAT) score at Week 24. Key secondary endpoints (Week 24) include changes from baseline in Inflammatory Rasch-built Overall Disability Scale score, Medical Research Council sum score, dominant hand grip strength, Timed Up and Go and time to reduction of ≥ 1 point versus baseline in aINCAT score.

Conclusion: These two global phase 3 studies will evaluate the efficacy and safety of empasiprubart in participants with CIDP. Similar designs and endpoints with complementary study populations support the potential role of empasiprubart as a reference treatment across patients with CIDP, regardless of treatment status.

Disclosure: Simon Rinaldi: Annexon, argenx, the Beijing Association of Holistic and Integrated Medicine, British Medical Association, CSL Behring, Dianthus, Excemed, Fresenius, GBS/CIDP Foundation International, Guillain-Barré Syndrome and

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EPO-0219 | NF 140 and NF 186 positive nodopathy masquerading as GBS

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Background and aims: Nodal and para-nodal disorders are often misdiagnosed as GBS or CIDP. Diagnosis relies on clinical suspicion and re-calibration of decisions in patients earlier diagnosed as GBS or CIDP not responding to treatment.

Methods: Nodal and para-nodal disorders can have clinical features and nerve conduction abnormalities that mimic GBS or CIDP but there is no demyelination. The pathology is at the level of the node of Ranvier. These patients have poor responses to IVIG. This case report describes a 19 year old boy who presented after 10 days of difficulty walking and a preceding history of respiratory infection. He was diagnosed initially with GBS based on nerve conduction studies and CSF analysis. 1 year later, on further evaluation considering poor response to IVIG he was found to be positive for antibodies to Neurofascin 140 and 186 and was diagnosed with nodopathy.

Results: The patient was treated with 2 doses of Rituximab and he improved neurologically from mobility with walker to independent ambulation.

Conclusion: Nodal and para-nodal disorders are now part of the spectrum of inflammatory neuropathies. This case reiterates the need to suspect nodopathies among other causes in patients earlier diagnosed with GBS/ CIDP with inadequate response to conventional IVIG or steroids. Whether antibody testing for nodopathy should be part of initial evaluation in patients with features of GBS/ CIDP needs further speculation.

Disclosure: Nothing to disclose.



FIGURE 1 EMNERGIZE study design.



FIGURE 2 EMVIGORATE study design.

EPO-0220 | Cerebellar cognitive affective syndrome in hypertrophic olivary degeneration associated progressive ataxia and palatal tremor – A case series

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Background and aims: Hypertrophic olivary degeneration (HOD) is a rare trans-synaptic degeneration of the inferior olivary nucleus caused by lesions within the Guillain–Mollaret triangle (GMT). This pathway, connecting inferior olivary nucleus, red nucleus, and dentate nucleus, plays a key role in cerebellar motor control. Lesions of the GMT may impair coordination, articulation, and precision movements. Previous studies have focused primarily on motor manifestations of HOD, particularly progressive ataxia and palatal tremor (PAPT), the characteristic clinical features. Potential cognitive consequences of cerebellar involvement remain largely unexplored. Only a single case report has described a cerebellar cognitive affective syndrome in HOD. We aimed to systematically assess cerebellar cognitive impairment in a series of three patients with HOD.

Methods: We conducted a monocentric retrospective case series of three patients with HOD who underwent multiparametric brain MRI including diffusion tensor imaging (DTI) and comprehensive neuropsychological assessment.

Results: All patients were male, aged 57–75 years. An autoimmune-paraneoplastic etiology was suspected in two patients, and one had a post-hemorrhagic structural lesion. Clinically, all presented with PAPT, with disease duration of 1.5–2 years. MRI revealed bilateral HOD in two patients. None of the patients reported subjective cognitive complaints, and global cognitive status was normal. However, all fulfilled criteria for definite cerebellar cognitive impairment on the CCAS-Scale. Notably, despite normal semantic fluency and absence of language impairment, all showed deficits in switching between semantic categories.

Conclusion: This case series suggests that HOD-associated PAPT may involve the GMT in cognitive functions, particularly executive aspects of language production, highlighting a broader cerebellar role beyond motor control.

Disclosure: The authors report nothing to disclose.

EPO-0221 | Emotion recognition deficits in frontotemporal degeneration: Exploring brain functional connectivity and emotional reserve

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Background and aims: By examining emotion recognition (ER), direct functional connectivity (dFC) of ER-related networks, and their relationship in frontotemporal degeneration (FTD), this study uncovers the neural underpinnings of socio-emotional variability and explores emotional reserve, defined as better-than-predicted ER based on dFC.

Methods: 109 FTD patients [46 behavioral (bvFTD) and 10 semantic behavioral (sbvFTD); 13 nonfluent (nfvPPA) and 18 semantic (svPPA) primary progressive aphasia; 22 progressive supranuclear palsy (PSP)] and 109 HC completed the Comprehensive Affect Testing System-abbreviated (CATS-A), yielding discrete and composite ER scores. Resting-state functional MRI was analyzed in 92 patients and 97 HC, by extracting dFC and nodal graph metrics across six ER-related networks [salience (SN), semantic appraisal, anterior default mode (aDMN), visuo-associative, sensorimotor (SMN), basal ganglia] reconstructed in 50 independent HC. Correlations between ER scores

and network properties were examined. Emotional reserve was evaluated using a residual-based approach.

Results: All FTD showed reduced positive and negative ER versus HC. SvPPA outperformed bvFTD and nfvPPA in happiness; sbvFTD performed worse than svPPA in disgust. FTDs exhibited altered dFC properties in SN, SMN, visuo-associative and aDMN. In FTD, lower negative ER correlated with reduced aDMN integration. Emotional reserve analysis revealed lower residuals in FTD versus HC, for comparable SN connectivity, reflecting reduced emotional reserve.

Conclusion: FTD showed widespread ER deficits, particularly for negative emotions, with distinct temporal-variants (svPPA vs sbvFTD) profiles. Findings highlight aDMN involvement in negative ER and identify reduced emotional reserve as a potential marker of socioemotional vulnerability in FTD.

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EPO-0222 | Identifying individuals at-risk for Alzheimer's Disease: The role of eye-tracking in the NAP4A digital platform

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Background and aims: Early identification of individuals with amnesic mild cognitive impairment (aMCI) at risk of Alzheimer's disease (AD) requires sensitive digital biomarkers beyond standard neuropsychological testing. Eye-tracking may detect subtle changes in attentional and mnemonic processing preceding overt cognitive decline. This study reports the first validation of the Neurocognitive Assessment Platform for Alzheimer's disease (NAP4A), integrating eye-tracking and cognitive assessment.

Methods: Twenty-one healthy controls were assessed at baseline, 6 months, and 12 months using the NAP4A platform, which collects physiological and behavioral data during cognitive tasks. Participants completed the CANTAB battery and a visual memory task involving personal/autobiographical and unfamiliar neuroaesthetic ("Neffie") images. Eye movements were recorded with a TOBII™ eye tracker (90 Hz). Fixation number, fixation duration, saccade number, and saccade distance were analyzed. Wilcoxon signed-rank tests compared encoding versus recall phases and stimulus types. Cognitive performance was examined longitudinally.

Results: CANTAB performance remained stable across time-points, with no significant cognitive changes ($p > 0.05$). Eye-tracking showed greater visual exploration during encoding than recall, with increased fixation number, fixation duration, saccade number, and saccade distance ($p < 0.001$). Neffie images elicited more fixations and shorter fixation durations than personal stimuli ($p < 0.001$), with no saccade differences.

Conclusion: Stable cognition over 12 months aligns with expected trajectories in healthy individuals. Eye-tracking findings indicate higher attentional demands during encoding and greater exploration of unfamiliar stimuli, supporting eye-tracking as a sensitive marker for early AD risk detection.

Disclosure: C. Tripodi, A. Gilioli, S. Musicco, L. Flematti, M. Nouri Janian, M. Zullo, A. Sanna report no competing interests; E. Canu received research support from the Italian Ministry of Health; F. Agosta is Associate Editor of NeuroImage: Clinical, has received speaker honoraria from Biogen Idec, Italfarmaco, Roche, Zambon and Eli Lilly, and receives or has received research supports from the Italian Ministry of Health, the Italian

Ministry of University and Research, AriSLA (Fondazione Italiana di Ricerca per la SLA), the European Research Council, the EU Joint Programme – Neurodegenerative Disease Research (JPND), and Foundation Research on Alzheimer Disease (France). M. Filippi is Editor-in-Chief of the Journal of Neurology, Associate Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmed Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla.

EPO-0223 | Feasibility study of the European inter-societal recommendations for the biomarker-based diagnosis of dementia: The multi-center EuroValiCo project

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Background and aims: The increasing availability of disease-modifying therapies for neurocognitive disorders has

intensified the need for accurate and timely diagnosis supported by biomarker-based approaches. Recent European intersocietal recommendations (Frisoni, 2023) proposed a structured, phenotype-driven diagnostic workflow embedding biomarker use within a stepwise clinical pathway. This preliminary analysis from the EuroValiCo study aim to characterise real-world diagnostic pathways in European memory clinics and to evaluate concordance between recommended and observed biomarker use.

Methods: EuroValiCo is an ongoing multicentre observational study, aiming at validating the European recommendations in clinical practice. Medical charts of consecutive patients at first clinical access for cognitive complaints in 2022 and 2024 are retrospectively reviewed, representing pre- and post-recommendation cohorts. Demographic, clinical, neuropsychological, imaging, and biomarker data are collected. Process discovery techniques were applied to reconstruct real-world diagnostic pathways and assess concordance between recommended and observed biomarker use.

Results: Recommendations indicate cerebrospinal fluid (CSF) analysis as the first-line biomarker for suspected Alzheimer's disease (AD), FDG-PET for frontotemporal lobar degeneration (FTLD) and tauopathies, and DAT-SPECT for Lewy body disease (LBD). In a preliminary cohort of 268 patients (49% women; median age 72 years, range 48–85), concordance with recommendations was observed in 71% of suspected AD cases, 57% of FTLD cases, 0% of tauopathy cases, and 50% of LBD cases.

Conclusion: These findings provide insight into the alignment between guideline-recommended and real-world biomarker use, revealing substantial condition-specific differences, with the highest concordance observed in suspected AD. Future analyses will assess adherence across the full diagnostic process, inter-centre variability, and the impact of recommendation implementation.

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EPO-0224 | Data-efficient AI models for predicting three-year cognitive decline in early Alzheimer’s disease

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Background and aims: Alzheimer’s disease has a prolonged preclinical phase in which pathological changes precede cognitive symptoms by many years. Blood-based biomarkers offer a scalable and minimally invasive opportunity for early risk stratification; however, small sample sizes and cohort heterogeneity limit the clinical translation of advanced machine learning models.

Methods: Using the Alzheimer’s Disease Neuroimaging Initiative (ADNI) dataset, Baseline blood biomarkers, including pTau217–amyloid beta interaction terms, along with demographics and cognitive assessments, were analysed. Cognitive decline was defined as a sustained decrease of 2 or more points on the Montreal Cognitive Assessment across two consecutive years. Artificial Neural Networks (ANNs) and Support Vector Machines (SVMs) were trained on original and synthetically augmented datasets using Conditional Tabular Generative Adversarial Networks (CTGAN), Tabular Variational Autoencoders (TVAE), and Synthetic Minority Over-sampling Technique for Nominal and Continuous features (SMOTENC). Model performance was evaluated using area under the curve (AUC), sensitivity, and specificity, with Shapley value analysis used to assess feature importance.

Results: CTGAN augmentation achieved optimal performance with AUC improvements of 5.6% (ANN) and 3.2% (SVM) vs non-augmented models. Models demonstrated the ability to predict cognitive decline outcomes over a three-year period. Shapley analysis identified pTau217–amyloid beta interaction terms, baseline diagnosis, and age as primary predictors, with significant biomarker interaction effects.

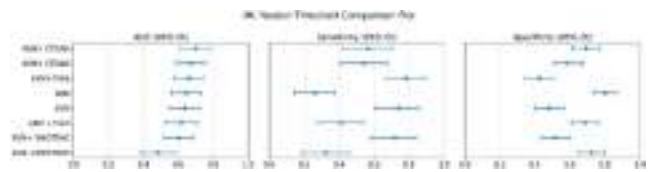


FIGURE 1 Youden-threshold performance comparison across models and augmentation strategies. ANN with CTGAN achieves the best balance of AUC, sensitivity, and specificity, while SMOTENC underperforms.

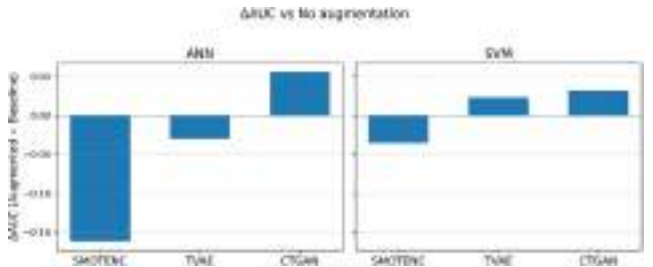


FIGURE 2 ΔAUC relative to baseline across augmentation methods. CTGAN consistently improves performance in both ANN and SVM, whereas TVAE shows minor gains and SMOTENC reduces AUC.

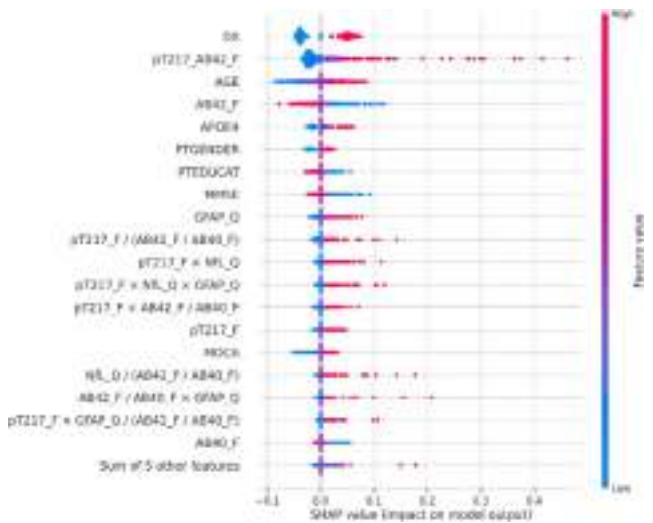


FIGURE 3 SHAP summary plot for the ANN model predicting three-year cognitive decline. Points represent individual participants; colors indicate feature values (blue = low, red = high).

Conclusion: Despite sample size limitations, synthetic data augmentation enables robust prediction of cognitive decline risk over three years using baseline blood biomarkers. This methodology supports translation of AI-based screening tools into clinical practice, facilitating early identification of at-risk patients and timely intervention strategies.

Disclosure: Nothing to disclose.

EPO-0225 | Associations between carotid atherosclerosis, dyslipidemia, activity of daily living and cognitive decline

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Background and aims: According to recent studies cardiovascular-risk-factors (CVRFs) and carotid-atherosclerosis may contribute to cognitive-decline (CD). The aim of the study

is to evaluate the CD, instrumental-activity-of-daily-living (IADL), signs of depression, subclinical-atherosclerosis (intima-media-thickness (IMT))/carotid-atherosclerosis < 50% stenosis in carotid-atherosclerosis/dyslipidemia patients versus patients without carotid-atherosclerosis/dyslipidemia.

Methods: We included in the study 156CVRFs-patients mean-age 70.83 ± 9.35 (92(59.00%) female/64 (41.00%) male), with/without carotid-atherosclerosis/dyslipidemia, without stroke/cardiovascular-disease/carotid-atherosclerosis > 50%stenosis. We compared the results from the CD, IADL and signs-of-depression-screening of the 79 (50.60%) carotid-atherosclerosis/dyslipidemia-patients versus 77 (49.40%) patients-without-carotid-atherosclerosis/dyslipidemia. They underwent clinical-examination, BP, HR, lipid-profile, Montreal-Cognitive-Assessment (MOCA), IADL and Geriatric-Depression-Scale (GDS-15). We measured IMT (mm)/carotid-plaque using a carotid-duplex-scan in both groups of patients.

Results: From 79 (50.60%) carotid-atherosclerosis/dyslipidemia patients (34 (43.00%) male/45 (57.00%) female), there were 71 (89.90%) hypertension-patients, 28 (35.40%) with obesity, 27 (34.20%) smokers, 43 (54.40%) with atrial-fibrillation, 42 (53.20%) diabetes-mellitus-type-II patients. MoCA, IADL, DBP (mmHg), LDL-cholesterol (mg/dl) significant-decrease and IMT (mm), GDS-15 significant-increase in carotid-atherosclerosis/dyslipidemia patients versus patients without-carotid-atherosclerosis/dyslipidemia ($p < 0.05$). In carotid-atherosclerosis/dyslipidemia patients we determined a significant-weak-negative-correlation between age and MoCA ($p = 0.027$), a weak-significant-positive-correlation with SBP (mmHg) ($p = 0.002$), IMT (mm) ($p < 0.05$) and moderate-significant-positive with GDS-15 ($p < 0.001$).We determined a moderate-positive-statistically-significant correlation between MoCA and IADL ($p < 0.001$) and weak-positive with total-cholesterol/LDL-cholesterol (mg/dl) ($p < 0.05$) and a strong-statistically-significant-negative correlation with GDS-15 ($p < 0.001$), moderate-negative with SBP (mmHg) ($p < 0.05$)/DBP (mmHg) ($p < 0.001$) and IMT (mm) ($p < 0.001$) and weak-negative with HR (b/min.) ($p < 0.05$).We found a moderate-statistically-significant-negative correlation between left/right IMT (mm) with MoCA, IADL ($p < 0.05$) and a moderate-positive-statistically-significant correlation between left/right IMT with GDS-15 ($p < 0.001$).

Conclusion: Early evaluation of CD, activities of daily-living, and depressive symptoms is essential in patients with carotid-atherosclerosis and dyslipidemia, as these patients are at increased risk of cognitive-impairment. Correlating these findings with carotid-atherosclerosis severity, age, BP, HR, lipid-profile alterations and CVRFs may improve early identification and management of CD.

Disclosure: Nothing to disclose.

EPO-0226 | Neuropsychiatric symptoms and structural MRI correlates in major dementia subtypes: A retrospective cohort study

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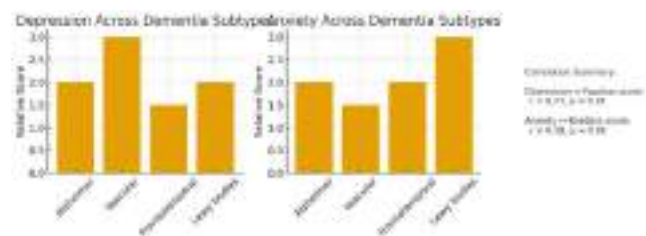


FIGURE 1 Depression and anxiety scores across dementia subtypes. Depression peaked in vascular dementia, while anxiety was highest in dementia with Lewy bodies. Source: Created by the authors. Author's own analysis and visualization.

Background and aims: This study aimed to investigate the distribution of depression and anxiety symptoms among major dementia subtypes and to explore their associations with structural magnetic resonance imaging findings.

Methods: We retrospectively analyzed clinical and imaging data from 1,450 patients followed in a tertiary dementia clinic. Diagnoses included Alzheimer disease, vascular dementia, frontotemporal dementia, and dementia with Lewy bodies. Depression and anxiety were measured using the Geriatric Depression Scale and Geriatric Anxiety Scale. Structural brain changes were rated with the Medial Temporal Atrophy scale, Fazekas score, orbitofrontal sulcus score, collateral sulcus score, and Koedam posterior atrophy score. Group comparisons were conducted using the Kruskal–Wallis test; correlations were assessed with Spearman coefficients. A p -value < 0.05 was considered statistically significant.

Results: Depression scores varied significantly across dementia types ($p = 0.043$), with the highest scores in vascular dementia. Anxiety scores also differed significantly ($p = 0.007$), being highest in dementia with Lewy bodies. Depression correlated positively with Fazekas score ($r = 0.27$, $p = 0.01$), while anxiety showed a weaker but significant association with Koedam posterior atrophy score ($r = 0.18$, $p = 0.04$).

Conclusion: Distinct dementia subtypes exhibit heterogeneous neuropsychiatric profiles and imaging markers. The findings suggest different underlying mechanisms, such as white matter changes linked to depression and posterior atrophy linked to anxiety. Integrating neuroimaging and symptom data may aid personalized dementia care.

Disclosure: Nothing to disclose.

EPO-0227 | Probiotic *Lactiplantibacillus plantarum* KABP051 prevents depressive-like symptoms in *drosophila melanogaster* and murine models of depression

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Background and aims: Previous clinical evidence indicated that *Lactiplantibacillus plantarum* KABP051 may alleviate stress and anxiety in adults in association to modulation of serotonergic and kynurenine pathways. This study investigated its potential to prevent depressive-like behaviors using two validated animal models.

Methods: Adult germ-free flies and flies mono-colonized with different probiotics (10⁸ CFU) were subjected to a 4-day consecutive mechanical stress protocol to induce depression-like states. Subsequently, voluntary behavioural responses were assessed using the gap climbing (GAP) and stop-for-sweet (SFS) test. Adult male C57BL/6 mice were administered with probiotic formulations (10⁹ CFU/day) for 43 days. From day 22, all groups except placebo-unstressed underwent water avoidance stress (WAS) for seven days. Depressive-like behaviors were evaluated using the open field (OFT) and forced swim (FST) tests on days 39 and 40.

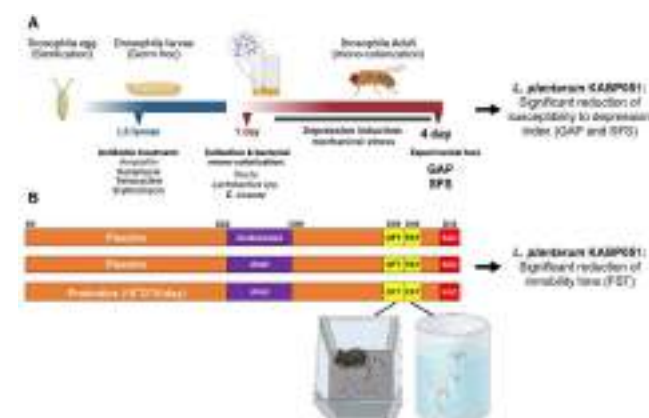


FIGURE 1 Graphical abstract of the *D. melanogaster* (A) and mice (B) models of depression. *L. plantarum* KABP051 showed the greatest efficacy in preventing depressive-like symptoms in both animal models.

Results: Mechanical stress and WAS induced depressive-like phenotypes in flies and mice, respectively. Among the tested probiotics (including additional *L. plantarum* strains), strain KABP051 showed the strongest reduction of the susceptibility to depression index in both GAP and SFS tests ($p < 0.0001$ vs stressed germ-free flies). In mice, all probiotics showed minor effects on OFT, but only KABP051 significantly reduced immobility time in FST ($p < 0.05$ vs stressed group), mirroring unstressed animals.

Conclusion: *L. plantarum* KABP051 exhibits cross-species depression-preventive activity, likely via conserved pathways such as kynurenine modulation. Further research should elucidate underlying mechanisms and confirm translational benefits in humans.

Disclosure: PH and JE-M are full time employees of AB-BIOTICS S.A. (Kaneka Corporation).

EPO-0228 | Early identification of cognitive impairment in elderly patients with HFpEF: A simple clinical risk score

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Background and aims: Cognitive impairment is a frequent yet under-recognized comorbidity in elderly patients with heart failure with preserved ejection fraction (HFpEF), contributing to functional decline and poor outcomes. Simple and clinically applicable tools for early identification of patients at risk are currently lacking. We aimed to develop a practical clinical risk score for predicting cognitive impairment in elderly patients with HFpEF.

Methods: We conducted a cross-sectional observational study including 326 HFpEF patients aged ≥ 65 years. Cognitive function was assessed using the Mini-Mental State Examination-2 (MMSE-2), with cognitive impairment defined as a score < 24 . Clinical, laboratory, vascular, and functional parameters were collected. Independent predictors were identified using multi-variable logistic regression, and a point-based clinical risk score was derived. Discriminative performance was evaluated using receiver operating characteristic (ROC) analysis.

Results: Cognitive impairment was identified in 9.8% of patients. Independent risk predictors included diabetes mellitus, prior stroke or transient ischemic attack, carotid artery disease, elevated NT-proBNP levels, and reduced estimated glomerular filtration rate. Higher Kansas City Cardiomyopathy Questionnaire (12-KCCQ) scores and anticoagulant therapy for atrial fibrillation were independently associated with lower cognitive risk. The clinical risk score demonstrated strong discriminative ability (AUC=0.84). A threshold of ≥ 2 points identified cognitive impairment with 75% sensitivity and 83% specificity.

Conclusion: We propose a simple, clinically applicable risk score that enables early identification of cognitive impairment in elderly HFpEF patients and may support targeted cognitive screening within an integrated heart-brain care approach.

Disclosure: Nothing to disclose.

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Background and aims: Complaints about cognitive performance in the absence of cognitive impairment represent the core feature of subjective cognitive decline (SCD), a common complaint in older adults and a potential risk factor for future cognitive impairment. This study examined clinical and psychological correlates of self- and partner-reported cognitive complaints, as well as awareness of personal cognition, in SCD.

Methods: Data of 789 participants with SCD were obtained from the Alzheimer's Disease Neuroimaging Initiative 2 (ADNI2). Cognitive complaints were assessed using the Everyday Cognition scale, completed by both participants (ECog-P) and

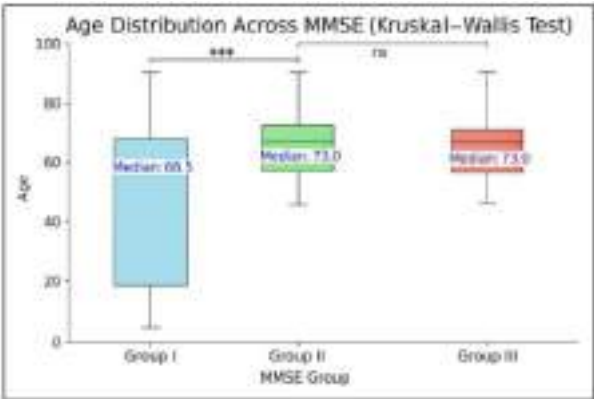


Figure 1 Box plot showing the distribution of age according to Mini-Mental State Examination-2 (MMSE-2) score categories. The central line within each box indicates the median; the box limits represent the interquartile range (IQR); whiskers denote the range excluding outliers. Bonferroni-corrected p-values are displayed for each pairwise comparison between MMSE groups. *** = $p < 0.005$, ns = not significant.

FIGURE 1 Age distribution.

Table 1. Univariate logistic regression results predicting cognitive impairment (MMSE < 24).

Univariate Regression Analysis			
Variable	OR	95% CI	p-Value
Age	1.07	1.05–1.12	0.005
ECOG	0.18	0.08–0.39	<0.001
TIA/stroke	2.44	1.17–5.11	0.01
Stroke/TIA	4.78	2.08–10.91	<0.001
Carotid artery disease	5.43	2.12–13.67	0.001
Depression	3.16	1.81–11.45	<0.05
eGFR mL/min/1.73 m ²	0.95	0.93–0.98	<0.05
Total Cholesterol	1.01	0.66–1.59	<0.05
Triglycerides	1.02	1.01–1.09	0.03
Uric acid	2.44	2.13–8.90	0.04
NT-proBNP	1.02	1.0–1.03	<0.0001
Anticoagulants for AIT	0.47	0.18–1.19	<0.04

Abbreviations: Depressive variable: MMSE group: 0 = normal, 1 = mild cognitive impairment, 2 = suspected dementia; Alzheimer's Disease Neuroimaging Initiative 2 (ADNI2); ECOC, Kansas City Cognitive Quality Questionnaire; eGFR, estimated glomerular filtration rate; TIA, transient ischaemic attack; MMSE, Mini-Mental State Examination; NT-proBNP, N-terminal pro-B-type natriuretic peptide; AIT, atrial fibrillation or flutter; TIA, transient ischaemic attack; eGFR, estimated glomerular filtration rate.

FIGURE 2 Logistic regression.

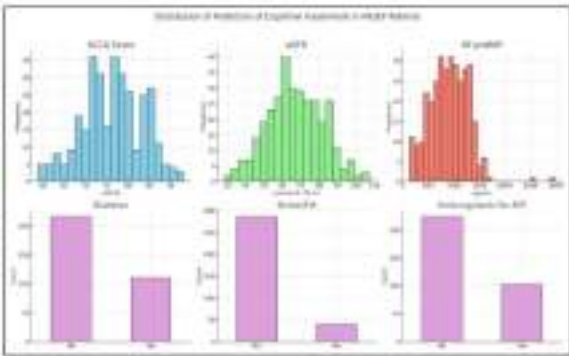


Figure 3 The distribution of the key predicting factors among the 1191 participants. Histograms (top row) display the distribution of continuous variables: ECOC score, eGFR, and NT-proBNP. ECOC scores are generally high, with most patients scoring above 40, indicating preserved quality of life. eGFR values show a moderate spread, reflecting variation in renal function across the cohort. NT-proBNP levels exhibit a right-skewed distribution, consistent with clinical variability observed in heart failure. The plots (bottom row) show the frequency of binary variables: diabetes mellitus, history of stroke or transient ischaemic attack (TIA), and anticoagulant use for atrial fibrillation. These categorical predictors demonstrate distinct group distributions, supporting their relevance in the assessment of cognitive risk in HFpEF.

FIGURE 3 Distribution of key predicting factors.

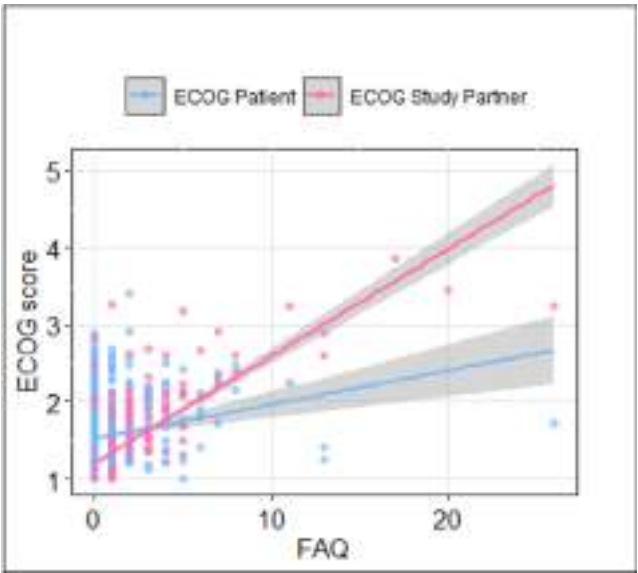


FIGURE 1 Correlations of ECOG Patient (blue circles) and ECOG Study Partner (red triangles) scores with FAQ.

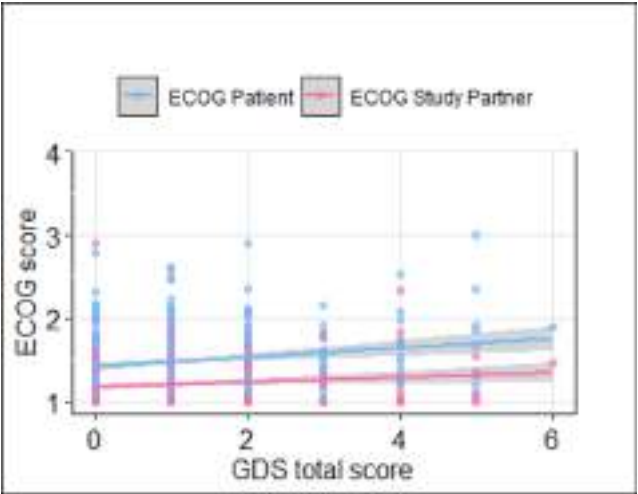


FIGURE 2 Correlations of ECOG Patient (blue circles) and ECOG Study Partner (red triangles) scores with FAQ.

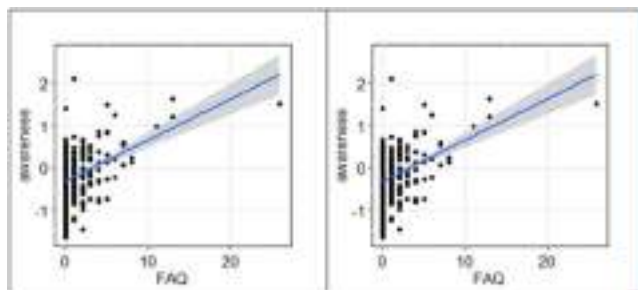


FIGURE 3 Correlation of awareness with FAQ and GDS.

study-partners (ECog-SP). Awareness of cognitive functioning was operationalized as the difference between ECog-P and ECog-SP scores (awareness). Additional measures included the Geriatric Depression Scale (GDS) and the Functional Activities Questionnaire (FAQ). Descriptive statistics summarized demographic and clinical characteristics. Linear regression models, adjusted for age, sex, and education, were used to examine associations between ECog-P, ECog-SP, awareness, and the clinical variables.

Results: The mean age was 71.0years (SD=6.3), with a mean education of 16.7years (SD=2.4). Higher FAQ scores were significantly associated with worse ECog-P ($\beta=0.04$, $p < 0.001$), ECog-SP ($\beta=0.14$, $p < 0.001$) (Figure 1), and greater awareness ($\beta=0.10$, $p < 0.001$) (Figure 3). Depressive symptoms were positively associated with both ECog-P ($\beta=0.05$, $p < 0.001$) and ECog-SP ($\beta=0.03$, $p=0.02$) (Figure 2). No correlation was found with cognitive awareness (Figure 3).

Conclusion: Cognitive complaints SCD are influenced by multiple factors. Understanding the clinical correlates of perceived cognitive decline can offer valuable insights into the complexity of SCD, supporting both prognosis and management.

Disclosure: Nothing to disclose.

EPO-0230 | Persistent cognitive, functional, and fatigue deficits in post-COVID-19 individuals: A comparative study

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Background and aims: The long-term impact of COVID-19 extends beyond respiratory symptoms. This study aimed to compare cognitive function, functional capacity, fatigue, and healthy lifestyle behaviors between individuals with a history of COVID-19 and healthy controls

Methods: We included 40 individuals with a history of COVID-19 and 40 healthy controls. Cognitive status was assessed using the Montreal Cognitive Assessment (MoCA), and functional capacity was measured via the 2-Minute Walk Test. Additional evaluations included the Fatigue Severity Scale,

Pittsburgh Sleep Quality Index, Beck Depression Inventory, and Healthy Lifestyle Behaviors Scale II (HLBS-II).

Results: The clinical characteristics of COVID-19 infection in the study group are presented in Table 1. The study group demonstrated significantly lower scores on the MoCA and 2-Minute Walk Test compared to controls ($p < 0.05$) (Table 2). Similarly, they reported higher fatigue levels and lower scores in the physical activity, stress management and interpersonal relations subdomains of the HLBS-II ($p < 0.05$). Conversely, sleep quality and depression scores showed no significant differences between the groups ($p > 0.05$).

Conclusion: Individuals with a history of COVID-19 exhibit persistent deficits in cognitive function and functional capacity, accompanied by increased fatigue. These impairments appear independent of sleep quality or depression. Clinicians should prioritize long-term monitoring and implement targeted rehabilitation strategies focused on neuro-cognitive recovery and lifestyle modification for this population.

Disclosure: Nothing to disclose.

TABLE 1 Clinical characteristics of COVID-19 infection in the study group.

Clinical Characteristics	Study Group (n=40)
Age (years), Mean $\pm$ SD	35.95 $\pm$ 12.85
Time since infection (month), Mean $\pm$ SD	10.8 $\pm$ 6.9
Disease Severity, n (%)	
Mild	16 (40.0)
Moderate	16 (40.0)
Severe	8 (20.0)

SD: Standard deviation

TABLE 2 Comparison of variables between groups.

Variables	Research Group (n=40)	Control Group (n=40)	p-value
MoCA Score	23.40 $\pm$ 5.58	24.95 $\pm$ 2.75	0.045*
2-Minute Walk Test (m)	146.0 $\pm$ 25.68	161.1 $\pm$ 35.27	0.026*
FSS Score	4.31 $\pm$ 1.77	3.34 $\pm$ 1.73	0.014*
PSQI Score	6.85 $\pm$ 3.86	5.87 $\pm$ 3.23	0.312
BDI Score	13.05 $\pm$ 10.10	10.15 $\pm$ 9.37	0.066
HLBS-II			
Health Responsibility	20.50 $\pm$ 4.81	22.63 $\pm$ 5.27	0.064
Physical Activity	18.73 $\pm$ 5.04	23.15 $\pm$ 4.48	<0.001*
Nutrition	20.25 $\pm$ 4.09	23.80 $\pm$ 4.37	0.130
Spiritual Growth	26.13 $\pm$ 4.74	28.20 $\pm$ 4.72	0.054
Interpersonal Relations	24.15 $\pm$ 4.22	28.35 $\pm$ 4.89	0.034*
Stress Management	18.45 $\pm$ 3.69	20.48 $\pm$ 4.89	0.040*
Total HLBS-II Score	128.90 $\pm$ 20.74	142.60 $\pm$ 23.36	0.005*

MoCA: Montreal Cognitive Assessment; FSS: Fatigue Severity Scale; PSQI: Pittsburgh Sleep Quality Index; BDI: Beck Depression Inventory; HLBS-II: Healthy Lifestyle Behaviors Scale II. * $p < 0.05$.

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Background and aims: Neuropsychological impairments are common in glioma patients, but their links to specific clinical and molecular features, beyond tumor location, remain unclear. This study examined whether molecular profiles influence pre-operative neuropsychological function more than tumor location alone.

Methods: We prospectively assessed 99 preoperative glioma patients and 52 healthy controls with a comprehensive neuropsychological battery. After controlling for demographics, patients with insular (deep) and fronto-temporo-parietal (superficial) tumors were compared. Subgroup analyses were then conducted separately for insular and frontal gliomas to examine associations with molecular markers and clinical factors.

Results: Patients showed broad cognitive-emotional impairments compared to controls. However, no significant difference was found between insular and superficial tumor groups.

TABLE 1 Demographic, Clinical, Molecular Characteristics and Cognitive and Emotional Measures of Glioma Patients by Lesion Location.

Variables	Insular (n=24)	Fronto-Temporo-Parietal (n=75)	t/F score/t/2	p-value
Demography				
Gender (M)	12 (50.0%)	43 (57.3%)	0.158	0.890
Age	43.54±9.66	41.39±11.33	-0.957	0.354
Education (y)	14.00±4.00	14.00±5.00	-0.446	0.658
Smoking	9 (25.0%)	19 (25.3%)	0.001	0.974
Alcohol consumption	6 (25.0%)	20 (26.7%)	0.777	0.378
Clinical feature				
Hypertension	3 (12.5%)	13 (17.3%)	1.025	0.313
Diabetes	3 (12.5%)	9 (12.0%)	-0.061	1.000
Operation history	3 (12.5%)	24 (32.0%)	3.466	0.067
History of other system disease	6 (25.0%)	33 (43.0%)	2.748	0.087
Course of disease (mo)	2.50±5.00	2.16±5.00	-0.929	0.357
Epilepsy	1 (4.2%)	30 (40.0%)	0.970	0.325
Dominant hemisphere tumor	10 (41.7%)	40 (53.3%)	0.990	0.320
Tumor location (2)	54 (66.6%)	27 (32.0%)	-1.751	0.080
Pathology				
Grade (high)	6 (25.0%)	23 (30.7%)	2.072	0.160
1p-19q	8 (33.3%)	8 (10.7%)	-0.900	0.364
IDH mutation	16 (66.7%)	46 (61.3%)	0.106	0.746
1p/19q codeletion	12 (50.0%)	18 (24.0%)	4.404	0.006
MGMT promoter methylation	16 (66.7%)	43 (57.3%)	0.263	0.601
TERT promoter mutation	13 (54.2%)	39 (52.0%)	0.186	0.668
Cognitive-emotional assessment				
Global cognitive function				
MMSE	28.00±2.00	29.00±2.00	-0.322	0.747
MoCA	25.33±2.33	24.92±2.80	0.623	0.536
Attention				
TMTA	26.50±8.00	26.00±10.00	-1.419	0.156
ACS	45.88±7.22	45.64±6.33	0.154	0.878
Executive function				
TMTB	70.00±40.00	63.00±57.00	-1.511	0.133
FAB	18.00±1.00	18.00±1.00	-0.278	0.784
Memory				
DST-1	12.88±2.22	12.59±2.23	0.478	0.634
DST-b	16.38±2.68	16.28±2.93	0.143	0.888
DST-s	9.50±2.00	9.66±2.06	-0.433	0.666
DST	33.54±5.38	32.36±6.40	0.482	0.631
Anxiety				
HAMA	7.79±5.14	8.28±5.54	-0.392	0.708
Depression				
BDI	2.50±6.00	3.56±6.06	-0.839	0.400
Affection				
PANASp	27.79±7.81	27.33±7.53	0.257	0.797
PANASn	21.67±7.87	21.48±7.26	0.120	0.903
Empathy				
IRI	61.04±7.35	60.77±10.22	1.006	0.317

TABLE 2 Comparisons of Neuropsychological Test Scores by Clinical and Molecular Features in the Insular Lobe Subgroup (n = 24).

Influencing Factor (Group, n)	Domain & Test Showing Significant Difference	t/F score	p-value
Laterality Dominant hemisphere (n=10) Non-Dominant Hemisphere (n=14)	Global cognition-MoCA [†]	3.103	0.062
	Attention-ACS [‡]	6.225	<0.001
IDH Mutation Status Mutant [§] (n=16) Wild-type (n=8)	Affection-PANASp	-4.154	<0.001
	Affection-PANASn	3.009	0.061
	Empathy-IRI	2.209	0.038

[†] Dominant hemisphere group scored higher than non-dominant hemisphere group.
[‡] Wild-type group scored higher than IDH-mutant group.
[§] Wild-type group scored lower than IDH-mutant group.
^{||} The two groups showed no significant differences in demographic factors.

TABLE 3 Comparisons of Neuropsychological Test Scores by Clinical and Molecular Features in the Frontal Lobe Subgroup (n = 52).

Influencing Factor (Group, n)	Domain & Test Showing Significant Difference	t/F score	p-value
Epilepsy Yes (n=19) No (n=33)	Global cognition-MoCA [†]	2.434	0.021
	Memory-DST-s [‡]	2.765	0.066
	Executive function-FAB [‡]	2.273	0.028
	Affection-PANASp	2.708	0.010
Grade High (n=20) Low (n=32)	Empathy-IRI	4.008	<0.001
	1p19q Co-deletion Codeleted (n=14) Intact (n=26)	Attention-TMTA [†]	2.078
TERT Promoter Mutation Mutant (n=23) Wild-type (n=29)	Memory-DST-1 [‡]	-2.752	0.009
	Memory-DST-b [‡]	-2.245	0.031
	Memory-DST [‡]	-2.560	0.015
	Empathy-IRI	3.048	0.004
IDH Mutation Status Mutant (n=20) Wild-type (n=23)	Attention-TMTA [†]	2.710	0.010

Low-grade WHO grade 1-2; High-grade WHO grade 3-4.
[†] No-epilepsy group scored higher than epilepsy group.
[‡] Low-grade group scored higher than high-grade group.
^{||} Co-deletion group required less time to complete the task.
[†] Co-deletion group scored higher than non-co-deletion group.
[‡] Wild-type group scored higher than TERT-mutant group.
^{||} IDH-mutant group required less time to complete the task.
The two groups showed no significant differences in demographic factors.

Subgroup analysis revealed: (1) Within insular gliomas, dominant hemisphere involvement correlated with poorer global cognition. Notably, IDH mutation here associated with worse attention and empathy, yet higher positive and lower negative affect. (2) In frontal gliomas, molecular and clinical features showed stronger associations: absence of epilepsy, lower grade, 1p19q codeletion, lack of TERT mutation, and presence of IDH mutation each linked to better performance across multiple cognitive and emotional domains.

Conclusion: This study indicate that molecular profiles and key clinical factors outweigh tumor location in shaping preoperative neuropsychological function in glioma. This argues for a molecularly-informed framework to better understand neurobehavioral manifestations and guide patient assessment and intervention.

Disclosure: Nothing to disclose.

EPO-0232 | Systematic review of Epilepsia partialis continua: Clinical profile, neurophysiological findings and etiological spectrum in a cohort of 287 patients

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Background and aims: Epilepsia partialis continua (EPC), also known as Kojevnikov epilepsy, is a rare form of focal status epilepticus with an incidence greater in males than females [1], characterized by continuous or near-continuous focal motor seizures [2]. It affects both adults and children and is associated with diverse etiologies, including structural, inflammatory, and metabolic brain disorders [3].

Methods: We performed a systematic literature review following the PRISMA guidelines. S PubMed and BVS were searched using the keywords “Kojevnikov epilepsy”, “focal status epilepticus”, “physiopathology”, “continuous focal seizures”, “etiology”, and “neurophysiology”. Nonexperimental, nonanimal, English-written studies were included.

Results: We included 287 patients. The mean age was 31 years. Sex was reported in a minority of cases (33 females, 17 males), and was not reported for the remaining cases. Continuous focal epileptiform activity (97.6%) and focal epileptic status (99.3%) were more often reported in most of the cases, commonly involving the cortical regions (96.9%). Structural brain lesions represented the most frequent etiologies (86.7%), preceded by autoimmune diseases (21.1%), of which Rasmussen syndrome is the most frequent (20.2%). Metabolic (0.70%) and mitochondrial (5.2%) causes were less commonly identified. Genetic involvement was uncommon and included POLG (0.7%), GFAP (0.3%), and DNMT1L (0.3%), as well as ARF6 dysfunction. One case (0.3%) of anti-GABA-A receptor antibodies following alemtuzumab treatment was identified.

Conclusion: EPC is predominantly a cortical focal status epilepticus with mainly structural etiologies, followed by autoimmune and inflammatory causes. These findings support a diagnostic approach and highlight the need for further research into less common immunological and genetic mechanisms.

Disclosure: Nothing to disclose.

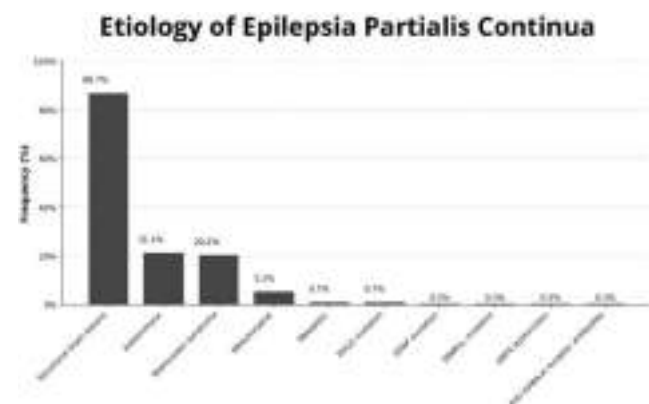


FIGURE 1 Frequency of the Etiologies in Epilepsia Partialis Continua.

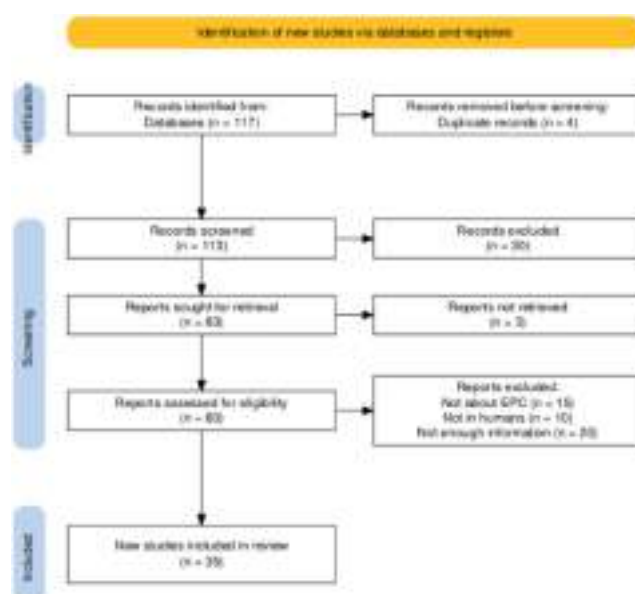


FIGURE 2 Prisma-P flowchart of the process of inclusion of studies in the systematic review.

EPO-0233 | Disconnection surgeries in refractory epilepsy: A retrospective study from a Tertiary Epilepsy Center

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Background and aims: Disconnection surgery is an established treatment option for selected patients with drug-resistant epilepsy in whom resective surgery is not feasible. This study aimed to evaluate postoperative prognosis according to different disconnection surgery subtypes.

Methods: Patients followed between 2000 and 2023 at the Epilepsy Unit of Istanbul University-Cerrahpasa Faculty of Medicine were retrospectively reviewed. Fifty-five patients who underwent disconnection surgery were included. Patients were classified into four subgroups based on surgical technique: Functional Hemispherectomy (FH), Posterior Quadrant Disconnection (PQD)—including temporo-parietal (TP-PQD) and parieto-occipital disconnection (PO-PQD)—and Subtotal Hemispherotomy (SH). Demographic and clinical characteristics were analyzed in relation to postoperative outcomes.

Results: The cohort consisted of 32 males and 23 females, with a mean age of 20.7 ± 12.5 years. Mean age at surgery was 14.2 ± 12.7 years, and the mean duration between seizure onset and surgery was 10.2 ± 10.8 years. FH was performed in 24 patients, PQD in 17 patients, and SH in 8 patients. At final follow-up, Engel class I outcome was achieved in 38 patients, class II in 7, class III in 5, and class IV in 5. No statistically significant differences were observed in postoperative Engel outcomes or complication rates among the different surgical subgroups.

Conclusion: Disconnection surgery is an effective therapeutic option for refractory epilepsy, with favorable seizure outcomes across different techniques. Surgical strategy should be

individualized based on patient characteristics and the anatomical localization of the epileptogenic zone.

Disclosure: Nothing to disclose.

EPO-0234 | A new non-invasive approach to quantify pharmacological modulation of cortical excitability

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Background and aims: Cortical excitability (CE)—the cortex's responsiveness to perturbation—is fundamental and altered in neurological and psychiatric disorders, making it a pharmacological target. Yet a robust, scalable, drug-sensitive marker is lacking. CE can be probed non-invasively with transcranial magnetic stimulation combined with electroencephalography (TMS-EEG), but standard comparisons of evoked amplitudes in predefined time windows are variable and may miss medication effects. We introduce and validate an excitability index (ExI) derived from TMS-EEG stimulus-response curves (SRCs) across intensities to quantify pharmacological modulation of CE.

Methods: In a placebo-controlled crossover study, 12 healthy men received levetiracetam (LEV; 3000 mg) and placebo (10 completed both). Single-pulse TMS over left M1 at seven intensities (70–130% resting motor threshold) was delivered before and 2 h after intake while EEG was recorded. Responses were quantified using global mean field amplitude (GMFA) and ExI (area under the normalized SRC using EEG line-length response magnitude). Wilcoxon signed-rank tests assessed within-session changes; precision-recall (PR) analyses compared ExI versus GMFA for discriminating LEV from placebo.

Results: LEV reduced GMFA in selected time windows with medium-to-large effect sizes, without consistent latency shifts. ExI significantly decreased following LEV ($p < 0.01$) but not placebo, and showed superior discrimination (AUC-PR 0.79, baseline prevalence = 0.27) versus GMFA (AUC-PR 0.27–0.43).

Conclusion: ExI is a scale-free, medication-sensitive marker of CE that outperforms standard TMS-EEG metrics, providing a non-invasive readout of pharmacological effects on human cortical physiology. ExI could serve as a translational biomarker to monitor drug engagement and optimize dosing for epilepsy and other excitability disorders.

Disclosure: Nothing to disclose.

EPO-0235 | Early use of Cenobamate in a case of non-convulsive status epilepticus in a woman with progressive multifocal leukoencephalopathy

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Background and aims: Non-convulsive status epilepticus (NCSE) is a type of status epilepticus (SE) with no prominent motor signs. In NCSE without coma that is refractory to first and second line treatment, therapy with anaesthetics is less appropriate. ASMs are therefore used sequentially. Cenobamate (CNB) efficacy in SE is supported by limited data. We present a case of NCSE in which CNB was used early.

Methods: A 54-year-old woman with multiple sclerosis received natalizumab until she was diagnosed with progressive multifocal leukoencephalopathy (mRS4). One year later, she was admitted with acute aphasia, right head deviation, and impaired consciousness (GCS10). Brain MRI revealed hyperintensities and edema in the right hemisphere. EEG showed right focal SE with prominent contralateral motor signs, resistant to benzodiazepines, levetiracetam, and lacosamide.

Results: After a few days, the EEG evolved into NCSE with preserved consciousness, repetitive right and head deviation, and an inability to walk (mRS5). Despite the introduction of a third ASM (perampanel, 8 mg/day), there was no electroclinical

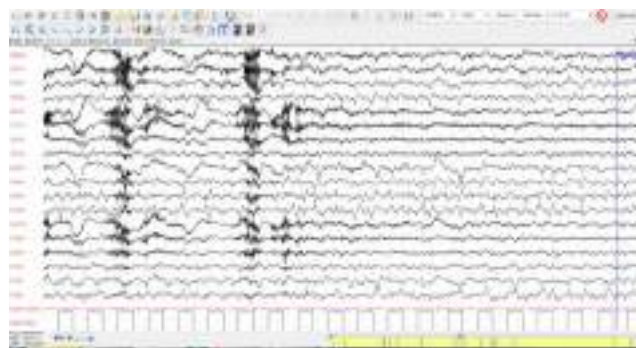


FIGURE 1 EEG performed while taking levetiracetam and lacosamide.



FIGURE 2 Follow-up EEG during levetiracetam and lacosamide treatment.

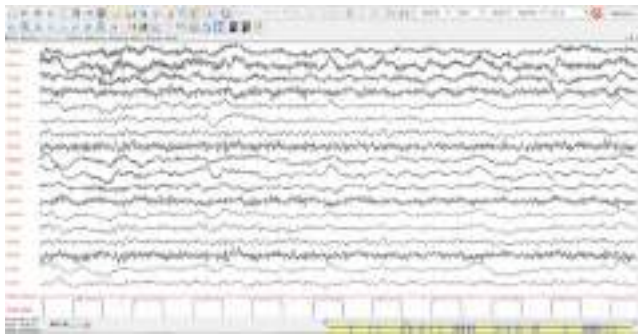


FIGURE 3 Follow-up EEG during treatment with levetiracetam, lacosamide, peramppanel, and cenobamate.

improvement until CNB was added and titrated up to 200 mg/day within one month. The MRI scan showed resolution of the perictal alterations in the right hemisphere. A two-month EEG follow-up excluded NCSE relapse.

Conclusion: Currently, it is not possible to recommend the use of cenobamate in NCSE, as only anecdotal cases have been reported to date. An important limitation is its slow titration, but it can be considered as a third-line therapy in selected cases with subtle clinical manifestations. Furthermore, CNB may modulate the GABAergic dysfunction described in NCSE.

Disclosure: Nothing to disclose.

EPO-0236 | A retrospective cohort study of valproate and infertility in men with epilepsy or bipolar disorder using international health data

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Background and aims: Against a background of limited animal data and existing restrictions, we assessed infertility in men with epilepsy or bipolar disorder exposed and unexposed to valproate.

Methods: We conducted a retrospective cohort study using data from TriNetX, an international network of healthcare organisations. Men aged <55 years were identified in three cohorts: (A) epilepsy or bipolar disorder, (B) epilepsy, and (C) bipolar disorder. Validated diagnostic and prescription codes were used. Valproate-exposed and unexposed men were compared for life-time risks of infertility outcomes. Follow-up began from the second valproate prescription (exposed) or second disease code (unexposed). Propensity-score matching balanced >140 baseline covariates. Survival analysis was undertaken using Cox proportional hazards models, generating hazard ratios (HRs) with 95% confidence intervals (CIs).

Results: Among 627,720 men with epilepsy or bipolar disorder, 91,917 were exposed and 535,803 unexposed to valproate. Serum valproate was detectable in 98% of tested exposed individuals. After matching, 78,971 men were included in each group. There was a <1% difference in infertility outcomes between those exposed and unexposed to valproate (not significant):

male infertility HR 1.057; CI 0.864–1.295, testicular hypofunction HR 0.916; CI 0.824–1.019, testicular atrophy HR 1.000; CI 0.682–1.465, and composite low sperm concentration, motility, vitality, normal forms, or semen volume HR 0.856; CI 0.613–1.196. Total testosterone was modestly lower in valproate-exposed men but remained within the normal range; other hormones did not differ. Analyses stratified by diagnosis were similarly null.

Conclusion: Our findings do not support an association between valproate and infertility in men with epilepsy or bipolar disorder in real-world settings.

Disclosure: A.G.M. declares (i) a UCB Pharma grant paid to University of Liverpool for the National Audit of Seizure Management in Hospitals (NASH) study, which is unrelated to the submitted work; (ii) an Angelini grant to be paid to University of Liverpool as co-applicant for A multi-method Project to maximise efficient and equitable pathways to support from a regional epiLepSy centre (PROPEL), which is unrelated to the submitted work; (iii) Honoraria paid to University of Liverpool for lectures unrelated to the submitted work given at educational events sponsored by Sanofi, Eisai, and GSK; (iii) Support from Angelini for attendance unrelated to the submitted work at the 2024 International League Against Epilepsy (ILAE) congress. G.K.M. declares an Angelini grant paid to the University of Liverpool as co-applicant on the PROPEL study, which is unrelated to the submitted work; (ii) Honoraria paid to the University of Liverpool for delivering a lecture at an educational event sponsored by Angelini, which was unrelated to the submitted work. G.Y.H.L. is a consultant and speaker for BMS/Pfizer, Boehringer Ingelheim, Daiichi-Sankyo, and Anthos, which is unrelated to the submitted work. No fees are received personally. G.Y.H.L. is a National Institute for Health and Care Research (NIHR) Senior Investigator and co-principal investigator of the AFFIRMO project on multimorbidity in AF, which has received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No. 899871, which is unrelated to the submitted work. T.S. is an employee of TriNetX, which routinely provides data for research studies, including the data used in this study. The involvement of T.S. as a co-author reflects her technical assistance as a data analyst and does not imply any compensation or influence from TriNetX on this study. The remaining authors declare no competing interests.

EPO-0237 | Beyond neuroprotection: A context-dependent view of GLP-1 receptor agonists and seizure susceptibility

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Background and aims: Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) are widely used in type 2 diabetes mellitus and obesity and are increasingly discussed for potential neuroprotective properties. Large-scale clinical and real-world studies from 2024/2025 report a predominantly neutral to protective association between GLP-1 RA use and seizure- or epilepsy-related endpoints. However, the effects of GLP-1 RAs on neuronal excitability in patients with established epileptogenic circuitry remain insufficiently characterized.

Methods: We conducted a narrative review critically examining experimental and clinical evidence regarding the impact of GLP-1 RAs on seizure propensity, with particular emphasis on discrepancies between population-level outcomes and individual network-level vulnerability.

Results: Preclinical studies demonstrate heterogeneous, network-dependent actions of GLP-1 RAs. Anticonvulsant properties are observed in chemically induced seizure models, whereas paradoxical pro-seizure responses have been reported in genetic absence epilepsy models, suggesting circuit-specific directionality. In contrast, population-level analyses consistently report no increased risk of incident seizure-related events under GLP-1 RA therapy. However, these findings are largely derived from administrative diagnostic codes, lack epileptological phenotyping and are not designed to capture transient, exposure-dependent alterations in ictogenic potential. Additional factors, including delayed gastric emptying affecting antiseizure medication absorption, rapid metabolic shifts during titration, blood-brain barrier dysfunction and agent-specific pharmacokinetics may further modulate individual susceptibility.

Conclusion: GLP-1 RA effects on neuronal excitability appear highly context-dependent, shaped by epilepsy subtype, network vulnerability, exposure dynamics and treatment phase. Prospective studies with higher temporal and clinical resolution, incorporating detailed phenotyping, therapeutic drug monitoring and longitudinal seizure assessment, are required to ensure neurological safety in patients with epilepsy.

Disclosure: Nothing to disclose.

EPO-0238 | A systematic review of Lamotrigine overdose: From uneventful to ICU admission and cardiac arrest

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Background and aims: Lamotrigine (LTG) is a widely used drug to treat epilepsy and depression. We performed a systematic review of studies reporting LTG overdose. Additionally, we reported an original case.

Methods: PubMed was searched without date limits. 103 records were screened, of which 40 studies were finally included in the systematic review, and results reported according to PRISMA guideline, adding seven studies identified from included articles. We extracted demographics, LTG indication, overdose reason, complications, EEG findings, and outcomes.

Results: Forty-seven patients (27 female; mean age 28.3 years) were included. Nineteen patients co-ingested substances in addition to lamotrigine. Intent was suicidal in 33 cases. Lamotrigine was mainly prescribed for bipolar disorder (55%) and epilepsy (28%). Seizures occurred in 45% of overdoses (5 single, 10 cluster, 11 status epilepticus), including 6/13 patients with epilepsy. Median ingestion was 4.7 g (IQR 2.7 to 9.5) and median plasma concentration was 35.75 mg/L (IQR 25.3 to 52.8). There were 3 deaths and 5 cardiac arrests. Twenty-five patients required ICU

admission. Electroencephalography was reported in 23/47: 3 showed ongoing seizure, 12 with slowing pattern or epileptiform abnormalities, and the remainder unremarkable. Personal case: 75-year-old man with temporal lobe epilepsy admitted for tonic-clonic status epilepticus. LTG concentration was 39.73 mg/L after erroneous intake. Patient required rehabilitation after prolonged ICU staying.

Conclusion: LTG overdose can result in severe neurotoxicity, coma, status epilepticus and death (6%). Cardiac arrest was reported 11% of cases. Suicidal ideation was the most common reason for overdose but also erroneous intake in children and elders.

Disclosure: Nothing to disclose.

EPO-0239 | Does time since diagnosis or the number of failed ASMs matter more in people with DRE receiving VNS therapy?

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Background and aims: Time since diagnosis and number of failed ASMs may have an impact of the outcomes of people who receive VNS Therapy for DRE.

Methods: Participants with DRE enrolled into the CORE-VNS study and who were naïve to VNS Therapy were grouped into early (EI), < 5 years; intermediate (II), 5 to < 10 years; or late (LI), > 10 years. Participants were also grouped by number of failed ASMs: < 3, 4–6, 7–10, 11–16, or > 16. Clinical outcomes were assessed across seizure-related (frequency) and non-seizure (quality of life) domains at 3, 6, 12, 24, and 36 months.

Results: EI tended to have greater seizure frequency reduction than LI: median percentage change from baseline in seizure frequency at 36 months for the EI group was –88.2% (95% CI –100% to –76.5%) versus –71.4% (95% CI –81.6% to –55.0%) in the LI group. 40.2% of EI achieved a 100% reduction in seizure frequency at 36 months, compared to 22.6% in the LI group. Despite this, participants treated with fewer ASMs had a significant greater likelihood of response than those with more failed ASMs when adjusted for covariates including age and time since diagnosis ($p < 0.05$). At 36 months, participants implanted after 2–3 failed ASMs had a median change from baseline in seizure frequency of –94.4% compared with a –57.4% who failed 11–16 ASMs.

Conclusion: The benefits of VNS Therapy on seizure outcomes are better when initiated promptly. Continued trials of ASMs may limit the benefit of early implantation following DRE diagnosis.

Disclosure: GG, MD, RV, CG, and KN are employees of LivaNova PLC (or a subsidiary) and hold stock or stock options with the company, who is the manufacturer of the VNS Therapy System. All other authors are investigators associated with the CORE-VNS Study, and in that capacity they or their institutions receive compensation from LivaNova for study-related activities. No author received direct compensation from LivaNova.

EPO-0240 | VNS therapy is associated with a decrease in health care resource utilization over 36 months in people with drug-resistant epilepsy

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Background and aims: VNS Therapy is important for people resistant to antiseizure medications and for those in whom surgery is not feasible. People with DRE have increased HCRU due to seizures and seizure-related health events. The impact of any therapy on HCRU is an important consideration when evaluating total benefit and costs of an intervention.

Methods: People with DRE were enrolled into a prospective, observational, multicenter, international study called the CORE-VNS (NCT03529045). HCRU was evaluated for 12 months before baseline and at 12-month intervals over the duration of the CORE-VNS study. For this analysis, the HCRU related to seizure activity in people with DRE from baseline to 36 months was analyzed.

Results: 526 participants met the study criteria. Participants who reported seizure-related ER visits and the number of events decreased from 32.3% at baseline ($n=599$) to 14.8% at 36 months ($n=128$). Participants with seizure-related ER visits due to status epilepticus decreased from 12.2% at baseline ($n=195$) to 3.6% at 36 months ($n=42$), and those with seizure-related ER visits leading to hospitalization decreased from 15.8% at baseline ($n=201$) to 6.2% at 36 months ($n=32$). Participants reporting seizure-related hospitalizations decreased from 27.0% at baseline ($n=312$) to 7.4% at 36 months ($n=45$). Participants reporting hospitalizations due to status epilepticus decreased from 9.5% at baseline ($n=129$) to 2.1% at 36 months ($n=11$).

Conclusion: The use of VNS Therapy is associated with a decrease in HCRU in people with DRE.

Disclosure: PT, GG, MD, GD, CG, and KN are employees of LivaNova PLC (or a subsidiary) and hold stock or stock options with the company, who is the manufacturer of the VNS Therapy System. All other authors are investigators associated with the CORE-VNS Study, and in that capacity they or their institutions receive compensation from LivaNova for study-related activities. No author received direct compensation from LivaNova.

EPO-0241 | First Antiseizure medication choice in older adults with seizures: An observational cohort study based on a claims database

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Background and aims: Data on first-line antiseizure medication (ASM) use in older adults with new-onset epilepsy are limited. Enzyme-modulating ASMs may affect cholesterol levels, which is relevant in patients with cardiovascular comorbidity. We examined sex differences and cardiovascular risk profiles, focusing on enzyme-modulating ASM use.

Methods: This nationwide, claims-based study included adults aged ≥ 50 years with seizures diagnosed in 2014, using Diagnosis Treatment Combination codes in the Dutch health insurance database (Vektis). Pharmacy claims documented ASM prescriptions. Analyses were restricted to incident cases with ASM after diagnosis. Descriptive statistics explored prescribing patterns and sex differences. Cox regression assessed associations between ASM class and initiation of lipid-lowering therapy. Consent was waived due to anonymization.

Results: Among 8728 adults with incident seizures, 3,583 received a single ASM after diagnosis. 42.5% ($n=1,524$) were female; median age was 70 years (range 60–102). Levetiracetam (53%) and valproic acid (30%) were the most prescribed ASMs across sexes. Enzyme-modulating ASMs were frequently used despite cardiovascular comedication. Carbamazepine was more often prescribed to women, while lamotrigine ranked fifth overall. Males had longer prescription intervals than females. Daily doses were similar between sexes, but lower in adults over 70. No

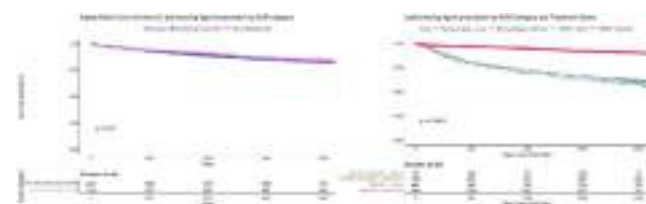


FIGURE 1 1A (left). Kaplan-Meier curves for lipid-lowering initiation by ASM class and 1B (right). active treatment in overall sample.

TABLE 1 ASM prescription frequency (N and percentage of the total number of prescriptions) and relative frequencies comparison across sex groups. *p*-values and adjusted *p*-values after correction for multiple testing, indicate the level of significance.

ASM	Total (N, frequency)	Male (N, relative frequency)	Female (N, relative frequency)	P-value	Adjusted P-value
Carbamazepine	183 (39%)	95 (44%)	88 (35%)	0.908	0.592
Valproic Acid	186 (2%)	83 (31%)	425 (28%)	0.952	0.367
Lamotrigine	77 (2%)	37 (2%)	40 (3%)	0.118	0.695
Phenytoin	75 (2%)	95 (2%)	25 (2%)	0.135	0.695
Phenylin	172 (3%)	95 (4%)	82 (3%)	0.187	0.748
Clozapine	54 (2%)	27 (1%)	27 (2%)	0.227	0.582
Larvalic acid	180 (34%)	101 (32%)	79 (34%)	0.768	1
Galopentol	40 (1%)	28 (1%)	20 (1%)	1	1
Phenobarbital	5 (0.1%)	3 (0.1%)	2 (0.1%)	—	—
Larvalic acid	4 (0.1%)	3 (0.1%)	1 (0.1%)	—	—
Carbamazepine	11 (0.3%)	8 (0.3%)	3 (0.2%)	—	—
Pyridoxine	4 (0.1%)	2 (0.1%)	2 (0.1%)	—	—
Topiramate	20 (1%)	7 (0.3%)	13 (0.5%)	—	—

TABLE 2 ASM prescriptions with the median prescribed daily doses ratio, and median prescription intervals length (in days). *p*-values and adjusted *p*-values for multiple testing indicate the level of significance.

ASM	Reference median (mg/day)				Effective median (mg/day)			
	Ratio	Median	P-value	Adjusted P-value	Ratio	Median	P-value	Adjusted P-value
Carbamazepine	0.11	100	0.04	0	100	0.04	0.04	0
Valproic acid	0	0	0.04	0	0	0	0.04	0
Lamotrigine	0	100	0.04	0	0	100	0.04	0.04
Phenytoin	0	0	0.04	0	0	0	0.04	0
Phenylin	0	0	0.04	0	0	0	0.04	0.04
Clozapine	0	0	0.04	0	0	0	0.04	0
Larvalic acid	0	0	0.04	0	0	0	0.04	0
Galopentol	0	0	0.04	0	0	0	0.04	0
Phenobarbital	0	0	0.04	0	0	0	0.04	0
Larvalic acid	0	0	0.04	0	0	0	0.04	0
Carbamazepine	0	0	0.04	0	0	0	0.04	0
Pyridoxine	0	0	0.04	0	0	0	0.04	0
Topiramate	0	0	0.04	0	0	0	0.04	0
Valproic acid	0	0	0.04	0	0	0	0.04	0

significant association was found between enzyme-modulating ASMs and time to lipid-lowering agent initiation.

Conclusion: Nationwide claims data offer insights into real-world ASM prescribing. Treatment remains conservative, with older ASMs prescribed despite cardiovascular co-medications. Women and the oldest adults may receive shorter treatment intervals and lower doses. Clinical linkage is lacking, but these findings may guide optimization of epilepsy management in specific subgroups.

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EPO-0242 | A ferroptosis-based diagnostic index for severity stratification in symptomatic epilepsy

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Background and aims: Reliable biomarkers for assessing disease severity in symptomatic epilepsy are limited. Given the emerging role of ferroptosis in neurological disorders,

integrating ferroptosis-related biomarkers into a composite diagnostic index may improve epilepsy severity stratification and support personalized management.

Methods: This prospective study included 103 patients with symptomatic epilepsy and 20 healthy individuals. Serum markers of iron metabolism (total iron, ferritin, transferrin), lipid peroxidation (MDA, 4-HNE), and antioxidant defense (GSH, GPX4, SOD) were measured using standardized biochemical methods. A ferroptosis index (FI) was calculated using a composite formula incorporating pro-oxidant and antioxidant parameters. Patients were stratified according to disease severity. Diagnostic performance was evaluated using ROC analysis.

Results: The ferroptosis index demonstrated progressive elevation with increasing epilepsy severity. Based on FI values, patients were classified into mild (FI < 2.5), moderate (FI 2.5–7.5), and severe epilepsy (FI > 7.5). ROC analysis revealed high diagnostic accuracy of the FI for identifying severe epilepsy, with an area under the curve of 0.89 (95% CI: 0.82–0.96), sensitivity of 87%, and specificity of 83%. Higher FI values were associated with increased seizure frequency and more pronounced cognitive impairment.

Conclusion: The proposed ferroptosis-based diagnostic index is a reliable and clinically applicable tool for severity stratification in symptomatic epilepsy and may facilitate individualized therapeutic decision-making.

Disclosure: Nothing to disclose.

EPO-0243 | Utility of the IsCHEMiA score in predicting post-stroke epilepsy after stroke reperfusion therapy

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Background and aims: The IsCHEMiA score is a newly developed, internationally validated tool for predicting post-stroke epilepsy (PSE) after acute ischemic stroke. We aimed to study its predictive performance in stroke patients after reperfusion therapy compared to existing risk scores.

Methods: We reviewed clinical and imaging features of patients with first-ever acute ischemic stroke who received intravenous thrombolysis (IVT) and/or endovascular thrombectomy (EVT) at Queen Mary Hospital (Hong Kong), Ruttonjee Hospital (Hong Kong), and National Cerebral and Cardiovascular Center (Japan). We calculated the IsCHEMiA, SeLECT, and SeLECT 2.0 scores, and compared their performances using discrimination (c statistic).

Results: We included 627 patients with a mean age of 72.1 years, where 421 (67.1%) received IVT and 349 (55.7%) underwent EVT (Table 1). PSE, defined as the occurrence or recurrence of unprovoked seizure > 7 days after stroke, occurred in 42 (6.7%) patients. Haemorrhagic transformation (of any grade) occurred in 154 (24.6%) patients. The IsCHEMiA score outperformed the SeLECT score (c=0.806 vs. 0.729, z=2.772; p=0.006)

TABLE 1 Baseline Characteristics of overall cohort.

	Overall cohort (n = 927)
Age at stroke	
< 50 years	12.1 ± 13.3
≥ 50 years	799 (85.4)
Gender	
Male	535 (57.8)
Female	392 (42.2)
First medical history	
Hypertension	380 (40.9)
Diabetes mellitus	251 (27.1)
Hyperlipidemia	176 (18.9)
Atrial fibrillation	173 (18.6)
Stroke recurrence	138 (14.8)
Major health history	
Coronary atherosclerosis	441 (47.6)
Other atherosclerotic disease	385 (41.5)
Previous major thrombotic risk factors	154 (16.6)
Mean follow-up duration (days)	722 ± 21.5
Any post-stroke outcome	417 (44.9)
First post-stroke outcome	
Early (< 7 days after stroke)	141 (15.1)
Late (≥ 7 days after stroke)	276 (29.7)
Early symptomatic chronic epileptiform (≥ 7 days after stroke)	145 (15.6)
Any stroke subcategory	349 (37.6)
Post-stroke mortality	42 (4.5)

Data presented as n (%) or mean ± standard deviation. Post-stroke outcome is defined as the occurrence of recurrent or prolonged stroke, TIA, or other stroke.

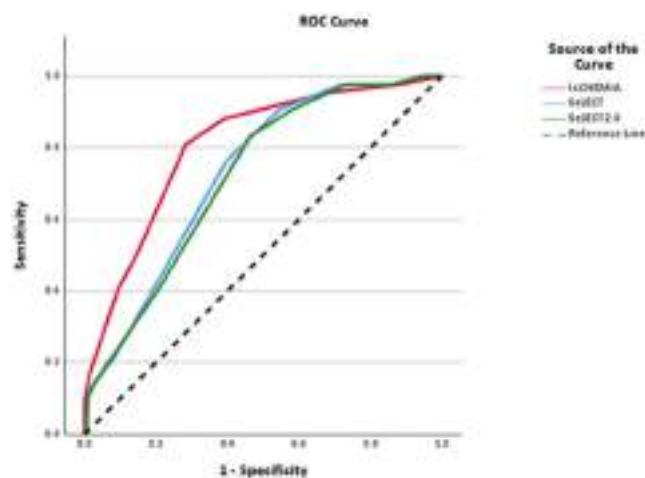


FIGURE 1 Receiver Operating Characteristic (ROC) Curves of IsCHEMiA, SeLECT, and SeLECT2.0.

and SeLECT 2.0 score ($c=0.806$ vs. 0.718 , $z=2.784$; $p=0.005$) (Figure 1).

Conclusion: The IsCHEMiA score further improved the prediction of PSE in the stroke population who received acute reperfusion therapy. This reflects the strength of the IsCHEMiA score by accessing the characteristics of the final infarct instead of stroke severity. Our findings further suggest an important role haemorrhagic lesions in epileptogenesis. Future research is warranted to study the role of preventing post-reperfusion haemorrhagic transformation in reducing the risk of PSE.

Disclosure: Nothing to disclose.

Headache 2

EPO-0244 | Consistency of effect of Atogepant in the acute treatment of migraine across multiple attacks: Results from the ECLIPSE trial

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Background and aims: Atogepant is an FDA/EMA approved calcitonin gene-related peptide receptor antagonist for migraine preventive treatment. This analysis evaluated within-participant consistency of effect (CoE) of atogepant 60 mg in the acute treatment of migraine headache and its effect across multiple migraine attacks.

Methods: ECLIPSE was a double-blind (DB), placebo-controlled, multiple-attack, crossover trial evaluating atogepant's efficacy, safety, and tolerability for acute treatment of migraine. Adults with 2–8 monthly moderate-to-severe attacks were randomised to receive atogepant for three qualifying attacks and placebo for one attack during the up to 16-week DB period. CoE was assessed by six prespecified supportive secondary endpoints, defined as achievement of key outcomes in ≥ 2 out of 3 atogepant-treated attacks, pain freedom at 2 hours postdose, pain relief at 2 hours, and sustained pain freedom/pain relief (from 2–24 to 2–48 hours postdose). Efficacy of atogepant versus placebo was evaluated for each of these endpoints across multiple attacks (Attacks 1–4).

Results: Atogepant demonstrated CoE for pain freedom and pain relief at 2 hours, as well as sustained pain freedom and relief from 2–24 to 2–48 hours (Table 1). Across all 4 treated attacks, atogepant demonstrated improvements versus placebo for pain freedom and pain relief at 2 hours, and sustained pain

TABLE 1 Supportive endpoints for consistency of effect: Responder rates across multiple attacks treated with double-blind atogepant.

Supportive Endpoints – Efficacy	Responder Rate n/N1 (%)	95% CI
Pain freedom at 2 hours after receiving DB atogepant for at least 2 out of 3 attacks	288/381 (75.6)	(73.7, 77.5)
Pain relief at 2 hours after receiving DB atogepant for at least 2 out of 3 attacks	375/487 (76.9)	(75.0, 78.7)
Sustained pain freedom 2 to 24 hours after receiving DB atogepant for at least 2 out of 3 attacks	151/191 (78.9)	(76.1, 81.6)
Sustained pain freedom 2 to 48 hours after receiving DB atogepant for at least 2 out of 3 attacks	145/188 (77.1)	(74.3, 79.9)
Sustained pain relief 2 to 24 hours after receiving DB atogepant for at least 2 out of 3 attacks	327/406 (80.5)	(78.7, 82.3)
Sustained pain relief 2 to 48 hours after receiving DB atogepant for at least 2 out of 3 attacks	304/399 (76.2)	(74.3, 78.1)

N1: number of participants who achieved consistent pain freedom or pain relief for the specified endpoint; N2: number of participants who experienced at least 2 successes (responses) or failures (non-responses) in the endpoints across all treated attacks treated with atogepant during the double-blind period; CI, confidence interval; DB, double-blind; Percentages are calculated as N1/N2 (95% CI) for the consistency of response endpoint

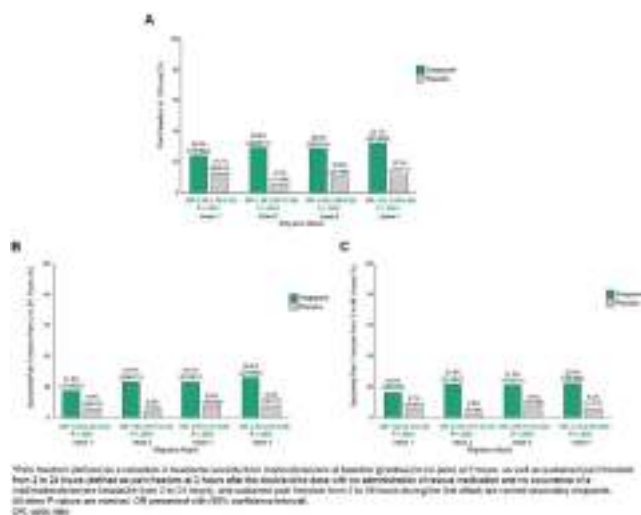


FIGURE 1: Proportion of participants achieving pain freedom (A) at 2 hours after double-blind dose, (B) from 2 to 24 hours after double-blind dose, and (C) from 2 to 48 hours after double-blind dose.

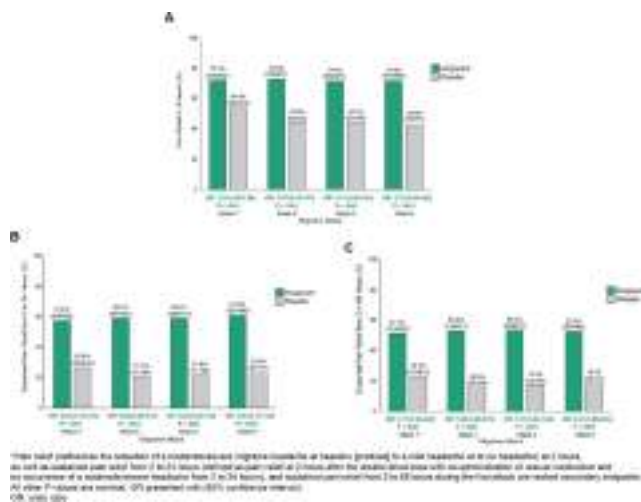


FIGURE 2: Proportion of participants achieving pain relief (A) at 2 hours after double-blind dose; (B) from 2 to 24 hours after double-blind dose, and (C) from 2 to 48 hours after double-blind dose.

freedom and relief from 2–24 to 2–48 hours (Attack 1, adjusted $P < 0.0001$; Attacks 2–4, nominal $P < 0.0001$) (Figures 1 and 2).

Conclusion: Among adults with migraine headache, atogepant demonstrated consistency of within-participant effect and showed reliable efficacy compared with placebo across multiple migraine attacks.

Disclosure: AVD has served on advisory/scientific boards and/or given lectures for AbbVie/Allergan, Angelini Pharma, Eli Lilly, Lundbeck, Neuraxpharm, Novartis, Organon, Pfizer, TEVA, and UCB, for which she has received honoraria. RO reports personal fees from AbbVie, Bayer, Eli Lilly, Lundbeck, Novartis, Organon, Pfizer, and Teva. He is a member of the Editorial Board of The Journal of Headache and Pain, International Journal of Stroke, Arquivos de Neuropsiquiatria, and Confinia Cephalalgia. MJL has received honoraria as a consultant or speaker for Eli Lilly, Teva, AbbVie, Pfizer, Organon Korea, SK pharm, and YuYu Pharma; has been the principal investigator or co-investigator in trials sponsored by Eli Lilly, Novartis, Teva (Otsuka), Allergan,

AbbVie, Lundbeck, Biohaven, Yuhan Company, Ildong Pharm, Samjin Pharm, and DongAST; and has received research support from the National Research Foundation of Korea and Korea Medical Device Development Fund. TT has been on the speakers' bureau for Amgen K.K., Daiichi Sankyo Co, Ltd, Eli Lilly Japan K.K., and Otsuka Pharmaceutical Co, Ltd, and has received research funding/collaborative research expenses from AbbVie GK, Amgen K.K., Eli Lilly Japan K.K., Eisai Co, Ltd, Lundbeck Japan K.K., and Pfizer Japan Inc. TT has also acted as an advisor to Hedgehog MedTech, Inc, Sawai Pharmaceutical Co, Ltd, and TEIJIN Pharma Ltd. DHL received honoraria for consulting from AbbVie/Allergan, Amgen, Eli Lilly, Novartis, Teva, Lundbeck, Hormosan, and Zuellig Pharma. CT reports, over the last 36 months, personal fees for participating in advisory boards or for lecturing at sponsored symposia for AbbVie, Dompé, Eli Lilly, Ipsen, Lundbeck, Medscape, Organon Pfizer, and Teva. She is principal investigator or collaborator in clinical trials sponsored by AbbVie, Eli Lilly, Ipsen, Lundbeck, Pfizer and Teva. She has received research grants from the European Commission, the Italian Ministry of Health, the Italian Ministry of University, the Migraine Research Foundation, and the Italian Multiple Sclerosis Foundation. EB-S, LL, KC, and KN are employees of AbbVie and may hold AbbVie stock.

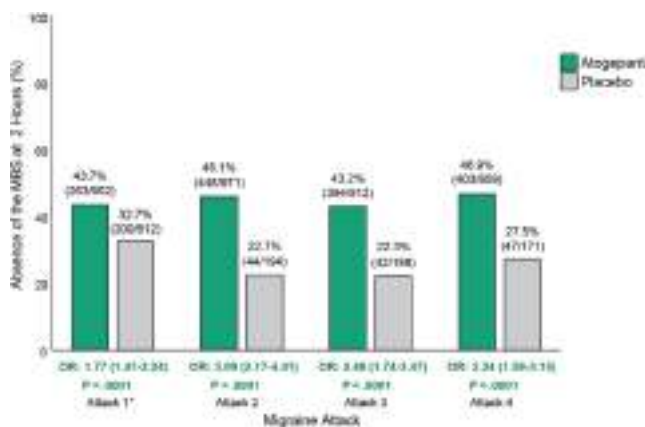
EPO-0245 | Efficacy of acute treatment with Atogepant on migraine symptoms and rescue medication use across multiple attacks: Results from the ECLIPSE trial

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Background and aims: Atogepant, an FDA/EMA approved migraine preventive treatment, has recently demonstrated efficacy for the acute treatment of a single migraine attack. This analysis evaluated the efficacy of atogepant 60mg versus placebo for the acute treatment of migraine-related symptoms and rescue medication use across multiple attacks in double-blind (DB) trial.

Methods: ECLIPSE was a DB, placebo-controlled, multiple-attack, crossover trial evaluating atogepant's efficacy, safety, and tolerability for acute treatment of migraine. Adults with 2–8 monthly moderate-to-severe attacks were randomised to receive atogepant for three qualifying attacks and placebo for one attack during the up-to-16-week DB period. This analysis evaluated the proportion of participants achieving absence of the



*Absence of the most bothersome symptom 2 hours postdose during the first treated attack is a secondary endpoint (co-primary for China). All other P-values are nominal. Participants were randomized in a 3:1:1:1 ratio to one of four treatment sequences in which each participant received placebo for 1 qualifying migraine attack and atogepant 80 mg for 3 qualifying migraine attacks during the double-blind period; participants were treated 1:1 with atogepant or placebo, respectively, for attack 1, and 3:1 with atogepant or placebo, respectively, for attacks 2-4. Odds ratio presented with 95% confidence interval; MBS, most bothersome symptom; OR, odds ratio.

FIGURE 1: Proportion of participants free from the most bothersome symptom at 2 hours postdose during the double-blind period across multiple attacks.

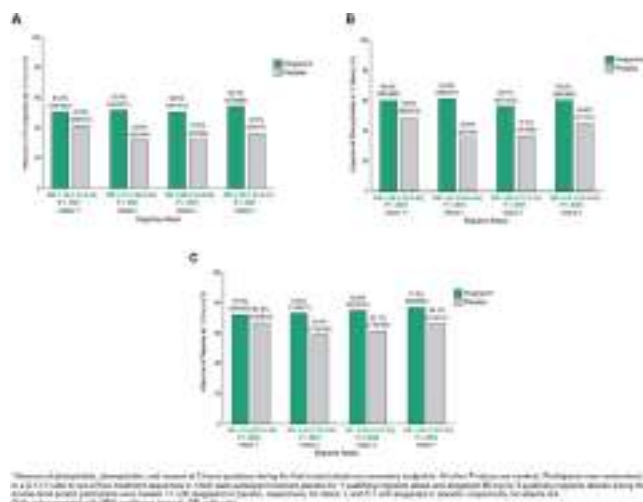


FIGURE 2: Proportion of participants with absence of photophobia, phonophobia, and nausea at 2 hours postdose during the double-blind period across multiple attacks.

	Atogepant	Placebo	Odds Ratio (95% CI)	P-value
Use of rescue medication within 24 hours, n/N (%)				
Attack 1	8/602 (1.3)	32/672 (4.8)	0.34 (0.11-0.99)	<.001
Attack 2	18/327 (5.5)	14/654 (2.1)	0.35 (0.11-0.99)	<.001
Attack 3	10/322 (3.1)	10/569 (1.8)	0.34 (0.10-0.99)	<.001
Attack 4	15/859 (1.7)	16/621 (2.6)	0.34 (0.10-0.99)	<.001
Use of rescue medication within 48 hours, n/N (%)				
Attack 1	18/602 (3.0)	33/672 (4.9)	0.39 (0.12-0.99)	<.001
Attack 2	18/327 (5.5)	14/654 (2.1)	0.35 (0.11-0.99)	<.001
Attack 3	10/322 (3.1)	10/569 (1.8)	0.34 (0.10-0.99)	<.001
Attack 4	15/859 (1.7)	16/621 (2.6)	0.34 (0.10-0.99)	<.001

Over the course of the study, 24-hour and 48-hour rescue medication rates were significantly lower in the atogepant group compared to the placebo group for all attacks (P < .001 for all comparisons). Participants who received atogepant for all attacks had significantly lower rates of rescue medication use compared to participants who received placebo for all attacks (P < .001 for all comparisons). Odds ratio presented with 95% confidence interval; OR, odds ratio.

FIGURE 3: Proportion of participants using rescue medication within 24 and 48 hours postdose during the double-blind period across multiple attacks.

most-bothersome symptom (MBS: photophobia, phonophobia, or nausea) at 2-hours postdose across attacks 1-4. Additionally, the absence of photophobia, phonophobia, and nausea at 2-hours

postdose, as well as rescue medication use within 24- and 48-hours postdose, were examined across attacks.

Results: Atogepant resulted in a significantly ($P < 0.0001$, nominal or ranked secondary as indicated) higher proportion of participants achieving absence of the MBS (OR: 1.77-3.09), photophobia (OR: 1.74-2.77), and phonophobia (OR: 1.96-3.43) 2 hours postdose across attacks 1-4 compared to placebo (Figures 1, 2). Participants had lower rates of nausea with atogepant, but these results were not significant for the ranked attack (Figure 2). Rescue medication use was lower with atogepant versus placebo across both 24 and 48 hours postdose period amongst all attacks (OR: 0.13-0.16; all $P < 0.0001$) (Figure 3).

Conclusion: Atogepant demonstrated reliable treatment effect compared to placebo by reducing migraine-related symptoms and the need for rescue medication use across multiple migraine attacks.

Disclosure: AVD has served on advisory/scientific boards and/or given lectures for AbbVie/Allergan, Angelini Pharma, Eli Lilly, Lundbeck, Neuraxpharm, Novartis, Organon, Pfizer, TEVA, and UCB, for which she has received honoraria. RO reports personal fees from AbbVie, Bayer, Eli Lilly, Lundbeck, Novartis, Organon, Pfizer, and Teva. He is a member of the Editorial Board of The Journal of Headache and Pain, International Journal of Stroke, Arquivos de Neuropsiquiatria, and Confinia Cephalalgia. ML has received honoraria as a consultant or speaker for Eli Lilly, Teva, AbbVie, Pfizer, Organon Korea, SK pharm, and YuYu Pharma; has been the principal investigator or co-investigator in trials sponsored by Eli Lilly, Novartis, Teva (Otsuka), Allergan, Abbvie, Lundbeck, Biohaven, Yuhon Company, Ildong Pharm, Samjin Pharm, and DongAST; and has received research support from the National Research Foundation of Korea and Korea Medical Device Development Fund. TT has been on the speakers' bureau for Amgen K.K., Daiichi Sankyo Co, Ltd, Eli Lilly Japan K.K., and Otsuka Pharmaceutical Co, Ltd, and has received research funding/collaborative research expenses from AbbVie GK, Amgen K.K., Eli Lilly Japan K.K., Eisai Co, Ltd, Lundbeck Japan K.K., and Pfizer Japan Inc. TT has also acted as an advisor to Hedgehog MedTech, Inc, Sawai Pharmaceutical Co, Ltd, and TEIJIN Pharma Ltd. DHL received honoraria for consulting from AbbVie/Allergan, Amgen, Eli Lilly, Novartis, Teva, Lundbeck, Hormosan, and Zuellig Pharma. CT reports, over the last 36 months, personal fees for participating in advisory boards or for lecturing at sponsored symposia for AbbVie, Dompé, Eli Lilly, Ipsen, Lundbeck, Medscape, Organon, Pfizer, and Teva. She is principal investigator or collaborator in clinical trials sponsored by AbbVie, Eli Lilly, Ipsen, Lundbeck, Pfizer and Teva. She has received research grants from the European Commission, the Italian Ministry of Health, the Italian Ministry of University, the Migraine Research Foundation, and the Italian Multiple Sclerosis Foundation. EB-S, LL, KC, and KN are employees of AbbVie and may hold AbbVie stock.

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Background and aims: Atogepant is approved for the preventive treatment of migraine in adults and has recently demonstrated its efficacy and safety for the acute treatment of migraine. This analysis evaluated the impact of atogepant 60mg administered as needed (PRN) for acute treatment of migraine on cognitive function assessed by the ability to think clearly.

Methods: ECLIPSE was a double-blind (DB), placebo-controlled, multiple-attack, crossover trial evaluating atogepant's efficacy, safety, and tolerability for acute migraine treatment. Adults with 2–8 monthly moderate-to-severe attacks were randomised to receive atogepant for three qualifying attacks and placebo for one attack during the up to 16-week DB period. Cognitive function scale is a single-item, patient-reported questionnaire measuring participants' difficulty in ability to think clearly, rated from 0 (not difficult at all) to 3 (very difficult). The proportion of participants achieving the ability to think clearly at 2-, 4-, and 8-hours postdose across attacks 1–4 was an additional (exploratory)

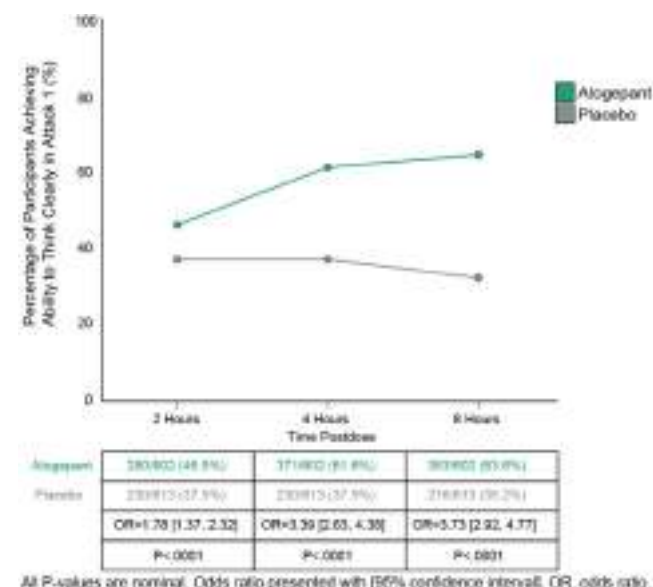


FIGURE 1: Proportion of participants who reported “not difficult at all” in ability to think clearly at 2-, 4-, and 8-hours postdose in the first migraine attack.

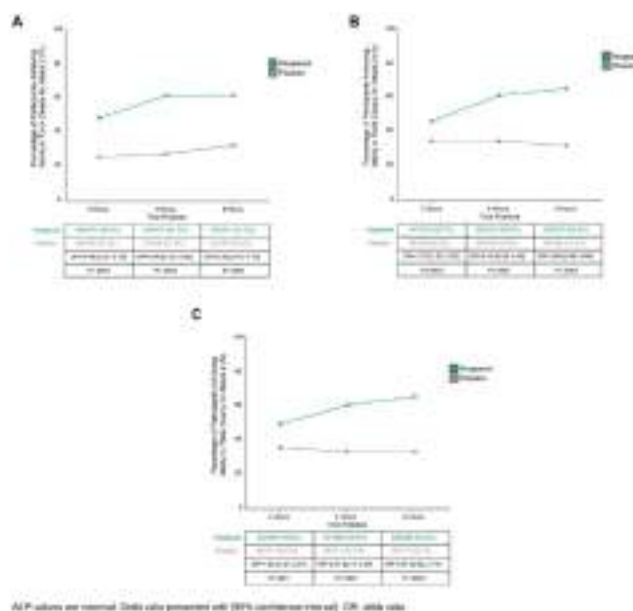


FIGURE 2: Proportion of participants who reported “not difficult at all” in ability to think clearly at 2-, 4-, and 8-hours postdose across attacks 2, 3, and 4 (A, B, and C, respectively).

efficacy endpoint. Data for atogepant and placebo are presented with nominal *p*-values.

Results: Atogepant treatment resulted in a nominally significant higher proportion of participants achieving the ability to think clearly compared with placebo as early as 2-hours postdose in the first treated attack (OR: 1.78, Figure 1); this effect increased over time through 4- and 8-hours postdose (OR: 3.39, 3.73, respectively; Figure 1). A similar reliable response was observed across attacks 2–4 (Figure 2).

Conclusion: A greater proportion of participants treated with atogepant achieved the ability to think clearly compared to placebo as early as 2-hours and up to 8-hours postdose across multiple attacks.

Disclosure: AVD has served on advisory/scientific boards and/or given lectures for AbbVie/Allergan, Angelini Pharma, Eli Lilly, Lundbeck, Neuraxpharm, Novartis, Organon, Pfizer, TEVA, and UCB, for which she has received honoraria. RO reports personal fees from AbbVie, Bayer, Eli Lilly, Lundbeck, Novartis, Organon, Pfizer, and Teva. He is a member of the Editorial Board of The Journal of Headache and Pain, International Journal of Stroke, Arquivos de Neuropsiquiatria, and Confinia Cephalalgia. RG-G has received honoraria for consultant and/or advisory boards from Allergan/AbbVie, Astra Zeneca, Almirall, Bial, Biogen, Bristol-Myers Squibb, CMBE, FLOAT, Lilly, Lundbeck, Merk, Novartis, Novo Nordisk, Organon, Pfizer, Roche, Sanofi, Tecnifar and Teva; research funding from Fundação Ciência Tecnologia (Project 29675, MigN2Treat, 02/SAICT/2017); Learning-Health, Luz Saúde (LiON, Luz Innovation on Neurosciences); Novartis-Sociedade Portuguesa de Cefaleias; Bolsa –Projecto Centro de Investigação Interdisciplinar em Saúde and UCP. PPR reports personal fees for consulting from AbbVie, Amgen, Dr. Reddy's, Eli Lilly, Lundbeck, Medscape, Novartis, Organon, Pfizer, and Teva in the last three years. She has received personal fees for speaking from AbbVie, Amgen, Dr. Reddy's, Eli Lilly, Lundbeck, Medscape, Novartis, Organon, Pfizer and Teva, and has had grants paid to her research group from AbbVie, AGAUR, EraNet Neuron, FEDER

RIS3CAT, Instituto Investigación Carlos III, Novartis, and Teva. UR has served on advisory boards for AbbVie, Eli Lilly, Lundbeck, Novartis, Pfizer, and Teva and has received institutional honoraria for lectures from AbbVie, Eli Lilly, Lundbeck, Novartis, StreaMedUp, Springer, and Teva, received institutional honoraria for consulting services from Lundbeck, Pfizer, and AbbVie; received research funding from Novartis (CHERUB01) and the German Federal Ministry of Education and Research; UR is an associate editor of the Journal of Headache and Pain, and is the president of the EHF. GA, EB-S, LL, CC, and KC are employees of AbbVie and may hold AbbVie stock.

EPO-0247 | Longitudinal shifts in migraine frequency patterns: Transition from chronic to episodic migraine in a pooled analysis of the PEARL and FINESSE studies

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Background and aims: Chronic migraine (CM) is associated with increased disability, greater reductions in health-related quality of life and higher rates of medication overuse compared with episodic migraine (EM). Consequently, achieving reversion from CM to EM is an important therapeutic goal. This analysis assessed long-term changes in migraine frequency among participants with CM in PEARL and FINESSE.

Methods: The 24-month, prospective, observational, real-world PEARL (pan-European) and FINESSE (Germany and Austria) studies evaluated the effectiveness and tolerability of fremanezumab in routine clinical practice among adults with migraine. Mean monthly migraine days (MMD) were assessed using daily headache diaries completed by participants throughout the study. In this pooled analysis, the proportions of participants shifting from CM (diagnosed at baseline) to high-frequency EM (HFEM; ≥ 8 and <15 MMD), moderate-frequency EM (MFEM; ≥ 4 and <8 MMD) and low-frequency EM (LFEM; <4 MMD) were assessed over time.

Results: Of 2145 participants included in the pooled analysis, 1212 had CM at baseline (Table 1). By Month 6, 20.4%, 25.0% and 39.5% of participants with available data ($n=1070$) had transitioned to HFEM, MFEM and LFEM, respectively (Table 2). At Month 24, 49.5% of participants with available data ($n=641$) had shifted to LFEM and only 7.6% of participants still had >15 MMD.

TABLE 1 Baseline characteristics.

	Participants in the PEARL & FINESSE studies N=2145
Age	
Mean (SD)	45.96 (12.17)
Median (Min, Max)	47 (18, 85)
Past preventive migraine treatment attempts, n (%)	
≤ 4	1397 (65.1)
≥ 4	748 (34.9)
Time from initial migraine onset date to fremanezumab initiation, years	
Mean (SD)	26.72 (13.53)
Median (Min, Max)	26 (0, 76)
Time from date of migraine diagnosis to first fremanezumab initiation, years	
Mean (SD)	16.12 (12.79)
Median (Min, Max)	13 (0, 65)
Presence of onabotulinumtoxinA failure as a past preventive treatment, n (%)	711 (36.4)
Migraine diagnosis, n (%)	
CM	1212 (56.3)
EM	933 (43.5)

CM, chronic migraine; EM, episodic migraine; Min, minimum; Max, maximum; SD, standard deviation.

TABLE 2 Proportion of participants transitioning from CM at baseline to HFEM, MFEM and LFEM at Months 6, 12, 18 and 24.

	Baseline	Month 6	Month 12	Month 18	Month 24
Participants with available data, n (%)	1212 (56.3)	1070 (50.1)	1070 (50.1)	1070 (50.1)	641 (30.0)
Participants with available data, n (%)	1212 (56.3)	1070 (50.1)	1070 (50.1)	1070 (50.1)	641 (30.0)
Participants with available data, n (%)	1212 (56.3)	1070 (50.1)	1070 (50.1)	1070 (50.1)	641 (30.0)
Participants with available data, n (%)	1212 (56.3)	1070 (50.1)	1070 (50.1)	1070 (50.1)	641 (30.0)
Participants with available data, n (%)	1212 (56.3)	1070 (50.1)	1070 (50.1)	1070 (50.1)	641 (30.0)

Conclusion: Fremanezumab facilitated meaningful reductions in migraine frequency over time, with nearly half of the participants with CM who persisted with treatment up to Month 24 transitioning to LFEM. These findings highlight the long-term benefit of fremanezumab and its potential to restore low disease burden in a relevant subset of patients.

Disclosure: MA has received personal fees and declares PI/collaborator in clinical trials for AbbVie, AstraZeneca, Eli Lilly, GlaxoSmithKline, Lundbeck, Novartis, Pfizer and Teva Pharmaceuticals; and research grants from the Lundbeck Foundation, the Novo Nordisk Foundation, Lundbeck and Novartis. GB is, or was, a member of the following advisory boards: Grünenthal, Lilly, Lundbeck, Novartis, Pfizer and Teva Pharmaceuticals; is a speaker or member of the following speaker boards: Grünenthal, Lilly, Lundbeck, Novartis, Pfizer and Teva Pharmaceuticals; has acted as a consultant for Grünenthal, Lilly, Novartis and Teva Pharmaceuticals; and received grant support for research or education from Teva Pharmaceuticals. CT declares advisory boards, symposia and/or PI/collaboration in clinical trials for AbbVie, Chordate, Dompé, Eli Lilly,

Ipsen, Lundbeck, Novartis, Pfizer and Teva Pharmaceuticals; and research grants from the European Commission, the Italian Ministry of Health, the Italian Multiple Sclerosis Foundation and the Migraine Research Foundation. CG is, or was, a member of the following advisory boards: AbbVie, Hormosan Pharma, Lilly, Lundbeck, Merz, Novartis Pharma, Organon, Sanofi-Aventis, Teva Pharmaceuticals and Vectura Fertin Pharma; is a speaker or member of the following speaker boards: AbbVie, Hormosan Pharma, Lilly, Lundbeck, Novartis Pharma, Reckitt-Benckiser, Sanofi-Aventis and Teva Pharmaceuticals; has acted as a consultant for Chordate and Pfizer; has served the following editorial boards: Acta Neurologica Scandinavia, Frontiers in Neurology and Medizinischer Sachverständiger; and reports author royalties for Novartis Pharma. PP-R has received honoraria as a consultant and speaker for AbbVie, Amgen, Eli Lilly, Lundbeck, Medscape, Novartis, Pfizer and Teva Pharmaceuticals; research grants from AbbVie, Novartis and Teva Pharmaceuticals; and clinical trial funding from AbbVie, Alder, Amgen, Biohaven, electroCore, Eli Lilly, Lundbeck, Novartis and Teva Pharmaceuticals. LN is, or was, a member of the following advisory boards: AbbVie, Lundbeck and Teva Pharmaceuticals; is a speaker or member of the following speaker boards: AbbVie, Lundbeck, Novartis Pharma and Teva Pharmaceuticals; has acted as a consultant for Perfood; has served the following editorial boards: Frontiers in Neurology. PK is an employee and/or shareholders of Teva Pharmaceuticals. XH is an employee of Teva GmbH. HA is an employee and/or shareholder of Teva Pharmaceuticals. AS is, or was, a member of the following advisory boards: Allergan, Lilly, Novartis, Sanofi and Teva Pharmaceuticals; and a speaker or member of the following speaker boards: Allergan, Lilly, Novartis, Sanofi and Teva Pharmaceuticals; has acted as a consultant for Sanofi and Teva Pharmaceuticals; received grant support for research or education from Novartis; and acted as an editorial board member for Lilly, Novartis and Sanofi.

EPO-0248 | **Changes in IHS-proposed migraine control categories over time with fremanezumab: A pooled longitudinal analysis of the PEARL and FINESSE studies**

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Background and aims: With advances in preventive therapy, treatment goals for migraine have evolved from reduced migraine frequency toward optimal disease control. To evaluate disease control among participants in the long-term, real-world PEARL and FINESSE studies, this pooled analysis assessed mean monthly migraine days (MMD) according to IHS-defined migraine control categories in adults treated with fremanezumab.

Methods: The prospective, observational PEARL (pan-European) and FINESSE (Germany and Austria) studies evaluated the effectiveness and tolerability of fremanezumab in routine clinical practice among adults with episodic or chronic migraine (EM; CM), over 24 months. MMD were assessed using daily headache diaries completed by participants throughout the study. In this pooled analysis, the proportions of participants achieving different treatment outcomes categories (migraine freedom [0MMD], optimal [<4MMD], modest [4–6MMD] or insufficient [>6MMD] control) were assessed over 24 months.

TABLE 1 Baseline characteristics.

	Participants in the PEARL & FINESSE studies N=2145
Age	
Mean (SD)	45.96 (12.17)
Median (Min, Max)	47 (18, 85)
Past preventive migraine treatment attempts, n (%)	
<4	1397 (65.1)
≥4	748 (34.9)
Time from initial migraine onset date to fremanezumab initiation, years	
Mean (SD)	20.72 (13.53)
Median (Min, Max)	26 (0, 70)
Time from date of migraine diagnosis to first fremanezumab initiation, years	
Mean (SD)	16.12 (12.79)
Median (Min, Max)	17 (0, 65)
Presence of onabotulinumtoxinA failure as a past preventive treatment, n (%)	791 (36.4)
Migraine diagnosis, n (%)	
CM	1212 (56.5)
EM	933 (43.5)

CM, chronic migraine; EM, episodic migraine; Max, maximum; Min, minimum; SD, standard deviation.

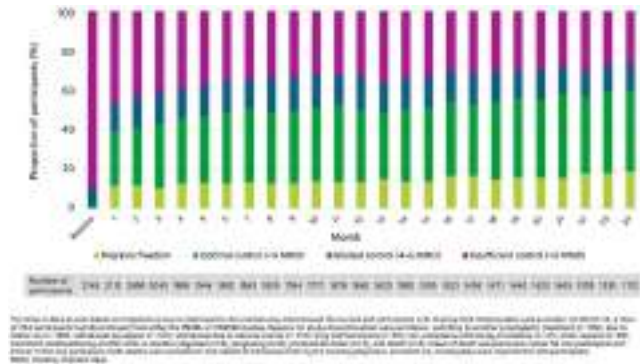


FIGURE 1 MMD control over time.

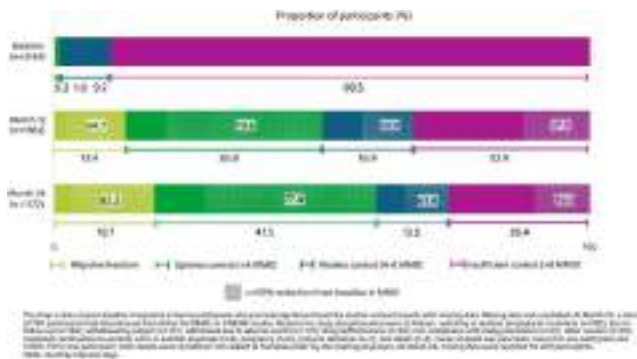


FIGURE 2 Participants achieving ≥ 50 reduction from baseline in MMD within each control category at Month 12 and Month 24

Results: A total of 2145 participants were included in the pooled analysis (Table 1). Optimal migraine control and migraine freedom was achieved by 28.0% and 11.2% of participants, respectively, as early as 1 month after starting fremanezumab and by 41.5% and 18.7% of participants at Month 24 (Figure 1). Among the subset of participants who had modest or insufficient control at Months 12 and 24 respectively, a substantive proportion achieved $\geq 50\%$ reduction from baseline in MMD (Figure 2).

Conclusion: Using an outcome measure that aligns with individualised treatment goals beyond traditional responder rates, this pooled analysis of the PEARL and FINESSE studies demonstrated the early and sustained effectiveness of fremanezumab in substantially improving migraine control.

Disclosure: MA has received personal fees and declares PI/collaborator in clinical trials for AbbVie, AstraZeneca, Eli Lilly, GlaxoSmithKline, Lundbeck, Novartis, Pfizer and Teva Pharmaceuticals; and research grants from the Lundbeck Foundation, the Novo Nordisk Foundation, Lundbeck and Novartis. GB is, or was, a member of the following advisory boards: Grünenthal, Lilly, Lundbeck, Novartis, Pfizer and Teva Pharmaceuticals; is a speaker or member of the following speaker boards: Grünenthal, Lilly, Lundbeck, Novartis, Pfizer and Teva Pharmaceuticals; has acted as a consultant for Grünenthal, Lilly, Novartis and Teva Pharmaceuticals; and received grant support for research or education from Teva Pharmaceuticals. CT declares advisory boards, symposia and/or PI/collaboration in clinical trials for AbbVie, Chordate, Dompé, Eli Lilly, Ipsen, Lundbeck, Novartis, Pfizer and Teva Pharmaceuticals; and research grants from the European Commission, the Italian Ministry of Health, the Italian Multiple Sclerosis Foundation and the Migraine Research Foundation. CG is, or was, a member of the following advisory boards: AbbVie, Hormosan Pharma, Lilly, Lundbeck, Merz, Novartis Pharma, Organon, Sanofi-Aventis, Teva Pharmaceuticals and Vectura Fertin Pharma; is a speaker or member of the following speaker boards: AbbVie, Hormosan Pharma, Lilly, Lundbeck, Novartis Pharma, Reckitt-Benckiser, Sanofi-Aventis and Teva Pharmaceuticals; has acted as a consultant for Chordate and Pfizer; has served the following editorial boards: Acta Neurologica Scandinavica, Frontiers in Neurology and Medizinischer Sachverständiger; and reports author royalties for Novartis Pharma. PP-R has received honoraria as a consultant and speaker for AbbVie, Amgen, Eli Lilly, Lundbeck, Medscape, Novartis, Pfizer and Teva Pharmaceuticals; research grants from AbbVie, Novartis and Teva Pharmaceuticals; and clinical trial funding from AbbVie,

Alder, Amgen, Biohaven, electroCore, Eli Lilly, Lundbeck, Novartis and Teva Pharmaceuticals. LN is, or was, a member of the following advisory boards: AbbVie, Lundbeck and Teva Pharmaceuticals; is a speaker or member of the following speaker boards: AbbVie, Lundbeck, Novartis Pharma and Teva Pharmaceuticals; has acted as a consultant for Perfood; has served the following editorial boards: Frontiers in Neurology. PK is an employee and/or shareholder of Teva Pharmaceuticals. XH is an employee of Teva GmbH. SZ is an employee and/or shareholder of Teva Pharmaceuticals. AS is, or was, a member of the following advisory boards: Allergan, Lilly, Novartis, Sanofi and Teva Pharmaceuticals; and a speaker or member of the following speaker boards: Allergan, Lilly, Novartis, Sanofi and Teva Pharmaceuticals; has acted as a consultant for Sanofi and Teva Pharmaceuticals; received grant support for research or education from Novartis; and acted as an editorial board member for Lilly, Novartis and Sanofi.

EPO-0249 | Eptinezumab reduced acute medication use in participants with chronic migraine and medication-overuse headache: 24-week data from the RESOLUTION trial

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Background and aims: This phase 4 trial evaluated the 24-week impact of eptinezumab on acute medication use in adults with chronic migraine (CM) and medication-overuse headache (MOH) who also received patient education.

Methods: RESOLUTION (NCT05452239) included a 12-week double-blind placebo-controlled period and 12-week open-label extension (OLE). At baseline, adults with CM and MOH were randomized 1:1 to IV eptinezumab 100mg or placebo; all participants received a brief educational intervention on MOH before infusion. At Week 12, all participants received eptinezumab 100mg. Acute medication use was captured daily via electronic diary. Analyses applied inferential statistics to the placebo-controlled analyses and descriptive methods to the OLE.

Results: Participants randomized: $n=608$; treated, baseline: $n=604$; full-analysis set, placebo-controlled period: $n=602$; treated/analyzed, OLE: $n=593$; completed: $n=584$. Baseline

mean monthly acute medication days: 20.1 across arms. Eptinezumab was more favourable than placebo in reducing monthly acute medication use days over Weeks 1–4 (–11.3 vs. –7.7) and 1–12 (–11.2 vs. –7.8), particularly triptan days ($p < 0.01$ all comparisons). During the OLE, improvements were sustained for participants continuing eptinezumab; participants started on placebo and switched to eptinezumab achieved similar changes as those continuing eptinezumab. Eptinezumab provided greater decreases in mean Severity of Dependence Scale for Headache medication score than placebo at Week 12 (–2.2 vs. –1.2; $p = 0.0002$).

Conclusion: In participants with CM and MOH who also received patient education, eptinezumab resulted in greater reductions than placebo in acute medication use and dependence over Weeks 1–12. Similar reductions in acute medication use were seen during open-label eptinezumab treatment, irrespective of treatment in the placebo-controlled period.

Disclosure: CL has participated on an advisory board and received payment for lectures arranged by AbbVie, Lundbeck, Novartis, Pfizer, and Roche, and has received research sponsorship from AbbVie. RHJ has given lectures for Allergan, ATI, Eli Lilly, Lundbeck, Merck, Novartis, Pfizer, and Teva; served as investigator in clinical trials with ATI, Eli Lilly, Lundbeck, Novartis, and Novo Nordisk; is the director of the Danish Headache Center, Lifting The Global Burden of Headache, and Founder of Master of Headache Disorders at University of Copenhagen; and has received research funding from ATI, Lundbeck Foundation, Rigshospitalet, The Medical Society in Copenhagen, Novo Nordisk Foundation, Tryg Foundation, and University of Copenhagen. HWS has received personal fees from AbbVie, Eli Lilly, Lundbeck, Novartis, and Teva, and has received research grants from Novartis and Novo Nordisk Foundation. CT in the past 3 years has received support (financial or drugs) from AbbVie and Novartis for an investigator-initiated trial; consulting fees for the participation in advisory boards for AbbVie, Dompé, Eli Lilly, Ipsen, Lundbeck, Organon, Pfizer, and Teva; honoraria for scientific lectures and presentations from AbbVie, Eli Lilly, Lundbeck, Pfizer, and Teva; support for attending meetings from AbbVie, Dompé, Eli Lilly, Ipsen, Lundbeck, Pfizer, and Teva; has been Principal Investigator in clinical trials sponsored by AbbVie, Biohaven, Chordate, Eli Lilly, Ipsen, Lundbeck, Pfizer, and Teva; and received grants from the European Commission, the Italian Ministry of Health, and the Italian Ministry of University. FV has received financial support from AbbVie, Angelini, and Lundbeck for investigator-initiated trials; consulting fees for the participation in advisory boards from AbbVie, Angelini, Eli Lilly, Lundbeck, Organon, Pfizer, and Teva; honoraria for scientific lectures and presentations from AbbVie, Eli Lilly, Lundbeck, Pfizer, and Teva; support for attending meetings from AbbVie, Eli Lilly, Lundbeck, Pfizer, and Teva; and has been Principal Investigator in clinical trials sponsored by AbbVie, Eli Lilly, Lundbeck, Pfizer, and Teva. ML-M reports personal fees for advisory boards, speaker panels, or investigation studies from Allergan, Amgen, Astellas, ATI, BMS, Boehringer, Boston Scientific, CoLucid, Convergence, Eli Lilly, GlaxoSmithKline, Grunenthal, Ipsen, Lundbeck, Medtronic, MSD, Novartis, Orion Pharma, Perfood, Pfizer, Reckitt Benckiser, St. Jude, Salvia BioElectronics, Sanofi-Aventis, Teva, UCB, UPSA, and Zambon. AB has served on advisory boards for AbbVie, Aeon, Alder, Amgen, Biohaven, Eaglet, Eli Lilly, Impel, Lundbeck, Novartis, Promius, Revance,

Supernus, and Teva; and has received funding for speaking from AbbVie, Amgen, Avanir, Biohaven, Depomed, Eli Lilly, Impel, Lundbeck, Pernix, Promius, Supernus, and Teva. [Disclosures for the remaining authors will be included in the presentation].

EPO-0250 | Real-world effectiveness and adherence of atogepant in chronic and episodic migraine: Results from the ATOMUR study

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Background and aims: Atogepant, an oral CGRP receptor antagonist, has shown efficacy in migraine prevention in clinical trials. Nonetheless, real-world data on its long-term effectiveness and treatment adherence in chronic (CM) and episodic migraine (EM) remain limited. We evaluated treatment compliance and changes in monthly migraine days (MMDs), MIDAS score and its side effects in patients with CM and EM treated with atogepant in clinical practice.

Methods: 101 patients diagnosed with chronic or episodic migraine who initiated preventive treatment with atogepant were included and followed at 3, 6, and 12 months.

Results: 57 (56.4%) patients had CM and 44 (43.6%) EM. Mean baseline MMDs were 18.6 ± 4.9 in CM and 9.8 ± 2.7 in EM patients. At 3 months, mean MMDs were reduced to 10.2 ± 4.6 in CM and 5.4 ± 2.9 in EM, corresponding to a reduction of 45% and 44%, respectively. These reductions were sustained at 6 months (CM: 9.6 ± 4.8 ; EM: 5.1 ± 2.6) and 12 months (CM: 9.1 ± 5.0 ; EM: 4.9 ± 2.8). At 12 months, 68% of patients achieved a $\geq 50\%$ reduction in MMDs. Mean MIDAS scores decreased significantly from baseline (48.2 ± 19.6) to 3 months (28.5 ± 17.3 ; $p < 0.001$), with further reductions at 6 months (24.1 ± 16.8 ; $p < 0.001$) and 12 months (21.6 ± 17.1 ; $p < 0.001$). Treatment adherence remained high, with 80% of patients continuing therapy at 6 months and 66% at 12 months. Discontinuations were mainly due to lack of effectiveness or mild adverse events (nausea).

Conclusion: Atogepant showed sustained effectiveness in reducing monthly migraine days and high adherence over 12 months in patients with chronic and episodic migraine. These findings support its role as a long-term preventive treatment.

Disclosure: Nothing to disclose.

M. Katsuki¹; M. Okada²; T. Shimazu³; T. Takeshima⁴; H. Igarashi⁵; M. Tatsumoto⁶; Y. Ito⁷; T. Mitsufoji²; M. Hirayama²; K. Ohbayashi⁷; N. Imai⁸; S. Kiku⁴; D. Danno⁴; J. Miyahara⁴; K. Murakata⁴; Y. Matsumori⁹; Y. Nakazato²; K. Fujita¹⁰; E. Hoshino³; Y. Kato¹¹; Y. Maruki³; R. Delyon¹²; I. Kizil¹²; U. Alexandre¹²; F. Cadiou¹²; G. Oyama²; S. O'Connor¹³; T. Ward¹⁴; K. Moran¹⁵; T. Yamamoto²

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Background and aims: The International Classification of Headache Disorders, 3rd edition (ICHD-3), defines migraine at the diagnostic level but is not suited for qualifying individual headache attacks, limiting accurate evaluation of acute treatments. This study aimed to construct an expert-derived ground-truth dataset of individual attacks and to compare three artificial intelligence (AI)-based classifications of each attack with specialist judgment.

Methods: Patients from headache-specialized clinics in Japan completed standardized clinical questionnaires and recorded attacks in a smartphone diary (Migraine Buddy) for 3 months. Three board-certified headache specialists independently evaluated 10 attacks from each of 10 patients (100 attacks total), using full clinical context and diary data. Each attack was rated on a five-level migraine-probability scale. Majority voting classified attacks as migraine or non-migraine and served as the ground truth. We evaluated three AI approaches against this reference: (1) a rule-based ICHD-3 decision tree, (2) large language models (LLMs), and (3) supervised machine-learning models trained on expert labels.

Results: Ninety-eight attacks were eligible for analysis; 82 were classified as migraine and 16 as non-migraine. The ICHD-3 rule-based approach showed high specificity (0.81) but low sensitivity

(0.43). LLMs demonstrated high sensitivity (0.95) and moderate specificity (0.69), with notable variability across repeated runs. XGBoost models achieved balanced performance, with sensitivity of 0.84 and specificity of 0.88.

Conclusion: Automated approaches can approximate specialist-level classification of individual headache attacks, with XGBoost showing the most robust performance. These findings support the use of AI for per-attack migraine qualification while reinforcing the continued role of expert clinical judgment.

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EPO-0252 | Magnetic resonance-guided focused ultrasound (MRgFUS) in pharmacoresistant trigeminal neuralgia: A single-center pilot feasibility and safety study

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Background and aims: Pharmacoresistant trigeminal neuralgia (TN) represents a severe form of neuropathic pain and poses a major therapeutic challenge. Functional neurosurgery has identified central targets involved in pain perception, such as the central lateral nucleus (CLN) of the thalamus. Magnetic resonance-guided focused ultrasound (MRgFUS) enables precise thermal lesioning of deep brain structures, including the thalamus, in a non-invasive manner. This technique is an innovative and expanding therapeutic option for refractory neuropathic pain.

Methods: This is a prospective, single-center, uncontrolled pilot study designed to assess the feasibility and safety of MRgFUS targeting the CLN. Patients with pharmacoresistant TN evaluated at our Headache Unit are included. Clinical screening, neuroimaging, psychopathological and neuropsychological assessments are performed. The procedure is conducted under conscious sedation, with monitoring of adverse events and postoperative MRI. Validated scales are used for baseline and follow-up assessments (CBD, DN4, PainDETECT, EQ-5D-5L, PDQ, PCS, PGIC, HADS).

Results: To date, one patient (male, 52years) has been successfully treated. One week after the procedure, complete resolution of trigeminal pain and paroxysms was observed, with no clinically relevant adverse events. Additional patients will be included in the coming months.

Conclusion: MRgFUS targeting the CLN demonstrated good tolerability, absence of complications, and a relevant analgesic effect in this first case. This ongoing study aims to confirm the feasibility of MRgFUS as a non-invasive therapeutic alternative for pharmacoresistant trigeminal neuralgia.

Disclosure: Nothing to disclose.

R. De Icco; F. Cammarota; V. Grillo; G. Vaghi; M. Corrado; B. Agostini; M. Giraudo; D. Martinelli; M. Allena; G. Sances; R. Greco; C. Demartini; M. Francavilla; S. Facchetti; C. Tassorelli

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Background and aims: Atogepant is an oral CGRP receptor antagonist approved for migraine prevention. The aim of this study was to investigate the biomolecular changes induced by atogepant in individuals with high-frequency episodic migraine (HFEM).

Methods: In this prospective study, 40 participants (age 35.5 ± 21.9 years, 30 females) underwent blood sampling in the interictal phase at baseline (T0) and after a 3-month-daily atogepant 60 mg treatment (T1). The biomolecular assessment included: (i) monoacylglycerol lipase (MAGL) and fatty acid amid hydrolase (FAAH) gene expression (mRNA) in peripheral blood mononuclear cells (PBMCs); (ii) microRNAs expression in PBMCs, namely miR-382-5p, miR-34a, and miR-155; and (iii) plasma levels of CGRP and PACAP (pg/mL).

Results: At T1, 70% of participants qualified as Responders ($> 50\%$ reduction in monthly migraine days). MAGL expression only decreased in Responders (Responders: T0: 9.8 ± 5.4 vs. T1: 13.2 ± 7.9 relative quantification – RQ, $p=0.021$, Non-Responders: T0: 7.4 ± 4.3 vs. T1: 8.3 ± 4.4 RQ, $p=0.552$). At T0 MAGL was higher in Responders compared to Non-Responders ($p=0.022$). At T1, we did not find modifications in FAAH and microRNAs expression or CGRP and PACAP levels.

Conclusion: After a 3-month atogepant treatment, we described a modulation of MAGL expression in Responders. Along with clinical benefit, atogepant treatment was associated with an improvement in the homeostasis of our pain-control system. It remains unclear whether the MAGL change reflects a direct pharmacological effect or, more plausibly, a downstream consequence of clinical improvement.

Disclosure: RDI received honoraria from AbbVie for advisory board participation and speaker invitation. The ATOM project was funded by a CORE – AMD Resident Research Grant (application ID#: 1273396).

EPO-0254 | Indomethacin for the symptomatic treatment of migraine: A clinical reappraisal

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Background and aims: Indomethacin is a potent NSAID with distinctive neurovascular pharmacology, including interactions with nitric oxide pathways and significant effects on intracranial pressure regulation. Despite long-standing clinical use in headache disorders, evidence supporting indomethacin for the symptomatic treatment of migraine remains limited. This study

aims to provide new clinical data on its effectiveness and tolerability in the symptomatic treatment of migraine.

Methods: This is a pilot, observational, open-label, head-to-head study. We enrolled adults with low-frequency episodic migraine (2–8 attacks/month). Participants were sequentially assigned in alternating order to indomethacin 50 mg or ibuprofen 600 mg, taken at attack onset. The primary endpoint was 2-hour pain freedom during the first treated attack. We also recorded the rate of adverse events.

Results: A total of 120 participants were included (indomethacin $n=60$; ibuprofen $n=60$). Two-hour pain freedom occurred more frequently with indomethacin (43.3%) than with ibuprofen (21.7%) ($p=0.01$). Adverse events were numerically more common with indomethacin (36.7%) than with ibuprofen (21.7%), although without a statistically significant difference ($p=0.07$).

Conclusion: Indomethacin showed superior outcomes versus ibuprofen, supporting a clinical reappraisal of this drug as an acute migraine option. Its higher efficacy might relate not only to anti-inflammatory activity but, importantly, also to modulation of nitric oxide-mediated neurovascular signaling and intracranial pressure dynamics. Although not statistically significant, indomethacin was associated with a higher number of adverse events, so its use should be preferably considered in more severe or refractory cases.

Disclosure: Nothing to disclose.

EPO-0255 | Erenumab versus OnabotulinumtoxinA for reducing triptan use: A target trial emulation study

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Background and aims: To compare the real-world effectiveness of Erenumab and OnabotulinumtoxinA in reducing triptan use among patients with migraine

Methods: We used nationwide Norwegian health registry data to emulate a target trial comparing initiation of Erenumab with OnabotulinumtoxinA. Propensity score overlap weighting was applied to balance baseline characteristics. Outcomes were assessed over 12 months and included absolute change in triptan defined daily doses (DDD) and achievement of $\geq 50\%$ reduction in triptan use. Weighted linear mixed effects models were used for continuous outcomes, and weighted logistic mixed effects models for binary outcomes. Both intention-to-treat and per-protocol analyses were conducted.

TABLE 1 Primary outcome: Absolute change in triptan daily defined doses (DDD).

Follow-up interval	ITT: Difference in DDDs (Erenumab - Botex)	95% CI	p-value	PP: Difference in DDDs	95% CI	p-value
0-3 months	-6.36	-10.69 to -2.01	<0.001	-6.59	-12.22 to -0.97	<0.001
3-6 months	-6.38	-10.65 to -2.11	<0.001	-6.37	-12.05 to -0.68	<0.001
6-9 months	-5.21	-9.72 to -0.69	<0.001	-7.09	-12.93 to -1.25	0.001
9-12 months	-4.24	-8.91 to -0.57	0.002	-6.01	-12.34 to -0.28	0.013

TABLE 2 Secondary outcome: $\geq 50\%$ reduction in triptan use.

Follow-up interval	ITT: OR (Erenumab vs Botex)	95% CI	p-value	PP: OR	95% CI	p-value
0-3 months	1.49	1.31 - 1.69	<0.001	1.66	1.42 - 1.93	<0.001
3-6 months	1.61	1.42 - 1.84	<0.001	1.81	1.55 - 2.17	<0.001
6-9 months	1.39	1.18 - 1.64	<0.001	1.63	1.32 - 2.02	<0.001
9-12 months	1.18	1.02 - 1.35	0.027	1.55	1.29 - 1.86	0.001

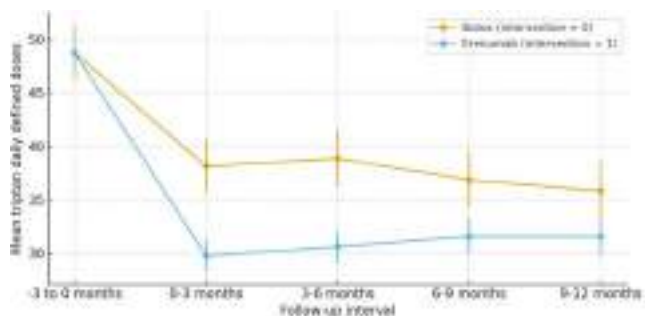


FIGURE 1 Weighted mean triptan daily defined doses over time (ITT).

Results: A total of 3,062 Erenumab users and 2,431 OnabotulinumtoxinA users were included. After weighting, baseline characteristics were identical. As shown in Table 1, Erenumab produced greater reductions in triptan DDDs. In the intention-to-treat analysis, adjusted between-group differences ranged from -8.35 (95% CI -10.69 to -6.01) at 0–3 months to -4.24 (-6.91 to -1.57) at 9–12 months; per-protocol differences ranged from -9.59 to -6.01 . For the secondary outcome (Table 2), Erenumab users had higher odds of achieving a $\geq 50\%$ reduction in triptan use, with intention-to-treat ORs from 1.49 to 1.18 and per-protocol ORs from 1.66 to 1.55. Triptan utilization trajectories are shown in Image 1.

Conclusion: Erenumab showed a consistent advantage over OnabotulinumtoxinA in reducing triptan use, particularly among adherent patients. These findings support the superior real-world effectiveness of Erenumab.

Disclosure: The author receives a PhD stipend from Helse Vest and this is a part of the NorHead project.

EPO-0256 | Overtreatment and delays in antiviral therapy for suspected herpesvirus CNS infections

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Background and aims: Herpesviruses are a common cause of CNS infections. Guidelines recommend empirical antiviral therapy when herpesvirus encephalitis is suspected. This practice often results in treatment of patients without herpesvirus CNS infection.

Methods: We analysed adults with suspected CNS infection enrolled in two prospective cohorts (2012–2025). All patients receiving antiviral therapy for suspected CNS infection were included. All PCR-confirmed herpesvirus CNS infections were considered as appropriately treated, and all others were classified as overtreatment. We quantified overtreatment and undertreatment, and examined the clinical context of delayed therapy and prolonged therapy. In addition, the use of dexamethasone was evaluated in patients with confirmed herpesvirus CNS infection.

Results: Among 1,785 patients, 506 (28%) received antiviral therapy. Of these, 436 (86% of treated; 24% of total) did not have herpesvirus CNS infection, while 70 (14% of treated; 4% of total) had PCR-confirmed herpesvirus CNS infection. All patients with herpesvirus encephalitis received antiviral treatment. Delayed treatment (> 1 day after presentation) occurred in 9/70 (17%) herpesvirus CNS infection cases, mostly due to concurrent or alternative diagnoses. Continued treatment (≥ 7 days) in non-herpesvirus cases occurred in 18/436 (4%), predominantly while awaiting repeat PCR results. Dexamethasone was administered in 20/70 (29%) patients with herpesvirus CNS infection and was not associated with differences in GOS at discharge.

Conclusion: In order to ensure all herpesvirus encephalitis cases were treated, nearly one-quarter of patients with suspected CNS infection received antiviral therapy, exposing many to potential adverse effects. Nevertheless, delayed therapy remained

TABLE 1 Antiviral therapy duration and Glasgow outcome scale in patients treated with antiviral therapy, comparing patients with herpesvirus CNS infection to those with other diagnoses.

	All Patients	Herpesvirus CNS Infection	No herpesvirus CNS infection	p-value ²
	N = 1,785 ¹	N = 70 ¹	N = 436 ¹	
Antiviral therapy Duration (days)	0 [0, 1], n = 1785	14 [9, 21], n = 70	2 [1, 4], n = 436	<0.001
GOS at discharge				
Unfavorable	740 / 1,785 (42%)	32/70 (46%)	239 / 434 (55%)	0.2

¹ n (present) / N (%), Mean (SD) or Median [Q1, Q3], n = patient count with available data
² Pearson's Chi-squared test; Fisher's exact test; Welch Two Sample t-test; Mann-Whitney U tests

TABLE 2 Glasgow outcome scale in patients who used antiviral therapy with PCR-confirmed herpesvirus CNS infection (delayed start of antiviral therapy > 1 day vs no delay).

	No delay N = 45 ¹	Delay N = 9 ¹	p-value ²
Glasgow outcome score			
Favorable	27 / 45 (60%)	3 / 9 (33%)	0.165
Unfavorable	18 / 45 (40%)	6 / 9 (66%)	
¹ n / N (%)			
² Fisher's exact test			

TABLE 3 Glasgow outcome scale in patients who used antiviral therapy with PCR-confirmed herpesvirus CNS infection (dexamethasone vs no dexamethasone).

	No dexamethasone N = 48 ¹	Dexamethasone N = 20 ¹	p-value ²
Glasgow outcome score			
Favorable	25 / 48 (52%)	12 / 20 (60%)	0.165
Unfavorable	23 / 48 (48%)	8 / 20 (40%)	
¹ n / N (%)			
² Fisher's exact test			

common among herpesvirus CNS infections, highlighting the challenge of early identification.
Disclosure: Nothing to disclose.

EPO-0257 | **Optimal timing of ARV initiation among patients with toxoplasma encephalitis: A meta-analysis**

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Background and aims: Toxoplasma encephalitis (TE) affects 10–40% of AIDS patients worldwide, with mortality rates of 10–50%. The optimal timing of antiretroviral therapy (ART) initiation remains controversial, balancing early immune recovery benefits against immune reconstitution inflammatory syndrome (IRIS) risks.
Methods: A meta-analysis of six studies (2009–2025) encompassing 710 HIV patients with TE was conducted. Studies compared early (≤ 10 days) versus delayed (> 21 days) ART initiation after anti-toxoplasma treatment. Primary outcomes included mortality and neurological sequelae. Random-effects models were used with heterogeneity assessed by I^2 statistics.

TABLE 1 Critical appraisal of studies.

STUDY	STRENGTHS	LIMITATIONS	QUALITY SCORE
Bongers (2009)	Prospective, clear outcomes, Statistical analysis	Single-center, small sample size, (1) Selection bias, (unpublished follow-up data)	7/10
Li et al. (2016)	Large-sample size, Prospective, outcomes	Regional bias, (1) publication bias	8/10
Cohen-Vogel et al. (2009)	Long-term follow-up, clear definitions, Large sample size	Regional bias, (1) selection bias	8/10
Geethapriya et al. (2015)	Indian's population, randomized	Non-response, missing data, limited follow-up	6/10
Scholar et al. (2020)	Randomized Controlled Trial, multicenter	Loss of blinding	9/10 (1 score of bias)
Delgado et al. (2008)	Randomized Controlled Trial	Loss of blinding	9/10 (1 score of bias)

TABLE 2 PICOM table of Studies

	P	I	C	O	M
STUDY	Study population (P)	Intervention (I)	Comparison (C)	Outcomes (O)	Methodology (M)
STUDY 1	Population 1 (P1)	Intervention 1 (I1)	Comparison 1 (C1)	Outcomes 1 (O1)	Methodology 1 (M1)
STUDY 2	Population 2 (P2)	Intervention 2 (I2)	Comparison 2 (C2)	Outcomes 2 (O2)	Methodology 2 (M2)
STUDY 3	Population 3 (P3)	Intervention 3 (I3)	Comparison 3 (C3)	Outcomes 3 (O3)	Methodology 3 (M3)
STUDY 4	Population 4 (P4)	Intervention 4 (I4)	Comparison 4 (C4)	Outcomes 4 (O4)	Methodology 4 (M4)
STUDY 5	Population 5 (P5)	Intervention 5 (I5)	Comparison 5 (C5)	Outcomes 5 (O5)	Methodology 5 (M5)
STUDY 6	Population 6 (P6)	Intervention 6 (I6)	Comparison 6 (C6)	Outcomes 6 (O6)	Methodology 6 (M6)

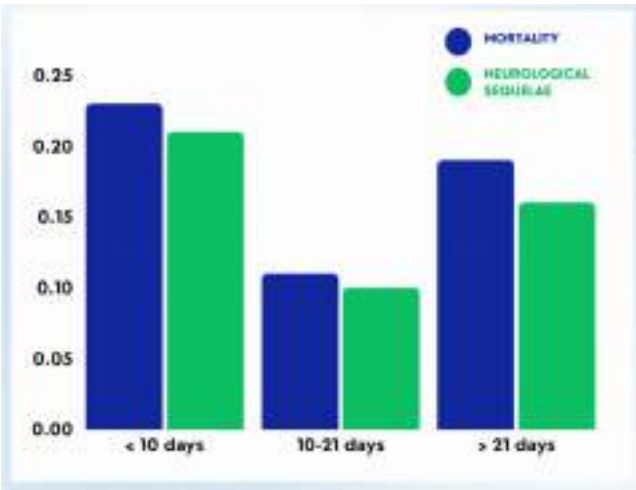


FIGURE 1 Optimal timing of antiretroviral therapy initiation based on mortality and neurological sequelae.

Results: Early ART initiation showed no significant overall mortality difference (pooled OR: 0.85, 95% CI: 0.42–1.72, $p=0.65$) but demonstrated significant mortality reduction at 24-week follow-up (pooled OR: 0.65, 95% CI: 0.50–0.83, $p<0.001$). Neurological sequelae increased non-significantly with early initiation (pooled OR: 1.28, 95% CI: 0.97–1.67, $p=0.07$). IRIS occurrence showed no significant difference between groups. Optimal outcomes occurred with ART initiation at 10–21 days post-diagnosis, balancing mortality reduction with minimized complications. The mortality benefit of early ART supports immune reconstitution advantages, while increased neurological sequelae raise safety concerns. The absence of significant IRIS differences suggests concurrent anti-toxoplasma therapy may reduce inflammatory responses. Study heterogeneity, timing definitions and geographic diversity affect interpretation.
Conclusion: ART initiation between 10–21 days after anti-toxoplasma treatment provides optimal risk-benefit balance for most patients. Clinical decisions should incorporate individual factors including CD4 count, neurological severity, and health-care resources. Standardized timing definitions and long-term outcome studies are needed.
Disclosure: The authors declare no conflicts of interest, external funding, and followed PRISMA guidelines. Ethical approval attained.

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Background and aims: Bacterial meningitis is a serious disease characterized by high rates of complications and adverse outcomes. Disfunction of the spleen is a known risk factor for developing bacterial meningitis.

Methods: Patients with cerebrospinal fluid culture-confirmed bacterial meningitis were enrolled in a nationwide prospective cohort study between March 1, 2006, and April 1, 2025. Splenectomy or conditions associated with functional hyposplenia were scored for each patient. Clinical characteristics and outcome of patients with a history of splenectomy or functional hyposplenia were assessed and compared with normosplenic patients.

Results: Hundred-four of 3165 patients (3.3%) had an abnormal splenic function: 52 had a history of splenectomy and 52 had functional hyposplenia. Vaccination status was known for 76 patients – 26 of 45 splenectomized (58%) and 4 of 31 hyposplenic patients (13%) were vaccinated. The most common causative organisms were *Streptococcus pneumoniae* in splenectomized patients (51 of 52 patients, 98%), while 18 of 52 hyposplenic patients were infected with *Listeria monocytogenes* (35%). Asplenic patients presented with higher serum leukocytes ($P < 0.01$) and C-reactive protein ($P < 0.01$), but lower levels of leukocytes in cerebrospinal fluid ($P < 0.001$) compared to normosplenic patients. Outcome was unfavorable in 47 patients (45%) and 17 patients died (16%).

Conclusion: Bacterial meningitis in splenectomized patients is caused by *S. pneumoniae* and is associated with a more severe systemic inflammatory response. In hyposplenic patients *L. monocytogenes* was a frequent cause of meningitis, likely due to a combination of immunocompromising factors and spleen dysfunction. Enhanced vaccination strategies should be employed to reduce the disease burden in these patients.

Disclosure: Nothing to disclose.

EPO-0259 | Influenza B-related mild encephalopathy with reversible splenial lesion Type II in an adult

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Background and aims: Mild encephalopathy with a reversible splenial lesion (MERS) is a rare clinico-radiological syndrome characterized by transient neurological symptoms and

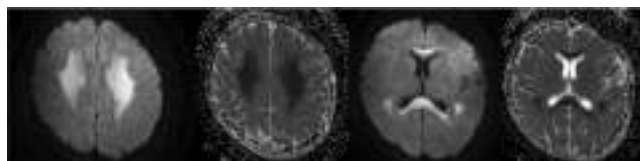


FIGURE 1 Initial brain MRI demonstrating DWI hyperintense and ADC hypointense lesions in the splenium of the corpus callosum and bilateral frontoparietal deep white matter.

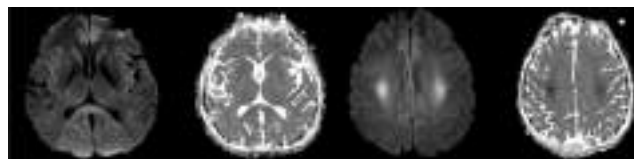


FIGURE 2 Follow-up brain MRI performed 4 days later demonstrating regression of the lesions observed on the initial MRI.

reversible lesions of the splenium of the corpus callosum. It is classified into Type I, limited to the splenium, and Type II, involving the entire corpus callosum and adjacent white matter. Adult cases associated with influenza infection are uncommon.

Methods: We report a case of influenza B-associated type II MERS in a 35-year-old woman.

Results: A previously healthy 35-year-old woman presented with acute upper respiratory tract symptoms and fever, followed by rapid onset of confusion on the same day. On examination, she was disoriented to time, place, and person, with a body temperature of 38°C. There were no signs of meningeal irritation. Cranial magnetic resonance imaging revealed hyperintense lesions on diffusion-weighted imaging involving the splenium of the corpus callosum and bilateral frontoparietal deep white matter. Cerebrospinal fluid analysis, routine laboratory investigations, and electroencephalography were unremarkable. Polymerase chain reaction testing of a nasopharyngeal swab was positive for Influenza B virus. The patient was managed with supportive treatment alone. Her neurological symptoms resolved completely within two days. A follow-up MRI demonstrated marked regression of the previously noted lesions.

Conclusion: Influenza B-associated MERS Type II is exceedingly rare in adults. This case highlights the importance of diffusion-weighted MRI in diagnosis and emphasizes that MERS typically follows a self-limiting course with excellent clinical and radiological recovery under supportive management.

Disclosure: Nothing to disclose.

EPO-0260 | Can we predict meningitis before the tap? A machine learning approach to lumbar puncture decision-making

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Background and aims: Diagnosis of meningitis in the emergency department (ED) presents a challenge due to variable clinical presentations that overlap with other neurological and systemic conditions. The diagnosis today relies mostly on lumbar puncture (LP). Our aim was to develop clinical decision support tool for managing suspected meningitis patients in the ED.

Methods: Retrospective data of patients who underwent LP in the ER for suspicion of meningitis between 2019 and 2024 was collected. We used clinical and laboratory data to train machine learning (ML) model to predict meningitis prior to LP results. Model performance was evaluated using accuracy, precision, recall, and the area under the curve (AUC). Shapley value was used to identify important features in meningitis prediction.

TABLE 1 Demographic and characteristics.

	Meningitis n=344	non meningitis n=1183	P value
Age, median (IQR)	36 (30-57)	52 (34-71)	>0.001
Sex (F), n (%)	159 (46.2)	581 (49.1)	0.377
Background, n(%)			
DM	42 (12.2)	222 (18.8)	0.006
Hypertension	50 (14.5)	322 (27.2)	>0.001
Dyslipidemia	43 (12.5)	225 (19)	0.007
Prior_Stroke	8 (2.3)	61 (5.2)	0.038
IHD	9 (2.6)	95 (8.)	0.001
Migraine	27 (7.9)	68 (5.8)	0.196
Epilepsy	11 (3.2)	50 (4.2)	0.483
Anxiety	3 (0.9)	25 (2.1)	0.2
Depression	5 (1.5)	34 (2.9)	0.202
Fibromyalgia	2 (0.6)	17 (1.4)	0.325

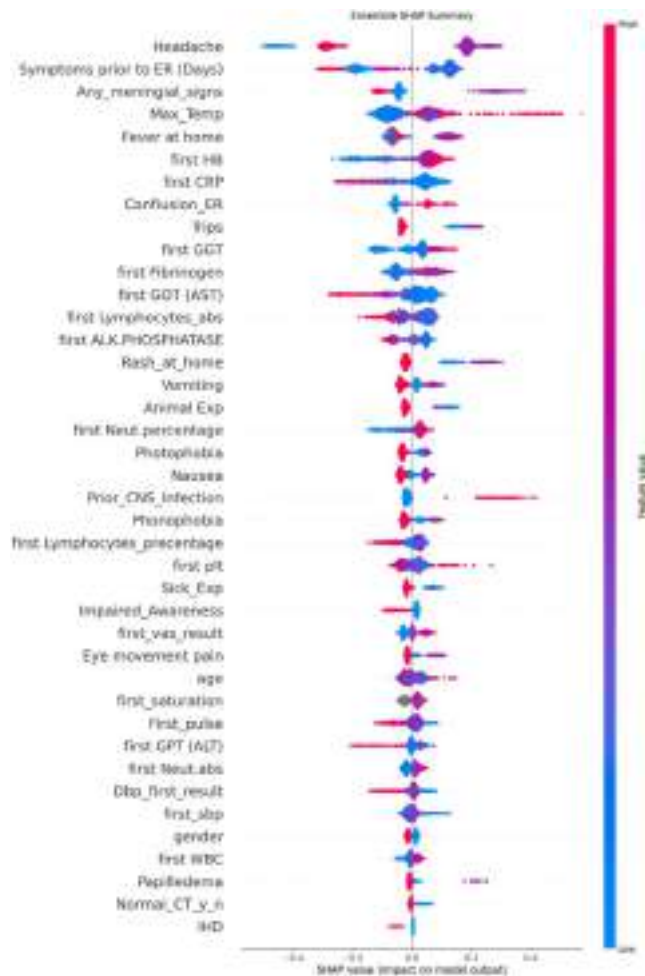


FIGURE 1

Results: 1526 patients were included, among them 344 (22.54%) had meningitis. Most were aseptic meningitis ($n=270$, 78.49%), followed by meningoencephalitis ($n=49$, 14.24%), bacterial ($n=24$, 6.97%) and brain abscess ($n=1$, 0.29%). Meningitis patients were younger compared to non-meningitis [median age of 36 (IQR30-57) and 52 (IQR34-71), respectively, $p < 0.001$] and had less cerebrovascular risk factors. Our ML model achieved AUC of 0.87 and accuracy of 0.85 for the diagnosis of meningitis.

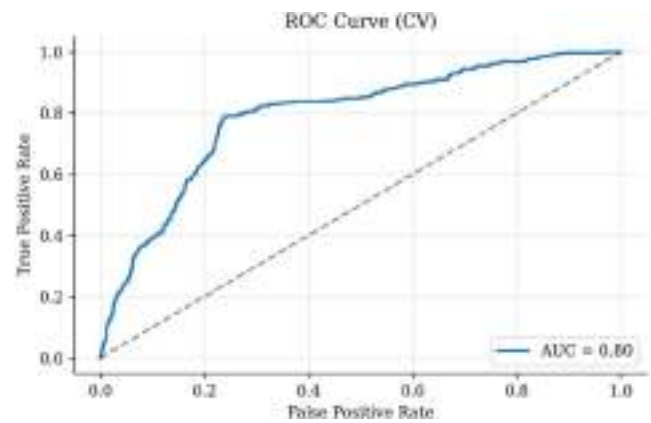


FIGURE 2

The most important features in our model were number of symptomatic days prior to ED visit, temperature, CRP, photophobia and absolute lymphocytes.

Conclusion: Our ML model was able to successfully predict patients with meningitis, providing a tool in clinical management of meningitis patients in the ED. Such tool can help determine whether a LP is indicated, avoiding unnecessary invasive tests.

Disclosure: Nothing to disclose.

EPO-0261 | **Beta-blockers in tuberculous meningitis associated cerebral salt wasting: An open label randomized controlled trial**

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Background and aims: More than 50% tuberculous meningitis (TBM) patients have hyponatremia. Cerebral salt wasting (CSW) is the commonest cause of hyponatremia, and has been attributed to higher catecholamine level as a result of stress response. Beta-blockers may reduce stress response, and may be effective in TBM associated CSW. We report the efficacy and safety of propranolol in TBM associated CSW compared to placebo in a randomized placebo-controlled trial.

Methods: Thirty-eight TBM associated CSW patients were included, and were randomized to tablet propranolol 10mg twice daily on day 1, and 20mg twice daily from day 2 till one month or placebo. Both the arms received sodium chloride 10gm/day and normal saline till the normalization of serum sodium and fluid deficit. The primary outcome was day of normalization of serum sodium, and secondary outcomes were day of correction of fluid deficit and polyuria, adverse effects and a modified Rankin Scale (mRS) score at 1, 3 and 6 months. Both the groups received standard antitubercular treatment.

Results: Nineteen patients each received propranolol and placebo, and their baseline clinical, MRI, and laboratory findings were comparable. The propranolol group had an earlier normalization of serum sodium (median days 8vs20; $P < 0.01$), fluid deficit (median days 11vs42; $P < 0.01$) and polyuria (median days 4vs16; $P < 0.01$) compared to the placebo group. Death at 1 month and mRS score at 3 and 6 months were comparable between the

two groups. Two patients required reduction of propranolol dose due to hallucinations and bradycardia.

Conclusion: Adjunctive propranolol results in earlier resolution of CSW in TBM patients

Disclosure: Nothing to disclose.

EPO-0262 | Plasma BD-tau in post-COVID neurological syndrome: A longitudinal study

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Background and aims: Post-COVID-19 condition (PCC) includes persistent neurological symptoms, raising concerns about long-term neurodegeneration. Brain-derived tau (BD-tau) is a novel plasma biomarker with improved brain specificity compared to traditional tau assays. This study evaluated BD-tau as a marker of cognitive impairment and symptom persistence in PCC.

Methods: We conducted a longitudinal analysis within the Norwegian NeuroCOVID cohort, including adults with SARS-CoV-2 infection and neurological symptoms ($n=112$) and recovered controls without persistent symptoms ($n=33$). Plasma BD-tau, p-tau217, p-tau181, and total tau were measured at 6 and 12 months using ultrasensitive immunoassays. Clinical outcomes included modified Rankin Scale and neuropsychological testing. Associations were assessed using regression and correlation analyses.

Results: BD-tau, p-tau217, p-tau181, and total tau remained stable between 6 and 12 months, regardless of symptom remission (43% at 12 months) or cognitive impairment (39% at 6 months; 25% at 12 months). None of the tau biomarkers correlated with cognitive domains. BD-tau showed strong correlations with total tau ($\rho=0.66$ at 6 months; $\rho=0.61$ at 12 months, $p<0.001$) and moderate associations with p-tau217 and p-tau181, but no significant relationship with cognitive scores or headache persistence. Higher BD-tau was predicted by age, creatinine levels,

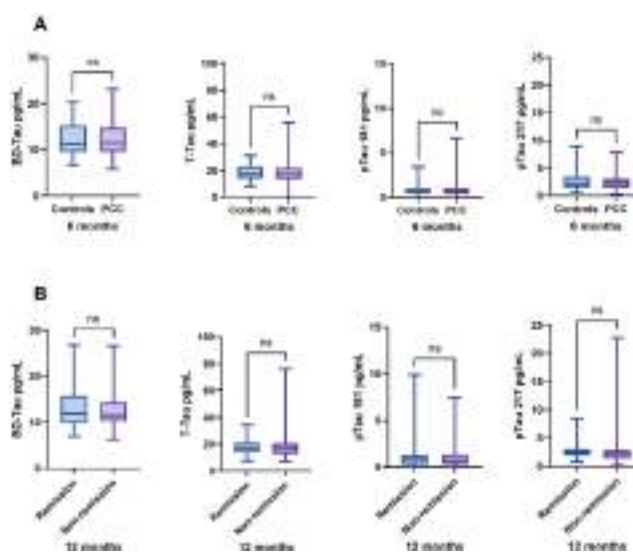


FIGURE 1 (A) Plasma concentrations of BD-tau, total tau (t-tau), phosphorylated tau 181 (pTau181), and phosphorylated tau 217 (pTau217) in individuals with post-COVID condition (PCC) compared to recovered controls at 6 months post-infection. (B) Distribution of plasma BD-tau, t-tau, pTau181, and pTau217 in participants with and without symptom remission at 12 months post-COVID-19 infection. Biomarker concentrations remained stable over time, with no significant differences between groups, despite clinical remission in nearly half of the cohort ($n=48$; 43.2%).

comorbidities, and COVID-19 severity, explaining up to 26% of variance at 6 months and 38% at 12 months.

Conclusion: Plasma BD-tau does not reflect remission or cognitive impairment in PCC, suggesting tau-driven neurodegeneration is not a dominant mechanism. Larger studies with extended follow-up are needed to clarify long-term neurocognitive risk.

Disclosure: The authors report no disclosures relevant to this study. A.H. Aamodt has received honoraria for advice or lecturing from BMS-Pfizer, Abbvie, Teva, Novartis, Roche and Lundbeck and research grant from Boehringer Ingelheim, EAH has received honoraria for advice or lecturing from Sanofi and research grant from MS union. ØT received honoraria for advice or lecturing from Merck, Sanofi and Novartis. MBA received honoraria for advice or lecturing from BMS-Pfizer, Abbvie, Teva, Novartis and Lundbeck.

EPO-0263 | Neurological manifestations of dengue: Clinical spectrum, imaging diagnosis, and response to immunomodulatory treatment

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Background and aims: Dengue virus infection is described as systemic febrile illness. Increasing evidence shows that the nervous system is a frequent and clinically relevant target. Neurological manifestations include encephalitis, encephalopathy, myelitis and demyelinating syndromes, cerebrovascular events, and immune-mediated neuropathies, occurring during acute infection or post-infectious complications, often

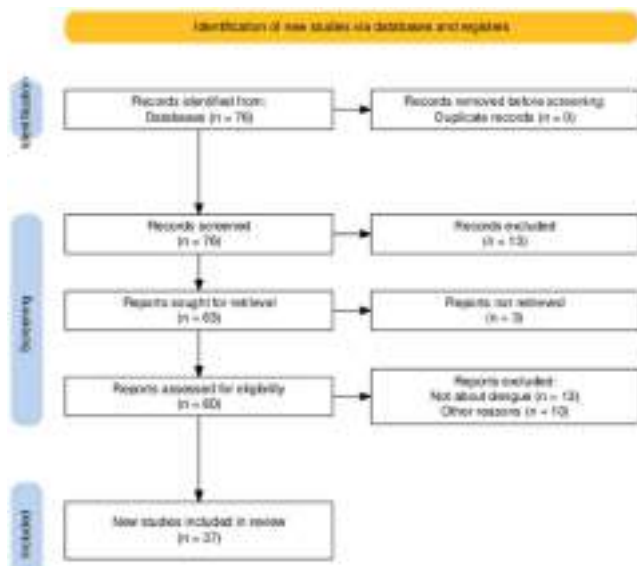


FIGURE 1 Prisma-P flowchart of the process of inclusion of studies in the systematic review.



FIGURE 2 Neurological manifestations of dengue, neuroimaging findings and immunomodulatory treatment.

remaining underrecognized [1–3]. This study describes clinical profile and symptomatic treatment of cases of neurological dengue, aiming to better define its spectrum and outcomes.

Methods: We conducted a PRISMA systematic review. Pubmed was searched using the keywords “dengue fever” and “neurological complications”. Studies reporting clinical, radiological, or laboratory data on dengue-associated neurological manifestations were included, excluding non-human and literature reviews studies.

Results: We analyzed 142 patients with neurological complications of dengue infection. Mean age was 28 years. Neurological symptoms developed at a mean of 10.9 days after fever onset. The most frequent manifestations were encephalopathy (40%), seizures (25%), and focal neurological deficits (20%). The overall mortality rate was 5.6%. Diagnosis was confirmed by serology (NS1 and/or IgM) in 90% of cases and by cerebrospinal fluid PCR in 30%. MRI revealed abnormalities in 70% of patients, most commonly thalamic edema, myelitis, and corpus callosum lesions. Treatment with corticosteroids or intravenous immunoglobulins was administered in 50% of cases, with complete recovery observed in 60%.

Conclusion: Neurological complications of dengue are frequent and diverse, with significant mortality. Diagnosis is based on serology and neuroimaging, and immunomodulatory treatment is associated with high rates of complete recovery, highlighting the need for early recognition.

Disclosure: Nothing to disclose.

EPO-0264 | How long MRI abnormality persists in Tuberculous meningitis?

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Background and aims: There is paucity of information regarding resolution of MRI abnormalities, and its clinical relevance in patients with Tuberculous meningitis (TBM). We report the resolution of MRI abnormality at 6 and 12 months of treatment, and association with admission clinic-laboratory findings.

Methods: 115 patients with TBM diagnosed on the basis of clinical, CSF and MRI findings were included during 2018–2024, and followed up clinically and radiologically at 3, 6 and 12 months. The baseline clinical severity, and MRI findings (exudates, Infarcts, hydrocephalous and granuloma) were noted. Patients were treated with four antitubercular drugs, prednisolone and aspirin. The outcome was assessed using modified Rankin Scale (mRS) and categorized into death, poor (3–5) and good (0–2). The predictors of complete resolution of MRI at one year was evaluated including baseline findings.

Results: Their median age was 25 years, and 45.2% were females. The median duration of illness was 60 days and majority were in stage II ($n = 78$ (67.8%) at admission). MRI revealed exudates in 103 (89.6%), Infarcts in 51 (44.3%), granuloma in 93 (80.9%) and hydrocephalous in 32 (27.8%) patients. The complete resolution occurred at 3 months in 10 (8.7%), 6 months in 25 (27.1%) and at 12 months 25 (27.1%) patients. Enhancing granuloma was seen at 12 months in 62 (53.9%), and exudates in 7 (6.1%) patients. On multivariate analysis, the predictors of abnormal MRI at one year included seizure ($p = 0.026$) and lymphopenia in blood ($p = 0.005$) at baseline.

Conclusion: Complete resolution of MRI abnormality at year occurs in 33.9% patients especially in nonlymphopenic and non-seizure patients

Disclosure: Nothing to disclose.

EPO-0265 | Opportunistic central nervous system infections and transplant rejection in solid organ transplant recipients: A scoping review

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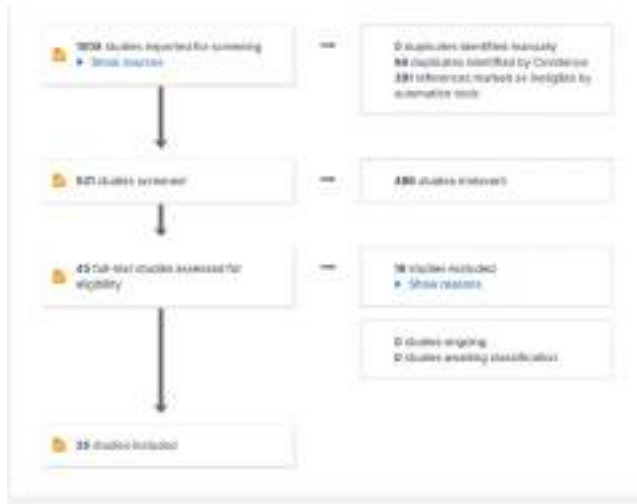


FIGURE 1 PRISMA flow diagram illustrating study identification, screening, eligibility assessment, and inclusion for the scoping review of opportunistic CNS infections and transplant rejection in solid organ transplant recipients.

Background and aims: Opportunistic central nervous system (CNS) infections are uncommon but devastating complications of solid organ transplantation (SOT), accounting for up to 10% of transplant-associated complications and carrying disproportionate morbidity and mortality. While prior studies have emphasized short-term neurologic outcomes, the impact of immunosuppressive modulation on graft rejection and longer-term outcomes remains poorly characterized. To map and synthesize the existing literature describing transplant rejection in the setting of CNS infections among SOT recipients.

Methods: A scoping review was conducted following the Arksey and O'Malley framework, refined by the Joanna Briggs Institute, and reported according to PRISMA-ScR guidelines (Figure 1). PubMed, EMBASE, and Scopus were searched from inception through October 2025. Eligible studies included human experimental and observational designs describing both opportunistic CNS infection and transplant rejection in SOT recipients of any age. Two reviewers independently screened studies and extracted data using a standardized form. Findings were synthesized narratively using the Population–Concept–Context framework.

Results: CNS infections frequently occurred years after transplantation, often following intensified immunosuppression for rejection. Immunosuppressive regimens including calcineurin inhibitors and corticosteroids were consistently associated with pathogen-specific CNS infections. Reduction of immunosuppression during infection management was followed by acute rejection in approximately 25–35% of patients within one year. Neurologic outcomes ranged from full recovery to permanent neurologic injury and graft dysfunction.

Conclusion: The literature addressing transplant rejection in the context of opportunistic CNS infections is limited and heterogeneous. Significant gaps remain regarding standardized immunosuppressive management and long-term outcomes, highlighting the need for prospective studies and consensus-based guidance.

Disclosure: Nothing to disclose.

Background and aims: Neurobrucellosis is a rare but severe complication of systemic brucellosis, accounting for 3–10% of cases. Clinical presentations range from meningitis to stroke, mimicking neuro-tuberculosis in endemic countries, leading to delayed and often misdiagnosis.

Methods: A 58-year-old man presented with intermittent fever, polyarthralgia, and headache for three weeks. General and neurological examinations were unremarkable. Evaluation for pyrexia of unknown origin revealed lymphocytic pleocytosis, elevated protein, and hypoglycorrhachia on cerebrospinal fluid analysis, suggestive of infection. Blood cultures grew *Brucella*, and serum agglutination testing was positive. MRI of the spine demonstrated thoracic discitis. Treatment with ceftriaxone, rifampicin, and doxycycline was initiated.

Results: Four weeks later, despite appropriate antimicrobial therapy, he developed recurrent transient neurological episodes followed by persistent dysarthria and right hemiparesis. Brain MRI revealed acute lentiform nucleus infarcts, and MR angiography showed focal stenosis of the left middle cerebral artery (M1 segment). Vessel wall imaging demonstrated concentric enhancement, consistent with cerebral vasculitis. High-dose corticosteroids were added while continuing antibiotics. The patient stabilized without further neurological deterioration. Steroids were gradually tapered over six months, ceftriaxone was continued for six months, and doxycycline with rifampicin for one year. At one-year follow-up, he had minimal residual dysarthria without limb weakness.

Conclusion: Vasculitis in neurobrucellosis is very rare, with no consensus on treatment protocol. Starting steroids is a cliff-hanger with the risk of worsening an active systemic infection and the presumed benefit of neurological improvement. Our experience highlights that steroids, when judiciously used in

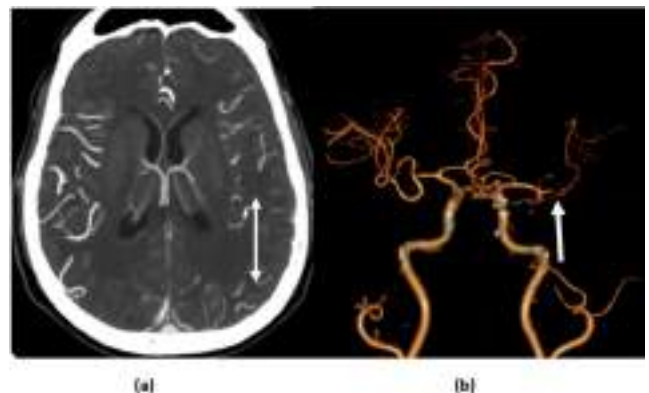


FIGURE 1 Axial CT sections of CT angiogram study (a) and 3D reconstruction of intracranial vessels show paucity of left middle cerebral artery (MCA) territory M3 and M4 cortical branches (double headed arrow) and stenosis at distal left M1 MCA on 3D images (arrow).



FIGURE 2 T1 pre contrast (a) and T1 post contrast sagittal (b) and T1 post contrast axial (c) images of dorsal spine show abnormal contrast enhancement (arrow) at D8 vertebral body.

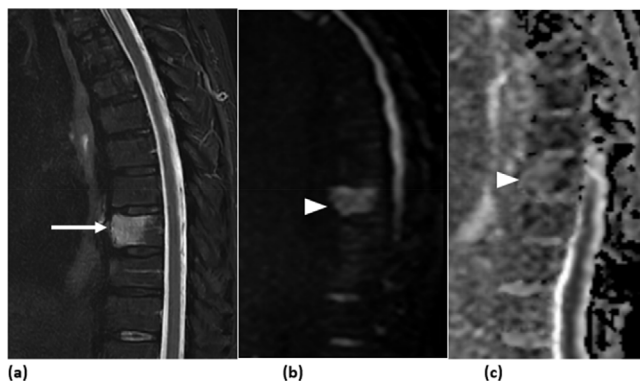


FIGURE 3 T2 STIR (a) image of dorsal spine show abnormal signal intensity (arrow) along the D8 vertebral body with DWI (b) hyperintensity (arrowhead) and ADC (c) image showing partial signal drop suggesting partial diffusion restriction.

selective complications like CNS vasculitis, along with antibiotics, can improve the outcome.

Disclosure: Nothing to disclose.

Motor neurone diseases 2

EPO-0267 | Functional status and prognostic predictors in the final six months of life in ALS: A retrospective analysis of 703 patients

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Background and aims: In advanced Amyotrophic Lateral Sclerosis (ALS), transition to palliative care often leads to formal neurological follow-up discontinuation. Consequently, revised ALS Functional Rating Scale (ALSFRS-R) data near the end of

life are scarce. This study aimed to characterize ALSFRS-R total and subdomain scores during the final six months of life and evaluate their independent prognostic value.

Methods: We retrospectively analysed 703 patients with ALS (including progressive muscular atrophy) who underwent ALSFRS-R assessment within six months of death. Clinical and functional data were analysed using descriptive statistics; scores were compared by onset site (spinal vs. bulbar) using the Mann-Whitney U test. Cox proportional hazards models were employed to evaluate the association between functional scores and mortality risk, adjusting for age at onset, sex, onset region, and the use of gastrostomy and non-invasive ventilation.

Results: Median age at onset was 66.7 years and median survival was 29 months. Near death, median ALSFRS-R total score was 20, with no significant differences between onset types. Notably, respiratory and bulbar subscores remained higher relative to limb and global domains. Multivariate analysis revealed that higher total ALSFRS-R scores (HR 0.988, 95% CI 0.979–0.998) and higher bulbar subscores (HR 0.965, 95% CI 0.939–0.992) were independently associated with reduced risk of death. Other subscores did not significantly predict survival in this late stage.

Conclusion: Quantifying functional decline in the terminal phase of ALS is vital for informed end-of-life clinical decision-making. In advanced disease, overall functional status and, specifically, bulbar dysfunction emerge as the most robust predictors of mortality.

Disclosure: The author has applied for a research grant related to this work and is currently awaiting the outcome.

EPO-0268 | Therapeutic comparison of edaravone and riluzole in amyotrophic lateral sclerosis: A systematic review and meta-analysis

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Background and aims: Amyotrophic lateral sclerosis (ALS) is a rapidly progressive neurodegenerative disorder. Riluzole is the standard therapy, while edaravone is an approved adjunct treatment; however, the clinical benefit of combination therapy remains uncertain. This meta-analysis evaluates the effect of riluzole plus edaravone versus riluzole alone on disease progression in ALS according to the Revised Amyotrophic Lateral Sclerosis Functional Rating Scale (ALSFRS-R).

Methods: A systematic search was performed for studies published through October 2025 comparing combination therapy with riluzole monotherapy in patients with ALS. Extracted outcomes included ALSFRS-R and all-cause mortality. Standardized mean differences (SMDs) and odds ratios (ORs) with corresponding 95% confidence intervals (CIs) were calculated using random-effects models. Statistical heterogeneity was assessed using the I² statistic. All analyses were performed using RStudio.

Results: Five studies comprising 548 patients were included, of whom 39.4% received riluzole plus edaravone. For functional progression, pooled analysis showed no significant

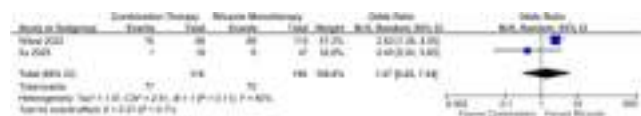


FIGURE 1 Forest plot of all-cause mortality comparing combination therapy (edaravone plus riluzole) versus riluzole monotherapy in patients with ALS.



FIGURE 2 Forest plot comparing functional progression (ALSFRS-R scores) between edaravone plus riluzole combination therapy and riluzole monotherapy.

difference between combination therapy and riluzole monotherapy (SMD=0.20, 95% CI -0.37 to 0.77; $p=0.49$), with moderate heterogeneity ($I^2=63\%$). Similarly, no significant difference in all-cause mortality was observed between treatment strategies (OR=1.37, 95% CI 0.25 to 7.44; $p=0.71$), also with moderate heterogeneity ($I^2=60\%$). Heterogeneity likely stemmed from differences in study design and disease stage at treatment initiation.

Conclusion: Riluzole plus edaravone did not demonstrate a significant advantage over riluzole alone in ALS. This quantitative synthesis clarifies the absence of a population-level benefit of combination therapy. However, larger and standardized randomized trials are required to inform future treatment recommendations.

Disclosure: Nothing to disclose.

EPO-0269 | NLRP3 inflammasome and pyroptosis as contributors to neuroinflammation in amyotrophic lateral sclerosis

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Background and aims: Amyotrophic lateral sclerosis is a fatal neurodegenerative disorder in which growing evidence points to a contributory role of innate immune mechanisms. Activation of inflammasome-dependent inflammatory pathways and pyroptotic cell death has been demonstrated in experimental models; however, clinical evidence in patients remains scarce. This study

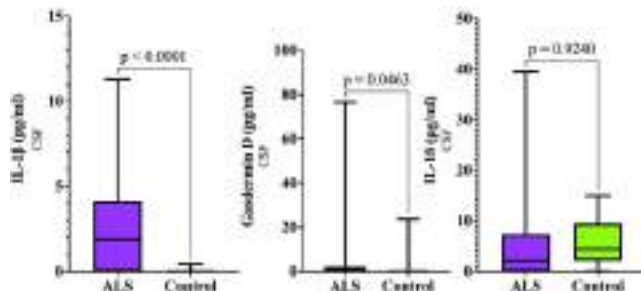


FIGURE 1 Cerebrospinal fluid levels of interleukin-1 beta, gasdermin D, and interleukin-18 in patients with ALS and controls.

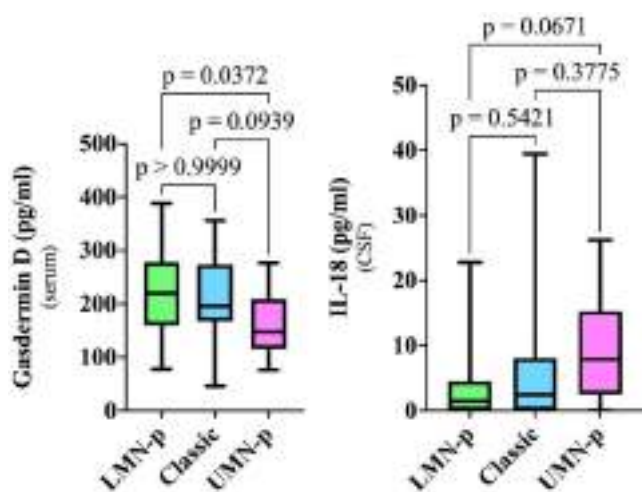


FIGURE 2 Serum gasdermin D and CSF interleukin-18 levels across ALS clinical phenotypes: lower motor neuron-predominant, classic, and upper motor neuron-predominant.

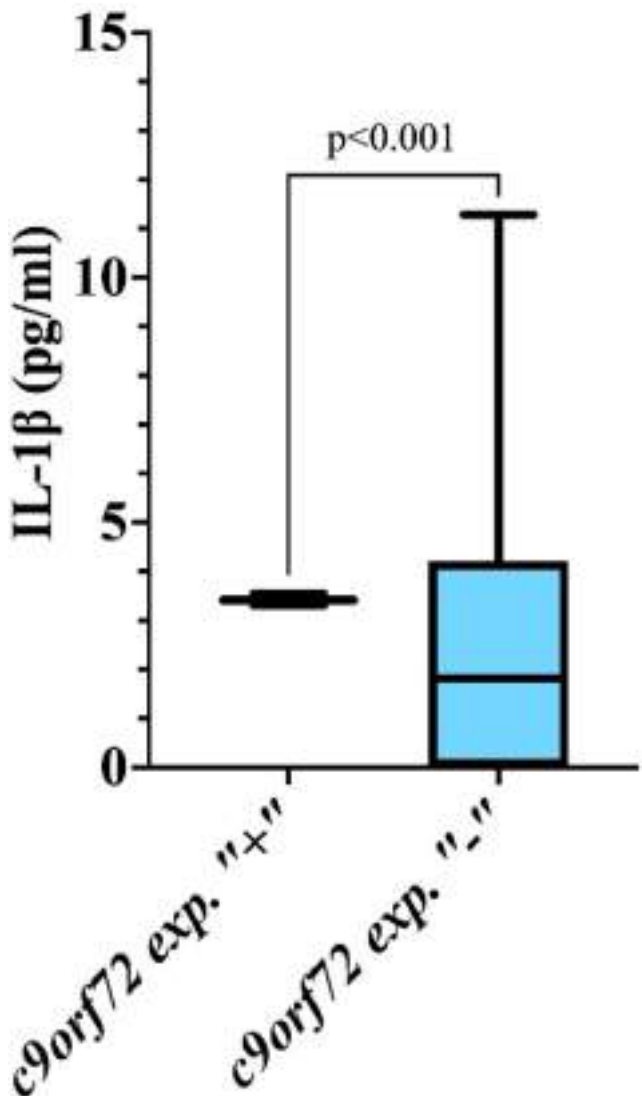


FIGURE 3 CSF interleukin-1 beta levels in ALS patients with and without c9orf72 repeat expansion.

aimed to evaluate circulating and cerebrospinal fluid markers related to NLRP3 inflammasome activation and pyroptosis in amyotrophic lateral sclerosis and to explore their clinical and genetic associations.

Methods: A total of 100 patients with amyotrophic lateral sclerosis and 36 neurologically healthy controls were enrolled. Serum and cerebrospinal fluid levels of interleukin-1 beta, interleukin-18, and gasdermin D were quantified using ELISA. Clinical phenotypes, disease staging, and progression characteristics were analyzed. Molecular genetic testing was performed to identify SOD1 mutations and c9orf72 repeat expansion. Group and subgroup comparisons were conducted using appropriate statistical methods.

Results: Compared with controls, patients with ALS exhibited higher serum concentrations of gasdermin D and increased cerebrospinal fluid levels of interleukin-1 beta. Elevated cerebrospinal fluid interleukin-1 beta was observed in carriers of c9orf72 repeat expansion. Serum gasdermin D concentrations varied across clinical phenotypes, with higher levels in patients with predominant lower motor neuron involvement. Interleukin-18 levels did not significantly differ between groups. Associations between peripheral and central inflammatory markers were identified.

Conclusion: The results indicate activation of inflammasome-related inflammatory pathways and pyroptosis in amyotrophic lateral sclerosis. These findings support a role for innate immune dysregulation in disease pathogenesis and suggest that inflammasome-associated molecules may represent promising biomarkers and therapeutic targets.

Disclosure: Nothing to disclose.

EPO-0270 | Efficacy of immunoglobulin in participants with post-polio syndrome: Results from the multicenter, randomized, placebo-controlled FORCE trial

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Background and aims: Post-Polio Syndrome (PPS) is a chronic, slowly progressive neuromuscular condition with no specific medical treatments. This study evaluated intravenous immunoglobulin (IVIG, Flebogamma® 5% DIF) in participants with PPS, based on observations of immune/inflammatory markers activation.

Methods: This Phase II/III multicenter, randomized, double-blind, placebo-controlled trial included adult participants with PPS across Europe, Canada, and the US. Participants received monthly infusions of IVIG (1 g/kg) or placebo for 52 weeks, followed by a 24-week observation. The primary endpoint was change from baseline in Two-Minute Walking Distance (2MWD) at Week 52. Secondary and exploratory endpoints included Six-Minute Walking Distance (6MWD), fatigue (Fatigue Severity Scale and Borg scale), muscle strength (Medical Research Council scale), and pain (Visual Analog Scale).

Results: 149 participants were randomized to IVIG 1 g/kg ($n=75$) or placebo ($n=74$). Mean age 63.2 years, 61.7% female. IVIG significantly improved 2MWD vs placebo at Week 52 ($p=0.0469$), which was further confirmed in a sensitivity analysis (least squares mean difference [LSMD]: +6.07 m; $p=0.0147$) (Figure 1). A clinically meaningful improvement in 6MWD was observed in the IVIG group vs placebo (+15.8 m; $p=0.1205$); IVIG improvement from baseline was twice that of placebo (Figure 2). Muscle strength decreased in placebo group and increased in IVIG group ($p=0.0220$). No notable differences were observed in fatigue or pain (Table 1). IVIG 1 g/kg was well tolerated.

Conclusion: Monthly IVIG infusions demonstrated statistically and clinically meaningful improvements in walking capacity in

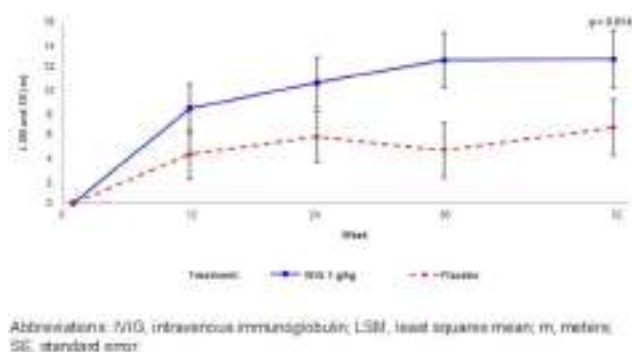


FIGURE 1 Least squares mean change in 2MWD from baseline to Week 52.

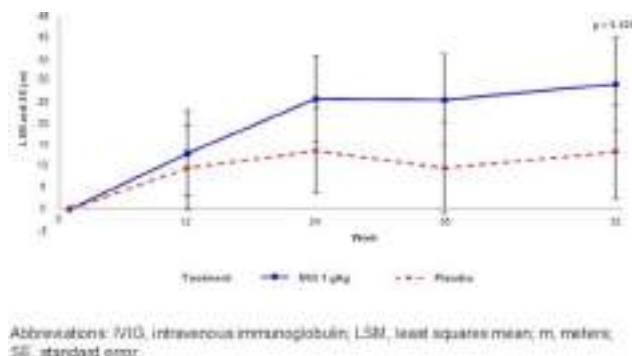


FIGURE 2 Least squares mean change in 6MWD from baseline to Week 52.

TABLE 1 Summary of efficacy outcomes: Change from baseline to Week 52.

Variable	IVIG (N=76)	Placebo (N=74)
2MWD*		
LSM (SE), m	12.75 (2.496)	6.89 (2.511)
LSMD from Placebo (SE), m		5.07 (2.455)
p-value		0.0147
6MWD		
LSM (SE), m	29.15 (10.937)	13.36 (10.918)
LSMD from Placebo (SE), m		15.80 (10.112)
p-value		0.1205
MMT**		
LSM (SE), points	0.58 (0.306)	-0.03 (0.306)
LSMD from Placebo (SE), points		0.61 (0.262)
p-value		0.0220
FSS		
LSM (SE), points	-0.57 (0.282)	-0.22 (0.285)
LSMD from Placebo (SE), points		-0.35 (0.197)
p-value		0.0503
Borg scale, difference post-prior 2MWD		
LSM (SE), points	-0.12 (0.183)	-0.18 (0.184)
LSMD from Placebo (SE), points		0.06 (0.200)
p-value		0.7867
Borg scale, difference post-prior 6MWD		
LSM (SE), points	-0.05 (0.327)	-0.02 (0.326)
LSMD from Placebo (SE), points		-0.03 (0.326)
p-value		0.9310
VAS for pain		
LSM (SE), mm	-3.2 (3.65)	-2.6 (3.67)
LSMD from Placebo (SE), mm		-0.5 (3.65)
p-value		0.8897

* Results from the primary endpoint sensitivity analysis.

** Using the Medical Research Council scale

2MWD, Two Minute Walking Distance; 6MWD, Six Minute Walking Distance; FSS, Fatigue Severity Scale; IVIG, intravenous immunoglobulin; LSM, least squares mean; LSMD, least squares mean difference; m, meters; mm, millimetres; MMT, manual muscle testing; SE, standard error; VAS, Visual Analog Scale.

participants with PPS, indicating IVIG 1 g/kg monthly for one year improves physical performance and endurance.

Disclosure: This study was funded by Grifols. S. Camprubi, B. Garcia, D. Whyms, and E. Mondou are full-time employees of Grifols and have no other competing interests to declare. F. Nollet and D. Trojan received institutional grants for participation as study site in the FORCE trial, for providing the training of outcome assessors in the FORCE trial, and fees for consultation services and management of study participants. D. Trojan receives research support from the Polio Quebec Association and The Robert Lassner Research Fund. H. Andersen received research support, travel support, speaker honoraria and served as consultant on advisory boards from CSL Behring, NMD Pharma, Pfizer, Novo, Lundbeck, Alexion, Sanofi Genzyme, Biogen, Merck, UCB Pharma. J. Jakobsen received speaker honoraria from Takeda. The rest of authors declare no conflict of interest.

EPO-0271 | Neutrophil percentage-to-albumin ratio as a possible prognostic biomarker in amyotrophic lateral sclerosis: A PRO-ACT study cohort

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Background and aims: Increasing evidence suggests that immune dysregulation contributes to the pathophysiology and progression of amyotrophic lateral sclerosis (ALS), underscoring the need for inflammatory biomarkers to monitor

disease progression. The neutrophil percentage-to-albumin ratio (NPAR) has been proposed as a marker of systemic inflammation and has shown prognostic relevance in ischemic stroke, intracerebral hemorrhage, and cognitive decline. However, its role in ALS remains unclear. This study aimed to investigate longitudinal changes in NPAR and to evaluate its prognostic value in ALS.

Methods: We conducted a retrospective longitudinal analysis of 917 ALS patients, comprising 2,168 NPAR measurements collected at baseline and at 6 and 12 months. NPAR values were log-transformed to improve normality. Longitudinal trajectories of log-NPAR were modeled using linear mixed-effects models with random intercepts and slopes, adjusted for age, sex, site of onset, riluzole treatment, baseline ALSFRS-R score, and body mass index. Survival was analyzed using Cox proportional hazards models with NPAR as a time-dependent covariate, considering a composite endpoint of death or tracheostomy. Data were obtained from the PRO-ACT database.

Results: Log-NPAR increased significantly over time across the entire cohort (slope: 0.036/year, $p < 0.001$). Patients who reached the endpoint of death or tracheostomy exhibited a steeper increase in log-NPAR ($p = 0.0003$). In survival analyses, higher time-dependent NPAR was associated with an increased risk of death or tracheostomy (hazard ratio [HR] 12.75, 95% [CI] 3.09–52.57, $p < 0.001$), with attenuation of the effect over time.

Conclusion: NPAR demonstrates longitudinal changes in ALS, particularly among patients with poorer outcomes, supporting its potential role as a prognostic blood biomarker.

Disclosure: Nothing to disclose.

EPO-0272 | Tofersen in the treatment of SOD1 ALS – A two year experience from the Polish Early Access Program

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Background and aims: Tofersen, an ASO against SOD1, is now registered by FDA and EMA. Some patients obtained tofersen in the EAP and/or rescue therapy. The aim of the study was to follow up on clinical outcomes and NfL concentration in patients participating in the EAP in Poland for 24 months.

Methods: 20 SOD1-ALS patients qualified to EAP (10 FALS and 10 SALS, a male:female ratio of 2.2:1) carried 11 SOD1 mutations (45% – L145S and 10% – K4E). Median age of onset was 49.1 years, age of introducing tofersen was 52.6 years. From the original cohort, 1 patient resign after the first dose of treatment, two withdrew (due to severe adverse event or respiratory insufficiency) and 2 passed away due to disease progression. The analysis included the demographic and genetic data, ALSFRS-R, delta ALSFRS-R, FVC and NfL CSF concentration.

Results: Mean treatment was 17 months (16–24 months), delta ALSFRS-R ranged from 0.46 prior to treatment to 0.15 at the last assessment. Seven patients (37%) showed increase of ALSFRS-R by 1–2 points in the treatment period, six patients (31%) stabilized, while 6 patients (31%) showed further progression. Mean ALSFRS-R was stable after 6 and 24 months, and increased after 12 and 18-months. NfL concentration decreased in 5/7 of analyzed patient. Treatment was well tolerated in 78% of patients.

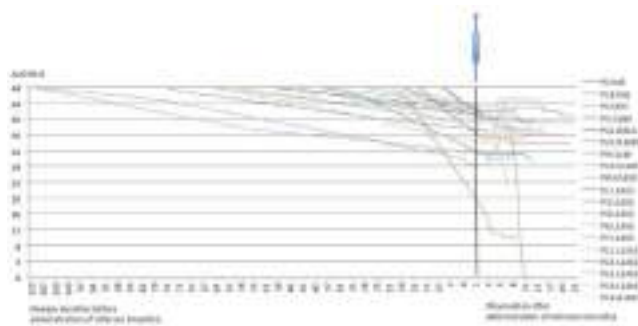


FIGURE 1 Disease progression prior and after tofersen treatment.

Three patients experienced SAE– two transverse myelitis, one exacerbation of colitis ulcerosa and one poliradiculopathy.

Conclusion: The data from a long term follow-up supports the efficiency of tofersen in SOD1-ALS.

Disclosure: MKK obtained lecture grants from Biogen.

EPO-0273 | Sensor-based real-world gait metrics as sensitive mobility outcomes in amyotrophic lateral sclerosis

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Background and aims: Amyotrophic lateral sclerosis (ALS) is a heterogeneous, rapidly progressive neurodegenerative disease. Clinical trials rely mainly on the Revised ALS Functional Rating Scale (ALSFRS-R), which is subjective and often insensitive to subtle changes in ambulation. This study aimed to evaluate the analytical validity, feasibility, and clinical validity of real-world gait outcomes derived from wearable digital health technology (wDHT) in ALS.

Methods: One analytical validation study and three longitudinal observational studies were conducted in Belgium and the United States. Ambulatory adults with ALS wore ankle-mounted wDHTs daily for repeated 30-day periods. Three digital

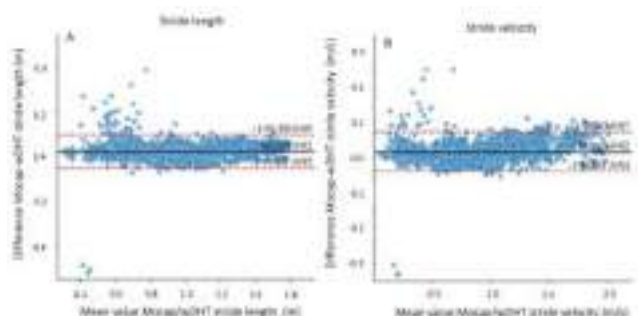


FIGURE 1 Bland Altman plots for the error of the stride velocity and length.

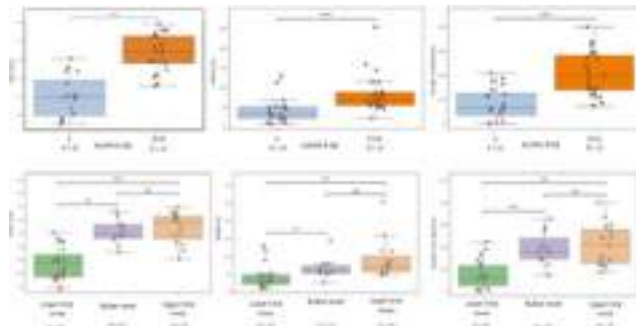


FIGURE 2 Digital outcome measures by ALSFRS-R walking score and disease onset type ($p < 0.05^*$, $p < 0.01^{**}$, $p < 0.001^{***}$, $p < 0.0001^{****}$, ns = not significant). ALSFRS-R Question 8 – Walking assessment: 4 = normal, 3 = early ambulation difficulties.

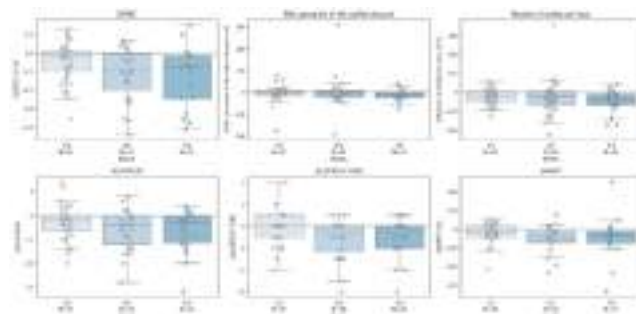


FIGURE 3 Box plots with change at 3, 6, and 9 months for each digital and clinical outcome ($p < 0.05^*$, $p < 0.01^{**}$).

mobility outcomes were derived: stride velocity 95th centile (SV95C), number of strides per hour, and peak walking distance. Analytical validation against optical motion capture assessed stride detection and gait parameter accuracy. Reliability, construct validity and sensitivity to change were evaluated.

Results: Analytical validation showed excellent precision and recall for stride detection (> 0.97), and mean error of 2.2cm and 1.7cm/s for stride length and velocity measurements, respectively ($N = 10$). In the longitudinal study ($N = 46$), adherence rate exceeded 80% over 9 months and SV95C demonstrated outstanding reliability (intraclass correlation coefficient of 0.99). SV95C correlated strongly with the ALSFRS-R gross motor subscore ($R = 0.67$) and 6MWT ($R = 0.89$; $p < 0.001$), but weakly with ALSFRS-R total score. SV95C declined significantly over 3, 6, and 9 months and showed greater sensitivity to change than ALSFRS-R and 6MWT.

Conclusion: Continuous real-world gait monitoring using wDHTs is feasible in ALS. SV95C provides a reliable and sensitive measure of ambulatory decline and represents a promising outcome for ALS clinical trials.

Disclosure: This project received funding from Belgian Association for Neuromuscular Disease (ABMM) and Muscular Dystrophy Association (MDA).

EPO-0274 | The role of hsa-miR-21-5p, hsa-miR-210-3p, hsa-miR-197-3p, hsa-miR-125a-5p and hsa-miR-206-3p as potential biomarkers in amyotrophic lateral sclerosis

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Background and aims: Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disease characterized by progressive loss of motor neurons. MicroRNAs (miRNAs) are small, non-coding RNA molecules that regulate gene expression. The purpose of this study is to identify selected miRNAs in the cerebrospinal fluid (CSF) and serum as biomarkers for ALS.

Methods: A total of 50 ALS patients and 50 age and sex-matched non-ALS controls were enrolled. MiRNAs were isolated from serum and CSF samples and quantified using quantitative reverse transcription polymerase chain reaction (qRT-PCR) with specific primers for hsa-miR-21-5p, hsa-miR-210-3p, hsa-miR-197-3p, hsa-miR-125a-5p and hsa-miR-206-3p. Internal and spike-in controls were used for normalization. ΔC_t values were compared between ALS and non-ALS groups using non-parametric tests with covariates for age, gender, amyotrophic lateral sclerosis functional rating scale – revised (ALSFRS-R), disease duration and progression rate.

Results: Hsa-miR-210-3p and hsa-miR-206-3p were markedly up-regulated in ALS CSF and serum and correlated with disease duration. Hsa-miR-210-3p also correlated with progression rate in the CSF ($p < 0.001$). Hsa-miR-210-3p exhibited the highest diagnostic performance, with an area under curve (AUC) of 0.925 in the serum and 0.902 in the CSF. Hsa-miR-21-5p showed up-regulation in ALS CSF and serum and strong association with poorer ALSFRS-R. Hsa-miR-125a-5p was significantly down-regulated in ALS serum but remained stable in the CSF. Hsa-miR-197-3p showed no diagnostic or prognostic value in either material.

Conclusion: Serum and CSF hsa-miR-210-3p and hsa-miR-206-3p could serve as strong diagnostic ALS biomarkers, while hsa-miR-21-5p as a robust disease-severity indicator. The combination of up-regulated and down-regulated miRNAs suggests a multimarker panel.

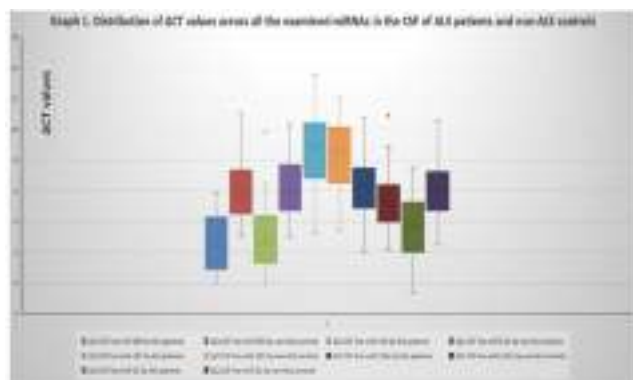


FIGURE 1 Distribution of ΔC_t values across all the examined miRNAs in the CSF of ALS patients and non-ALS controls.

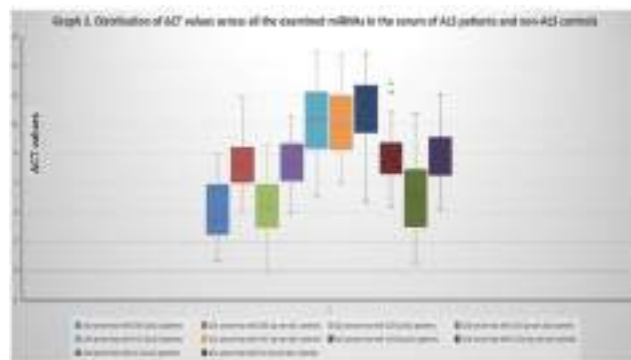


FIGURE 2 Distribution of ΔC_t values across all the examined miRNAs in the serum of ALS patients and non-ALS controls.

Disclosure: All authors declare that they have nothing to disclose and there is no conflict of interest.

EPO-0275 | Elevated CSF GFRA1 and NFL levels: A comparative study on ALS biomarkers

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Background and aims: Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disease characterized by complex axonal degeneration and impairments in neurotrophic support mechanisms, leading to the progressive loss of motor neurons.

Methods: In this study, cerebrospinal fluid (CSF) levels of GFAP, IL-1B, IL-22, IL-6, GFRA1, and NFL were investigated using the ELISA method in 6 patients with ALS (2F, 4M) and 6 healthy controls (3F, 3M).

Results: The median age of the ALS group was 67 years (range: 38–73), while the median age of the healthy control group was 51 years (range: 23–72), no significant difference in age was found between the groups ($p = 0.35$). In the ALS group, cerebrospinal fluid (CSF) levels of GFRA1 and NFL ($10.16 \text{ ng/L} \pm 1.16$ and 20.95 ± 6.29) were significantly higher compared to the control group ($5.30 \text{ ng/L} \pm 3.23$ and 13.37 ± 2.46) ($p = 0.01$, $p = 0.02$). No statistically significant differences were detected between the groups regarding CSF levels of GFAP, IL-1B, IL-22, IL-6, or total protein ($p = 0.85$, $p = 0.85$, $p = 0.68$, $p = 0.87$). Furthermore,

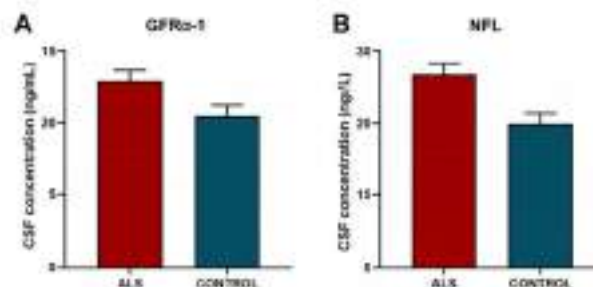


FIGURE 1 Comparative analysis of CSF GFRA1 and NFL levels between ALS patients and healthy controls.

a statistically significant, moderate-to-strong positive correlation was identified between GFRA1 and NFL levels ($r=0.64$; $p=0.03$).

Conclusion: The significant elevation of GFRA1 accompanying the increase in NFL, coupled with the identified positive correlation, reflects a dynamic neurotrophic response that intensifies as motor neuron destruction deepens. This increase may represent a compensatory mechanism aimed at protecting motor neurons from apoptosis. The absence of inflammation suggests that the pathology is driven by an axis of neuronal integrity rather than glia-mediated neuroinflammation. This pathogenic link holds significant potential for both understanding ALS pathogenesis and formulating novel therapeutic strategies.

Disclosure: Nothing to disclose.

EPO-0276 | Respiratory function improvement in spinal muscular atrophy patients after long-term risdiplam treatment: The experience of two Italian centers

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Background and aims: Risdiplam is an available treatment of all patients with spinal muscular atrophy (SMA). Although the respiratory impairment is inevitably progressive also in later-onset SMA, no studies report the effect of this drug on respiratory function.

Methods: We report a case series of 14 patients, followed at two different Italian Centre, with long-term disease duration, treated with risdiplam for more than 5 years. We describe changes in respiratory and motor function and the need for non-invasive ventilation (NIV).

Results: Patients (mean age: 19.7 years, range: 14–28), 11 SMA2 (78.6%) and 3 SMA3 (21.4 %), were sitter (57.1%) or non-sitter (43.9%). The Hammersmith Functional Motor Scale Expanded (HFMSE) score was ≤ 8 in 13/14 patients, and did not show significative changes after treatment ($p=0.9$). Mean FVC at baseline was 1.27 l (SD: 1.02). After more than 5 years (mean follow-up 5.3 years) the mean FVC improved significantly (1.41 l; SD: 1.06, $p=0.04$). Overall 4 patients (28%) showed minimal FVC decrease (-0.12 l; $p=0.09$), while the other 10 patients (72%) showed a significative improvement in FVC ($+0.24$; $p=0.009$). None of the patients on NIV at baseline ($n=8$) required changes in ventilation regime at follow-up.

Conclusion: We report longer-term respiratory follow-up in treated patients and, for the first time, an improvement in commonly used pulmonary function tests, in contrast to motor functional outcome measures. Therefore, we propose to explore a high responsiveness of the FVC, mostly in severely affected SMA patients, in which commonly used motor functional outcome measures might not detect changes.

Disclosure: The study has been founded by Italian Health Ministry project PNRR-MR1-2022-12376937. Nothing else to disclose.

EPO-0277 | Autologous SVF therapy modulates neuroinflammation in ALS: Phase I trial demonstrating safety and CSF biomarker dynamics

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Background and aims: Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disorder with limited treatments. Stromal vascular fraction (SVF), a cell population derived from autologous adipose tissue, exhibits multimodal immunomodulatory and neuroprotective properties, positioning it as a promising therapeutic candidate. This trial aimed to assess autologous stromal vascular fraction (SVF) safety and efficacy in patients with ALS.

Methods: This trial aimed to assess autologous stromal vascular fraction (SVF) safety and efficacy in patients with ALS. 26 patients received combined intravenous (0.5×10^6 cells/kg) and intrathecal (20×10^6 cells) autologous SVF.

Results: SVF administration was well-tolerated. Five mild adverse events (AEs) (subcutaneous bleeding, headache, and low-grade fever) occurred, with no serious AEs reported. Although ALSFRS-R scores showed non-significant improvement post-treatment, 15/26 participants (57.7%) self-reported symptomatic improvement after treatment. Critically, cerebrospinal fluid biomarker analysis revealed significant reductions in neurofilament light chain (NfL; $\Delta 530.29$ pg/mL, $P=0.039$) and glial fibrillary acidic protein (GFAP; $\Delta 622.23$ pg/mL, $P=0.038$), indicating attenuation of neuroaxonal degeneration and astroglial activation. While ALSFRS-R scores showed no significant change ($\Delta -0.53$, $P=0.384$), prognostic modeling identified female sex (OR=0.011, $p=0.008$) and shorter disease duration (OR=1.35/month, $P=0.005$) as predictors of response.

Conclusion: These findings support Autologous SVF therapy demonstrates an acceptable safety profile in patients with ALS. The significant reduction in CSF NfL and GFAP levels provides objective evidence of their potential neuroprotective effects that

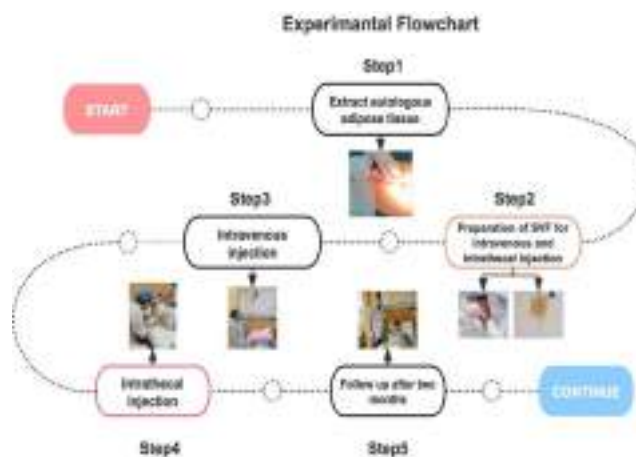


FIGURE 2 The experimental process of this study.

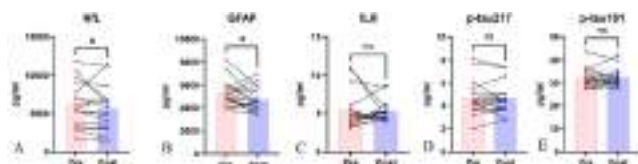


FIGURE 1 CSF biomarker changes after SVF therapy.

modulates ALS-relevant neuroinflammation pathways. Female participants and those with shorter disease duration may derive greater benefits.

Disclosure: Nothing to disclose.

Movement Disorders 2

EPO-0278 | Real-world outcomes of subcutaneous foslevodopa/foscarbidopa therapy in Parkinson's disease (DuoPark study)

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Background and aims: Subcutaneous foslevodopa/foscarbidopa (fLD/fCD) is a continuous infusion therapy for Parkinson's disease (PD) with motor complications. Real-world data are essential to assess its effectiveness and safety in clinical practice.

Methods: Interim analysis of a prospective, multicenter, observational study including people with PD treated with fLD/fCD between April/2024-December/2025. Clinical and socio-demographic variables, “off” and “on” times, and MDS-UPDRS scores (parts III–IV) at 3 and 6 months were analyzed. Infusion rates, levodopa equivalent daily dose, discontinuation of dopaminergic drugs, adverse events, and treatment discontinuation were also assessed. The effectiveness and safety of fLD/fCD for motor symptoms of PD were evaluated.

Results: Forty-one patients were included (48.8% women; age 75 ± 15 years; MDS-UPDRS-III score: “off” 56.16 ± 15.65 ; “on” 34.5 ± 13.34). Mean daily “off” time was 6.02 ± 2.69 h/day, decreasing by 20.32% (-2.38 h/day, $p=0.017$) and 14.10% (-2.08 h/day, $p=0.005$) at 3 and 6 months, respectively. “On” time without troublesome dyskinesia increased by 35.26% ($p=0.003$) and 33.08% ($p=0.002$) at 3 and 6 months. MDS-UPDRS-IV scores improved significantly at both time points, with no significant differences in MDS-UPDRS-III “on” scores. Adverse events occurred in 26 patients (63.4%), mainly infusion-site skin reactions, and neuropsychiatric symptoms. Seven patients (17.1%) discontinued treatment: 4 (9.8%) due to adverse events, 2 (4.9%) due to death, and 1 (2.4%) due to lack of family support.

Conclusion: fLD/fCD infusion significantly reduces “off” time and increases “on” time without troublesome dyskinesia during the first 6 months of treatment. Despite frequent but mostly mild to moderate adverse events, fLD/fCD demonstrated a favorable effectiveness and safety profile.

Disclosure: Nothing to disclose.

EPO-0279 | Neuroinflammatory marker MCP-1 and its correlation with motor and non-motor dysfunction in Parkinson's disease and atypical parkinsonism

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Background and aims: Parkinson's disease (PD) and atypical parkinsonism (AP) represent a heterogeneous group of neurodegenerative disorders with overlapping motor manifestations but markedly different disease trajectories and prognoses. Increasing evidence highlights neuroinflammation as a central mechanism driving disease progression.

Methods: A total of 163 patients were enrolled, including 80 with PD and 83 with AP (MSA, PSP, CBD, DLB). Comprehensive neurological assessments were conducted using the Unified Parkinson's Disease Rating Scale (UPDRS) and standardized non-motor symptom questionnaires. Serum MCP-1 concentrations were measured by enzyme-linked immunosorbent assay. Correlation and multivariate regression analyses were applied to explore associations between MCP-1 levels and clinical parameters. Statistical significance was defined as $p < 0.05$.

Results: MCP-1 levels were significantly higher in AP compared to PD patients ($p < 0.01$). Elevated MCP-1 concentrations demonstrated a strong positive correlation with non-motor symptom severity ($r=0.46$, $p < 0.001$), particularly cognitive decline and autonomic dysfunction. Moderate correlations were observed with motor disability ($r=0.29$, $p < 0.05$). Patients with high MCP-1 levels exhibited accelerated clinical progression and reduced functional independence. Multivariate analysis confirmed MCP-1 as an independent predictor of disease severity after adjustment for age, disease duration, and genetic status.

Conclusion: This study provides the first comprehensive evidence from a Central Asian cohort demonstrating that MCP-1 is a robust biomarker of neuroinflammatory activity and neurological deterioration in parkinsonian disorders. Our findings support the integration of MCP-1 into prognostic models for early risk stratification and personalized management. Presenting these results at an international congress will facilitate cross-regional collaboration, enhance global data diversity, and promote precision medicine approaches in neurodegenerative diseases.

Disclosure: Nothing to disclose.

EPO-0280 | Mapping spatial and temporal evolution of dystonia in atypical parkinsonisms: A retrospective longitudinal cohort study

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Background and aims: Dystonia is a frequent yet inconsistently characterized feature of atypical parkinsonian disorders (APDs), including progressive supranuclear palsy (PSP), multiple system atrophy (MSA), and corticobasal syndrome (CBS). The objective of this study was to provide a first systematic, longitudinal

comparison of dystonia across APDs focusing on its prevalence, topography, and timing of onset.

Methods: We retrospectively analysed longitudinal clinical data from a single-center cohort of 182 patients diagnosed with PSP, MSA, or CBS. A five-year subcohort with homogeneous follow-up from symptom onset ($n = 119$) was examined to estimate dystonia prevalence and spatiotemporal trajectories. Complementary time-to-event analyses were used to model the evolution of dystonia across the entire disease course. Associations between dystonia and disease-progression milestones, including dysphagia and loss of ambulation, were also explored.

Results: Within five years from symptom onset, dystonia occurred in 71% of PSP, 47% of MSA, and 86% of CBS patients. Cranial dystonia was most frequent in PSP (55.6%), whereas upper limb dystonia predominated in CBS (82.6%) (both $p < 0.001$). No significant differences were observed for cervical or truncal dystonia. Upper limb dystonia occurred earlier in CBS compared with PSP ($p = 0.025$). Dystonia-related pain was significantly more frequent in CBS than in MSA and PSP ($p < 0.001$). Dysphagia and loss of ambulation were not significantly influenced by cervical and trunk dystonia, respectively.

Conclusion: Dystonia is a common and early feature of APDs, with distinct spatial and temporal trajectories across syndromes. Systematic longitudinal assessment of dystonia may improve clinical characterization and support diagnostic and therapeutic strategies.

Disclosure: Nothing to disclose.

EPO-0281 | Recruitment barriers for Parkinson's disease research at a tertiary centre in Kenya: A TraPCAf sub-study

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Background and aims: Africa faces a rising burden of Parkinson's disease (PD), yet research is constrained by an unquantified failure to enrol hospital-identified patients into clinical trials, limiting data on disease phenotypes and treatment



FIGURE 1: Study site map, adapted from Step et al, Mov Disord, 2024.

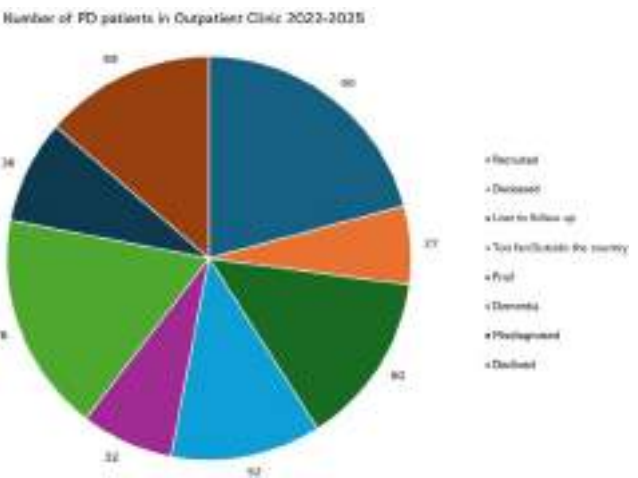


FIGURE 2: Patient final enrolment outcome.

needs. We have previously described these challenges in recruiting participants to a motor neurone disease (MND) study.

Methods: As part of the ongoing multinational Transforming Parkinson's Care in Africa (TraPCAf) study, we conducted a retrospective analysis of patient recruitment challenges at our study site in Nairobi, Kenya. Participants were chosen based on coding from our hospital's electronic health records.

Results: Out of 432 potential participants, 20.8% (90/432) were recruited; of the remaining: 25.0% (108/432) had frailty including dementia; 13.7% (59/432) declined; 13.8% (60/432) were lost to follow-up; 12% (52/432) lived too far, including outside Kenya; 8.3% (36/432) were misdiagnosed; and 6.3% (27/432) were deceased.

Conclusion: Our findings underscore that patient identification from EHR does not translate to successful study enrolment. The spread of challenges is similar to those we encountered in our MND study. Our study suggests these factors should be consistently accounted for when establishing sample sizes for research studies in neurological disease in our part of the world.

Disclosure: This work was conducted as part of the TraPCAf study, funded by the UK National Institute for Health and Care Research (NIHR) via Newcastle University in partnership with Aga Khan University. The views expressed are those of the author(s) and not necessarily those of the NIHR, the UK Department of Health and Social Care, or Newcastle University. The authors have no other conflicts of interest or competing financial interests to declare relevant to this work.

EPO-0282 | Beyond motor symptoms of cervical dystonia – is cognitive deficit primary or secondary feature of the disease?

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Background and aims: The study aimed to evaluate subtle cognitive impairment (CI) in patients with cervical dystonia (CD) and to determine whether it represents an independent non-motor feature of the disease or arises secondary to interactions between motor manifestations and other non-motor

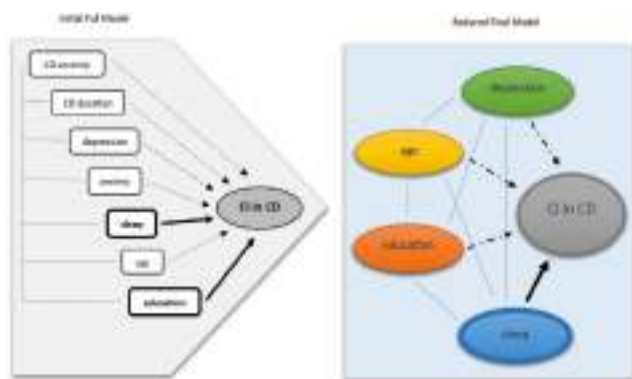


FIGURE 1 Multivariable models of factors associated with cognitive impairment in cervical dystonia. The initial full model and the reduced final model are presented. CI denotes cognitive impairment; CD severity refers to the severity of cervical dystonia.

symptoms (NMS). Although CI is increasingly recognized in dystonia, its associations with motor severity and concomitant NMS remain insufficiently explored.

Methods: Patients with CD underwent comprehensive clinical assessment of motor symptoms (CD severity) and NMS of dystonia (cognition, depression, anxiety, sleep, pain). Matched controls underwent a single assessment (cognition, depression, anxiety, sleep).

Results: Patients with CD demonstrated significantly lower total MoCA scores compared with controls ($p < 0.001$). Relative to controls, CD patients showed poorer performance across multiple cognitive domains: executive function ($p < 0.001$), visuospatial abilities ($p = 0.004$), language ($p < 0.001$), memory ($p = 0.002$), and attention ($p = 0.022$). Patients also exhibited higher rates of depressive symptoms, anxiety, and sleep disturbances. To explore contributing factors, a multivariable model incorporating NMS and socioeconomic variables was constructed, revealing a combined influence of these factors on the severity of CI in CD.

Conclusion: The study examined CI in CD in relation to motor severity and non-motor domains, with multivariable analysis indicating a potential contribution of sleep disturbances. Although a definitive distinction between cognitive dysfunction as a core feature of dystonia or a secondary effect and overlap of other NMS cannot be made, the findings highlight interdependent mechanisms underlying cognitive deficit in CD. These results underscore the importance of comprehensive assessment of non-motor domains in this population.

Disclosure: Nothing to disclose.

EPO-0283 | Investigation of SKY-0515 in patients with Huntington's disease: Interim results of a phase 1 study

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Background and aims: Huntington's disease (HD) is a neurodegenerative disorder caused by a trinucleotide (CAG) repeat expansion in the huntingtin (HTT) gene, resulting in a toxic mutant huntingtin (mHTT) protein. Disease progression correlates with the rate of somatic CAG expansion. SKY-0515 has the potential to provide disease modifying benefits through reduction

of toxic proteins, mHTT and PMS1, a key driver of expansion. The study aims to evaluate safety, PK/PD and efficacy of SKY-0515, an orally available, brain penetrant small molecule RNA splicing modulator, in HD patients.

Methods: SKY-0515-001-C is an ongoing 15-month Phase 1 study in HD. Patients were randomized to 3mg ($N = 10$), 9mg ($N = 10$) or placebo ($N = 6$) for 84 days, after which patients rolled over onto active drug, receiving 4mg or 9mg (blinded) for an additional 12 months. Key enrollment criteria include genetic diagnosis of HD (CAG ≥ 40) and clinical baseline of TFC ≥ 10 , IS ≥ 70 , and TMS ≥ 6 , reflecting an HD-ISS disease stage 2 or mild stage 3.

Results: 9-month interim results in patients who continuously received SKY-0515 (excluding placebo) show positive trends in HD efficacy outcomes compared to expected natural history disease progression. In HD patients treated with SKY-0515, early signs of disease stabilization are observed in components of the UHDRS, including function (TFC = +0.35 points), motor (TMS = -3.69 points), and cognition (Stroop = +1.0 points). SKY-0515 has been well tolerated up to 9 months.

Conclusion: These findings support further clinical evaluation of SKY-0515 as a potential disease-modifying therapy for HD. A confirmatory Phase 2/3 study (FALCON-HD) is now enrolling.

Disclosure: The authors are full time employees of Skyhawk Therapeutics, a privately owned company, and the sponsor of the referenced study.

EPO-0284 | Objective assessment of long- and short-term levodopa effects on hypomimia and head movement

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Background and aims: Hypomimia is a hallmark feature of Parkinson's disease (PD) that significantly impairs non-verbal communication. Although levodopa is known to improve motor symptoms, its effect on facial and head movements, particularly in the early phase of treatment, has not been objectively and quantitatively assessed.

Methods: Video recordings were obtained using a single-camera setup from 93 drug-naïve PD patients at baseline and one year later after initiation of stable pharmacological treatment. At follow-up, patients were examined in a defined OFF state (≥ 12 hours without dopaminergic medication) and in the ON state one hour after medication intake. Facial and head movements were quantified using a state-of-the-art facial landmark tracking system and derived movement markers. A control group of 50 age- and sex-matched healthy participants was also recorded.

Results: At baseline, PD patients showed significantly reduced facial and head movements compared with healthy controls ($p < 0.001$). After one year of treatment, a significant improvement was observed across all evaluated areas ($p < 0.001$) nearly matching performance of control group. However, no significant differences were found between the OFF and ON states during the short-term levodopa challenge at follow-up.

Conclusion: Levodopa treatment is associated with a significant long-term improvement of hypomimia across facial and

head movement domains in early PD. The absence of short-term OFF–ON differences suggests that a 12-hour washout period is insufficient to fully eliminate the sustained beneficial effects of stable dopaminergic treatment.

Disclosure: The authors report no disclosures. This study was supported by the Ministry of Health of the Czech Republic, grant No. NU23J-04-00042.

EPO-0285 | **Unilateral MR-guided laser interstitial thermal therapy thalamotomy for essential tremor: A 12-month follow-up study**

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Background and aims: Essential tremor (ET) often resists medical therapy. Magnetic Resonance-guided Laser Interstitial

TABLE 1 Clinical characteristics and longitudinal outcomes. Data presented as mean ± SD or n (%). *p*-values compare baseline vs. 12 months for primary metrics (FTM-TRS, QUEST-SI, MoCA, MMSE). Adverse events decreased significantly by month 12.

Parameter	Baseline	3 Months	12 Months	p-value
DEMOGRAPHICS				
Age (years)	67.2 ± 12.8	–	–	–
Sex (Male / Female)	12 / 9	–	–	–
Handedness (Right / Left)	17 / 4	–	–	–
Thalamotomy Side (Left / Right)	17 / 4	–	–	–
TREMOR SEVERITY (FTM-TRS)				
Global Score (Treated Limb)	14.0 ± 3.1	3.9 ± 2.4	3.5 ± 1.6	< 0.001
Resting Tremor (Treated Limb)	0.2 ± 0.5	0.0 ± 0.0	0.0 ± 0.0	–
Postural Tremor (Treated Limb)	2.3 ± 0.9	0.1 ± 0.3	0.1 ± 0.3	–
Action Tremor (Treated Limb)	2.0 ± 0.7	0.3 ± 0.5	0.6 ± 0.6	–
Drawing Score (Treated Limb)	9.4 ± 2.4	3.5 ± 2.3	2.8 ± 1.2	–
QUALITY OF LIFE (QUEST)				
Summary Index (%)	41.2 ± 15.7	19.2 ± 16.0	18.0 ± 14.0	< 0.001
Communication Domain (%)	29.0 ± 25.5	15.1 ± 22.7	15.8 ± 20.9	–
Work / Daily Activities (%)	18.7 ± 20.6	14.3 ± 24.7	7.5 ± 11.5	–
Leisure Activities (%)	41.7 ± 32.0	23.0 ± 30.4	15.5 ± 24.5	–
Physical Domain (%)	63.6 ± 18.2	21.8 ± 21.7	25.9 ± 23.3	–
Psychosocial Domain (%)	52.8 ± 25.0	21.8 ± 18.1	22.9 ± 20.2	–
COGNITION & SATISFACTION				
MoCA Score	25.4 ± 3.1	–	23.9 ± 3.5	0.026
MMSE Score	27.2 ± 2.3	–	26.5 ± 2.6	0.158
Patient Satisfaction (P-CGI %)	–	–	82.0 ± 20.7%	–
SAFETY & COMPLICATIONS				
Adverse Events (n, %)	–	15 (71.4%)	1 (4.8%)	< 0.001
Rehospitalization (n, %)	–	0 (0%)	3 (7%)	–

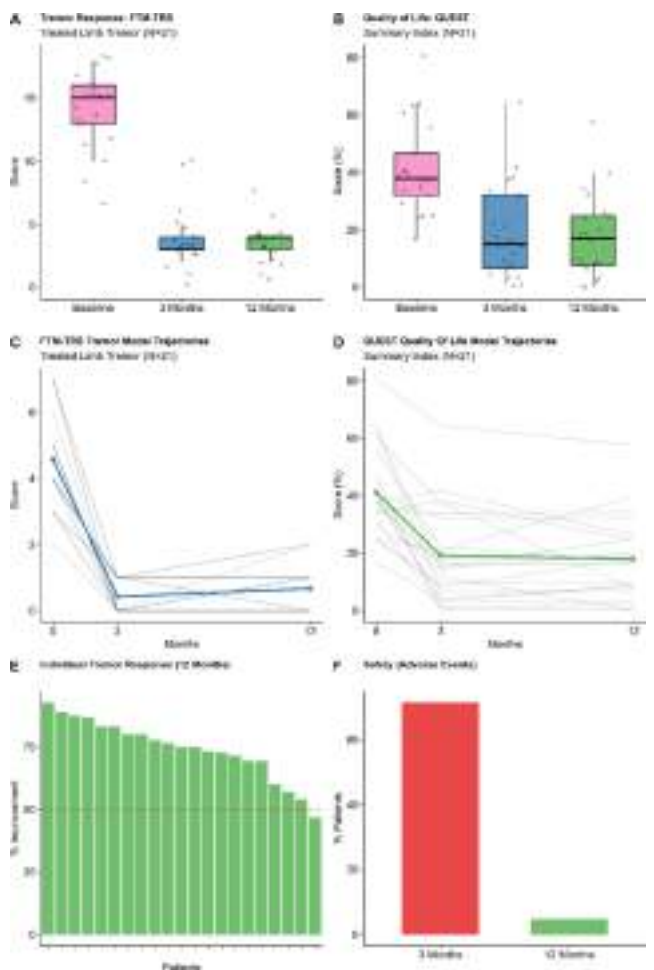


FIGURE 1 Unilateral MRgLITT thalam outcomes. (A-B) Group improvements in FTM-TRS and QUEST-SI. (C-D) Longitudinal model trajectories. (E) Waterfall plot of 12-month individual responders. (F) Transition from transient (M3) to resolved (M12) adverse events.

Thermal Therapy (MRgLITT) is a minimally invasive technique allowing real-time MRI thermometry for precise thalamotomy. We evaluated the 12-month efficacy and safety of unilateral MRgLITT thalamotomy in 21 pharmacoresistant ET patients.

Methods: Patients underwent unilateral MRgLITT thalamotomy (ROSA robot, Visualase system). Assessments at baseline, 3, and 12 months used the Fahn-Tolosa-Marin Tremor Rating Scale (FTM-TRS). Primary outcome was the treated limb FTM-TRS score (rest, posture, action, drawing). Secondary outcomes included Quality of Life in Essential Tremor Questionnaire (QUEST-SI), Patient Global Impression of Change (P-CGI), Montreal Cognitive Assessment (MoCA), Mini-Mental State Examination (MMSE), and adverse events (AE). Linear mixed-effects models were used for analysis.

Results: Twenty-one patients (mean age 67.2 ± 12.8 years; 12 males; 17 right-handed) underwent unilateral thalamotomy (17 left). Treated limb FTM-TRS scores decreased by 75.1% (14.0 ± 3.1 baseline vs 3.5 ± 1.6 at 12 months; *p* < 0.001). All patients (100%) achieved > 50% improvement. QUEST-SI improved from 41.2 ± 15.7% to 18.0 ± 14.6% (*p* < 0.001). Mean satisfaction (P-CGI) was 82.0 ± 20.7%. Transient AEs (3 months: 71.4%) primarily included proprioceptive and balance disorders; only one patient (4.8%, gait ataxia) had persistent symptoms at 12 months. MoCA decreased slightly (25.4 ± 3.1 to 23.9 ± 3.5; *p* = 0.024) while MMSE remained stable (27.2 ± 2.3 vs 26.5 ± 2.6; *p* = 0.158).

Conclusion: Unilateral MRgLITT thalamotomy provides robust, sustained tremor relief and improved quality of life for

pharmacoresistant ET. Despite the small cohort, it represents a safe, promising alternative to other surgical techniques. Larger longitudinal studies are warranted to establish its long-term role.
Disclosure: Nothing to disclose.

EPO-0286 | **SWITCH-ON: Prospective, observational study of LECIG efficacy in Parkinson's disease patients previously treated with subcutaneous foslevodopa**

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Background and aims: Levodopa–entacapone–carbidopa intestinal gel (LECIG) is a device-aided therapy (DAT) for advanced Parkinson's disease (PD) patients who have severe motor fluctuations and hyperkinesia/dyskinesia that persists despite treatment with optimised oral therapy combinations (Figure 1). Subcutaneous (SC) infusion of foslevodopa/foscarbidopa (Fos) is an alternative DAT option in this setting. However, real-world data suggest patient discontinuation from SC Fos is high, so other DAT options need to be considered. The SWITCH-ON study (NCT07313176; Figure 2) will evaluate whether transition to intestinal infusion of LECIG is beneficial for those who discontinue SC Fos.

Methods: Target recruitment is ~215 adult PD patients from 30 centres in Spain, the UK, Germany, Romania and the Netherlands undergoing routine clinical care who will be followed-up for 12-months. Patients will have had prior SC Fos for ≥1 month. Data collection is planned from January 2026, with an interim analysis once 100 patients have completed ≥1 follow-up visit. Data completion is planned for January 2028.

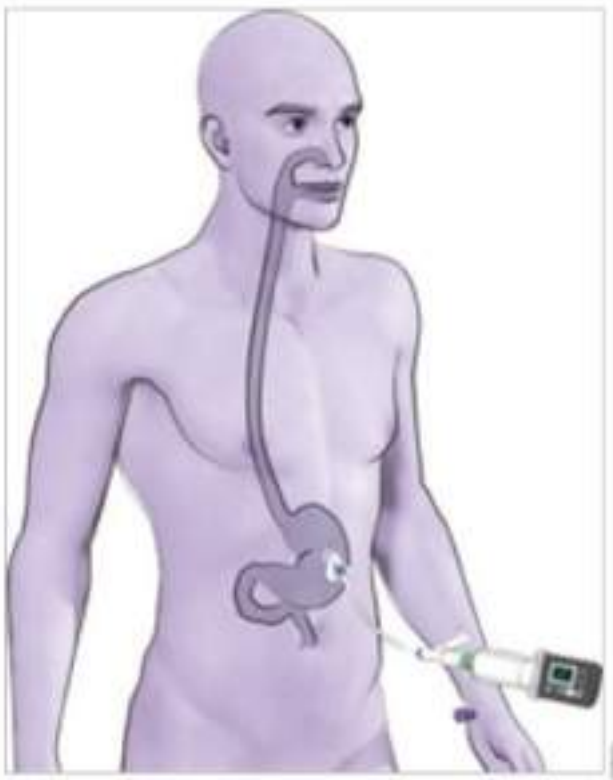


FIGURE 1 Illustration of the placement of levodopa–entacapone–carbidopa intestinal gel (LECIG) infusion as a device-aided therapy for advanced Parkinson's disease.



FIGURE 2 SWITCH-ON Study logo.

TABLE 1 SWITCH-ON Study secondary endpoints.

Secondary endpoints and their assessment	
Off-time reduction	- At 6 months using the Movement Disorder Society Unified Parkinson's Disease Rating Scale (MDS-UPDRS) part IV - Up to 12 months using Hauser patient diaries
Treatment pattern analysis	Assessed from daily levodopa dose (mg/day) up to 12 months using total daily dose (morning bolus, continuous flow, extra boluses, and oral doses) and pump multi-rate programming usage
Other endpoints assessed	Up to 12 month change in: - Clinician Global Impression–Corrections and Patient Global Impression–Corrections - Non-motor experiences of daily living using MDS-UPDRS part I - Motor complications using MDS-UPDRS part IV - Quality of life using the Parkinson's Disease Quality of Life Questionnaire 8-item (PDQ-8) - Sleep using the Parkinson's Disease Sleep Scale 2 (PDSS-2) - Treatment satisfaction, assessed using a questionnaire - Body mass index (BMI)
Safety and tolerability	Long-term safety by reporting of adverse drug reactions and severe adverse drug reactions; gastrointestinal adverse events and severe adverse events and special situations

Results: The primary outcome is OFF time reduction with LECIG treatment at 12 months using the Movement Disorder Society Unified Parkinson's Disease Rating Scale part IV, with multiple secondary endpoints (Table 1). SWITCH-ON aims to generate valuable real-world evidence on the long-term effectiveness, safety and tolerability of LECIG in routine clinical practice for advanced PD patients transitioning from SC Fos therapy.

Conclusion: SWITCH-ON hopes to demonstrate that intrajejunal therapy can be a feasible option after Fos, and that prior SC treatment failure is not the end of the road for patients.

Disclosure: The SWITCH-ON study is sponsored by Britannia Pharmaceuticals Ltd. SS, NS and BA are employees of Britannia Pharmaceuticals Ltd.

EPO-0287 | **Patient perceptions of the Crono® LECIG pump device in advanced Parkinson's disease treatment – data from the ELEGANCE observational study**

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Background and aims: Levodopa–entacapone–carbidopa intestinal gel (LECIG) infusion is a treatment option for advanced Parkinson's disease (PD). It is delivered using the specifically designed Crono® LECIG pump (Figure 1), which aims to provide convenience and usability for patients. ELEGANCE is an international, non-interventional study (NCT05043103) capturing efficacy, safety and patient-reported outcomes data from advanced PD patients treated with LECIG in routine clinical practice.

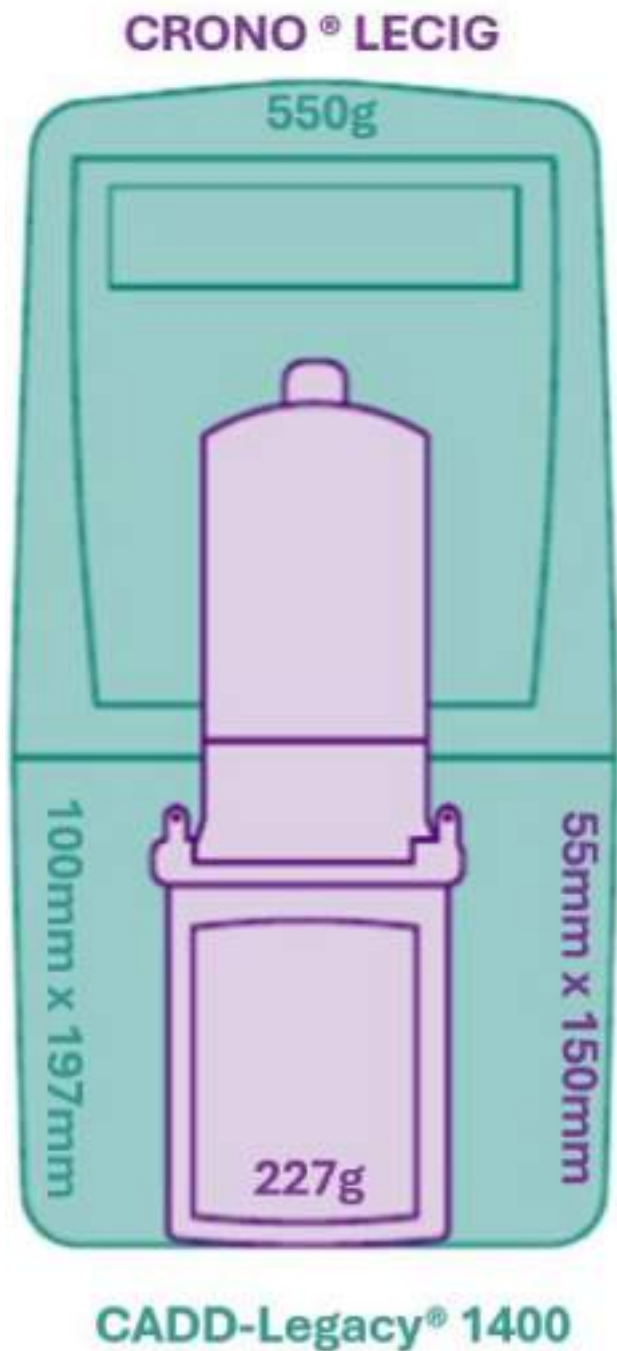


FIGURE 1 Comparison of the Crono® LECIG pump and the Smith Medical CADD-LEGACY® 1400 pump. Silhouettes show size and weight with full cassette (references: Crono® LECIG pump user manual and Smith Medical CADD-LEGACY® 1400 pump user manual).

Methods: Here we report an analysis of interim data regarding patient perceptions of the Crono® LECIG pump for 161 patients treated with LECIG infusion for up to 24 months. Patient-reported satisfaction with the device, as well as its handling, weight, size and noise, were assessed on scale of 1 = absolutely dissatisfied to 10 = absolutely satisfied, with a total score for all parameters (possible total score: 50).

Results: Mean satisfaction scores were high (≥ 8.2 for individual parameters), giving a mean total satisfaction score of 43.5.

Table 1. Perceptions of the Crono® LECIG pump according to previous treatment at Visit 5 (up to 24 months of LECIG treatment). Total score: maximum score = 50; individual attributes: maximum score = 10.

Previous treatment	Weight		Size		Handling		Noise		Overall handling		Total score	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Did not switch from another pump system (n=143)	8.52	0.82	8.18	0.94	8.65	1.25	9.39	1.58	8.73	1.25	43.34	4.84
Switched from another pump system (n=18)	9.17	1.20	8.72	2.00	8.39	2.14	9.56	0.74	8.83	1.51	44.67	6.48
Switched from LECIG (n=10)	9.30	0.99	9.00	1.49	8.70	1.69	9.50	1.25	9.00	1.34	45.50	5.53
Switched from APO (n=8)	9.00	1.99	8.38	2.19	8.00	1.80	9.63	1.35	8.63	1.52	43.63	7.27
All groups (n=161)	8.59	1.91	8.24	2.13	8.62	1.79	9.41	1.34	8.74	1.49	43.49	7.10

APO, subcutaneous apomorphine infusion LECIG, levodopa-carbidopa intestinal gel infusion; SD, standard deviation.

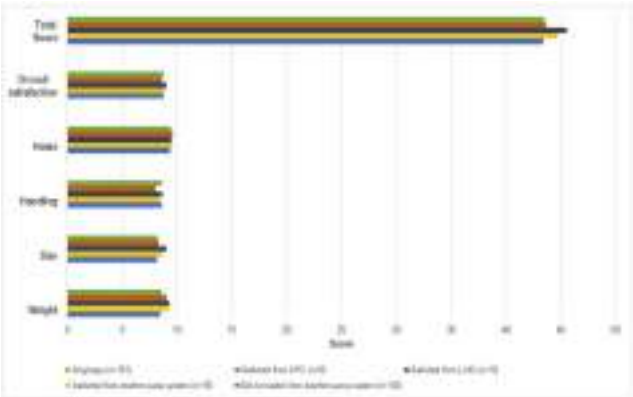


FIGURE 2 Perceptions of the Crono® LECIG pump according to previous treatment at Visit 5. Total score: maximum score = 50; individual attributes: maximum score 10.

Scores stratified by previous treatment confirm high satisfaction, even in those who switched from another infusion therapy (Table 1; Figure 2). Levodopa infusion switch patients reported the highest satisfaction scores for most attributes, suggesting that the smaller, lighter design of the pump is meaningful. APO infusion switch patients had lower satisfaction scores which may reflect differences in device expectations.

Conclusion: Higher device satisfaction scores in those who switched from another pump therapy, suggest the Crono® LECIG pump may address some limitations of other devices. These findings highlight the critical role of patient-reported outcomes in evaluating drug-delivery developments in PD.

Disclosure: The ELEGANCE study is sponsored by Britannia Pharmaceuticals Ltd. SS, NS and BA are employees of Britannia Pharmaceuticals Ltd.

EPO-0288 | Eslicarbazepine acetate shows anti-tremorogenic properties in the mouse harmaline-induced tremor model

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Background and aims: Essential tremor (ET) is a common movement disorder, affecting around 5% of individuals over 65.

Its hallmark feature is an 8–12 Hz kinetic tremor of upper limbs, often accompanied by head and voice tremor. Current treatment relies on pharmacological agents, yet almost 55% of patients respond inadequately. This study evaluated the anti-tremorgenic effect of eslicarbazepine acetate (ESL), an antiseizure medication approved for treatment of focal-onset epilepsy, in the harmaline-induced tremor mouse model.

Methods: Quantitative electroencephalography (EEG) was recorded for 6 hours in freely moving mice (previously implanted with electrodes on motor cortex and cerebellum) under a cross-over design. Tremor activity was assessed for 18 minutes in a tremor monitor chamber. Animals received ESL (75 and 150 mg/kg, gavage), propranolol (10–20 mg/kg, intraperitoneally) as reference, or vehicle-control, 20–30 min before harmaline (20mg/kg, intraperitoneally).

Results: In vehicle-treated ($n=10$), harmaline induced sustained EEG low gamma oscillation increase in both brain regions. Similarly to propranolol, ESL treatment significantly, and dose-dependently, prevented harmaline-induced low gamma oscillation increase vs vehicle (ESL 75mg/kg $p < 0.05$ in motor cortex, ESL 150mg/kg $p < 0.01$ in both brain regions, at 2hours time-point). In vehicle-treated, harmaline induced a generalized tremor activity ($n=12$) peaking at 12–14 Hz, whilst with ESL, similarly to propranolol, it was reduced (–18% by ESL 75mg/kg $p=0.002$, –21% by ESL 150mg/kg $p=0.0002$, vs vehicle).

Conclusion: ESL reversed harmaline-induced EEG alterations and attenuated tremor in a mouse model of ET. This effect, possibly attributed to a T-type calcium channel inhibition, suggests a therapeutic potential for this movement disorder.

Disclosure: Supported by BIAL – Portela & C^a, S.A. Ana Coelho, Valentina di Foggia, Joerg Holenz and Nuno Pires are current BIAL employees.

EPO-0289 | Functional bowel disorders in idiopathic Parkinson's disease: A Rome IV-based controlled study

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Background and aims: Functional bowel disorders (FBDs) are common non-motor manifestations of idiopathic Parkinson's disease (iPD). Although constipation is well recognized, the prevalence and clinical relevance of broader Rome IV-defined FBDs in iPD remain insufficiently characterized. This study aimed to compare FBD frequency between iPD patients and controls and to examine their clinical associations within iPD.

Methods: This cross-sectional controlled study included 64 patients with idiopathic Parkinson's disease and 64 age- and sex-matched controls without neurological disease. Functional bowel disorders were assessed using the Rome IV Diagnostic Questionnaire. Non-motor symptoms, mood, and quality of life were evaluated using the Non-Motor Symptoms Questionnaire, Hamilton Depression Rating Scale, Hamilton Anxiety Rating Scale, Parkinson's Disease Questionnaire-39, and the Gastrointestinal Quality of Life Index. Multivariable logistic regression was used to assess factors associated with FBD presence, and multivariable linear regression to identify predictors of gastrointestinal quality of life.

Results: The prevalence of functional bowel disorders did not differ significantly between iPD patients and controls. Within

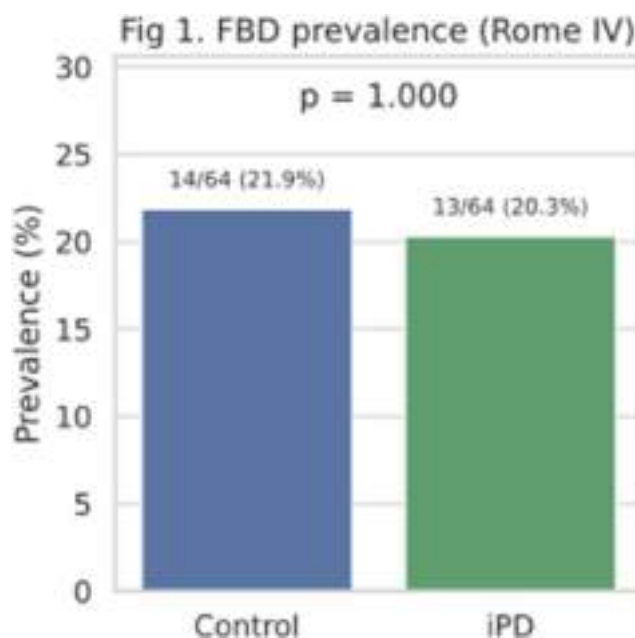


FIGURE 1 Prevalence of Rome IV-defined functional bowel disorders in idiopathic Parkinson's disease patients and controls ($p=1.000$).

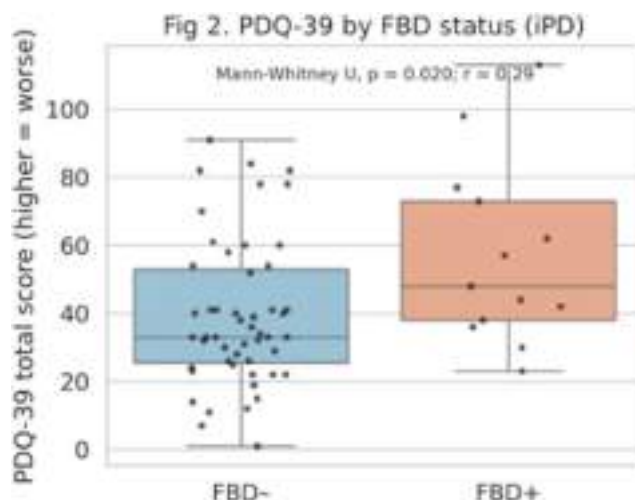


FIGURE 2 Parkinson's Disease Questionnaire-39 total scores according to functional bowel disorder status in idiopathic Parkinson's disease patients.

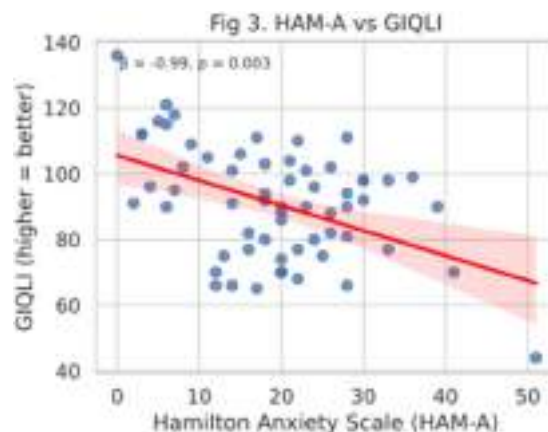


FIGURE 3 Relationship between Hamilton Anxiety Scale scores and Gastrointestinal Quality of Life Index in idiopathic Parkinson's disease patients.

the iPD group, FBD positivity was associated with higher non-motor symptom burden and poorer quality of life. Cognitive and sexual domains of the Parkinson's Disease Questionnaire-39 were particularly affected. Anxiety-related measures were significant predictors of reduced gastrointestinal quality of life.

Conclusion: Functional bowel disorders do not appear to be disease-specific markers in idiopathic Parkinson's disease but rather phenotype modifiers associated with increased non-motor symptom burden. Targeting anxiety-related factors may improve gastrointestinal well-being in iPD.

Disclosure: Nothing to disclose.

MS and Related Disorders 2

EPO-0290 | Microstructural damage and disability associations of paramagnetic rim and T1-hypointense lesions in multiple sclerosis

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Background and aims: Recent microglia PET studies in MS show that most white matter (WM) T1-hypointense lesions exhibit activated microglia within lesion core or at the border. Lesions with activated microglia, such as paramagnetic rim lesions (PRLs), are associated with smoldering inflammation and greater clinical disability. However, whether PRLs and non-PRL-T1-hypointense lesions share similar tissue-disrupting properties remains unclear. This diffusion MRI study investigated lesion-level microstructural damage in PRLs, non-PRL-T1-hypointense lesions, and T1-isointense lesions, and assessed the relative contribution of their lesion burden to clinical disability.

Methods: In 34 newly diagnosed, treatment-naïve patients with relapse-onset MS (mean age 38.4 years; mean disease duration 0.65 years) WM lesions were classified using FLAIR, T1-weighted and susceptibility-weighted imaging as PRLs, non-PRL-T1-hypointense lesions, or T1-isointense lesions. Fractional anisotropy (FA), mean diffusivity (MD), and neurite density index (NDI) were extracted for each lesion class. Regression models compared diffusion metrics across lesion classes and evaluated associations between lesion burden and Expanded Disability Status Scale (EDSS) scores.

Results: Among 638 lesions, 23 were PRLs (4%), 586 non-PRL-T1-hypointense (91%), and 29 T1-isointense lesions (5%). PRLs showed lower FA than non-PRL-T1-hypointense ($p=0.045$) and T1-isointense lesions ($p=0.005$). Both PRLs and non-PRL-T1-hypointense lesions had higher MD and lower NDI compared with T1-isointense lesions (all $p < 0.002$). At patient level, PRL volume was more strongly associated with clinical disability ($p < 0.001$) than non-PRL-T1-hypointense ($p=0.334$) and

T1-isointense lesion volume ($p=0.737$), independently of age and disease duration.

Conclusion: Non-PRL-T1-hypointense lesions represent over 90% of MS WM lesions and exhibit intermediate tissue disruption. Despite their low prevalence, PRL burden most strongly affects clinical disability.

Disclosure: Nothing to disclose.

EPO-0291 | Identifying MS subtypes using MRI-based machine learning

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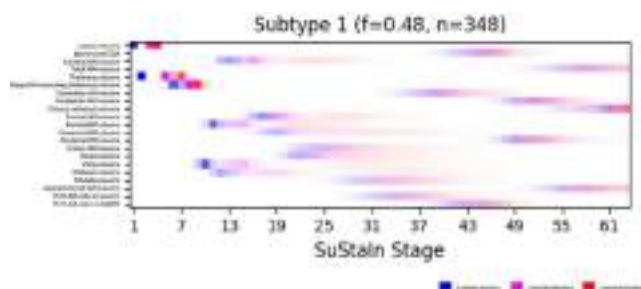


FIGURE 1 Progression pattern of MRI abnormalities in MRI-driven subtype 1 identified by SuStaIn. Subtype 1 was lesion-driven, characterised by early lesion accumulation, followed by the involvement of deep grey matter structures.

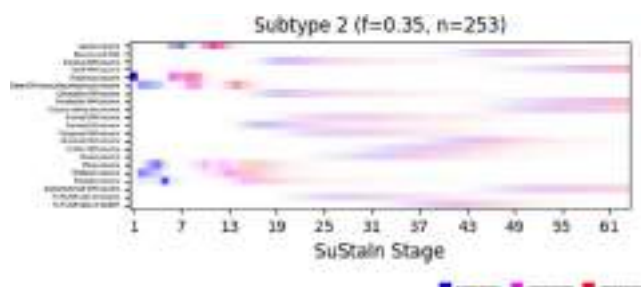


FIGURE 2 Progression pattern of MRI abnormalities in MRI-driven subtype 2 identified by SuStaIn. Subtype 2 showed diffuse and early deep grey matter and brainstem damage.

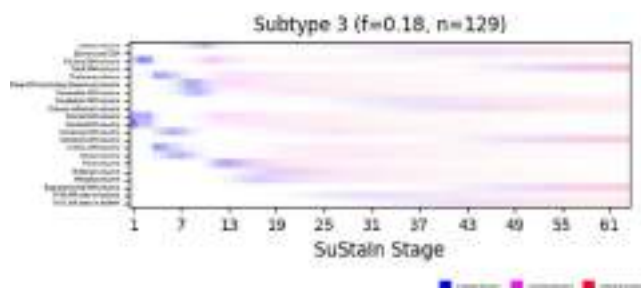


FIGURE 3 Progression pattern of MRI abnormalities in MRI-driven subtype 3 identified by SuStaIn. Subtype 3 was characterised by prominent cortical grey matter damage with early deep grey matter involvement.

Background and aims: Multiple sclerosis (MS) is a heterogeneous disorder whose clinical classification does not fully capture its pathological complexity. Machine learning (ML) approaches applied to research data have identified MRI-based MS subtypes. The aim of this study is to validate this ML approach using real-world data.

Methods: We analysed cross-sectional MRI data from a multicentre MAGNIMS cohort of patients with MS (pwMS) and healthy controls (HC), including a training cohort (746 pwMS, 114 HC) and an independent validation cohort (199 pwMS). T1-weighted and T2-FLAIR MRI data were processed to extract measures of lesion burden, tissue integrity, brain and spinal cord volumes. MRI variables distinguishing pwMS from HC were normalised using Bayesian linear regression. The unsupervised ML algorithm SuStaIn was used to identify MRI-driven subtypes and stages, and their associations with clinical phenotypes and disability were assessed using appropriate regression models.

Results: Three distinct MRI-driven MS subtypes were identified and replicated across training and validation cohorts: (1) a lesion-driven subtype, (2) a subtype characterised by early deep grey matter and brainstem damage, and (3) a cortical grey matter-driven subtype. Clinical phenotype distributions were consistent across cohorts, with no significant associations between MRI-driven subtypes and standardised clinical phenotypes. ML-derived stages correlated with disease duration ($b=0.072$, $p<0.001$) and were associated with clinical phenotypes (LR $\chi^2=90.03$, $b=0.545$, $p=0.002$) and disability ($b=0.263$, $p<0.001$).

Conclusion: These findings confirm that ML applied to MRI data can identify MS subtypes and stages of progression in real-world multicentre cohorts, correlating with but extending beyond clinical phenotypes within a data-driven classification framework.

Disclosure: ABia received research funding from the Italian Society of Neurology, a MAGNIMS-ECTRIMS fellowship (2023), and honoraria/travel support from Merck Serono and Biogen. RC received honoraria/travel support from Roche, Merck Serono, UCB, Sanofi-Genzyme, Alexion, Novartis and Janssen, and research funding from the Italian Ministry of University and Research. FB serves/served on steering committees, DSMBs, advisory boards or as consultant for multiple pharmaceutical and research organizations, has research agreements with industry partners, and is co-founder/shareholder of Queen Square Analytics Ltd. ABis, BB, ACag, MC, MF, CGa, SL, CL, MM, MAR, SR, BS, EMMS, CT, TU, MV, ATo, OC and NDS received honoraria, consulting fees, travel support and/or research funding from pharmaceutical companies active in MS research. Several authors (FB, MF, MMS, EMMS, ATo, MAR) hold editorial roles in scientific journals. CGr reports institutional research support and fees received by her employer. DRN, FP and ACaI received fellowship or grant support from public or charitable bodies. MB, AE, GB, LL, FS, GG, FC, ML, BB, EC, SC, AG, MK, AL, EMH, GP, ATa and PV declare no disclosures. All the authors declare no disclosures for this work.

EPO-0292 | Brain lesion topography relates to cognitive functions in multiple sclerosis: Cross-sectional and longitudinal analysis

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Background and aims: Cognitive impairment (CI) is a common and disabling feature of multiple sclerosis (MS), affecting

multiple domains. Understanding the biological mechanisms underlying CI in MS is key research goal. Conventional MRI measures have long been associated with cognitive deficits, reflecting the impact of white matter disconnection on large-scale brain networks. This study investigated cross-sectional and longitudinal associations between brain lesion distribution and domain-specific cognitive performance in MS.

Methods: We analyzed longitudinal data from the Amsterdam MS cohort. Cognitive performance was assessed at baseline and 5-year follow-up using the Brief Repeatable Neuropsychological Battery. Longitudinal cognitive decline was defined using a regression-based Reliable Change Index. Baseline T2-weighted lesion masks were processed and analyzed using voxel-based lesion-symptom mapping. All models were adjusted for demographic and clinical covariates, total lesion volume.

Results: Cross-sectional analyses identified lesion-symptom associations across major white matter pathways. Processing speed and attention were associated with lesions in the corpus callosum, cingulum, and internal capsule, while memory and language domains involved temporo-occipital and frontotemporal tracts. Baseline lesion distribution predicted cognitive performance at 5 years across several domains. Longitudinal decline occurred in a subset of patients and was specifically associated with lesions in the left superior temporal pole for executive

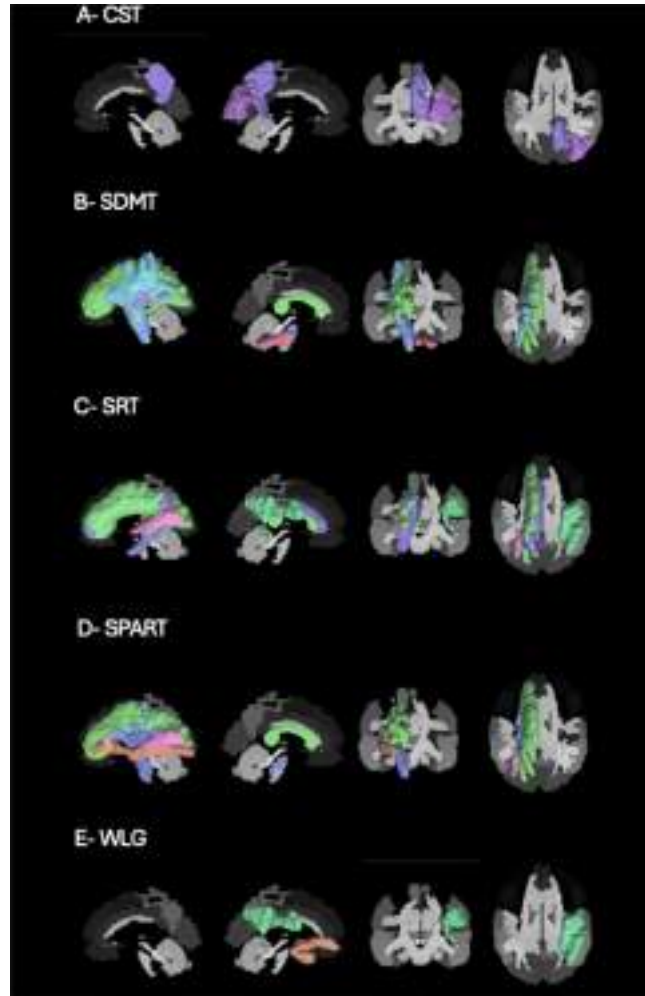


FIGURE 1 Atlas-based visualization of regions of interest across neuropsychological tests at year 0. Different colors indicate distinct anatomical regions across neuropsychological tests.

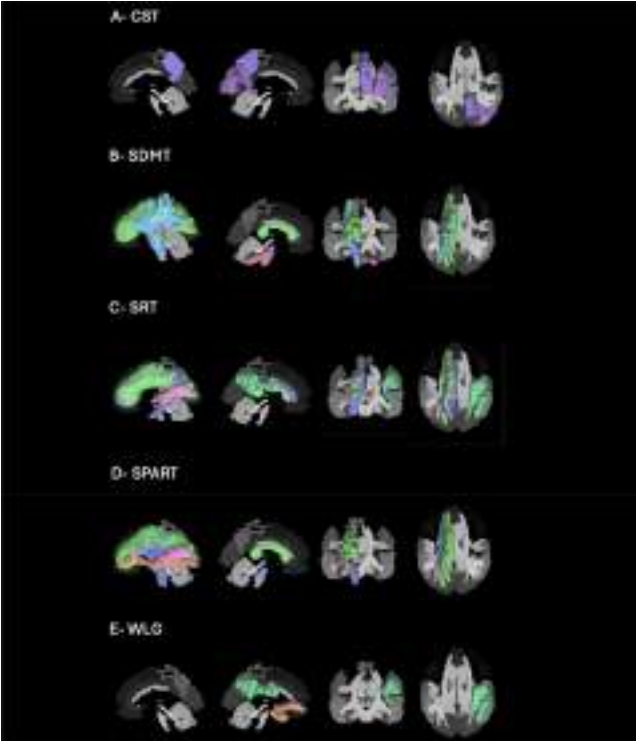


FIGURE 2 Atlas-based visualization of regions of interest across neuropsychological tests at year 5. Different colors indicate distinct anatomical regions across neuropsychological tests.

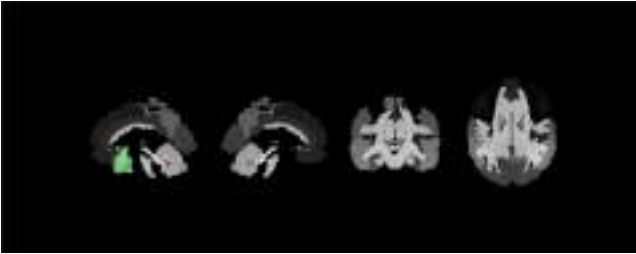


FIGURE 3 Atlas-based visualization of regions of interest associated with CST-decliners.

function. These associations were visualized using atlas-based representations of affected regions across cognitive domains.

Conclusion: Lesion location is a key determinant of current and future CI in MS. Shared involvement of major white matter tracts supports domain-general disconnection mechanism, while selective regions contribute to domain-specific vulnerability. Lesion mapping may provide prognostic information beyond global lesion measures.

Disclosure: Nothing to disclose.

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Background and aims: Multiple sclerosis (MS) is a chronic immune-mediated disease of the central nervous system in which aging-related mechanisms are thought to contribute to disease activity. Telomere length is a well-established biomarker of biological aging and has been reported to be shortened in MS. However, the contribution of telomere-regulating genes such as TERF1 and TERF2 to telomere dynamics in MS remains unclear.

Methods: This case-control study included 40 patients with relapsing-remitting MS (RRMS) and 39 age- and sex-matched healthy controls aged 18–40 years. Relative leukocyte telomere length was measured by qPCR using the telomere-to-single copy gene ratio. The TERF1 polymorphisms rs10107605 and rs1545827, and the TERF2 polymorphism rs251796 were genotyped using high-resolution melting analysis.

Results: Leukocyte telomere length was significantly shorter in patients with MS compared with healthy controls ($p=0.005$). Telomere length was not associated with age, sex, smoking status, disease duration, or other clinical variables within the MS group. Receiver operating characteristic analysis demonstrated a moderate discriminatory capacity for telomere length between patients and controls (AUC=0.70). All investigated single nucleotide polymorphisms were in Hardy-Weinberg equilibrium, and no significant associations were observed between TERF1 or TERF2 variants and telomere length across any inheritance model.

Conclusion: Accelerated leukocyte telomere shortening appears to reflect biological aging in multiple sclerosis, independent of common TERF1 and TERF2 genetic variants. These findings support the relevance of cellular aging mechanisms in early MS and highlight telomere length as a potential component of multimodal biomarker strategies. Longitudinal and multi-omics studies are warranted to further clarify telomere dynamics in MS.

Disclosure: Nothing to disclose.

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Background and aims: Heterogeneous registry structures limit cross-border real-world evidence generation in multiple sclerosis (MS). The European Medicines Agency calls for standardised, interoperable data approaches to support post-authorisation safety studies and rare outcome detection. We developed and validated a common data model (CDM) within the Big MS Data (BMSD) Network to enable reproducible federated analyses while preserving data sovereignty across registries.

Methods: Six MS registries (Czech, Danish, French, Italian, Swedish, MSBase) collaboratively defined 120 standardised variables covering demographics, disease history, relapses, treatments, comorbidities, disability outcomes, and paraclinical data. Registry-specific datasets were mapped to CDM format using automated transformation scripts with error logging. Validation included transformation success metrics, demographic plausibility checks, and pilot federated logistic regression analyses.

Results: The CDM successfully harmonised 348,371 patient records (ranging from 19,097 to 111,727 patients per registry). Transformation achieved 98–100% information retention across core domains (patients, visits, treatments, medical conditions). Demographic validation confirmed expected distributions:

mean age at MS onset ranged 31.8–35.4 years across registries with approximately 70% female representation. Federated logistic regression demonstrated consistent effect estimates across registries without patient-level data transfer. The framework now supports ongoing European research initiatives investigating treatment patterns and rare adverse events.

Conclusion: This CDM represents a scalable solution for privacy-preserving multi-registry MS research aligned with European regulatory requirements. By enabling federated analyses across six registries, the framework facilitates detection of rare outcomes and treatment patterns undetectable in single-registry studies, with potential applicability to other neurological conditions.

Disclosure: Supported by Ministry of Health of the Czech Republic, grant nr. NW26J-08-00103.

EPO-0295 | High levels of p-STAT1 are associated with lower brain volumes at baseline in patients with early Multiple Sclerosis

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Background and aims: The role of oxidative stress has been suggested in Multiple Sclerosis (MS) pathogenesis. Phosphorylated Signal Transducer and Activator of Transcription 1 (pSTAT1) is a key transcription factor that promotes proinflammatory processes in response to oxidative stress. This study investigates the association of blood levels of pSTAT1 with measures of disease severity in patients with early MS.

Methods: 29 patients aged 18–40 years, recently (≤ 2 years) diagnosed with MS, were consecutively enrolled in an observational study at Verona University Hospital; a brain MRI scan performed between 6 months before and 1 month after inclusion was mandatory. A clinical and MRI follow-up was performed after 10–48 months in a subgroup of 20 of patients. We assessed the association of baseline values of pSTAT1 with clinical variables, global and regional brain volumes and their annualized variations.

Results: At inclusion, patients with medium-to-high compared to low levels of p-STAT1 showed lower median volumes of global brain (1.13×10^7 vs 1.033×10^7 mm³ $p = 0.024$), cerebellar cortex (101.7 vs 112.2 cm³, $p = 0.028$), thalamus (132.4 vs 150 cm³, $p = 0.028$), caudate (60.9 vs 69.5 cm³, $p = 0.007$) and putamen (87.56 vs 94.96 cm³, $p = 0.041$). In the longitudinal study, we didn't find any significant association between pSTAT1 and any of the imaging and clinical variables.

Conclusion: High pSTAT1 expression is associated with low brain volumes in early MS, to be confirmed in larger longitudinal studies.

Disclosure: Nothing to disclose.

EPO-0296 | Fatigue control: 24-month real-world MoOzaRt study Shows 88.8% stabilization and strong correlation with reduced MS burden

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Background and aims: Fatigue is considered the most common and one of the most debilitating symptoms in multiple sclerosis (MS). MoOzaRt assesses the impact of ocrelizumab on patient-reported persistent (trait) and transient (state) fatigue in patients with relapsing MS (RMS).

Methods: The ongoing non-interventional MoOzaRt study (ISRCTN55332718) recruited 272 RMS patients initiating ocrelizumab. The primary combined endpoint is a clinically meaningful reduction (≥ 9 points) or stabilization (± 8 points) from baseline in trait fatigue, measured by the Fatigue Scale for Motor and Cognitive Functions (FSMC) total score over 24 months. Secondary endpoints include state fatigue (Visual Analogue Scale (VAS)), Expanded Disability Status Scale (EDSS), Patient Reported Outcomes (PROs) and safety.

Results: The third interim analysis (cut-off Dec 10, 2025, snapshot) included 261 patients (Table 1 67.4% female, mean age 38.3 years, mean EDSS 2.43). Over the 24-month observation period, 88.8% of patients showed stable or reduced FSMC total scores, of which 30.1% achieved a clinically meaningful improvement. Treatment-naïve patients exhibited the highest proportion of fatigue stabilization or improvement (90.4%). Lower fatigue scores strongly correlated with reduced physical and psychological disease burden (MSIS-29, WAPI:MS, PDQ-20, HADS).

Conclusion: This analysis confirms that ocrelizumab treatment is associated with stable or even improved fatigue in a very high proportion of RMS patients, particularly in treatment-naïve patients (90.4%). Given the strong correlation between reduced fatigue scores and reduced disease burden, these findings suggest that early high-efficacy therapy (HET) is a valuable strategy for relieving and preventing disease burden.

Disclosure: IKP: Almirall, Biogen, BMS, Celgene, Genzyme, Janssen, Merck, Novartis, Roche, Teva//speakers bureau or advisory board, consulting fees; The German MS Society, Celgene, Novartis, Roche, Teva//research grants. TW: Roche//employee HS: Almirall, Biogen, BMS, Genzyme, Janssen, Merck, Novartis, Roche, Sanofi, Teva//speakers bureau or advisory board, consulting fees, travel reimbursement; Biogen, Novartis,

TABLE 1

Baseline characteristics	Total (N=261)
Sex, female, N (%)	176 (67.4)
Age at baseline [years], mean (SD)	38.3 (10.6)
Any medication history, N (%)	164 (62.8)
EDSS score, mean (SD)	2.43 (1.52)
Time since first MS symptoms [years], mean (SD)	8.86 (8.75)
Time since first MS diagnosis [years], mean (SD)	7.37 (8.88)
Previous treatments and therapies, N (%)	185 (70.9)

EPO-0297 | Diagnostic guidelines for identifying and monitoring cognitive impairment in multiple sclerosis: An IMSCOGS and ECTRIMS Delphi consensus statement

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Background and aims: CI occurs frequently in MS, is a common cause of loss of employment and can have a significant impact on quality of life. The aim was to develop international guidelines to assist in the identification of cognitive impairment (CI) among adult persons with MS (PwMS) for both clinical and research applications. The three topics for these guidelines were: 1) cognitive screening, 2) cognitive measurement and diagnosis and 3) monitoring and diagnosing change over time.

Methods: The International Multiple Sclerosis Cognition Society (IMSCOGS) and European Committee for Treatment and Research in MS (ECTRIMS) partnered with Cochrane Multiple Sclerosis and Rare Diseases to conduct a scoping literature review to support the formation of guidelines. Based on this review, statements were formulated. Next, a modified Delphi approach was used to establish consensus via multiple virtual meetings and rounds of anonymous voting by a large group of clinical and research experts in this area with global representation. A minimum response and consensus of 80% was required for a statement to be accepted.

Results: Our scoping review provided an evidence gap map, identifying key inconsistencies in the assessment of CI in PwMS. Delphi voting resulted in a clear set of criteria and guidelines. Results on aims 1, 2 and 3: Cognitive screening; cognitive measurement and diagnosis; monitoring/diagnosing change over time will be presented.

Conclusion: These guidelines permit reliable diagnosis of CI and cognitive decline in MS, allowing generalizability of research findings in future studies of CI in MS and consistency across research and clinical settings.

Disclosure: IP, SM, LH, DL, MPA and MS are members of the board/steering committee of IMSCOGS. This project was financially supported by ECTRIMS.

EPO-0298 | Modifications of time-varying thalamic connectivity underpin fatigue development in multiple sclerosis

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Background and aims: Fatigue is common in multiple sclerosis (MS) and occurs independently from disease stages. Resting state (RS) functional connectivity (FC) studies showed an implication of thalamic circuits dysfunction in MS-related fatigue pathogenesis. Here, we investigated changes over time of thalamic time-varying (TV) FC and its association with fatigue development in MS.

Methods: Ninety-two right-handed MS patients and 55 healthy controls (HC) underwent clinical and 3.0T RS fMRI assessment at baseline and after 1.27-year median follow-up. The modified fatigue impact scale (MFIS) assessed fatigue at both visits; patients were considered as fatigued (F) if MFIS was >38. TVFC of left/right thalamus was analyzed using sliding-window seed-voxel correlation.

Results: At baseline, 26 patients (28%) were F and 66 not fatigued (NF). Of these, 23 (35%) developed fatigue (devF) at follow-up and 43 remained NF. At baseline, F-MS patients exhibited increased TVFC ($p < 0.001$, conjunction analysis) between the left thalamus and hippocampus, and between the right thalamus, right insula, and bilateral frontal regions. Conversely, NF-MS patients showed reduced baseline thalamic TVFC ($p < 0.001$, conjunction analysis) with temporal, occipital and cingulate regions. At follow-up, NF-MS patients showed increased thalamic TVFC with parietal, temporal and occipital regions. Conversely, devF-MS patients showed increased TVFC over time ($p < 0.001$, time-by-group interaction) between the right thalamus and bilateral cingulate, left insular, left superior frontal and left inferior temporal regions. Finally, F-MS patients presented decreased TVFC over time ($p < 0.001$, time-by-group interaction) between right thalamus and left insula.

Conclusion: Distinct patterns of thalamic TVFC changes characterized MS patients based on their fatigue status.

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Sanofi. She receives research support from the MS Society of Canada, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. She is Associate Editor for Multiple Sclerosis and Related Disorders; and Associate Co-Editor for Europe and Africa for Multiple Sclerosis Journal. M. Filippi is Editor-in-Chief of the Journal of Neurology, Associate Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmaceuti Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla.

EPO-0299 | MRI-derived subtypes identified by sustain capture distinct clinical, cognitive, and disability profiles in Multiple Sclerosis

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Background and aims: Clinical multiple sclerosis (MS) classifications incompletely capture disease heterogeneity. Data-driven subtyping may better reflect MS pathobiology and clinical variability. We applied SuStaIn to identify MRI-based MS subtypes.

Methods: We retrospectively analyzed 1017 MS patients and 548 healthy controls with standardized clinical, neuropsychological, and brain 3T MRI assessments. Z-scores of 21 MRI features, including white matter (WM) lesion volume major WM tract microstructural abnormalities, and global and regional normalized brain volumes, were input into SuStaIn to derive subtypes and stages. Associations with clinical phenotype (relapsing-remitting [RR] vs progressive), age at onset (AAO) (pediatric [POMS], adult [AOMS], late [LOMS]), disability

(Expanded Disability Status Scale [EDSS]), and cognition (Brief Repeatable Battery of Neuropsychological Tests) were explored.

Results: SuStaIn identified four MRI subtypes: lesion-led (44%), cortex-led (23%), tract-led (23%), and deepGM-led (10%). Subtypes differed by clinical phenotype and AAO ($p < 0.001$). Cortex-led and deepGM-led were enriched in progressive forms (47% and 56%); lesion-led and tract-led were mainly RRMS (66–71%). Tract-led was overrepresented in POMS (38%) and underrepresented in AOMS (19%), lesion-led predominated in AOMS (48%), whereas deepGM-led was more frequent in LOMS (14%). Disability milestones were reached more often in cortex-led and deepGM-led patients (EDSS ≥ 4.0 : 45% and 56%; EDSS ≥ 6.0 : 28% and 29%) than in lesion-led (34% and 20%) or tract-led (32% and 19%) ($p < 0.001$). Cognitive performance did not differ across subtypes, but within each subtype higher SuStaIn stage correlated with worse global and domain-specific performance ($p \leq 0.030$).

Conclusion: SuStaIn-derived MRI subtypes capture clinically relevant heterogeneity in MS and may support personalized therapeutic strategies.

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Background and aims: The long-term prognostic value of gray matter (GM) damage in multiple sclerosis (MS) is unclear; we examined MRI markers of GM and white matter (WM) damage associated with long-term outcomes in MS.

Methods: Seventy-three relapse-onset MS patients underwent clinical and brain MRI at baseline and 12 months, including WM lesion volumes, brain, GM, WM, and thalamic volume fractions, and GM, thalamic, normal-appearing (NA) WM, and WM lesions MTR. High-efficacy disease-modifying therapies (HE-DMTs) escalation was recorded. Long-term associations with Expanded Disability Status Scale (EDSS) worsening, evolution to a more severe disease stage, MS-related death, and cognitive deterioration were analyzed using LASSO-regularized logistic regression with bootstrap validation.

Results: Over a median 25.9-year follow-up, 79.5% patients experienced EDSS worsening, 72.6% evolved to a more severe disease stage, 24.7% died from MS, and 39.5% exhibited cognitive deterioration. EDSS worsening was associated with lower baseline GM and thalamic fractions, HE-DMT escalation, lower baseline NAWM MTR peak height, and higher 12-month decline in GM MTR histogram peak height (AUC=0.843). Evolution to a more severe disease stage was associated with higher 12-month EDSS score change and lower baseline GM fraction (AUC=0.793). MS-related death was associated with male sex, higher baseline EDSS score, lower baseline GMF, lower baseline GM MTR peak height, and greater 12-month thalamic MTR decline (AUC=0.887). Cognitive deterioration was associated with older age, lower baseline brain parenchymal fraction, and lower baseline mean GM MTR (AUC=0.891).

Conclusion: GM damage is associated with worse 26-year clinical outcomes in relapse-onset MS, supporting the relevance of GM-focused MRI markers.

Disclosure: P. Preziosa received speaker honoraria from Roche, Biogen, Novartis, Merck, Bristol Myers Squibb, Genzyme, Horizon and Sanofi. A. Meani has nothing to disclose. N. Tedone has nothing to disclose. I. Gattuso has nothing to disclose. M. Rovaris received speaker honoraria from Merck and Novartis, he has received research support from Italian Ministry of University and Research and Fondazione Italiana Sclerosi Multipla (FISM). M.A. Rocca received consulting fees from Biogen, Bristol Myers Squibb, Roche; and speaker

honoraria from Alexion, Biogen, Bristol Myers Squibb, Celgene, Horizon Therapeutics Italy, Merck Serono SpA, Mitsubishi-Tanabe Pharma, Neuraxpharm, Novartis, Roche, Sandoz, and Sanofi. She receives research support from the MS Society of Canada, the Italian Ministry of Health, the Italian Ministry of University and Research, and FISM. She is Associate Editor for Multiple Sclerosis and Related Disorders; and Associate Co-Editor for Europe and Africa for Multiple Sclerosis Journal. M. Filippi is Editor-in-Chief of the Journal of Neurology, Associate Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmed Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and FISM.

Muscle and Neuromuscular Junction Disorder 2

EPO-0301 | Comparative efficacy of anti-C5 vs. anti-FcRn therapy in thymoma-associated myasthenia gravis: A multicentre retrospective study

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Background and aims: Thymoma-associated Myasthenia Gravis (TAMG) represents a challenging clinical phenotype. While anti-complement therapy (C5IT) and neonatal-Fc-receptor inhibitors (aFcRn) have expanded the therapeutic landscape, comparative data regarding their efficacy in TAMG

[illegible]

Methods: We conducted a retrospective study of 93 AChR-positive TAMG patients (58 C5IT maintenance; 35 aFcRn cycles) across eight Italian centers. The primary endpoint included longitudinal changes in MG-ADL, QMG, daily CS dosage over 12 months (Table 1). LMM was performed to assess treatment-by-time interactions. aFcRn patients were stratified into “short responders” (mean cycles-intervals <8 weeks) and “long-responders” (mean cycles-intervals $\geq$ 8 weeks).

Conclusion: In TAMG patients, chronic C5IT maintenance regimen provides a greater overall clinical improvement and daily corticosteroids compared to aFcRn cycle-based treatment schedule in TAMG patients. aFcRn patients requiring frequent cycling (<8 weeks) had suboptimal responses, whereas those with sustained durability mirrored C5IT efficacy. These findings highlight the necessity of predictive factors to identify potential “short responders” and optimize personalized treatment selection and administration schedules.

or for advisory boards from: Alfa Sigma, Angelini, Argenx, Amgen, Alnylam, Alexion, CLS Behring, Lusofarmaco, Johnson & Johnson, Merck, Roche, and Takeda. Raffaele Iorio has received consultancy fees and speaker honoraria from Alexion, Amgen, Argenx, Dianthus Therapeutics, Jonhson&Johnson, Merck, UCB. Silvia Falso has received consultancy fees and speaker honoraria from Alexion, Argenx, UCB. Sofia Marini has received consultancy fees and speaker honoraria from Alexion, Argenx, UCB. Michelangelo Maestri Tassoni has received personal fees from Alexion, UCB, Amgen and J&J. Matteo Gastaldi has received public speaking honoraria for Alexion, UCB, J&J. Alba Cepele has received personal fees from UCB. Melania Guida has received public speaking honoraria, advisory board and fees from Alexion, Argnx, UCB, Amgen, J&J.

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Methods: Peripheral blood mononuclear cells were isolated from MuSK-MG patients ($n=36$), acetylcholine receptor myasthenia gravis patients as disease controls ($n=7$), and age- and sex-matched healthy controls ($n=12$). Genomic DNA was extracted and after sodium bisulfite conversion, DNA methylation of the promoter regions of interleukin (IL)-4, IL-10, and IL-13 was assessed using methylation-sensitive high-resolution melting (MS-HRM) analysis. Methylation levels were estimated by comparing the melting profiles of subjects' DNA samples with those obtained from DNA standards with known methylation percentages.

Conclusion: Selective hypomethylation of IL-4 and IL-13 promoters is observed in MuSK-MG, while IL-10 promoter methylation remains unaltered. This potentially facilitates increased cytokine expression, potentially favoring IgG4 subclass switching, although additional regulatory mechanisms are likely involved.

Disclosure: This project is conducted within the IgG4-TREAT consortium, a Marie Skłodowska-Curie Actions doctoral

EPO-0303 | Clinical features and outcomes of very late-onset myasthenia gravis: Insights from a greek center of reference

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Background and aims: The demographic profile of myasthenia gravis (MG) has shifted in recent decades, with increasing prevalence among older individuals. This study investigates the clinical characteristics and outcomes of patients with very late-onset MG (VLOMG), defined as disease onset at ≥ 65 years.

Methods: We retrospectively reviewed patients with VLOMG registered in the Specialized Myasthenia Clinic database between Jan-2008 and Dec-2025.

Results: A total of 102 patients were included (72 males, 30 females), with a mean follow-up of 47.86 months. Mean age at diagnosis was 73.88 ± 6.65 years (range: 65–91), with a mean diagnostic delay of 5.1 months. Thymoma was identified in 3.92%, autoimmune disease in 11.76%, malignancies in 17.65%, and other comorbidities in 91.18%. Overall, 99% were seropositive; 94.11% AChR-Abs(+) and 4.9% MuSK-Abs(+). AChR-Abs titers ranged from 0.8 to 433 nM/L (mean 49.07 ± 76.28 nM/L). Generalized MG was present in 80.39%, most commonly with oculo-bulbar involvement. Rescue therapies were required in 63.72%; 12.74% required intensive care admission and 5.88% mechanical ventilation. Long-term immunosuppression was used in 93.13%. Advanced immunotherapies included rituximab (17.65%), FcRn antagonists (6.86%), ravulizumab (1.96%), and tocilizumab in one patient with concomitant thyroid eye disease. At last follow-up, 68.62% achieved minimal manifestations or better and 19.6% showed clinical improvement, on a mean daily prednisolone dose of 6.75 mg.

Conclusion: VLOMG represents a distinct MG subgroup characterized by male predominance, predominant bulbar phenotype, rare thymoma and high AChR positivity. Despite a significant comorbidity burden, early immunosuppression enables effective disease control with low maintenance corticosteroid doses. Novel and targeted immunotherapies may be safe and effective in elderly patients.

Disclosure: Nothing to disclose.

EPO-0304 | Electrophysiological assessment of motor unit loss in adult spinal muscular atrophy Types III/IV: A national study comparing MUNIX, CMAP, and MUSIX

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Background and aims: Spinal muscular atrophy (SMA) types III–IV are the most common late-onset forms and progress slowly, making sensitive biomarkers essential. The Motor Unit

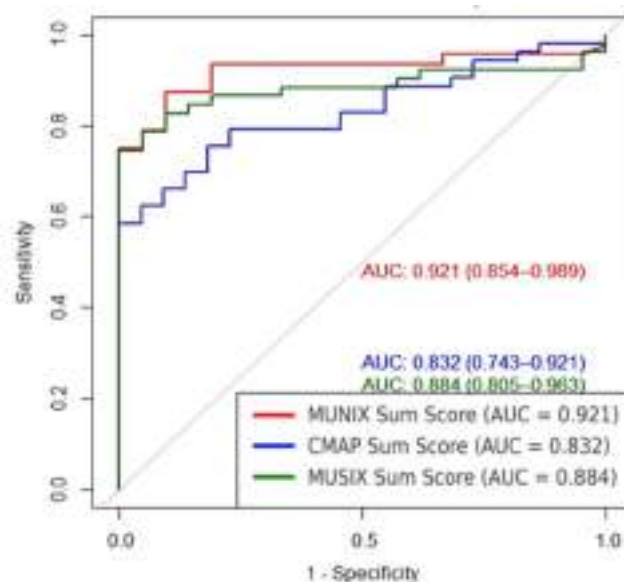


FIGURE 1 ROC curves of Sum Scores (MUNIX, CMAP, MUSIX) discriminating patients with SMA from healthy controls.

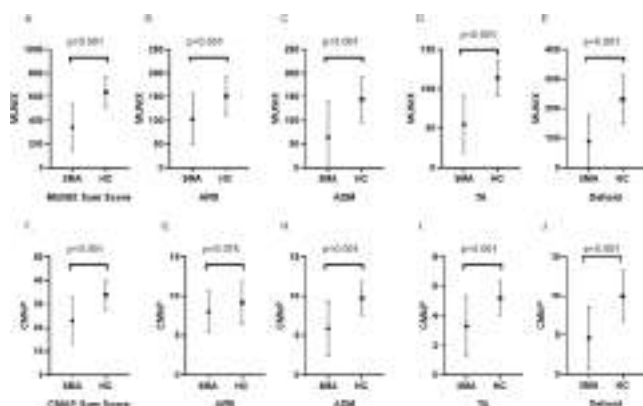


FIGURE 2 MUNIX and CMAP differences between patients with SMA and healthy volunteers (HC). Upper row shows MUNIX differences and lower row CMAP differences; for MUNIX Sum Scores, CMAP Sum Scores, and MUNIX and CMAP individual muscle scores.

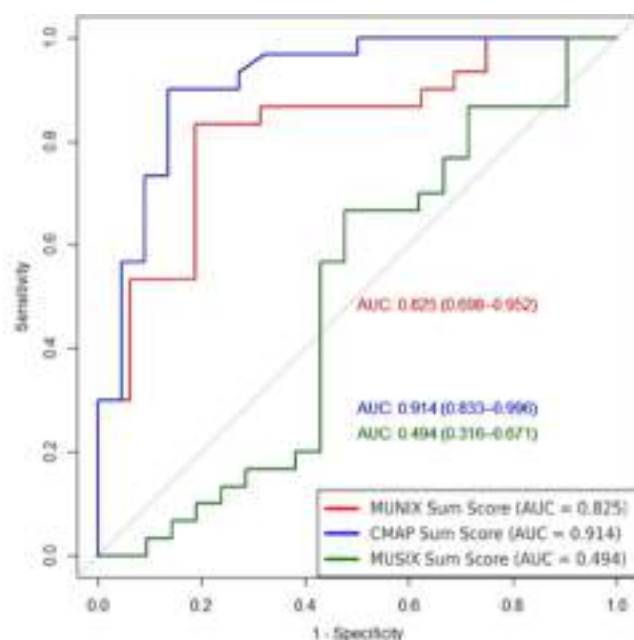


FIGURE 3 ROC curves of MUNIX, CMAP, and MUSIX Sum Scores discriminative performances between ambulant and non-ambulant patients with SMA.

Number Index (MUNIX) estimates motor unit loss and may complement traditional electrophysiological measures such as Compound Muscle Action Potential amplitudes (CMAP). However, their respective performances have not been directly compared in adult SMA.

Methods: In a French multicentric study (NCT04690998), 71 adults with SMA and 24 healthy controls underwent clinical and electrophysiological assessment. MUNIX, CMAP, and Motor Unit Size Index (MUSIX) were recorded in four muscles, and sum scores (SumMUNIX, SumCMAP, SumMUSIX) were computed. Test-retest reliability was evaluated using intraclass correlation coefficients (ICCs). Associations with functional outcomes and strength measures were analysed, including multivariate models.

Results: MUNIX, MUSIX and CMAP effectively distinguished SMA patients from controls. MUNIX showed the highest discriminative performance (AUC=0.92), while CMAP demonstrated the strongest and most consistent associations with clinical severity. In multivariate analyses, only CMAP remained independently associated with all functional and strength measures, whereas MUNIX and MUSIX lost significance. CMAP showed the strongest ability to discriminate between ambulant and non-ambulant SMA patients.

Conclusion: MUNIX was the most discriminative biomarker for differentiating SMA from controls, detecting motor unit loss even when CMAP remained within normal limits. CMAP better reflected overall disease burden, likely because it integrates both motor unit loss and reinnervation. These complementary profiles support the concurrent use of MUNIX and CMAP in adult SMA. Longitudinal studies are needed to determine their responsiveness and suitability as endpoints in future clinical trials.

Disclosure: This study was supported by Biogen. The funder had no role in the design, data collection, analysis, interpretation of data, or in the writing of the manuscript.

EPO-0305 | Assessment of sustained health-related quality of life in Phase 3 Vivacity-MG3 Trial of Nipocalimab versus Placebo in Generalized Myasthenia Gravis

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Background and aims: Generalized myasthenia gravis (gMG), an autoantibody-mediated disease causes fluctuating muscle symptoms and substantially impairs health-related quality-of-life (HRQoL). In Vivacity-MG3 (NCT04951622), nipocalimab+standard-of-care (SOC) demonstrated improved and sustained disease control versus placebo+SOC. HRQoL changes during the 24-week (W) double blind phase were evaluated using patient reported MGQoL-15r and EQ-5D visual analogue scale (EQ-5D-VAS).

Methods: Mean change-from-baseline (CFB) between groups in MGQoL-15r and EQ-5D-VAS over 24W were compared using ANCOVA models. Meaningful-within-person-improvement (MWPI) response was a change of one-half a standard deviation on MGQoL-15r (> = 4-points) and EQ-5D-VAS (> = 10-points). Proportion of patients achieving MWPI and time-to-MWPI over 24W were examined using Chi-square test and Kaplan-Meier analyses. Logistic regression evaluated likelihood of achieving MWPI over 24W and sustaining MWPI for > = 8, 12, 16, and 20W between groups.

Results: LS-mean (95% CI) difference in CFB on MGQoL-15r and EQ-5D-VAS was greater with nipocalimab+SOC versus placebo+SOC (-1.38 [-3.14, 0.37], not significant [ns] versus 6.86

[2.07, 11.66], $p < 0.05$, respectively) at 4W and sustained (−1.61 [−3.55, 0.33], ns; 6.47 [1.05, 11.88], $p < 0.05$) at 24W. At 24W, 67.2% (45/67) versus 56.5% (35/62) achieved MWPI on MGQoL-15r (absolute difference=10.7%[ns]) and 55.2% (37/67) versus 37.1% (23/62) patients achieved MWPI on EQ-5D-VAS (absolute difference=18.1%, [$p < 0.05$]) in nipocalimab+SOC versus placebo+SOC. For both measures, nipocalimab+SOC patients were twice more likely to achieve MWPI over 24W with median time-to-response approximately 4W versus 8W with placebo+SOC ($p < 0.05$). Similarly, nipocalimab+SOC patients were approximately twice more likely to sustain MWPI for $> 8, 12, 16, 20W$ ($p < 0.05$).

Conclusion: Nipocalimab+SOC-treated patients achieved meaningful HRQoL improvements as early as 4W and were significantly more likely to achieve and sustain these improvements over 24W versus placebo+SOC.

Disclosure: This work was funded by Johnson & Johnson. John Vissing: Consultant on advisory boards or received speaker honoraria related to myasthenia gravis from Roche, Regeneron, Toleranzia, Hansa Biopharma, argenX BVBA, UCB Biopharma SPRL, Amgen, Dianthus Therapeutics, NMD Pharma, Alexion Pharmaceuticals, Novartis Pharma AG, Johnson & Johnson and is principal investigator on trials in myasthenia gravis for Roche, argenX BVBA, Toleranzia, Hansa Biopharma, Novartis Pharma AG, Alexion Pharmaceuticals, UCB Biopharma SPRL, Regeneron, Johnson & Johnson, Dianthus Therapeutics. Kavita Gandhi, Sheryl Pease, Ibrahim Turkoz, Marie Fitzgibbon, Wisam Karmous, Nolan Campbell, and Sindhu Ramchandren: Employees of Johnson & Johnson may hold stocks/stock options in Johnson & Johnson. Geoffroy Coteur: Contractor for Johnson and Johnson. Elena Cortés-Vicente: Received consulting/advisory from argenX BV, Alexion Pharmaceuticals Inc, Janssen Pharmaceuticals Inc, UCB Pharma SA.

EPO-0306 | Safety and effectiveness of efgartigimod in Japanese patients with generalized myasthenia gravis by serostatus: Post-marketing surveillance analysis

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Background and aims: Efgartigimod for intravenous infusion (efgartigimod-IV) is approved in Japan for generalized myasthenia gravis (gMG) regardless of auto-antibody profiles. Post-marketing surveillance for efgartigimod-IV in gMG was performed to assess the safety and effectiveness in real-world settings in Japan.

Methods: Patients with gMG who received efgartigimod-IV between May 2022 and September 2023 were registered. Effectiveness was evaluated by Myasthenia Gravis-Activities of Daily Living (MG-ADL) total and subdomain (ocular, bulbar, limb/gross motor, respiratory) scores. The change of oral glucocorticoid dose was also evaluated.

Results: The safety analysis set consisted of 469 patients: 57.1% ($n=268$) anti-acetylcholine receptor antibody positive (AChR-Ab+), 13.4% ($n=63$) anti-muscle-specific receptor kinase antibody positive (MuSK-Ab+), and 29.4% ($n=138$) double-seronegative. Adverse drug reaction and serious adverse drug reaction were reported in 22.6% and 4.2% of patients,

respectively. No specific safety signals were observed in any of the serological profiles. The effectiveness analysis set consisted of 307 patients with a similar composition ratio of serological profile to the safety analysis set. After three weeks from the first administration, mean MG-ADL total score improved from 7.2 to 4.2: −3.0 points (standard deviation: 2.98, $p < 0.001$). Substantial improvements were observed in all subdomain scores regardless of serological profiles. Mean dose of oral glucocorticoid (standard deviation) (mg/day) at baseline and one year later were 10.7 (7.42) and 7.7 (5.41), respectively.

Conclusion: In real-world settings, efgartigimod-IV was well tolerated and effective in patients with gMG regardless of serological profiles. Oral glucocorticoid dose reduction was observed after efgartigimod administration.

Disclosure: This surveillance was funded by argenx Japan K.K. The writing support was also funded by argenx Japan K.K. Hirofumi Teranishi, Ryohei Aoyagi, and Daisuke Harada are fulltime employees of argenx Japan K.K. Mitsuru Watanabe has received speakers' honoraria, manuscript fee, or research support from argenx Japan K.K, Alexion Pharma, Novartis Pharma, UCB Japan, Johnson&Johnson, Mitsubishi Tanabe Pharma, Chugai Pharmaceutical, Biogen Japan, Viatris, Daiichi Sankyo, and Takeda Pharmaceutical.

EPO-0307 | Efficacy and tolerability of nipocalimab in patients with generalized myasthenia gravis: Real-world experience from an Academic Center

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Background and aims: Nipocalimab, a neonatal Fc receptor (FcRn) inhibitor, was approved in April 2025 for treatment of antibody positive generalized myasthenia gravis (gMG). However, data from clinical practice is limited. Here we describe a single academic institution's experience with nipocalimab.

Methods: A retrospective review of 11 gMG patients who received nipocalimab between May–November 2025 was conducted. Data was collected regarding demographics, comorbidities, antibody status, treatment history, Myasthenia Gravis Activities of Daily Living (MG-ADL) scores, side effects, and corticosteroid dose.

Results: Most patients were antibody positive ($n=9$ acetylcholine receptor, $n=1$ muscle-specific tyrosine kinase, $n=1$ seronegative). Mean baseline MG-ADL was 8 (± 3.98). In all patients, nipocalimab was started due to refractory symptoms and/or intolerable side effects with prior treatments. MG-ADL scores improved by a mean of 1.9 points ($p=0.017$), and 63.6% (7/11) of patients had a clinically significant improvement (reduction ≥ 2 in MG-ADL score) at follow up 10.6 (± 4.7) weeks after treatment initiation. Nine patients previously failed one or more FcRn inhibitors ($n=6$ efgartigimod, $n=1$ rozanolixizumab, $n=2$ both) due to inefficacy ($n=2$) or wearing off between cycles ($n=7$). 77.8% (7/9) tolerated nipocalimab, and the reduction in MG-ADL score remained statistically significant ($p=0.034$). Only 1 patient reported wearing off. 100% (7/7) of patients on corticosteroids successfully lowered or discontinued dosage without symptom worsening.

Conclusion: We found nipocalimab to be effective, fast-acting, and well tolerated in patients with refractory gMG, leading to modest clinical improvement and steroid tapering in most patients. Nipocalimab was effective in patients who had previously failed other FcRn inhibitors and wearing off between doses was uncommon.

Disclosure: Dr. Greenberg has no disclosures. Dr. Seyam has no disclosures. Dr. Muppidi has served on the advisory board for Alexion, UCB, Johnson & Johnson, Amgen, Kyverna, and Arcellex. Dr. Goyal has served as a consultant for Argenx, Alexion, UCB, Janssen, Amgen, Novartis, Immunovant, Dianthus, Annexon, Seismic, Cartesian, EMD Serono, and has grant funding from Argenx.

EPO-0308 | Progression and exacerbation timing in myasthenia gravis: Real-world evidence from France (RELIEF study)

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Background and aims: Understanding the timing of progression and acute events in myasthenia gravis (MG) is critical for optimizing care. This study aimed to estimate the time-to-progression from MG to its generalized (gMG) and refractory forms, and to assess the time-to-first exacerbation or crisis.

Methods: Adults with incident MG between 2014 and 2022 were identified in the French health claims database (SNDS) using algorithms based on ICD-10 codes and treatment patterns, including first- and second-line maintenance therapies and crisis treatments (IV immunoglobulins or plasma exchange). Patients were classified as MG, gMG, or refractory gMG accordingly. Exacerbations were defined as first acute immunoglobulin/plasma exchange, hospitalization for MG or MG crisis; crises

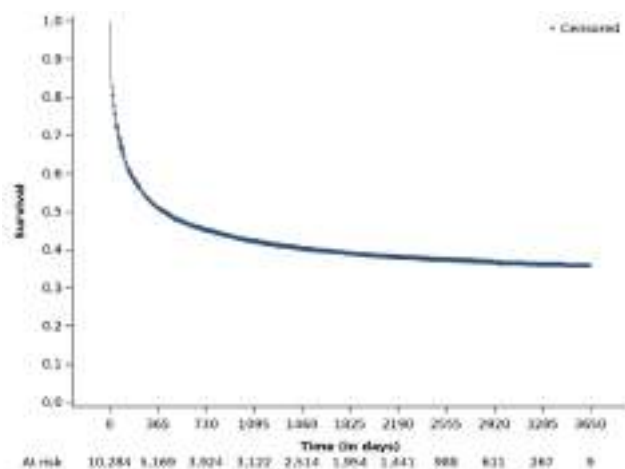


FIGURE 1 Kaplan Meier curve for the occurrence of gMG in the incident population of MG adults with index date between 2014 and 2022 and at least one year of FU available (except in case of death).

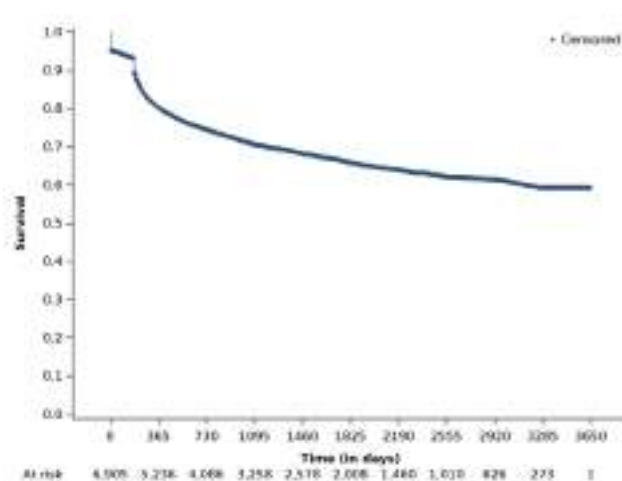


FIGURE 2 Kaplan Meier curve for the occurrence of refractory gMG in the incident population of gMG adults with first diagnosis of gMG between 2014 and 2022 and at least one year of FU available (except in case of death).

included hospitalization with respiratory failure, intubation, ventilation, or enteral feeding. Kaplan-Meier analyses estimated progression and first exacerbation/crisis event timing.

Results: Among 10,284 MG patients (49.3% males, mean age: 61.3 ± 18.2) the probability of gMG diagnosis was 49.0% within 12 months and 60.9% within 60 months (MST: 413 days [369; 459]) (see Figure 1). Of 6,905 gMG patients, the probability of refractory disease was 19.8% at 12 months and 34.1% at 60 months (see Figure 2). In MG patients, probability of exacerbation was 55.1% [54.1; 56.1] at 12 months and 63.5% [62.5; 64.5] at 60 months (probability of crisis: 6.1% [5.6; 6.6] and 10.0% [9.4; 10.7], respectively).

Conclusion: These real-world findings demonstrate rapid progression to gMG in majority of patients, with a significant proportion of these developing refractory disease. Most also experience exacerbation (including crisis) within one year of diagnosis, underscoring the need for early and sustained disease control.

Disclosure: Thierry Gendre has received consultancy fees from J&J, UCB, and Amgen; payments for oral communications from Argenx and CSL Behring; supports for congress from Alexion. Didier Theis, and Jean-Baptiste Noury have received consultancy fees from J&J. Erika Guyot, Maeva Nolin, and Manon Belhassen are employees of Epimentis (PELyon). Ingrid Rodriguez, Julia Meijer, Laurene Gautier, and Julien Dupin are employees of J&J Innovative Medicine. This study was funded by J&J Innovative Medicine.

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Background and aims: Acute events drive the short-term burden in myasthenia gravis (MG). This study evaluated the one-year risk of MG crises (MGc) and exacerbations (MGe) after diagnosis of MG and its generalized form (gMG), and characterized the nature of these acute episodes.

Methods: This French health claims database (SNDS) study included patients diagnosed with MG or gMG in 2022 using algorithms based on ICD-10 codes and treatment dispensing, including first- and second-line maintenance therapies and crisis treatments (IVIg or plasma exchange). MGc were defined as hospitalization with respiratory failure, intubation, mechanical ventilation, or enteral feeding. MGe included MGc, acute IVIg/plasma exchange, or MG hospitalization. Analyses reported patients with ≥ 1 event within one year, incidence rates per 100 patient-years (100PY) [95% CI], and event components.

Results: There were 1,197 patients with MG diagnosed in 2022 (50.7% males, age: 62.4 ± 18.0), and 859 patients with gMG. MGc occurred in 76 MG (6.3%) and 89 gMG (10.4%) patients. MGe affected 685 MG (57.2%) and 619 gMG (72.1%) patients. In MG, the incidence rates were 6.8/100PY [5.4;8.5] for MGc and 117.7/100PY [109.2;126.8] for MGe, while they were 11.7/100PY [9.5;14.5] and 236.3/100PY [218.4;255.6], respectively in gMG. Of MG patients with MGc, 81.6% had mechanical ventilation, 46.1% intubation, and 44.7% respiratory failure.

Conclusion: MGe are frequent in the first year following the diagnosis, particularly in gMG patients. MGc is a life-threatening acute event whose incidence is not negligible in the first year. These results highlight the importance of early prevention and prompt therapeutic intervention in gMG patients.

Disclosure: Jean-Baptiste Noury and Didier Theis have received consultancy fees from J&J. Thierry Gendre has received consultancy fees from J&J, UCB, and Amgen; payments for oral communications from Argencx and CSL Behring; supports for congress from Alexion. Erika Guyot, Maeva Nolin, and Manon Belhassen are employees of Epimentis (PELyon). Ingrid Rodriguez, Julia Meijer, Laurène Gautier, and Julien Dupin are employees of J&J Innovative Medicine. This study was funded by J&J Innovative Medicine.

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Background and aims: This post hoc analysis aimed to evaluate the domain-specific clinical efficacy of telitacicept and to explore its differential effect in the clinically relevant subgroup of patients with a history of thymoma.

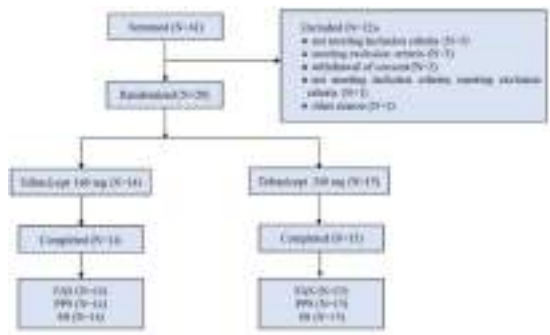


FIGURE 1 Consort flow diagram. Summary of patients screened and randomized for the two telitacicept treatment arms. FAS, full analysis set; PPS, per protocol set; SS, safety set.

TABLE 1 Demographics and clinical characteristics of the patients at baseline.

	Telitacicept 160 mg (N=14)	Telitacicept 240 mg (N=6)	All patients (N=20)
Age, yr., mean (SD)	42.6 (11.78)	45.5 (10.60)	44.1 (12.18)
Sex, n(%)			
Male	6 (42.9)	7 (46.7)	13 (64.8)
Female	8 (57.1)	8 (53.3)	16 (55.2)
Duration of MG, Mon	50.1 (50.323)	73.56 (90.068)	62.24 (71.338)
QMG score ¹ , mean (SD)	18.4 (6.01)	18.3 (4.43)	18.9 (5.17)
MG clinical absolute score ² , mean (SD)	23.1 (8.47)	24.8 (8.48)	24.0 (8.88)
MGFA clinical classification			
Class II, n (%)	1(9.0)	6(60.0)	16(55.2)
Class II, n (%)	1(9.0)	6(40.0)	13(46.8)
ACHR-Ab			
Negative, n (%)	0(0)	0(0)	0(0)
Positive, n (%)	14(100.0)	15(100.0)	29(100.0)
Any concomitant MG medication, n (%)	14(100.0)	15(100.0)	-
Cholinesterase inhibitor			
Pyridostigmine bromide, n (%)	14(100.0)	15(100.0)	29 (100.0)
Stigmid	8 (57.1)	8 (53.3)	16 (55.2)
Immunosuppressant, n (%)	4 (28.6)	8 (53.3)	12 (61.6)
History of thymoma	9 (64.3)	5 (33.3)	14 (48.3)

Abbreviations: QMG-quantitative myasthenia gravis; MGFA-myasthenia gravis foundation of America.

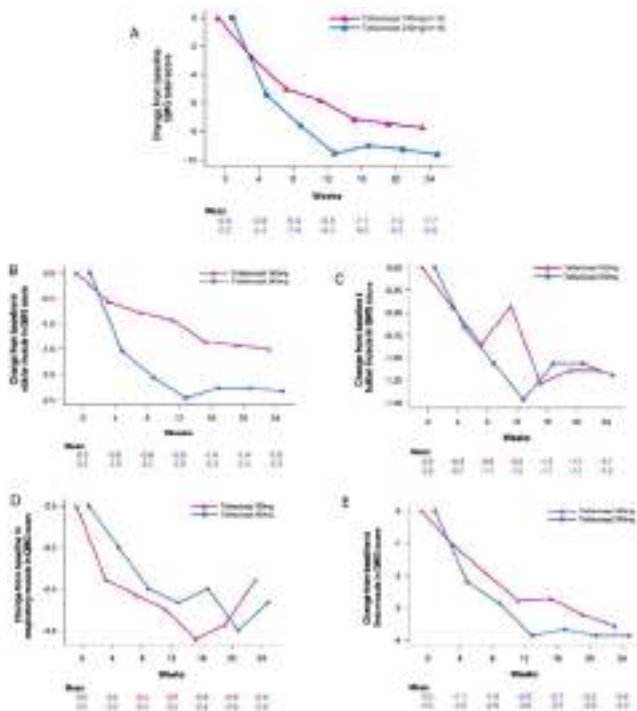


Figure 2 Changes in (A) QMG mean total score from baseline to week 24. Changes in QMG domain scores from baseline to week 24 for (B) ocular, (C) bulbar, (D) respiratory, and (E) limb domains.

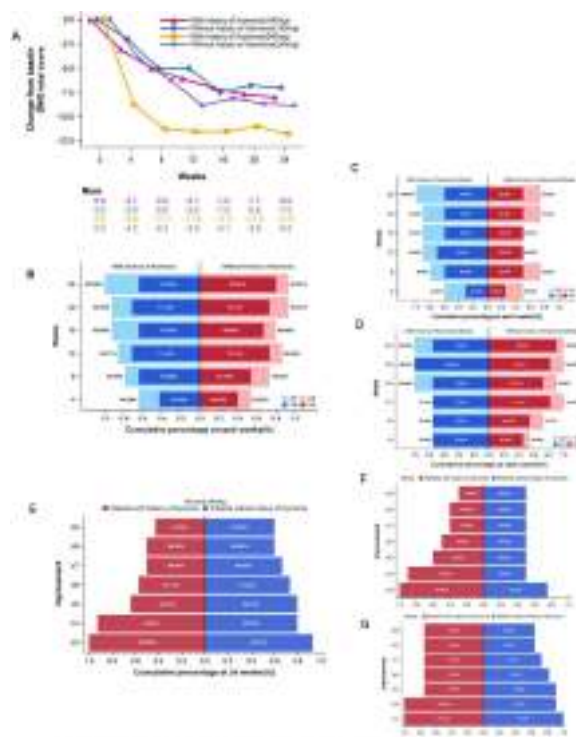


Figure 3 Changes in (A) QMG mean total score from baseline to week 24 in all patients with generalised myasthenia gravis and in subgroups of patients with and without a history of thymoma. QMG response rate in (B) all patients, (C) patients treated with 160mg Telitacicept and (D) patients treated with 240mg Telitacicept from baseline to week 24. Improvement in QMG mean total score at week 24 in (E) all patients, (F) patients treated with 160mg Telitacicept and (G) patients treated with 240mg Telitacicept.

Methods: Eligible patients from a phase 2 trial (NCT04302103) were randomized to receive weekly subcutaneous telitacicept at 160 mg ($n=14$) or 240 mg ($n=15$). Key changes from baseline

to Week 24 in the total Quantitative Myasthenia Gravis (QMG) score and its bulbar, ocular, limb, and respiratory subdomain scores were analyzed. Outcomes were also assessed based on thymoma history.

Results: Both doses induced early (from Week 4) and sustained improvements across all QMG domains. The 240-mg dose resulted in a significantly greater improvement in ocular scores at Weeks 8 and 12 compared to the 160-mg dose ($p=0.037$). Patients with a history of thymoma ($n=14$) showed a numerically greater reduction in the total QMG score than those without ($n=15$). The 240-mg dose demonstrated superior efficacy regardless of thymoma status.

Conclusion: Telitacicept provides rapid and sustained improvement in muscle strength across all domains in generalised myasthenia gravis. The 240-mg dose appears particularly beneficial for ocular involvement and in patients with a history of thymoma. These promising subgroup findings warrant further investigation in larger confirmatory studies.

Disclosure: Nothing to disclose.

EPO-0311 | Efficacy of long-term rozanolixizumab treatment cycles in patients with generalised myasthenia gravis: A subgroup analysis of Phase 3 studies

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Background and aims: In MycarinG (Phase 3; NCT03971422), one 6-week cycle of rozanolixizumab (7 mg/kg or 10 mg/kg) improved myasthenia gravis (MG)-specific outcomes versus placebo. Here, we evaluate the long-term efficacy of repeated rozanolixizumab treatment cycles across MycarinG and its

open-label extension studies in subgroups of patients with generalised MG (gMG).

Methods: Following MycarinG, patients could enrol in MG0004 (NCT04124965) to receive once-weekly rozanolixizumab for ≤ 52 weeks, or MG0007 (NCT04650854), which comprised an initial 6-week rozanolixizumab cycle, with further cycles administered upon symptom worsening. Final efficacy data were pooled across MycarinG, MG0004 (first 6 weeks) and MG0007 for patients with ≥ 2 symptom-driven rozanolixizumab cycles. Mean change from baseline (CFB) in MG Activities of Daily Living (MG-ADL) score to Day 43 in Cycles 1–13 was assessed. Subgroups were prespecified according to baseline characteristics.

Results: Overall, 129 patients received ≥ 2 symptom-driven cycles. Mean (standard deviation) CFB in MG-ADL score at Day 43 across Cycles 1–13 for the overall population ranged from -3.2 (3.3) in Cycle 3 ($n=113$) to -6.0 (3.9) in Cycle 12 ($n=24$). Improvements from baseline in MG-ADL scores at Day 43 were consistent in subgroups of autoantibody status (anti-muscle-specific tyrosine kinase and anti-acetylcholine receptor antibody positive), disease duration (< 5.4 and ≥ 5.4 years), age (< 65 and ≥ 65 years), MG-ADL score (< 5 and ≥ 5) and thymectomy status. Treatment-emergent adverse events occurred in 93.1% ($n=175/188$) of patients with ≥ 1 treatment cycle across MycarinG and MG0007; most were mild/moderate.

Conclusion: Repeated rozanolixizumab treatment cycles demonstrated long-term efficacy in a broad range of patients with gMG.

Disclosure: This study was funded by UCB. Full disclosure of all industry relationships will be made during congress presentation if accepted.

EPO-0312 | Aspects of molecular genetic polymorphism of the dystrophin gene in Uzbekistan

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Background and aims: To investigate the genetic polymorphism of the dystrophin gene among patients with Duchenne muscular dystrophy and to determine the frequency of dystrophin gene mutation carriage among female relatives in families affected by DMD in the population of Uzbekistan.

Methods: Molecular genetic studies were conducted MLPA diagnostics to detect deletions and duplications in patients with DMD. MLPA diagnostics was performed in 91 patients with DMD from 81 families. The analysis covered all 79 exons of the dystrophin gene.

Results: deletions were not detected in 33 patients (36.3%) from 32 families (39.5%), while deletions of the dystrophin gene of varying length—from one to nine exons—were identified in 58 patients (63.7%) from 49 families (60.5%). Extended deletions were verified in 65.3% of families, whereas single-exon deletions were observed in 34.7% of families. The main deletion spectrum was located in the distal part of the dystrophin gene at the 3' end (deletions of exons 40–60), accounting for 81.6% (40 families, 47 patients).

Conclusion: According to MLPA diagnostics in patients with Duchenne muscular dystrophy, mutations were identified in 16 of the 79 analyzed exons, which provides a basis for using the

DMD-del1 and DMD-del2 kits for the diagnosis of “major” deletions in the dystrophin gene in the population of Uzbekistan.

Disclosure: Nothing to disclose.

Neurogenetics 2

EPO-0313 | Binaural hearing in spinocerebellar ataxia type 3

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Background and aims: Brainstem degeneration in spinocerebellar ataxia type 3 (SCA3) affects binaural structures such as the medial superior olive (Faber et al, 2021), potentially impairing binaural processing. Huggins Pitch (HP), a psychoacoustic phenomenon dependent on binaural synchronization, may thus serve as a marker of early dysfunction. Previously, we demonstrated that the upper limit of Huggins Pitch (HP) differed significantly between SCA3 mutation carriers and age-matched controls (Jacobi et al, 2024). Here, we investigated longitudinal HP processing in 18 SCA3 mutation carriers (pre-ataxic and ataxic) to assess its potential as a biomarker for disease progression.

Methods: HP thresholds were determined via a 3-alternative forced-choice behavioral task, and cortical N100 responses were recorded with MEG. Ataxia severity was assessed using the SARA scale.

Results: HP thresholds declined significantly from baseline to follow-up (1.8195 vs. 1.4375 kHz; $p < 0.05$), with ataxic carriers showing a stronger decline (1.6511 vs. 1.2976 kHz; $p = 0.039$) and a trend in pre-ataxic carriers (2.1983 vs. 1.7522 kHz; $p = 0.09$).

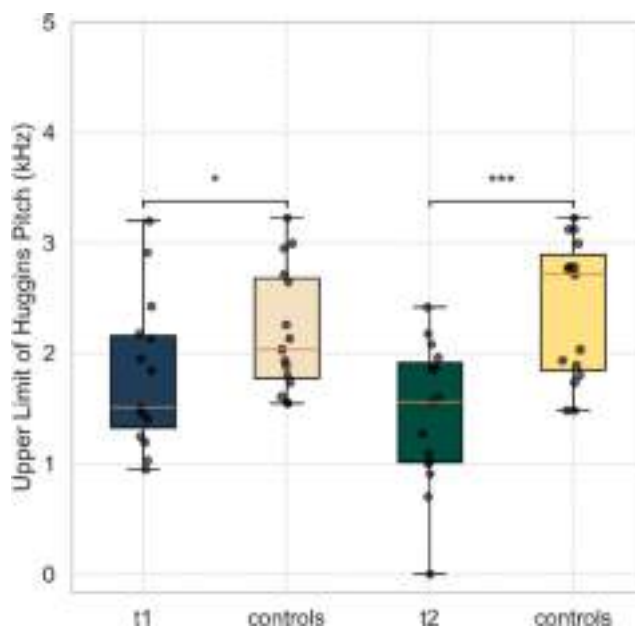


FIGURE 1.1 Boxplots show Huggins Pitch Thresholds in SCA3 carriers ($N=15$) vs healthy controls ($N=15$) at baseline and at follow-up. **FIGURE 1.2** Boxplot show HP Thresholds in SCA3 carriers at baseline vs follow-up (1.80 ± 0.67 vs 1.49 ± 0.53 kHz, $p = 0.019$).

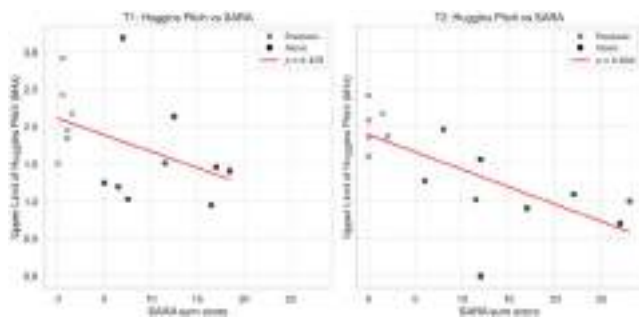


FIGURE 2 Upper limit of Huggins Pitch frequency (kHz) vs SARA sum score in pre-ataxic ($n=7$) and ataxic ($n=9$) SCA3 mutation carriers at baseline (T1, $r = -0.44$, $p=0.105$) and at follow-up (T2, $r = -0.84$, $p < 0.001$).

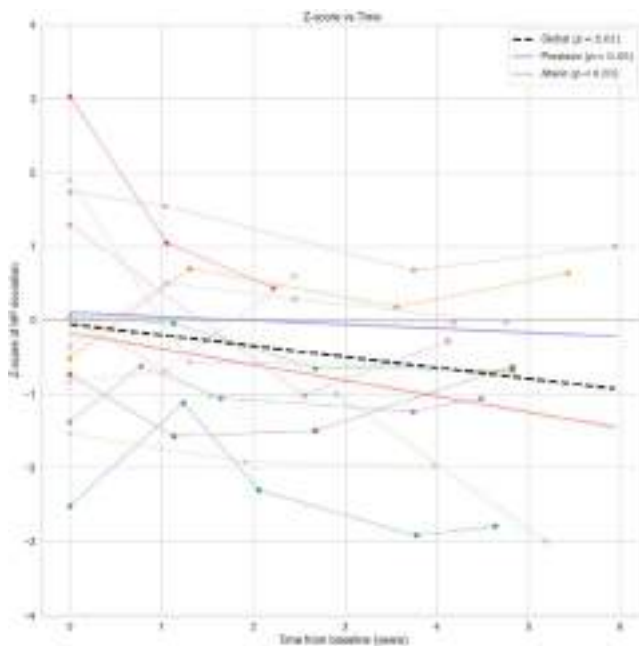


FIGURE 3 LME of longitudinal development of Carriers–Controls Z-scores shows a progressive decline over time for all carriers ($\beta_1 = -0.138 \pm 0.055$, $p=0.012$), preataxic carriers ($\beta_1 = -0.054 \pm 0.079$, $p=0.499$), and ataxic carriers ($\beta_1 = -0.195 \pm 0.075$, $p=0.009$).

Thresholds correlated strongly with SARA sum score at follow-up ($r = -0.82$, $p < 0.001$) and were predicted by time to onset in linear mixed-effects models. Carriers–control Z-scores declined significantly over time, and N100 amplitude was reduced compared to controls ($p < 0.05$). Linear mixed-effects modeling showed progressive HP decline over time for all carriers ($\beta = -0.173$ kHz/year, $p=0.004$).

Conclusion: These findings indicate that HP thresholds and cortical responses reliably decline with SCA3 progression, suggesting HP as a sensitive marker for early detection and longitudinal tracking, including in pre-ataxic mutation carriers.

Disclosure: Nothing to disclose.

EPO-0314 | KMT2B-related disorders in Austria: Clinical features and long-term outcome

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Background and aims: Since its initial description in 2016, DYT-KMT2B has emerged as one of the most common genetic causes of early-onset dystonia. Subsequent reports have expanded its phenotypic spectrum, frequently including neurodevelopmental features. Deep brain stimulation of the globus pallidus internus (Gpi DBS) has become a therapeutic mainstay; however, most published data are based on single cases or short-term observations, and long-term outcomes remain poorly characterized.

Methods: We report the clinical course and response to Gpi DBS in nine patients with KMT2B variants prospectively followed at two Austrian national reference centers for rare movement disorders. Long-term follow-up data (range: 5–20 years) were available for six patients. Clinical features and treatment outcomes were compared with previously published cohorts.

Results: Non-motor features such as developmental delay, intellectual disability, and epilepsy were more frequent in our cohort than in earlier reports. All patients developed generalized dystonia and laryngeal involvement over time, emphasizing the progressive nature of the disease. Despite secondary symptom worsening during long-term follow-up, Gpi DBS preserved ambulation in three patients and enabled sustained recovery of walking ability in two, maintaining functional independence. Surgical correction of foot deformities further supported mobility. Notably, KMT2B variants were identified upon genetic re-evaluation in two patients previously diagnosed with dyskinetic cerebral palsy.

Conclusion: Our long-term data underscore the progressive but heterogeneous course of DYT-KMT2B. Gpi DBS offers durable clinical benefits, particularly when initiated before loss of ambulation. Early surgical intervention and multidisciplinary management are essential to optimize long-term outcomes.

Disclosure: Nothing to disclose.

EPO-0315 | The first libyan patient diagnosed with POLR3B-related leukodystrophy: A case study

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Background and aims: Leukodystrophies are rare inherited white matter disorders with significant clinical and genetic heterogeneity. POLR3B-related hypomyelinating leukodystrophy is typically diagnosed in childhood, while adult-onset presentations are rare and often overlooked.

Methods: We report a 19-year-old Libyan male presenting with a two-year history of progressive ataxia and tremor. Detailed neurological examination, laboratory testing, neurophysiological studies, brain magnetic resonance imaging (MRI), and genetic analysis were performed. Autoimmune, infectious, paraneoplastic, and post-COVID-19 etiologies were systematically excluded.

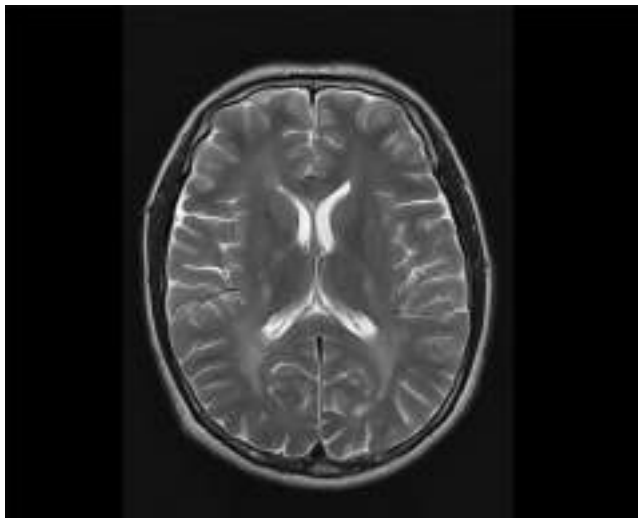


FIGURE 1 Left/Axial T2-weighted MRI of the brain showing confluent hyperintense lesions in the parieto-occipital deep white matter, extending through the splenium of the corpus callosum.



FIGURE 2 Right/Sagittal T2-weighted MRI of the brain demonstrating widespread hypomyelination with subtle involvement of the cerebellar peduncles, sparing the cerebellar tonsils.



FIGURE 3 POLR3B gene variant detected on genetic testing.

Results: Neurological examination demonstrated ataxia, dysarthria, tremor, nystagmus, myoclonus, and impaired coordination. Brain MRI revealed diffuse hypomyelinating white matter abnormalities involving the parieto-occipital regions, corpus callosum splenium, corticospinal tracts, and cerebellar peduncles without diffusion restriction or contrast enhancement. Genetic testing identified heterozygous variants in the POLR3B gene, confirming the diagnosis of POLR3B-related hypomyelinating leukodystrophy. The progressive clinical course, characteristic imaging findings, negative immunological and

oncological workup, and lack of response to immunotherapy unequivocally excluded post-COVID-19 neurological involvement.

Conclusion: This case highlights the diagnostic challenges of adult-onset POLR3B-related leukodystrophy and emphasizes the importance of genetic testing in young adults with progressive ataxia. To our knowledge, this represents the first reported case from Libya.

Disclosure: AI Use Disclosure: Artificial intelligence tools were used solely for language editing, grammar correction, and improvement of writing clarity. All data, analyses, interpretations, and conclusions presented in this work are entirely original and were generated by the authors. The use of AI did not influence the study design, data collection, data analysis, or scientific content.

EPO-0316 | Genetic landscape of pediatric epilepsy associated with focal cortical dysplasia

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Background and aims: Focal cortical dysplasia is a major structural cause of epilepsy in children and is frequently associated with drug-resistant seizures. Increasing evidence indicates that genetic factors contribute significantly to the pathogenesis of focal cortical dysplasia-related epilepsy. The objective of this study was to characterize the genetic spectrum of pediatric epilepsy associated with focal cortical dysplasia and to describe associated clinical features.

Methods: We analyzed a cohort of 36 children with epilepsy associated with focal cortical dysplasia evaluated at a national tertiary pediatric center between 2021 and 2025. All patients underwent whole-exome sequencing (WES). Available clinical data, electroencephalography (EEG), and magnetic resonance imaging (MRI) reports were reviewed. Genetic variants were interpreted according to current pathogenicity criteria. Demographic characteristics, including parental consanguinity, were assessed.

Results: Whole-exome sequencing data were available for 100 percent of patients. Pathogenic and likely pathogenic variants were identified in genes associated with epilepsy and cortical development, including SCN1A, DEPDC5, PCDH19, SCN2A, SCN8A, and TSC2. Both ion channel genes and genes related to cortical development and mammalian target of rapamycin pathway regulation were represented. Parental consanguinity was documented in 5.6 percent of cases. MRI reports were available in 77 percent of patients, EEG reports in 58 percent, and detailed clinical discharge summaries in 97 percent.

Conclusion: This study demonstrates a heterogeneous genetic landscape underlying pediatric epilepsy associated with focal cortical dysplasia. Whole-exome sequencing provides valuable diagnostic information even in settings with incomplete neuroimaging or electrophysiological data and supports its integration into routine clinical evaluation.

Disclosure: The author reports no conflicts of interest and no external funding related to this study.

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Background and aims: The Cerebellar Cognitive Affective Syndrome (CCAS) scale screens for cognitive and affective impairments in cerebellar disorders, but its prevalence in real-world settings remains underexplored.

Methods: The Italian CCAS scale was administered to 68 patients from a single-center (22 Friedreich ataxia (FA), 13 SCA2, 12 RFC1-related disease, 4 SCA27B, 4 SPG7, 3 SCA8, 3 SCA42, 2 SCAR32, 2 HSD17B4, 1 each of GSS, PEX2, ATM, and SCA45) and 30 age-, sex-, and education-matched healthy controls. CCAS was classified as definite (≥ 3 failed items), and a total sum score calculated. Motor impairment was evaluated with the SARA and 9-Hole Peg Test (9HPT). Dysarthria was assessed with the PATA rate, and its correlation with verbal fluency tasks analyzed. Subtest comparisons among genotypes were adjusted for age, education, and SARA score. Neurodegenerative liquid biomarkers were also measured.

Results: Definite CCAS was present in 68% of patients. By genotype, prevalence was 100% in SPG7, 74% in FA, 62% in SCA2, 50% in SCA27B, and 20% in RFC1-related disease. CCAS total score did not correlate with SARA ($p = -0.075$, $p = 0.54$) or 9HPT ($p = -0.18$, $p = 0.14$). Patients with FA, SCA2, and SPG7 performed worse in executive function, short-term memory, and abstract reasoning than those with RFC1-related disease and SCA27B. Word-fluency subtest had the strongest discriminative ability between patients and controls.

Conclusion: Cognitive impairment is frequent in HCAs and varies by genotype. The lack of correlation with motor severity suggests cognitive deficits may occur independently of ataxia progression.

Disclosure: Nothing to disclose.

EPO-0318 | Cortical atrophy mediates the link between clinical severity and retinal neurodegeneration in spinocerebellar ataxia type three

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Background and aims: SCA3 pathology extends beyond the cerebellum, and OCT reveals retinal thinning. We tested whether cortical atrophy mediates the association between clinical severity and retinal neurodegeneration, consistent with a cortical-retinal axis.

Methods: Ninety genetically confirmed SCA3 patients and 32 age- and sex-matched controls underwent OCT, structural MRI, and neurological assessment (SARA, ICARS). OCT metrics were compared between groups. In SCA3, correlations linked

retinal thickness with disease duration, clinical scores, and cortical volumes. Mediation analyses evaluated 20 cortical regions.

Results: SCA3 patients showed significant retinal layer thinning versus controls ($p < 0.05$). Longer disease duration and higher SARA scores correlated with reduced perifoveal thickness. Six significant indirect pathways were detected. Left calcarine atrophy mediated the link between SARA and right-eye inferior perifoveal thickness ($p = 0.04$) and mediated associations between disease duration and perifoveal thinning ($p < 0.001$). Right lingual and left precentral atrophy further mediated effects of disease duration on parafoveal thickness ($p < 0.001$).

Conclusion: Visual and motor cortical atrophy partially explains retinal thinning in SCA3, supporting OCT-derived retinal measures as noninvasive indicators of cortical integrity and disease progression.

Disclosure: Nothing to disclose.

EPO-0319 | Broadening the clinical spectrum of TGM6-related spinocerebellar ataxia: A pediatric case

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Background and aims: Spinocerebellar ataxia type 35 (SCA35), caused by pathogenic variants in the TGM6 gene encoding transglutaminase 6, is classically a late-onset cerebellar disorder characterized by progressive gait ataxia, tremor, and mild oculomotor abnormalities. Dystonia, myoclonus, and parkinsonism are less frequent, while neurodevelopmental impairment and epilepsy are rare. Although increasing evidence suggests broader phenotypic variability, early-onset presentations remain poorly characterized. Here, we describe an early-onset phenotype of SCA35 with objective kinematic characterization of associated movement disorders.

Methods: A 15-year-old boy with early developmental delay, focal epilepsy since infancy, and progressive gait impairment underwent neurological examination, longitudinal brain MRI review, and quantitative kinematic assessment of tremor and bradykinesia during repetitive finger tapping. Genetic testing was performed using a targeted next-generation sequencing panel for ataxia and movement disorders.

Results: The patient carried a heterozygous pathogenic TGM6 c.980A > G (p.Asp327Gly) variant, also detected in his mildly affected mother, supporting intrafamilial variability. Serial brain MRI (2013–2021) showed no cerebellar or brainstem atrophy. Neurological examination revealed bilateral irregular tremor at rest, posture, and action, mild cerebellar dysidiadochokinesia, and upper limb bradykinesia with stable clinical course. Quantitative kinematic analysis demonstrated a right-hand resting tremor peaking at ~8 Hz, a bilateral low-amplitude irregular postural tremor, and bradykinesia with sequence effect (Figure 1).

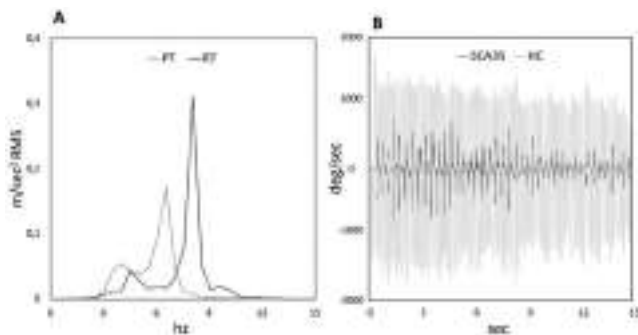


FIGURE 1 Quantitative kinematic characterization of tremor and bradykinesia.

Conclusion: This case expands the phenotypic spectrum of TGM6-related disease, showing that pathogenic variants may manifest in childhood with neurodevelopmental involvement, epilepsy, and complex movement disorders even in the absence of structural neuroimaging abnormalities. These findings support a network-based pathophysiological mechanism potentially involving cerebellar–basal ganglia circuits.

Disclosure: Nothing to disclose.

EPO-0320 | Long-term (30 Months) outcomes of N-Acetyl-L-leucine in adults and children with niemann-pick disease type C

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Background and aims: Niemann-Pick disease type C (NPC) is a rare lysosomal storage disorder. IB1001-301 is a Phase 3, placebo-controlled, crossover trial encompassing a Parent Study and an ongoing Extension Phase (EP) evaluating the long-term effects of N-acetyl-L-leucine (NALL) in people with NPC.

Methods: IB1001-301 enrolled participants with a genetically confirmed diagnosis of NPC. Participants received a total of 2–4 g/day orally administered NALL. The primary endpoint of the EP is the modified 5-domain NPC Clinical Severity Scale (5-D-NPC-CSS). Exploratory endpoints include the 15-D-NPC-CSS (excluding hearing), 4-D-NPC-CSS, and Scale for Assessment and Rating of Ataxia (SARA). Interim 30-month results are reported here.

Results: Forty participants completed Visit 12 (30-months) of the EP. The mean ($\pm$ SD) baseline score on the 5-D-NPC-CSS was 10.50 ($\pm$ 4.46). After 30 months, the mean ($\pm$ SD) change from baseline in the 5-D-NPC-CSS was -0.20 ($\pm$ 2.60) with NALL vs 3.75 ($\pm$ 7.9), i.e. worsening, in the historical cohort; mean difference between cohorts: -3.95 ; 95% CI -6.60 to -1.30 ; $P=0.0041$. The mean ($\pm$ SD) baseline score on the 15-D-NPC-CSS was 17.48 ($\pm$ 7.15). After 30 months, the mean ($\pm$ SD) change from baseline in the 15-D-NPC-CSS was 0.38 ($\pm$ 4.00) with NALL vs $+4.68$ ($\pm$ 2.74) in the historical cohort. The mean ($\pm$ SD) baseline scores on the 4-D-NPC-CSS and SARA were 7.65 ($\pm$ 3.32) and 15.0 ($\pm$ 7.68), respectively. After 30 months, the mean change from

baseline ($\pm$ SD) on the 4-D-NPC-CSS and SARA were -0.58 ($\pm$ 2.04) and -1.39 ($\pm$ 3.71), respectively.

Conclusion: Treatment with NALL demonstrated significant and meaningful long-term improvement, i.e. disease-modifying effects. NALL was well tolerated, and no serious adverse events occurred.

Disclosure: Tatiana Bremova-Ertl received speaker's honoraria and consultancy fees from Actelion, Sanofi-Genzyme and Zevra as well as blinded video-rater fees from IntraBio. Jorgji Kerthi, Bethany Zandrucha, Janelle Raymond, Asante Hatcher, Taylor Fields, Ian Billington and Marc C Patterson are employees of and may have stock option ownership in IntraBio Inc. Michael Strupp received speaker's honoraria from Abbott, Actelion, Auris Medical, Biogen, Eisai, Grünenthal, GSK, Henning Pharma, Interacoustics, MSD, Otometrics, Pierre-Fabre, TEVA, UCB, Viatrix. Consultant for Abbott, Actelion, Auris Medical, Decibel, Heel, and Sensorion. Scientific founder and shareholder of IntraBio.

EPO-0321 | Expanding the phenotypic spectrum of FAT2-associated disorders: An Italian case series

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Background and aims: Spinocerebellar ataxias (SCAs) are a clinically and genetically heterogeneous group of autosomal dominant neurodegenerative disorders with more than 50 distinct subtypes described to date. Among them, SCA45, associated with FAT2 gene mutation, was first described in 2017. Reported cases are rare and typically exhibit a pure cerebellar phenotype with late onset. FAT2 is highly expressed in cerebellar granule cells and is thought to play a role in neuronal migration and cerebellar development. We describe four patients from three unrelated families, expanding the clinical spectrum of SCA45, with two cases showing additional pyramidal involvement.

Methods: Three patients were evaluated at the Neurogenetics Clinic of Polo Pontino/ICOT, Sapienza University of Rome, and one at the IRCCS Stella Maris Foundation. Genetic analyses were performed via targeted NGS panels.

Results: We identified four patients with adult-onset gait disturbances and rare heterozygous FAT2 variants. P1: Ataxic gait after age 60; cerebellar atrophy on MRI; c.8218C > T (p.Phe2740Leu). P2: Spastic-paretic gait after 50; cerebellar atrophy and axonal neuropathy; c.5240A > G (p.Asp1747Gly). P3: Sister of P2; ataxic gait after 60; same variant c.5240A > G. P4: Spastic-paretic gait, hyperreflexia; SPRS 28/52, SARA 17/40, SPATAX stage 5, FARS 4.0, FARS-ADL 15/36; c.5416G > A (p.Glu1806Lys).

Conclusion: These cases broaden the phenotypic spectrum of FAT2-related ataxia, highlighting the possible presence of spasticity and pyramidal signs in addition to cerebellar features. FAT2 variants should be considered in patients with adult-onset cerebellar ataxia, especially when common SCA repeat expansions are excluded.

Disclosure: Nothing to disclose.

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Background and aims: Friedreich's Ataxia is a rare autosomal recessive neurodegenerative disease that typically presents in childhood or adolescence. It is caused by a GAA repeat expansion in the FXN gene, leading to frataxin deficiency, mitochondrial dysfunction, and progressive neurological decline. Impaired Nrf2 signaling exacerbates oxidative stress. Omaveloxolone is an oral Nrf2 activator. Following the MOXIe trials, it received FDA approval in 2023, and France initiated a national early access program in January 2024.

Methods: This study reports real life data from a French cohort treated with Omaveloxolone at CHU Bordeaux, with up to 18 months of follow up. Clinical evaluations included the Scale for the Assessment and Rating of Ataxia (SARA) and SF12. Blood samples for hepatic, lipid, and cardiac monitoring were collected at baseline and scheduled time points.

Results: The cohort initially included 32 patients, with a mean baseline SARA score of 22.8. After 18 months, 23 patients remained on treatment. The average SARA score at one year was 23. All patients received 150 mg/day of Omaveloxolone. Adverse events, particularly transient elevations in liver enzymes, were most frequent within the first 12 weeks and subsequently decreased or returned toward baseline, supporting good tolerability.

Conclusion: After 12 months, the mean change in SARA score from baseline was -0.02 SD 2.74 , indicating overall stability. This finding is consistent with a slowing of disease progression and contrasts with natural history data from the EFACTS cohort, where ambulant patients show an average annual SARA increase of $+0.82$ SD 0.05 , suggesting a potential attenuation of disease progression

Disclosure: Nothing to disclose.

EPO-0323 | Deep pheno-genotyping of NDUFV1-related mitochondrial disease

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Institute of Child Health – Lahore, Pakistan; ⁹Sobell Department of Motor Neuroscience and Movement Disorders – UCL Queen Square Institute of Neurology – London, UK; ¹⁰Department of Clinical and Movement Neurosciences – UCL Queen Square Institute of Neurology – London, UK

Background and aims: NDUFV1 encodes a 51-kDa core subunit of mitochondrial complex I. We aim to expand the phenotypic and genotypic spectrum of NDUFV1-related mitochondrial disease.

Methods: We screened international next-generation sequencing datasets for biallelic NDUFV1 variants. Detailed phenotypic data were collected for identified individuals. Haplotype analysis was performed for recurrent variants. A PubMed search (Dec 2025, keyword “NDUFV1”) was conducted to extract published genetic and clinical data.

Results: We identified 28 new affected individuals (16 males) from 22 families of Indian, Pakistani, Iranian, Malaysian, and Egyptian ancestry and 68 previously reported cases. In our cohort age at onset ranged from birth to 10years, with gait impairment and seizures being the most common initial symptoms. Core clinical features are summarized in Figure 1. We also document orofacial dystonia and sleep disturbances as newly associated features. Oculomotor abnormalities, as well as respiratory insufficiency, frequently reported in the literature, were rare in our cohort. We identified 27 homozygotes

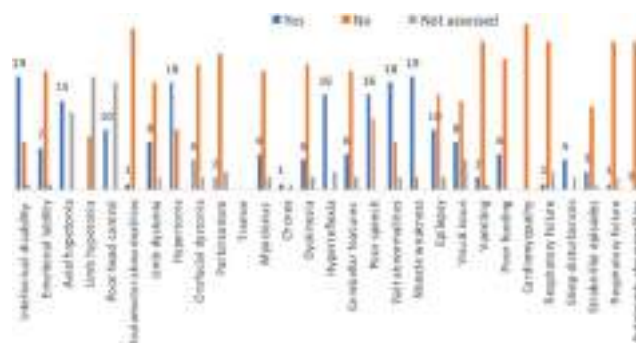


FIGURE 1 Clinical features of the 28 patients of our cohort.



FIGURE 2 NDUFV1 gene schematic with introns to scale; literature variants in black, known/cohort variants in blue, novel variants in red.

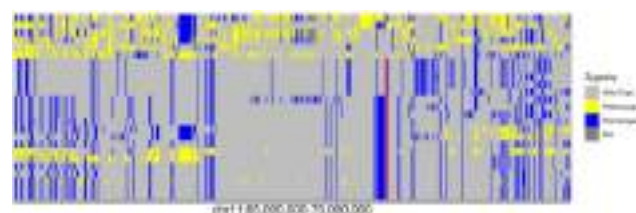


FIGURE 3 Haplotype analysis on exome files performed in patients carrying the NM_007103.4:c.1156C > T variant.

and 1 compound heterozygote. Among six identified variants, two were novel: NM_007103.4:c.597delC p.(Ala201LeufsTer25) and NM_007103.4:c.466A > G p.(Ile156Val). The most frequent was NM_007103.4:c.1156C > T p.(Arg386His), found in 78% of patients (Fig. 2). Although it was found recurrently in individuals from neighboring Asian regions, haplotype analysis showed distinct genetic backgrounds, supporting the view that it is not a founder effect (Fig. 3).

Conclusion: This study broadens the phenotypic spectrum of NDUFV1-related mitochondrial disease, highlighting orofacial dystonia and sleep disturbances as newly phenotypic features, and expands the genotypic landscape by identifying two novel variants.

Disclosure: Nothing to disclose.

Sunday, June 28 2026

Ageing and Dementia 3

EPO-0324 | Skin biopsy for detection of phosphorylated alpha-synuclein by indirect immunofluorescence in a cohort of patients with cognitive impairment

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Background and aims: Techniques for detection of Dementia with Lewy Bodies (DLB) and distinction from other causes of cognitive impairment are in rapid establishment. Among these are Indirect Immunofluorescence (IF) for identification of phosphorylated α -synuclein (p-syn) in skin tissue and Seeding Amplification Assays (SAAs), including Real Time Quaking-Induced Conversion (RT-QuIC).

Methods: patients referred to our Clinic for major or minor cognitive impairment were prospectively recruited from 2015 to 2025, undergoing full neurological and neuropsychological examination. Subsequent examinations were guided by the clinical presentation and the final diagnosis, serving as gold standard, was established by experienced clinicians. Skin biopsy for detection of p-syn by IF was performed in all patients at distal leg and cervical sites. Diagnostic accuracy (sensitivity, specificity) of skin IF was assessed, along with the correlation of quantitative p-syn measures with clinical measures.

Results: 50 patients with DLB (syn group) diagnosis and 74 fulfilling different diagnoses (non-syn cognitive impairment) were

recruited. Skin biopsy detected p-syn in 46 syn patients, resulting negative in all non-syn cases. Quantitative measures did not correlate with clinical measures.

Conclusion: Skin biopsy is a promising technique for detection of p-syn in patients with DLB diagnosis. The results suggest significant specificity. Current quantitative p-syn measures do not correlate with disease parameters in DLB, warranting development of more specific indexes.

Disclosure: Nothing to disclose.

EPO-0325 | Speech biomarkers derived from explainable AI reveal therapeutic effects of transcranial pulse stimulation in Alzheimer's disease

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Background and aims: We develop interpretable AI models using spontaneous-speech digital biomarkers to support the differential diagnosis of Alzheimer's disease (AD) and to quantify individual therapeutic effects of transcranial pulse stimulation (TPS) on disease progression. TPS is a CE-certified, non-invasive, shockwave-based neuromodulation technique. It enables neuronavigation-guided targeting and modulation of cortical gray matter as well as deep brain structures.

Methods: Automated classification models were trained on spontaneous speech from 40 AD patients and 27 socio-demographically matched healthy controls. Diagnosis followed German S3

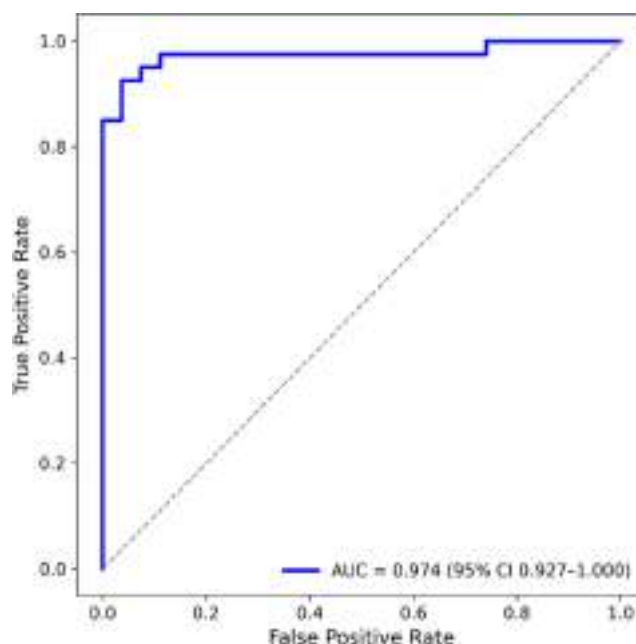


FIGURE 1 Performance of the AD classification model. Receiver operating characteristic (ROC) curve for the final model trained on linguistic features using the full dataset. The model achieved an area under the curve (AUC) of 0.974 [95% CI: 0.927-1.000].

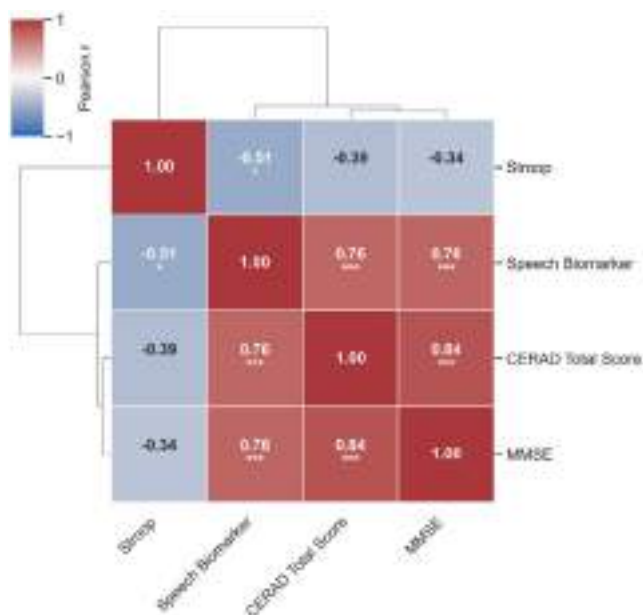


FIGURE 2 Speech-based biomarkers track global cognitive decline. Hierarchical clustering of Pearson correlation coefficients reveals a high-fidelity association between speech-biomarker and global cognitive measures (CERAD, $r=0.76$; MMSE, $r=0.76$).



FIGURE 3 Longitudinal trajectories of cognitive change captured by an AI-derived speech biomarker. Waterfall plot showing annualized rates of change in speech biomarker indexing cognitive functioning for a representative sub-cohort (AD group).

guidelines and was supported by CERAD Plus (including MMSE), Stroop test and structural MRI. Interpretable AI identified the most discriminative speech features. Biomarkers were then used to quantify TPS-associated modulation in 14 patients who received six sessions over 14 days, with 6000 pulses per session delivered bilaterally to the frontal and parietal lobes and the precuneus, using an energy level of 0.25 mJ/mm² and a frequency of 4 Hz.

Results: Speech biomarkers correlated with cognitive performance and captured impairment better than the Stroop test. AD versus control classification achieved F1=0.81. Group-level linguistic performance was stable or slightly improved during follow-up, contrasting with the 3.3-point annual MMSE decline expected in untreated AD. Individual t0/t1 biomarker trajectories varied markedly, indicating heterogeneous responsiveness. Discriminative speech features provided mechanistic insights into TPS-related neurophysiological effects.

Conclusion: Interpretable AI with speech-derived digital biomarkers provides a precision framework for AD diagnosis and treatment monitoring. TPS produces individualized, quantifiable effects on disease progression detectable with sensitive speech-based measures that outperform conventional cognitive assessments.

Disclosure: Nothing to disclose.

EPO-0326 | Semiquantitative neuropathologic thresholds for cerebrospinal fluid biomarker alterations in Alzheimer's disease

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Background and aims: Validation of Alzheimer's disease (AD) cerebrospinal fluid (CSF) biomarkers against pathology is limited by small sample cohorts and long CSF-death intervals. Moreover, few studies assessed the burden of brain A β and tau pathologies using semiquantitative scoring. Using a large cohort, we defined the brain A β and tau burden threshold associated with significant alterations in CSF markers.

Methods: We studied 233 subjects with post-mortem neuropathology (164 prion, 69 non-prion) and antemortem CSF samples. A β and tau burden were semi-quantitatively scored across nine and six brain regions, respectively. Thal phase, Braak stage and AD neuropathologic change (ADNC) category were assigned. CSF and plasma biomarkers were measured by the Lumipulse platform. Associations between pathology and log-transformed biomarkers were tested using linear regression models. Age, sex, and CSF-death interval were considered as covariates.

Results: Median sampling-death interval was 1.5 months (IQR 0.5–5.1). CSF A β 42/A β 40 was already significantly reduced at Thal phase 2 ($p=0.019$) and low-ADNC ($p<0.001$) when compared to non-AD. The first significant reduction in CSF A β 42/A β 40 occurred at an A β density score > 15 (moderate neocortical and amygdala pathology; $p=0.047$), with increasing pathological burden associated with greater biomarker changes ($p<0.001$). Values of CSF A β 42/A β 40 ≤ 0.068 had 92% specificity and 90% sensitivity for distinguishing an A β density score > 25. In non-prion cases, A β 42/p-tau181 was earliest reduced at intermediate-ADNC and A β density-score > 35 ($p<0.001$).

Conclusion: Group-alterations of CSF A β 42/A β 40 appear at a relatively early stage of AD continuum, although initial A β deposits remain below detection. In contrast, a reduction in CSF A β 42/p-tau181 emerges only at higher levels of beta-amyloid burden.

Disclosure: Nothing to disclose.

EPO-0327 | Validation of CogniPaz: A rapid, ecologically-oriented digital biomarker for detection of mild cognitive impairment

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Background and aims: Differentiation between amnesic mild cognitive impairment (aMCI), subjective cognitive decline



FIGURE 1 9-task CogniPaz protocol and interface (translated from the original Spanish).

TABLE 1 Demographic and clinical characteristics and mean scores of the study population.

Characteristic	HC (n=24)	SCD (n=17)	MCI/aMCI (n=39)	Total (n=70)
Age (mean ± SD)	71.2 (7.1)	71.2 (7.1)	71.2 (7.1)	71.2 (7.1)
Gender (n)	12 M, 12 F	10 M, 7 F	18 M, 21 F	40 M, 30 F
Education (mean ± SD)	12.5 (1.5)	12.5 (1.5)	12.5 (1.5)	12.5 (1.5)
MoCA score (mean ± SD)	28.5 (1.5)	26.5 (1.5)	24.5 (1.5)	26.5 (1.5)
MMSE score (mean ± SD)	28.5 (1.5)	26.5 (1.5)	24.5 (1.5)	26.5 (1.5)
CDR-SB score (mean ± SD)	10.0 (0.0)	9.0 (0.0)	8.0 (0.0)	9.0 (0.0)
Significance (p-value)				

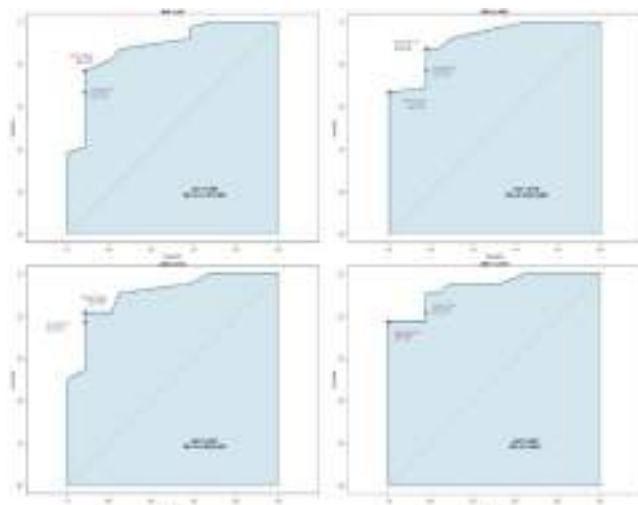


FIGURE 2 Cognipaz ROC curves. MCI: mild cognitive impairment. aMCI: amnesic mild cognitive impairment. SCD: subjective cognitive decline. HC: healthy controls.

(SCD) and healthy controls (HC) is a diagnostic challenge. We developed CogniPaz, an ecologically oriented digital cognitive assessment composed of nine tasks evaluating memory and executive functions. We evaluated its diagnostic accuracy as a digital biomarker in discriminating MCI/aMCI from SCD and HC. **Methods:** We conducted a prospective observational study including participants with subjective cognitive complaints or asymptomatic controls recruited between April 2025 and December 2025. Participants were classified based on clinical evaluation and Montreal Cognitive Assessment (MoCA) scores into HC (asymptomatic; MoCA ≥ 26), SCD (cognitive complaints; MoCA ≥ 26) and MCI/aMCI (cognitive complaints without functional impairment; MoCA < 26). CogniPaz was administered after MoCA by a blinded evaluator, following a standardized protocol. We assessed correlation with MoCA score (Pearson), diagnostic accuracy (ROC analysis) and usability (satisfaction questionnaire).

Results: 80 participants were included: 39 MCI (22 aMCI), 17 SCD and 24 HC. Mean administration time was 9.65 minutes (SD 3.72). CogniPaz total scores correlated strongly with MoCA

($r=0.83$) and memory tasks correlated with MoCA memory domains ($r=0.69$). Diagnostic accuracy was excellent for distinguishing MCI from HC (AUC=0.882), MCI from SCD (AUC=0.916), aMCI from HC (AUC=0.907) and aMCI from SCD (AUC=0.937). CogniPaz also discriminated suspected neurodegenerative from non-neurodegenerative cases (AUC=0.835). Usability was rated highly (mean score 8.11 out of 9).

Conclusion: CogniPaz shows strong correlation with MoCA and excellent diagnostic accuracy. Its brevity and ease of use support its utility as an efficient digital screening tool for distinguishing MCI/aMCI from SCD and HC.

Disclosure: The authors declare that this study was conducted with institutional research support. No commercial sponsorship influenced the design or results of this study.

EPO-0328 | Lobar Cerebral microbleeds and white matter hyperintensities differentially associate with neuropsychiatric vulnerability in dementia with Lewy bodies

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Background and aims: To explore the relationship between markers of small vessel disease (SVD)—lobar cerebral microbleeds (CMB) and white matter hyperintensities (WMH)—and neuropsychiatric symptoms (NPS) in dementia with Lewy bodies (DLB). Markers of SVD are associated with cognitive impairment in older adults, but their contribution to early neuropsychiatric manifestations in DLB remains insufficiently characterized.

Methods: Clinically diagnosed DLB cases ($n=70$) were analyzed. CMB and WMH were assessed using standardized visual rating scales. NPS, including depression, anxiety, apathy, hallucinations and delirium, was assessed retrospectively. NPS other than delirium were evaluated at the first cognitive visit. Pre-diagnostic delirium was defined via the Confusion Assessment Method scale within the year preceding diagnosis. Descriptive analyses were performed. Cross-sectional associations between SVD imaging markers and NPS were examined using regression models adjusted for age, sex, and educational level.

Results: CMB were present in 17/54 (31.5%) cases, and 45/69 (65.2%) had at least one documented delirium episode in the year preceding diagnosis. CMB were selectively associated with depression (OR=6.80, 95% CI 1.60–28.87, $p=0.009$) and pre-diagnostic delirium (OR=5.11, 95% CI 1.02–25.71, $p=0.048$). CMB were not associated with anxiety, apathy, or hallucinations. WMH showed no significant association with any NPS outcome.

Conclusion: In contrast to WMH, CMB were selectively associated with depression and pre-diagnostic delirium in clinically diagnosed DLB. These findings suggest diverging pathophysiological mechanisms of SVD comorbidity in LBD and support a differential contribution of cerebrovascular markers to prodromal neurocognitive symptoms and clinical expression in DLB.

Disclosure: Funding: L.S. is supported by the Alzheimer's Association grant (AACSf-22-922907) and clinician-scientist award at the University of Geneva.

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Background and aims: To assess clinical/neuropsychological, neurophysiological, neuroimaging, genetic, and fluid biomarker profiles of individuals attending a Brain Health Service (BHS) designed for early identification of Alzheimer's disease (AD).

Methods: Fifty-two participants were referred to the BHS and underwent clinical/neurological and neuropsychological evaluations, brain MRI, electroencephalography (EEG), optical coherence tomography (OCT), APOE genotyping, and, for research purposes, blood-sampling for plasma AD-biomarker-analysis. Participants were classified as A+ ($n=22$) or A- ($n=30$) based on p-tau217, and clinically stratified into cognitively normal (CN=15; CN+ =6; CN- =9), subjective cognitive decline (SCD=18; SCD+ =3; SCD- =15), and mild cognitive impairment (MCI=19; MCI+ =13; MCI- =6). We analyzed: gray matter (GM) atrophy, white matter (WM) hyperintensities, EEG features and OCT data.

Results: A+ cases were older than A-, reported higher alcohol consumption, showed poorer cognitive performances and engaged in less physical activity. CN+ showed reduced semantic fluency compared to SCD. Compared to A-, A+ showed reduced GM volume in the left medial temporal lobe, left fusiform gyrus, right precuneus and anterior temporal gyrus, and increased WM burden in periventricular and deep regions. Compared to A-, in A+ EEG analysis revealed lower frequencies in temporal and frontal regions, and OCT revealed increased global retinal nerve fiber layer thickness.

Conclusion: Our findings support the feasibility and clinical relevance of plasma biomarkers, offered within a research framework, for the early detection of AD in individuals accessing a BHS. Early biomarker identification enables timely lifestyle interventions that may delay symptoms onset. The BHS provides a promising framework to detect at-risk individuals and guide preventive strategies.

Disclosure: E. Canu has received research support from the Italian Ministry of Health; B. Lamorgese, E. Sibilla, V. Castelnovo, G. Rugarli, A. Ghirelli, R. Santangelo, J. Lanzone, M. Cursi, L.

Zanchi, report no competing interests; E.G. Spinelli received honoraria from Eli Lilly; G. Cecchetti received honoraria from Eli Lilly and Neopharmed Gentili; F. Agosta is Associate Editor of *NeuroImage: Clinical* and the *European Journal of Neurology*; has received speaker honoraria from Biogen Idec, Bristol Myers Squibb, Eisai, Eli Lilly, GE Healthcare, Neuraxpharm, and Roche; and receives or has received research supports from the Italian Ministry of Health, the Italian Ministry of University and Research, AriSLA (Fondazione Italiana di Ricerca per la SLA), the European Research Council (ERC), the EU Joint Programme – Neurodegenerative Disease Research (JPND), and Foundation Research on Alzheimer Disease (France); M. Filippi is Editor-in-Chief of the *Journal of Neurology*, Associate Editor of *Human Brain Mapping*, *Neurological Sciences*, and *Radiology*; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmed Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla.

EPO-0330 | **Greater burden of Alzheimer's co-pathology in women with Parkinson's disease**

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Background and aims: Alzheimer's disease (AD) and Parkinson's disease (PD) frequently co-occur in older adults, yet the influence of sex on AD pathology in the context of PD remains unexplored. The objective of this study is to investigate sex differences in AD related pathology among autopsy-confirmed PD cases.

Methods: All subjects were enrolled in the Arizona Study of Aging and Neurodegenerative Disorders (AZSAND) and Brain and Body Donation Program (BBDP) and had annual standardized research clinical assessments by neuropsychologists, subspecialty behavioral and movement disorders neurologists, as well as comprehensive neuropathological examinations after death

Results: Among 230 autopsy-confirmed PD cases, females exhibited greater amyloid plaque pathology burden than males, regardless of a co-occurring AD diagnosis. Specifically, females with PD had significantly higher mean cortical total plaque scores (mean 6.5/15 vs. 4.9/15, $p=0.045$) and greater CERAD neuritic plaque density (mean 1.7/3 vs. 1.3/3, $p=0.035$). Females were also more likely to have a higher cortical plaque burden (plaque total ≥ 5 : 56.8% vs. 39.7%, $p=0.015$). In multivariable logistic regression models, female subjects showed greater than twice the odds of having an amyloid plaque burden ≥ 5 compared to males (OR = 2.18; 95% CI = 1.17 – 4.06; $p=0.014$), when controlling for ApoE $\epsilon 4$ status and age at death.

Conclusion: Female sex is associated with increased amyloid plaque pathology in PD, independent of ApoE ϵ 4 status. These findings highlight a sex-specific vulnerability to AD pathology in PD patients and support the need for sex-informed approaches to diagnosis and treatment in mixed neurodegenerative disease.

Disclosure: Nothing to disclose.

EPO-0331 | Cognitive improvement following transcranial pulse stimulation (TPS) in patients with dementia: A randomized, sham-controlled study

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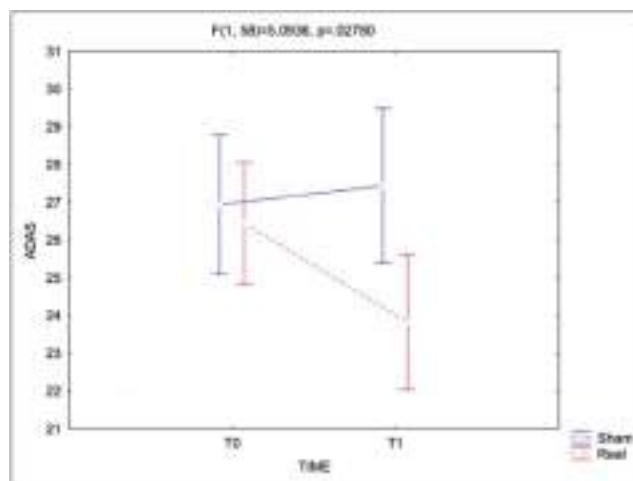
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Background and aims: The burden of dementia is increasing worldwide, resulting in a substantial social and healthcare burden. Hence, the increasing interest in both pharmacological and non-pharmacological treatments, with potential to slow the clinical course of the disease. The objective of this study was to investigate whether Transcranial Pulse Stimulation (TPS) may induce cognitive improvement in patients with an early stage of dementia.

Methods: A double-blind, sham-controlled study was conducted, with patients randomly assigned to receive either TPS or sham TPS for four weeks in parallel groups. Patients received either TPS or sham treatment once daily, five days per week. TPS was applied to bilateral frontal cortex, bilateral parietal cortex and precuneus cortex via a neuronavigation system based on individual MRI spatial coordinates. Clinical assessments were performed through the Alzheimer's Disease Assessment Scale-Cognitive Subscale (ADAS-Cog) at baseline (t0), two weeks after the start of TPS (t1), one month after the start of TPS (t2), two months after the start of TPS (t3), and six months after the start of TPS (t4).



FIGURE 2 TPS device (by courtesy of Storz Medical).



Pre- vs post-stimulation ADAS-Cog score comparison.

Results: 82 patients were enrolled. Sixty (mean age $\pm$ SD 74.3 $\pm$ 8; 20 males) completed assessments at baseline and after one month of stimulation. A significant improvement in ADAS-Cog scores at one month was detected in patients receiving the active treatment ($n=34$ mean age $\pm$ SD 73.2 $\pm$ 9; 11 males) compared with those receiving sham treatment ($n=26$ mean age $\pm$ SD 75.8 $\pm$ 6.5; 9 males) ($p < 0.009$). No significant side-effects were reported.

Conclusion: Preliminary findings suggest that TPS may be a promising tool for enhancing neuroplasticity-related recovery in dementia.

Disclosure: This research was funded by the Italian Ministry of Health under the National Recovery and Resilience Plan (PNRR), Mission 6 – Component 2 – Investment 2.1 “Enhancement and strengthening of biomedical research within the National Health Service (SSN)”, Project code PNRR-MCNT2-2023-12377235 (CUP: E13C24000930001), financed by the European Union – NextGenerationEU.

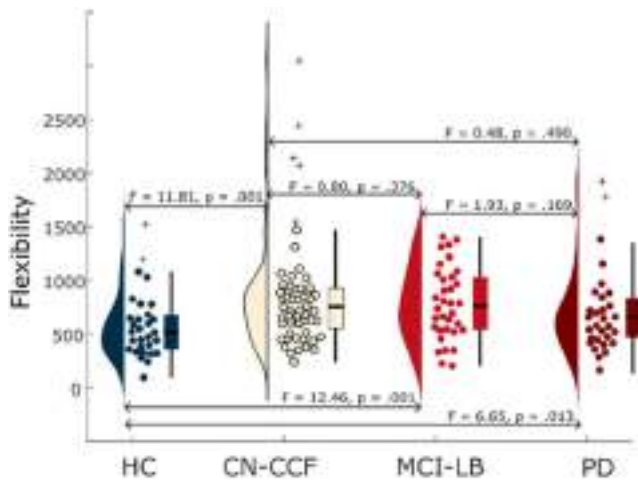
EPO-0332 | EEG dynamics in Lewy body diseases is related to clinical fluctuations and degeneration of locus coeruleus

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Background and aims: The complex relationships between the focal neurodegeneration, dynamics of the brain activity and cognitive decline are not well understood in the context of Lewy body diseases. Quantitative assessment of the transient cascades of increased brain activity (neuronal avalanches) has proven useful in capturing meaningful information across conditions. Here we examined whether the richness of brain dynamics was associated with clinical symptoms and with the microstructural integrity of catecholaminergic nuclei – the right caudal locus coeruleus (rLC) and the substantia nigra pars compacta (SNpc).

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Group differences in flexibility. Note: Crosses (+) mark 9 outliers that were removed before statistical analysis; $n(\text{HC}) = 28$, $n(\text{CN-CCF}) = 51$, $n(\text{MCI-LB}) = 34$, $n(\text{PD}) = 28$.

Methods: The sample comprised 30 HC, 56 cognitively normal individuals with core clinical features of dementia with LB (CN-CCF), 34 individuals with mild cognitive impairment with LB (MCI-LB), and 30 advanced PD patients. Neuronal avalanches were extracted from source-reconstructed resting-state EEG and described by their count, the number of unique spatial patterns (flexibility), hemispheric symmetry of flexibility, and avalanche transition matrices. LC and SNpc integrity were assessed using neuromelanin-sensitive MRI and free-water diffusion imaging, respectively.

Results: Lewy body disease groups exhibited increased flexibility relative to HC, which was, in prodromal groups, associated with cognitive fluctuations and REM sleep behavior disorder symptoms, but not parkinsonism. Flexibility was negatively related to rLC integrity, which was lowest in MCI-LB.

Conclusion: These findings suggest that elevated flexibility reflects dysregulated brain dynamics linked to malignant non-motor features of LB diseases and to the caudal LC involvement.

Disclosure: This work has received funding from the project nr. LX22NPO5107 (MEYS): Financed by European Union – Next Generation EU; from the European Union's Horizon Europe research and innovation programme under grant agreement No 101147319 (EBRAINS 2.0); from the Ministry of Education, Youth and Sports of the CR (Joint Programme Neurodegenerative Disease), project nr. 9F25001 (TRACE-PD): Tracking the mechanisms of disease progression and functional compensation in the early phase of Parkinson's disease; from EU Joint Programme Neurodegenerative Disease (JPND) project entitled 'TACKLing the Challenges of PREsymptomatic Sporadic Dementia (TACKL-PRED),' project nr. 8F22005. This work was supported by Ministry of Health of the Czech Republic, grant nr. NNW26J04-00157; by the European Regional Development Fund project LangInLife, reg. no.: CZ.02.01.01/00/23_025/0008726.

Background and aims: Alzheimer's disease (AD) is a progressive neurodegenerative disorder posing global health challenges. Accessible, non-invasive early biomarkers are urgently needed. Plasma-derived extracellular vesicles (EVs) cross the blood-brain barrier, carrying CNS cargo and offering biomarker potential. We aim to identify, validate, and determine the cellular origins of a novel plasma EV protein panel with high diagnostic accuracy for AD.

Methods: A two-phase nested case-control study was used. Discovery: untargeted 4D-Label-free proteomics (LC-MS/MS) on plasma EVs from 25 probable AD patients and 25 matched healthy controls (HC). Bioinformatic analyses integrated public brain scRNA-seq data to infer cellular origins. Validation: candidate panel quantified by targeted mass spectrometry (SRM) in an independent multi-center cohort (30 AD, 25 MCI, 30 HC). Models adjusted for age and systolic blood pressure. Machine learning built and evaluated a multi-marker diagnostic model.

Results: Several proteins were differentially expressed; genes encoding them were enriched in brain-specific cells, notably endothelial cells and microglia. In validation, PRKCSH was upregulated in AD; ANGPT1, NCKAP1, and FLJ78516 were downregulated vs. controls (all $p < 0.001$). MCI showed intermediate levels, suggesting a disease continuum. A diagnostic model with these four biomarkers achieved AUC 0.95 (95% CI 0.91–0.99) using Random Forest, with 92% sensitivity and 91% specificity for AD vs. controls.

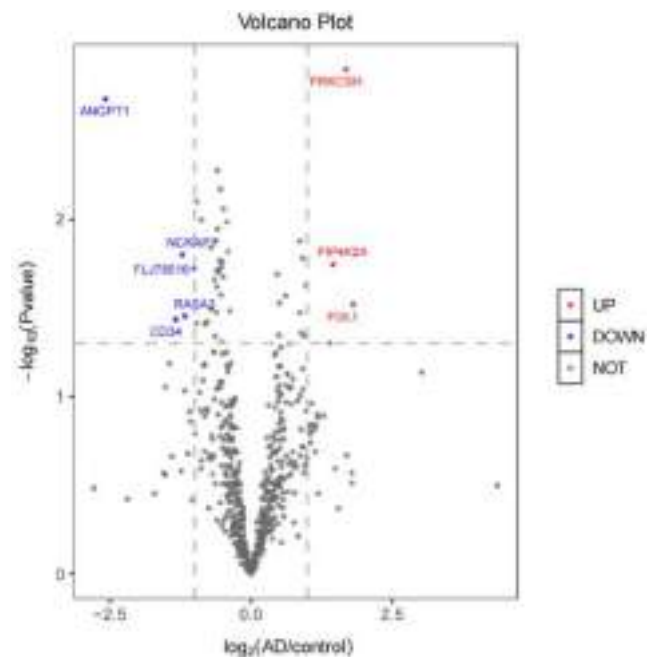


FIGURE 1 Volcano plot of differentially expressed EV proteins in the discovery cohort (AD vs. HC). Proteins with $\text{FC} > 2$ and $p < 0.05$ are shown in red (upregulated), while those with $\text{FC} < 0.5$ and $p < 0.05$ are in blue (downregulated).

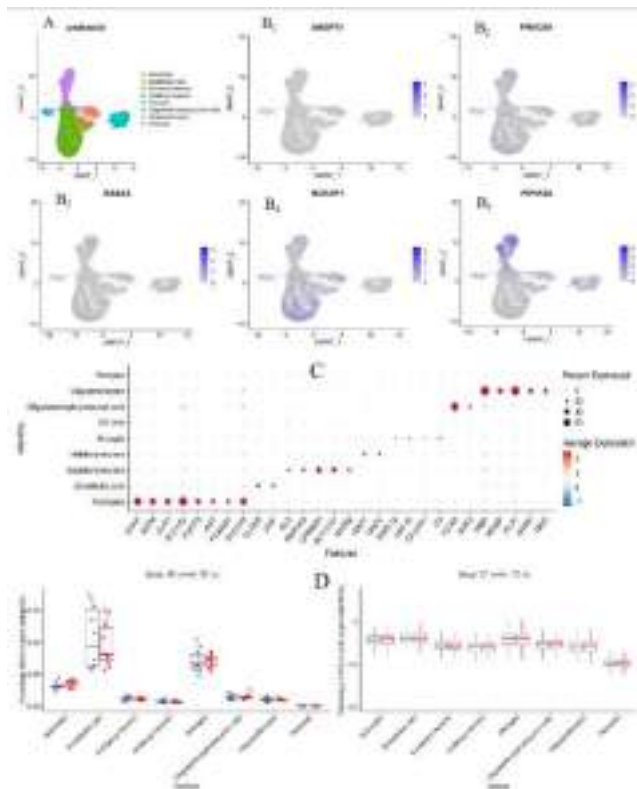


FIGURE 2 Results of tissue origin tracing by cumulative calculation of AD exosome markers. *A: UMAP plot of cell clustering; B1-5: UMAP plots of single gene expression distribution; C: Gene bubble plot; D: Box plot of cell proportion.

TABLE 1 Diagnostic Performance of Multi-Marker Models (AD vs. HC, Validation Cohort)

Model	AUC (95% CI)	Sensitivity (%)	Specificity (%)	Accuracy (%)
Logistic Regression (LR)	0.87 (0.81–0.93)	95	98	96
Support Vector Machine (SVM)	0.87 (0.81–0.93)	95	98	96.5
Random Forest (RF)	0.88 (0.82–0.94)	96	99	97

Conclusion: A four-protein plasma EV panel (PRKCSH, ANGPT1, NCKAP1, FLJ78516) shows high diagnostic potential, likely originating from brain endothelial cells and microglia. This non-invasive panel advances toward a clinically applicable AD blood test.

Disclosure: Nothing to disclose.

EPO-0334 | Alzheimer’s disease, environment and genes – looking at the unexpected

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Background and aims: Atypical forms of Alzheimer’s disease (AD) are characterized by predominant symptoms in nonmemory cognitive domains. Apart from different clinical manifestations, these variants also have a younger age of onset, which

may suggest a higher genetic contribution. With this work we aimed to study family history in AD subtypes, as a proxy of genetic contribution to their development.
Methods: Family history and APOE status in patients with typical and atypical AD diagnosis supported by biomarkers. Non-parametric tests (χ^2) were used to verify differences between groups. Logistic regression models were performed to investigate the association between family history and APOE status and the development of an atypical variant.
Results: The study included 392 patients: 293 with typical AD and 99 with atypical AD (53 frontal, 33 logopenic, and 14 posterior variants). A positive family history was more frequent in typical AD (48.5%) than in atypical AD (33.3%; $\chi^2 = 6.775$, $p = 0.009$). Family history prevalence did not differ significantly among atypical variants ($\chi^2 = 1.107$, $p = 0.575$). APOE $\epsilon 4$ carriage was also more common in typical AD (50.5%) compared to atypical AD (38.4%; $\chi^2 = 4.852$, $p = 0.028$). In logistic regression analyses, the presence of an atypical AD variant was inversely associated with a positive family history ($\beta = -0.548$, 95% CI 0.347–0.963, $p = 0.035$) and APOE $\epsilon 4$ status ($\beta = -0.495$, 95% CI 0.371–1.000, $p = 0.050$).
Conclusion: Our results suggest that genetic factors have a lower contribution to the development of atypical variants than typical AD, suggesting the contribution of other factors, namely environmental, warranting further studies.
Disclosure: Nothing to disclose.

EPO-0335 | Effects of different exercise modalities on cognitive function in older adults: A systematic review and network meta-analysis

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Background and aims: Globally, one in four older adults experiences cognitive impairment. Physical exercise has positive cognitive effects; however, the optimal exercise modality remains to be determined. Therefore, we aimed to systematically review and analyse the effects of different exercise modalities on cognitive function domains in older adults.

Methods: We included published randomised clinical trials from seven different databases on April 8, 2025, involving older adults (≥ 60 years), with or without cognitive impairment, randomised to supervised resistance (RE), aerobic (AE) and/or combined resistance and aerobic exercise (COMB) for at least 4 weeks. Outcomes included global cognition, attention, executive function and memory. Meta-analysis was undertaken with a three-level mixed effects model, and results were expressed as standardised mean difference (SMD) and 95% confidence intervals.

Results: We included 127 randomised controlled trials ($n = 10,433$) comprising 780 cognitive outcomes. Exercise significantly improved overall cognition (0.24 [0.17–0.30], $p < 0.001$). Similar benefits were observed following COMB (0.27 [0.09–0.45], $p = 0.005$) and RE (0.26 [0.14–0.37], $p < 0.001$), with

smaller effects for AE (0.18 [0.11–0.26], $p < 0.001$). Exercise improvements were mainly for global cognition (0.42 [0.26–0.57], $p < 0.001$), followed by complex attention (0.18 [0.09–0.27], $p < 0.001$), executive function (0.14 [0.07–0.21], $p < 0.001$) and learning and memory (0.14 [0.05–0.24], $p < 0.001$). No significant effects were observed for language (0.09 [–0.03–0.21], $p = 0.130$) or perceptual-motor function (0.05 [–0.11–0.22], $p = 0.510$).

Conclusion: Our findings indicate that resistance-based exercise programs significantly improve specific cognitive domains in older adults, particularly attention, executive function, and learning/memory. These results underscore the importance of exercise to prevent cognitive decline and to support individuals presenting with cognitive impairment.

Disclosure: Nothing to disclose.

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EPO-0336 | Cognitive and structural brain changes in ofatumumab-treated multiple sclerosis: A longitudinal study

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Background and aims: Anti-CD20 therapies control inflammatory activity in multiple sclerosis (MS), but their effects on neurodegeneration and cognition in real-world settings remain uncertain. We aimed to characterize longitudinal cognitive and regional brain structural changes in ofatumumab-treated MS patients. We also evaluated structure–cognition associations over time.

Methods: We conducted a longitudinal observational study of relapsing MS patients treated with ofatumumab. Cognitive performance was assessed using the Brief International Cognitive Assessment for Multiple Sclerosis (BICAMS), with processing speed as the primary outcome, and Magnetic Resonance Imaging (MRI) measures included thalamic volume, deep gray matter volume (DGM), and cortical thickness (Cth). Longitudinal changes and structure–cognition associations were analyzed using linear mixed-effects models.

Results: Eighty-five RMS patients treated with ofatumumab (mean age 37.9 ± 9.9 years; 69.4% female; 222 MRI scans) were included. Brain atrophy persisted but attenuated after 12 months: whole-brain volume -0.15% vs -0.09% ; DGM -1.01% to -0.36% ; thalamus -0.94% to -0.77% ; CTh -1.67% to -0.62% (all p for slope changes > 0.05). Cognitive performance improved (Symbol Digit Modalities Test, SDMT $+3.79$ points/year, 95% CI 1.35–6.23; $p = 0.003$), with improvements in verbal and visuospatial memory (all $p < 0.05$). No global longitudinal structure–cognition association was observed. In subgroups, thalamic atrophy was associated with SDMT decline in patients with Expanded Disability Status Scale ≥ 3 ($\beta = -54.31$; $p = 0.013$), and DGM atrophy with cognitive worsening in patients with baseline SDMT $z \leq -1$ ($\beta = -55.91$; $p = 0.012$).

Conclusion: In ofatumumab-treated relapsing MS, neurodegeneration persists but slows over time while cognition is preserved, and structural–functional coupling emerges only in vulnerable patients.

Disclosure: Nothing to disclose.

EPO-0337 | From intravenous to subcutaneous ocrelizumab in a high-volume infusion center: A time-and-motion-informed capacity and service-flow analysis

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Background and aims: Subcutaneous (SC) ocrelizumab offers a streamlined alternative to intravenous (IV) infusion in the management of multiple sclerosis (MS). Building on time-and-motion (T&M) methodologies validated in oncology, we conducted a single-center operational study to quantify procedure-level time savings, estimate the resulting care-time dividend, and assess changes in patient flow following SC implementation.

Methods: We employed a cross-sectional observational design in a five-chair day hospital treating ~600 patients with MS treated with infusive therapies. T&M data were collected for 15 IV ocrelizumab administrations (April 2025) and 15 SC administrations (November 2025), capturing active clinical time across four phases: premedication, administration, observation, and checkout. Service-flow metrics (monthly visit volume, mean waiting time) were extracted from the electronic queue system for May 2025 (early SC implementation) and November 2025 (mature SC adoption). A deterministic operational model projected capacity gains under different SC adoption scenarios.

Results: SC ocrelizumab reduced total active clinical time from 320 to 60 minutes (-81%), driven by administration (210→10 min) and observation (60→15 min) reductions. At 15 SC administrations/month, this translated into 65 clinical hours and 50 chair-hours released—equivalent to 14 IV-equivalent infusion slots. Despite unchanged ocrelizumab volumes, monthly visits increased ($+7.9\%$) and mean waiting time decreased (-30%) from May to November 2025. Scenario modeling projected 100–133 chair-hours released at 30–40 SC administrations/month.

Conclusion: Transitioning from IV to SC ocrelizumab yields substantial procedural efficiencies and frees infusion-unit capacity. The care-time dividend enables expanded access to complex therapies without additional staff or infrastructure, positioning SC ocrelizumab as a structural efficiency tool in MS care.

Disclosure: Nothing to disclose.

EPO-0338 | Serum neurofilament light chain levels are associated with increased disability and impairment of specific cognitive domains in neuro-Behçet disease

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Background and aims: Neurological symptoms may emerge in around 10% of Behçet disease (BD) patients. Biomarkers for

monitoring the clinical outcome of neuro-BD have not been properly investigated.

Methods: Levels of neurofilament light chain (NFL), biomarker of neuroaxonal degeneration, were measured in sera of 27 patients with parenchymal neuro-BD (16 men, 48 ± 9 -year-old, 11 ± 5 years disease duration) and age/gender-matched 30 patients with relapsing-remitting multiple sclerosis (RRMS) and 25 healthy controls by the Siemens Healthineers Atellica Analyzer. Sera of all patients were obtained during remission. Potential correlations were sought between NFL levels and clinical-cognitive aspects of neuro-BD.

Results: NFL levels of neuro-BD patients (13 ± 6 pg/mL; range, 6–31 pg/mL) were comparable with RRMS patients and significantly higher than healthy individuals ($p < 0.0001$). In neuro-BD, NFL levels were correlated with EDSS ($p = 0.019$, $R = 0.445$), 9-hole peg test ($p = 0.002$, $R = 0.603$), timed 25-foot walk ($p = 0.001$, $R = 0.612$), visual-spatial domain of Addenbrooke's cognitive examination revised (ACE-R; $p = 0.015$, $R = -0.490$), Stroop interference test ($p = 0.016$, $R = 0.506$), complex figure test ($p = 0.039$, $R = -0.422$) and Wisconsin card sorting test (WCST) scores ($p = 0.004$, $R = 0.592$). No significant correlation was found between NFL levels versus disease duration, scores of MMSE and tests assessing verbal/visual memory, language and psychiatric symptoms.

Conclusion: Neuroaxonal degeneration is intimately involved in development of somatic disability, visual-spatial and executive dysfunction in neuro-BD, whereas memory impairment and psychiatric symptoms appear to occur via divergent disease mechanisms. ACE-R, Stroop and WCST tests might potentially be used to monitor cognitive impairment in neuro-BD.

Disclosure: Nothing to disclose.

EPO-0339 | Serum galectin-3 as a biomarker in acute inflammatory polyradiculoneuropathies: A cohort study

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Background and aims: Guillain-Barré Syndrome (GBS) refers to a group of acute inflammatory polyradiculopathies characterized by a diffuse inflammatory attack on the peripheral nervous system. In this study, we propose galectin-3 (Gal-3), a novel serum molecule identified expressed by Schwann cells and macrophages, as a potential biomarker in patients with GBS.

Methods: Serum levels of Gal-3 and neurofilament light chain (NfL) were measured using the Simple Plex cartridge-based immunoassay on the Ella platform in serum samples from a cohort of patients with GBS ($n = 19$) and in a validation cohort of healthy control subjects ($n = 13$). We also collected data on clinical history and patient outcomes.

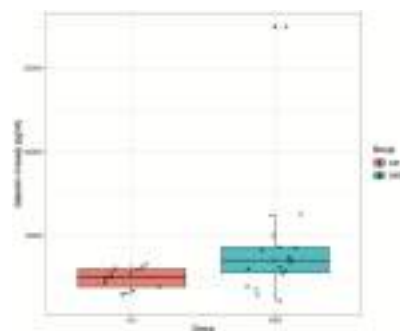


FIGURE 1 Boxplot of serum levels of Gal-3 in GBS patients and in control subjects.

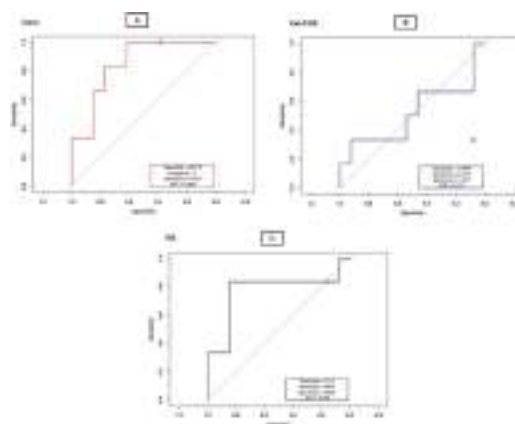


FIGURE 2 Discriminatory performance of Galectin-3 and Gal-3/NfL Ratio Between Axonal and AIDP GBS Patients. ROC curves demonstrating the diagnostic performance of serum Gal-3 levels (A), Gal3/NfL ratio (B), and serum NfL levels (C) in distinguishing the axonal variant.

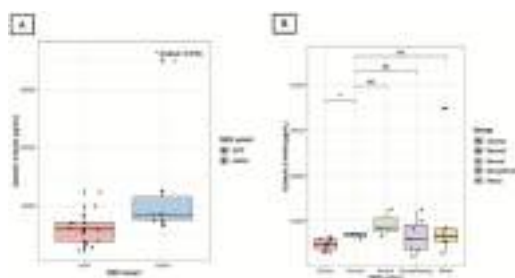


FIGURE 3 Boxplot of serum levels of Gal-3 in subgroup based on NCS findings firstly in axonal GBS variant (AMAN or AMSAN) and AIDP patient (A) and in relation to electrophysiological findings (B).

Results: Gal-3 levels were significantly higher in patients with GBS (median: 7041 pg/mL; IQR: 5576–8561) compared to healthy controls (median: 5030 pg/mL; IQR: 3877–5998). Within the GBS cohort, Gal-3 levels were significantly elevated in patients with the axonal variant compared to those with classical AIDP variant (median: 8561 pg/mL vs. median: 5998 pg/mL, $p < 0.05$).

In multivariate logistic regression, Gal-3 remained significantly associated with the axonal variant of GBS after adjustment for age at sampling, diarrhea, GBS Disability Score at admission, and log-transformed NfL. In ROC analysis, Gal-3 demonstrated good discriminatory performance between axonal GBS and AIDP (AUC=0.84), outperforming NfL (AUC=0.76). Gal-3 threshold of 6457.5 pg/mL distinguish axonal GBS with 100% sensitivity and 61.5% specificity

Conclusion: In our cohort, Gal-3 was associated with both diagnostic and prognostic features in patients with GBS. These findings suggest that Gal-3 may serve as a potential biomarker for differentiating axonal subtypes within the GBS spectrum.

Disclosure: Nothing to disclose.

EPO-0340 | Frequency of anti-neural antibodies and autoimmune epilepsy in focal epilepsy of unknown etiology: An observational study in a singaporean cohort

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Background and aims: Detection of anti-neural autoantibodies for autoimmune epilepsy (AES) is crucial for early immunotherapy for improved outcomes. Conventional cell-based assays (CBA) test for limited panel of known autoantibodies. Immunohistochemistry (IHC) using rodent brain tissue represents a more comprehensive repertoire. Prevalence of AES and anti-neuronal antibodies in this cohort is unknown in Singapore. We describe here 1. Establishment of IHC assay and comparison to CBA sensitivity and prevalence 2. Comparing clinical characteristics between seropositive and seronegative patients 3. Clinical utility of the ACES (Antibodies Contributing to Focal Epilepsy Signs and Symptoms)score.

Methods: This is a cross-sectional, prospective, single-center study performed at the National Neuroscience Institute (NNI), Singapore. Inclusion criteria were patients with focal epilepsy of unknown etiology; able to provide informed consent; ≥18years old and had at least 1 seizure in the last 1 year prior to recruitment. Demographic data, medical history and epilepsy information were collected. ACES scores were calculated. Patients were stratified into definite/probable/possible/non-AES using IHC and CBA. Clinical characteristics, MRI, EEG and ACES scores were analyzed.

Results: 120 patients were included. Demographics and clinical data are detailed in Table 1. Figure 1 shows selected staining patterns and table showing seropositive IHC and/or CBA. Clinical characteristics of seropositive patients are detailed in Table 2A. Comparative analysis of possible AES and non-AES patients are shown in Table 2B. Diagnostic performance of ACES score is shown in Table 2C.

Table 1. Demographic and clinical characteristics of the study population (n=120)

Demographics	
Age (mean ± SD), median (IQR), range	46.0 (10.0, 11.0)
Female, n (%)	41 (34.2)
Educational level (years), median (IQR), range	12.0 (9, 16)
Race	
Chinese, n (%)	96 (80.0)
Malay, n (%)	10 (8.3)
Indians, n (%)	11 (9.2)
Others, n (%)	3 (2.5)
Past medical history	
Age of 1st seizure (years), median (IQR), range	4.0 (3, 11)
Subsequent seizures, n (%)	97 (81.7)
Current seizure, n (%)	7 (5.8)
Posterior reversible amnesia, n (%)	1 (0.8)
Epilepsy history	
Seizure frequency (per year), median (IQR), range	2.0 (0.5, 14.0)
Age of seizure onset (years), median (IQR), range	10.0 (7.0, 11.0)
Age diagnosed with epilepsy (years), median (IQR), range	12.0 (9.0, 14.0)
Frequency of seizures in the last 12 months, median (IQR), range	2.0 (0.5, 11.0)
Frequency of seizures in the last 4 months, median (IQR), range	2.0 (0.5, 5.0)
Previous status epilepticus, n (%)	9 (7.5)
Medically refractory epilepsy, n (%)	18 (15.0)
History of current antiepileptic medications, median (IQR), range	1.0 (0.5, 10.0)
Seizure semiology	
Social history without impairment of consciousness, n (%)	90 (75.0)
Social history with impairment of consciousness, n (%)	30 (25.0)
Social history resulting in behavioral modification, n (%)	10 (8.3)
Has behavioral epileptic seizure, n (%)	10 (8.3)
Others, n (%)	4 (3.3)
Other associated clinical features	
Cognitive impairment, n (%)	8 (6.7)
Depressive disorder, n (%)	9 (7.5)
Autism spectrum disorder, n (%)	6 (5.0)
Speech problems, n (%)	1 (0.8)
ADHD, n (%)	10 (8.3)
Medication findings	
Alleviated, n (%)	40 (33.3)
Medication resistant (CR), n (%)	80 (66.7)
Clinical response to AEDs, n (%)	80 (66.7)
Pharmacological enhancement, n (%)	80 (66.7)
Pharmacological enhancement, n (%)	80 (66.7)
CR, n (%)	80 (66.7)
IHC findings	
Seropositive, n (%)	9 (7.5)
Seropositive, n (%)	9 (7.5)
Seropositive, n (%)	9 (7.5)
Seropositive, n (%)	9 (7.5)

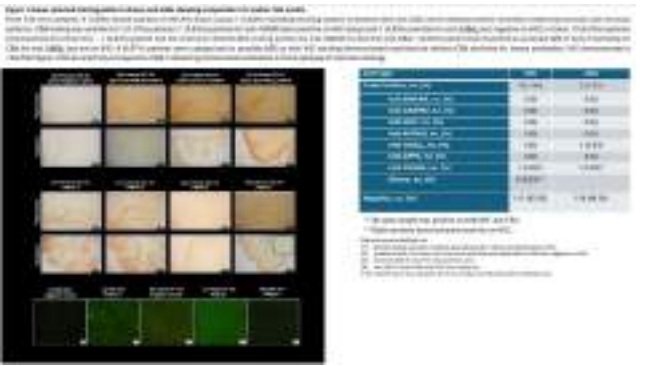


FIGURE 1 shows selected staining patterns shown and table showing seropositive IHC and/or CBA results.

Conclusion: The combination of clinical characteristics, IHC screening, and ACES scoring can improve AES detection, enabling earlier diagnosis and immunotherapy even in patients with negative CBA results.

Disclosure: This study was funded by the NNI RIE2025 CG Pilot Grant award to Jeanne May May Tan, AM-ETHOS grant awarded by Duke-NUS Medical School to Seong Jin Park and National Medical Research Council (Singapore) Research Training Fellowship grant awarded to Tianrong Yeo. Jeanne May May Tan is funded by the Singhealth CIVA award (06/2024/CIVA/04-A04). Tianrong Yeo is funded by the Singapore Ministry of Health's National Medical Research Council Clinician Scientist Award (CSAINV24jul-0004). Jeanne May May Tan has received honoraria from AstraZeneca, Merck, and Roche for speaker's fees, and research grants from the National Medical Research Council (NMRC Singapore) and Singhealth Pte Ltd. She has also received travel grants from

Roche and Merck. Tianrong Yeo has received honoraria from ASNA, AstraZeneca, Edanz Pharma, Euroimmun AG, Merck, Novartis, Roche, Terumo BCT for consulting services and speaker's fees, and research grants from the National Medical Research Council (NMRC Singapore), AstraZeneca and Roche. He has also received travel grants and awards from PACTRIMS, ACTRIMS, ECTRIMS, Orebro University, UCB, and Merck. The other co-authors declare no competing interest. This study was conducted in Singapore under local ethics approval (CIRB 2016/2469). Written informed consent was obtained from every study participant.

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Methods: Mechanistic studies used a dual-antigen (AQP4+MOG) EAE mouse model and SH-SY5Y cells. Interventions included GLP-1R antagonist Exendin-(9–39), agonist Exendin-4, and IL-6 inhibitor Satralizumab. Analyses comprised metabolomics, patch-clamp electrophysiology, and molecular assays.

Conclusion: GLP-1/GLP-1R-mediated glycolytic reprogramming via cAMP-PKA pathway drives AP neuronal hyperactivity in NMOSD-APS. Targeting GLP-1R signaling or IL-6-mediated



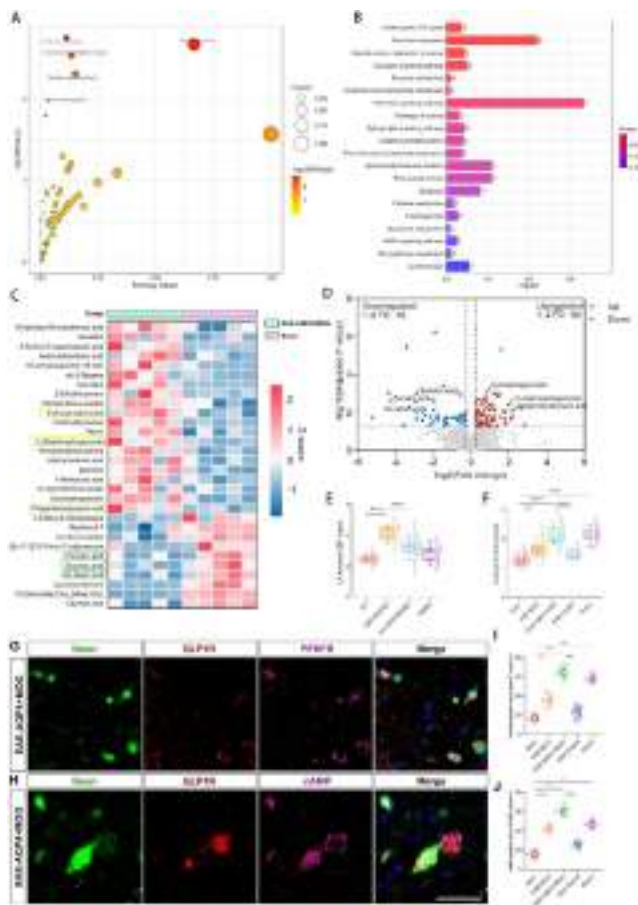


FIGURE 2 EAE mice metabolomics.

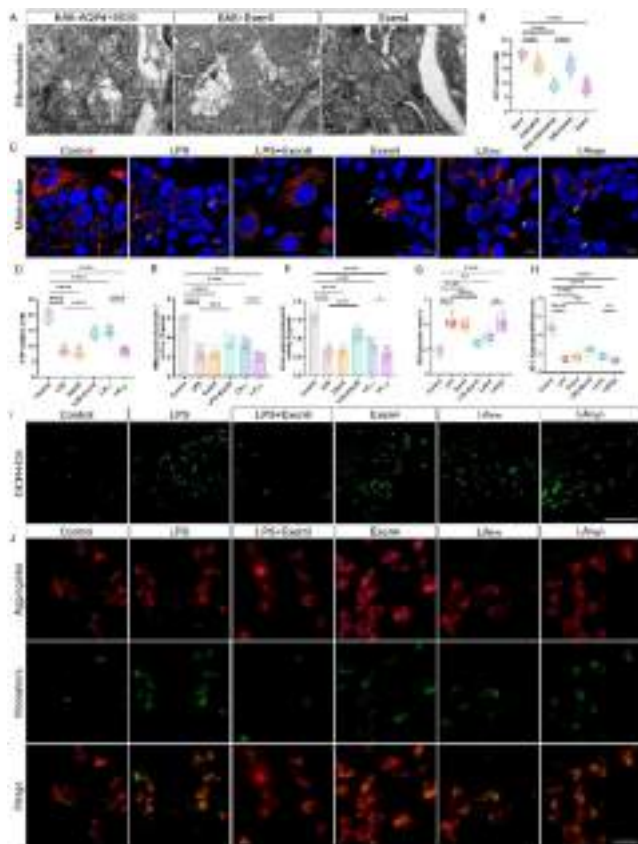


FIGURE 3 EAE mice mitochondrial dysfunction.

inflammation offers a novel therapeutic strategy by correcting both metabolic dysregulation and neuronal dysfunction. This study highlights the importance of metabolic-immune cross-talk in autoimmune neurological disorders and provides new insights into NMOSD pathophysiology.
Disclosure: Nothing to disclose.

EPO-0342 | Long-term and patient-reported outcomes (PROs) in patients with autoimmune encephalitis: A prospective cohort study

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Background and aims: Autoimmune encephalitis (AE) is a disorder presenting with acute neuropsychiatric manifestations and seizures. While AE may be associated with favorable recovery, prognosis can be variable. Outcomes in AE are traditionally assessed using the modified Rankin Scale (mRS) which fails to capture long-term effects on mood, sleep, and quality of life (QoL). Here, we evaluated a cohort of patients with AE using patient-reported outcomes (PROs) to provide a wider assessment of long-term prognosis.
Methods: We prospectively enrolled adult patients previously diagnosed with AE according to Graus criteria, with a minimum follow-up of two years. After collection of electroclinical data, the following validated PROs were administered: the Pittsburgh Sleep Quality Index (PSQI), Beck Depression Inventory-II (BDI-II), Hospital Anxiety and Depression Scale (HADS), and the Short Form Health Survey (SF-36).
Results: Thirty-two patients were enrolled (median age 67 years [IQR: 55–77]; 82% males), with a median follow-up of 7.5 years. At last follow-up, mRS scores ranged from 0 to 1 in 60% of patients. In contrast, the SF-36 revealed impaired quality of life in up to 80% of patients. Clinically relevant anxiety and/or depressive symptoms were observed in 71% of patients based on BDI-II and HADS scores, while 67% showed reduced sleep quality according to the PSQI. Diagnostic delay was associated with worse BDI-II ($p=0.046$) and PSQI scores ($p=0.022$).
Conclusion: Although most patients with AE show improvement on traditional mRS assessment at follow-up, a substantial proportion continues to experience mood disturbances and sleep impairment, which significantly affect their quality of life.
Disclosure: Nothing to disclose.

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Background and aims: Chronic Inflammatory Demyelinating Polyradiculoneuropathy (CIDP) is a heterogeneous immune-mediated neuropathy marked by diagnostic complexity and variable treatment responses. Reliable biomarkers for diagnosis and disease stratification are lacking. Lipoproteins have emerged as regulators of neuroinflammation and potential autoimmune targets. However, their role in CIDP is unknown. We investigated the lipid profiles in serum and cerebrospinal fluid (CSF) treatment-naïve CIDP patients.

Methods: Serum and CSF samples from CIDP patients at initial diagnosis and controls were analyzed using mass spectrometry-based lipidomics targeting glycerophospholipids and sphingomyelin species ($n=26$). Patients with idiopathic intracranial hypertension and diabetic neuropathy served as control groups ($n=18$, $n=22$). Lipidomic findings were integrated with proteomic data and longitudinal clinical scores, including therapy response and disease severity, and confounders like age, gender, and total CSF protein.

Results: Preliminary proteomic data reveal a differential increase in proteins involved in lipid metabolism in the CSF of CIDP patients (including APOA2, APOA4, and APOA1). The preliminary lipidomic analysis shows a significant decrease of phosphatidylinositol 32:1 in CSF. In contrast, the sphingomyelin 18:1 CSF-to-serum-ratio is markedly elevated, with significant increases in CSF and decreases in serum ($p < 0.05$).

Conclusion: Our findings provide evidence for lipid dysregulation in CIDP patients. Especially the altered CSF-to-serum ratio of 18:1 sphingomyelin species warrants further investigation. Further correlation with clinical measures and validation in independent cohorts are required to assess the potential of lipids as mechanistic biomarkers in CIDP.

Disclosure: The authors declare no conflicts of interest.

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Background and aims: To assess feasibility of a cognitive telerehabilitation protocol (CTR) in patients with autoimmune encephalitis (AE).

Methods: Patients with confirmed diagnosis of AE and neuropsychological impairment were prospectively recruited from November 2022 to November 2025. Participants were evaluated through a battery of neuropsychological testing and a comprehensive neurological evaluation at baseline (T0) and after 3 months (T1). At T0, patients and caregivers received a portable touch screen computer installed with a tailored telerehabilitation program (3 weekly, 25 min-sessions for a total of 12 weeks). User experience (UX) was assessed using the Italian-validated User Experience Questionnaire (UEQ) and the System Usability Scale (SUS). Health status was evaluated with the 12-Item Short Form Health Survey questionnaire (SF-12).

Results: Twelve subjects were included (4 LGII-AE, 2 GAD-AE, 3 CASPR2-AE, 1 seronegative-AE, 1 GABA-B-AE, 1 NMDAR-AE). A median of 100% of the sessions and 94.57% of the tasks were completed with a median SUS of 78.75. UEQ scores were above the benchmark level for all the items included in the analysis. Median MoCA scores were similar between T0 (21.5, range 14.6–30) and T1 (23, range 18–28), but cognitive performances significantly improved in attention as assessed by faster TMT-A ($p < 0.05$). In addition, patients showed an improved perception of physical health on the SF-12 questionnaire ($p < 0.05$).

Conclusion: CTR is a feasible and potentially effective approach to treat cognitive dysfunction in AE. Further prospective studies are needed to define specific protocols and timing of administration.

Disclosure: Nothing to disclose.

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Background and aims: This study evaluated the impact of intravenous/subcutaneous efgartigimod (EFG) on treatment burden in Japanese patients with generalized myasthenia gravis (gMG) between heavily pre-treated (HPT) and less heavily pre-treated (LHPT) cohorts.

Methods: We conducted a retrospective cohort analysis using hospital-based claims from Medical Data Vision of adult gMG patients initiating EFG between May 2022, and April 2025. Eligible patients had 12-month baseline and follow-up observation, oral glucocorticoids (GC) or non-steroidal immunosuppressants within 90-days pre-EFG initiation, and no other biologics during follow-up (Figure 1). HPT was defined as ≥ 2 baseline fast-acting treatment (FT) cycles (intravenous immunoglobulin, plasma exchange, intravenous methylprednisolone)



FIGURE 1 Patient Selection Criteria and Study Design. Eligible patients in the study must meet all the criteria listed for both baseline and the follow-up period.

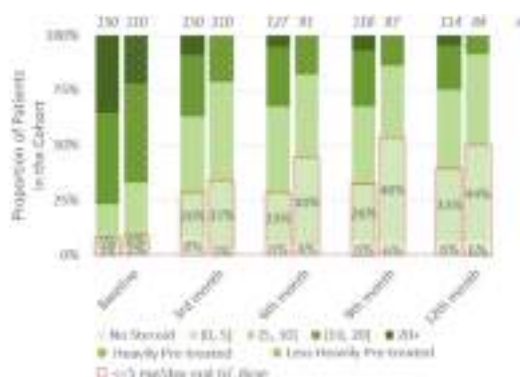


FIGURE 2 Oral GC ADD in Patients with Continuous EFG Use. ADD within 3rd, 6th, 9th, and 12th month follow-up was measured for the HPT (left) and LHPT cohorts (right), focusing on patients who continued EFG until the end of the months of measurement.

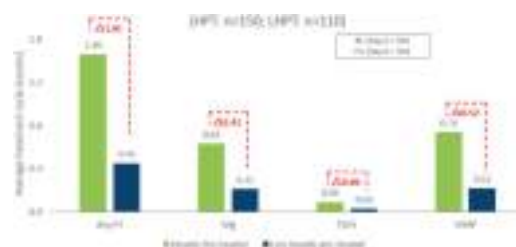


FIGURE 3: Average FT cycle count per patient in follow-up. Average FT (IVIg, PLEX, IVMP, sum of all) during 12-month follow-up was measured for the HPT (left) and LHPT cohorts (right).

or ≥ 20 mg/day prednisolone-equivalent oral GC dose within 90 days pre-EFG initiation; all others were LHPT. Outcomes were oral GC average daily dose (ADD) and FT cycles counts.

Results: Among 260 patients identified, 58% were HPT and 42% were LHPT. Mean duration from first recorded gMG diagnosis to index, and EFG treatment duration did not differ between cohorts (HPT: 5.7 years, 455 days; LHPT: 5.9 years, 447 days; $p=0.635$, $p=0.816$ respectively). Mean oral GC ADD approximately halved after EFG initiation in both cohorts (HPT: 19.1 to 9.6 mg/day, LHPT: 14.2 to 7.1 mg/day, baseline to follow-up). LHPT had higher proportion of patients achieving ≤ 5 mg/day throughout follow-up (Figure 2) and one-third as many FT cycles per patient versus HPT during follow-up (0.45 vs. 1.46, Figure 3).

Conclusion: LHPT consistently showed lower treatment burden, while effective oral GC tapering was observed in both cohorts. EFG initiation in earlier disease management may improve real-world treatment outcomes.

Disclosure: RA, HT, KT, and CB are employees of argenx. MPT reports unrestricted research grants from Japan Blood Products Organization and Astellas Pharma outside the submitted work, has served as a paid consultant for Alexion, Argenx, Hanall BioPharma, and UCB Pharma, and received honoraria for lectures from Argenx, Alexion Pharmaceuticals, and UCB Pharma. YZ, JL, and SI are employees of ZS Associates and serve as paid consultants for argenx.

EPO-0346 | Conformational antibodies to PLP1/DM20 define a clinical subgroup within immune mediated neuropathies

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Background and aims: We have previously identified conformational antibodies to proteolipid protein-1 (PLP1) and its peripheral isoform DM20 in patients with combined demyelinating disorders of the peripheral and central nervous systems. However, their presence in IMN has never been investigated.

Methods: We implemented a DM20 live cell-based assay and used it to screen a retrospective exploratory cohort of defined IMN ($n=294$), a prospective validation cohort of suspect IMN ($n=324$), and controls ($n=81$). Clinical features were compared with a cohort of DM20-IgG-negative chronic inflammatory demyelinating polyneuropathy (CIDP, $n=43$).

Results: DM20-IgG were identified in 20 of 537 cases (3.7%) of IMN and in none of the controls. DM20-IgG-positive IMN fulfilled the criteria for CIDP ($n=12$) or Guillain-Barré syndrome (GBS; $n=8$). Most presented with acute onset (17/20) and had a preceding trigger (11/20, of which 5 SARS-CoV-2 vaccination). Compared with "typical CIDP", DM20-IgG positive patients showed frequent cranial nerve involvement (12/20 vs 10/43, $p=0.009$), pain (12/20 vs 11/43, $p=0.012$) and higher disability at acme (INCAT total score 4 [1–9] vs 2 [0–8], $p=0.06$). A satisfactory response to intravenous immunoglobulin was obtained in 17/18 patients (94%). DM20-IgG predominantly pertained to IgG1 (12/20) and bound myelin on rat teased nerve fibres, live oligodendrocytes and myelinated Schwann cell segments.

Conclusion: DM20-IgG defines a subgroup of IMN characterized by acute onset, cranial nerve involvement, severe disability and a favorable response to immunotherapy. Their ability to bind their target on live cultures might suggest pathogenicity, that needs to be explored in future studies.

Disclosure: Nothing to disclose.

EPO-0347 | Abstract withdrawn

Cerebrovascular Diseases 4

EPO-0348 | Is selective screening safe for stroke prevention? High rates of missed severe stenosis in Pre-CABG: Validating ESVS 2023

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Background and aims: Perioperative stroke is a devastating complication of coronary artery bypass grafting (CABG), frequently driven by occult carotid atherosclerosis. The 2023 ESVS guidelines advise against routine carotid screening, favoring selective screening only for high-risk subsets (Age > 70, Stroke/TIA, Left Main Disease). However, the safety of this restrictive strategy in populations with high metabolic burden is unproven. We validated whether following these guidelines leaves patients at risk of undetected carotid stenosis and subsequent cerebrovascular events.

Methods: We analyzed 203 consecutive patients scheduled for CABG at King Abdullah University Hospital (Jordan). All patients underwent comprehensive neurovascular imaging (Duplex/CTA). We stratified the cohort based on ESVS selective criteria to determine the "miss rate" for hemodynamically significant internal carotid artery (ICA) stenosis ($\geq 50\%$). Multivariate logistic regression identified independent predictors of carotid disease relevant to neurological risk.

Results: The cohort had a high metabolic burden (Diabetes: 67%, Hypertension: 85%). Overall Carotid Artery Disease prevalence was 54.2%, with significant ICA stenosis ($\geq 50\%$) in 17.7% ($n=36$). Crucially, strict adherence to ESVS 2023 selective criteria would have failed to detect 11.1% ($n=4$) of patients with significant stenosis. These "missed" patients were asymptomatic and younger (< 70 years) yet possessed critical plaque burden. In multivariate analysis, Left Main Coronary Disease (OR 1.66, 95% CI 1.05–2.8) and Hypertension (OR 2.47) were independent predictors of significant carotid stenosis.

Conclusion: In a high-risk cohort, reliance on "selective screening" misses > 11% of patients with significant carotid stenosis, leaving them vulnerable to preventable perioperative stroke. To optimize neuroprotection, we propose that Left Main Disease should trigger mandatory carotid screening.

Disclosure: Nothing to disclose.

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Background and aims: Acute ischemic stroke (AIS) is a time-critical emergency in which rapid diagnosis within established therapeutic windows is essential to optimize outcomes. Fluid biomarkers offer a promising adjunct to clinical and neuroimaging assessment but their temporal dynamics in the acute phases remain incompletely characterized.

Methods: We performed an umbrella review of systematic reviews and meta-analyses evaluating fluid biomarkers in AIS versus controls or stroke mimics. Quantitative synthesis of primary studies (random-effects meta-analysis of standardized mean differences) was stratified by clinically relevant time windows. Heterogeneity, small-study effects, and excess significance bias were assessed.

Results: We included 27 publications (18 biochemistry, 1 metabolomic, 10 transcriptomic, 5 cell-free-DNA [cfDNA]). Across all time-points the largest effect sizes were observed for neuron-specific enolase (NSE), ischemia-modified albumin (IMA), d-Dimer, S100B, GFAP, and IL-6. Looking at metabolites, studies revealed early accumulation of lactate, succinate, glutamate and lysophosphatidylcholines, alongside depletion of arginine, citrulline and citrate. A catalogue of 220 micro-RNAs

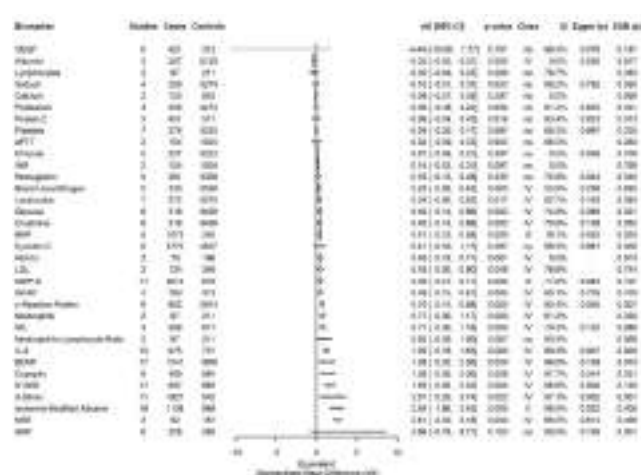


FIGURE 2 Forest plot of the meta-analyses for biochemistry biomarkers, comparing acute ischemic stroke (AIS) vs controls (HCs and mimics) across all time-points.

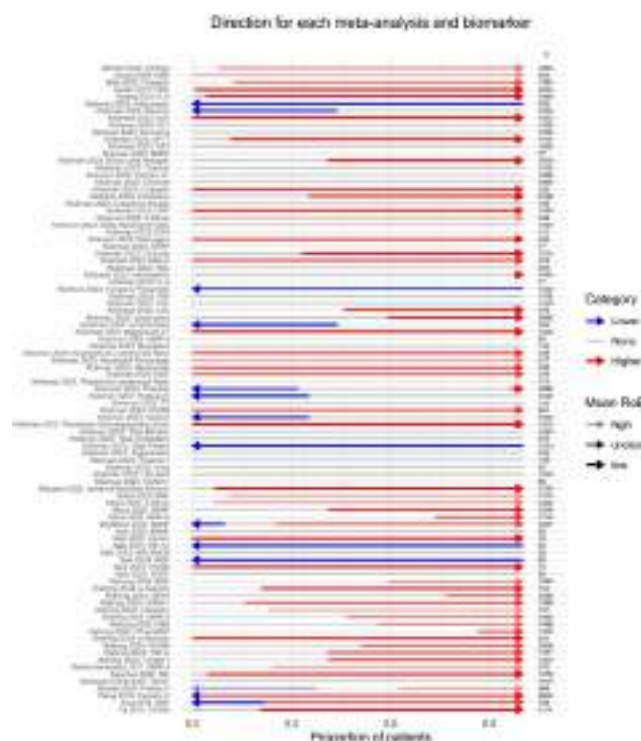


FIGURE 3 Arrow plot of the biochemistry biomarkers, comparing acute ischemic stroke (AIS) vs controls across all time-points. For each biomarker we reported the direction of the studies and the total number of patients across studies.

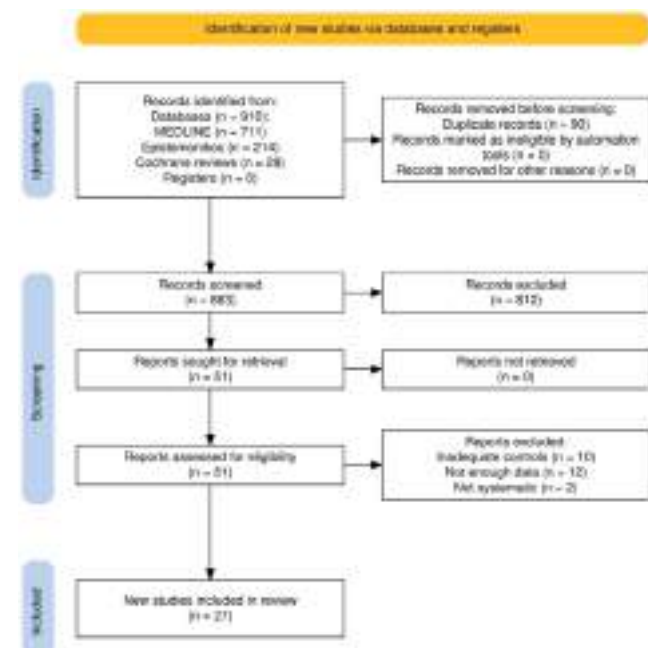


FIGURE 1 Prisma flowchart of the selected studies.

(132 upregulated; 108 downregulated) identified robust markers (miR-16-5p, let-7e-5p, miR-107, miR-451a, miR-126-3p). 46 circulating-RNAs, and 55 long-non-coding-RNAs were consistently dysregulated. Five studies reported elevated nuclear and mitochondrial cfDNA within 6h.

Conclusion: Fluid biomarkers exhibit a temporally evolving signature: early coagulopathy (D-dimer), glial activation (GFAP, S100B) and inflammation (IL-6), followed by neuronal necrosis (NSE) and oxidative stress (IMA) within 24 h. Multi-omic integration, including metabolomics, transcriptomics and cfDNA,

highlights convergent pathways (PI3K/Akt, NF-κB, immunometabolism) and supports the development of rapid, point-of-care panels. Standardized sampling windows and harmonized assay protocols are essential for clinical translation and prospective validation in prehospital settings.

Disclosure: Nothing to disclose.

EPO-0350 | Why genetics can't be ignored in secondary stroke prevention: The CYP2C19 challenge in Asia

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Background and aims: Stroke is a leading cause of death and disability globally, with Asia disproportionately affected. A critical barrier to effective secondary prevention is the high prevalence of CYP2C19 loss-of-function alleles, present in almost 75% of South and East Asians, which reduce clopidogrel efficacy.

Methods: Evidence from trials including CHANCE-2, shows that genotype-guided alternatives, including ticagrelor and cilostazol, substantially lower recurrent stroke risk in loss-of-function carriers. Yet, clinical adoption remains limited by insufficient genetic testing infrastructure, cost, guideline gaps, and clinician training.

Results: Despite these challenges, genotype-guided therapy is both feasible and cost-effective, with potential to reduce recurrent strokes, disability, and healthcare burden.

Conclusion: Urgent action is required to implement precision antiplatelet strategies, update guidelines, and ensure equitable access, making pharmacogenomics a central component of stroke care in Asia.

Disclosure: Nothing to disclose.

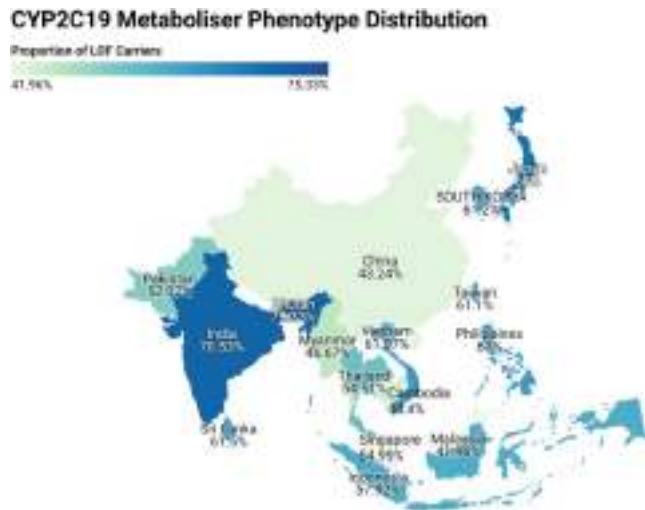


FIGURE 1 Geographic distribution of CYP2C19 intermediate (IM) and poor metabolisers (PM) rates across selected Asian countries. 3, 9, 11 Shading intensity corresponds to the combined prevalence of these phenotypes.

EPO-0351 | Cerebral small vessel disease in early- vs late-onset Alzheimer's disease in a memory clinic cohort

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Background and aims: Cerebral small vessel disease (CSVD) frequently co-occurs with Alzheimer's disease and increases with age. Here, we evaluated CSVD burden in early onset and late-onset Alzheimer's disease (EOAD and LOAD), distinguishing between hypertensive arteriopathy (HTNA) and cerebral amyloid angiopathy (CAA) co-pathology, as they have different underlying mechanisms.

Methods: We retrospectively included 59 EOAD and 162 LOAD (clinically-biologically diagnosed), retrieving clinical features including vascular risk factors. CSVD features were rated on MRI using established criteria, deriving HTNA and CAA prevalence. CSVD burden was compared between EOAD and LOAD, adjusting for sex and the difference between age at MRI and age at symptom onset.

Results: A probable CAA was present in 15.3% of EOAD and 29.6% of LOAD, while a moderate/severe HTNA was observed in 10.2% of EOAD and 30.2% of LOAD. LOAD present with higher rates of hypertension, diabetes, and atrial fibrillation ($p < 0.05$). LOAD showed a greater CSVD burden, driven primarily by more severe white matter hyperintensities, lacunes and perivascular spaces load ($p < 0.05$). HTNA severity and probable CAA prevalence were higher in LOAD ($p < 0.001$ and $p = 0.035$, respectively), despite the absence of significant differences in hemorrhagic features.

Conclusion: LOAD is associated with heavier vascular risk and CSVD co-pathology, in terms of both HTNA and CAA. The difference between groups seems to be mainly driven by CSVD features affecting the white matter, more severe in LOAD. However, CSVD burden in EOAD is not negligible, supporting routine vascular co-pathology assessment across age.

Disclosure: Nothing to disclose.

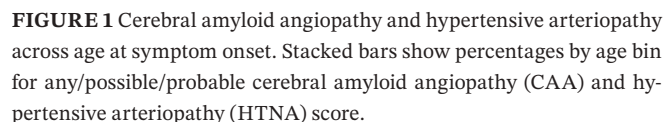
TABLE 2 Magnetic resonance imaging (MRI) markers of cerebral small vessel disease in early-onset ($n=59$) and late-onset ($n=162$) Alzheimer's disease.

Values are mean ± SD, median (SD), or n (%)				
	Early (n = 99)	Late (n = 162)	P-value	Sig.
Peripartum WMI fascicle score				
	1 (3-1)	1 (1-2)	<0.001*	***
Deep WMI fascicle score				
	1 (3-1)	1 (1-2)	<0.001*	***
PfV CSD score				
	2 (1-3)	3 (2-3)	<0.001*	***
PfV CSD (n)				
	21/29 ± 13.47	25/51 ± 10.08	0.0035	**
PfV B6 score				
	1 (1-2)	2 (1-3)	<0.001*	***
PfV B6 (n)				
	8/80 ± 5.14	12/38 ± 7.28	<0.001*	***
Deep L axon (n)				
	0.03 ± 0.18	0.22 ± 0.46	0.0065	**
Other L axons (n)				
	0.00 ± 0.00	0.14 ± 0.44	0.0155	*
aUS fetal number				
	0.03 ± 0.18	0.19 ± 0.38	0.1895	
aUS focal presence				
	2 (3.4%)	8 (4.9%)	0.818*	
aUS disseminated presence				
	0 (3.4%)	5 (3.1%)	0.904*	
Other MB (n)				
	0.05 ± 0.23	1.58 ± 0.58	0.1345	
Cardiac/vascular MB (n)				
	0.01 ± 0.23	1.75 ± 0.51	0.1735	
Deep MB (n)				
	0.00 ± 0.00	0.28 ± 0.28	0.0885	
Subcortical MB (n)				
	0.00 ± 0.20	0.18 ± 1.25	0.4825	

n = values for non-included patients are listed (1/Minors) CSD region

MADE-derived p-values are presented * actual made; * logistic codes; 3 lines count

MADE data: PfV CSD score (n), PfV CSD (n), PfV B6 score (n), PfV B6 (n), (n)



EPO-0352 | Medium-vessel occlusion recanalisation after medical management, and clinical outcome

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Methods: From our local stroke registry we included all consecutive patients with MeVO-AIS receiving CTr or IVT. IPR was defined as complete recanalisation on 12–24-hour CTA/MRA. Clinical outcome was 24h-NIHSS and 3-month modified-Rankin-Scale (mRS). The association of treatment (CTr vs. IVT) and recanalisation (IPR vs. no IPR) with clinical outcomes was assessed using propensity-score-weighted regression models. Weighting accounted for pre-stroke mRS, baseline NIHSS, age, sex, onset-to-door time, MeVO localisation, vascular risk factors.

Results: Among 229 patients with MeVO, 52 received CTr and 177 IVT. Overall, 114 had IPR. Patients with IPR had more M3 occlusions, fewer P1-P3 occlusions, and shorter onset-to-door times. IVT was significantly associated with IPR (aOR = 16.8,

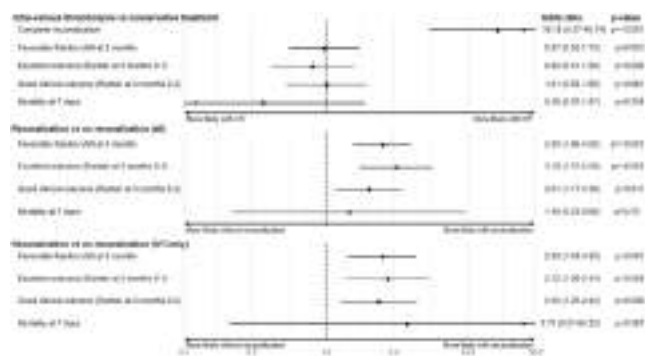


FIGURE 1 Forest plot of binary/categorical outcomes.

95% CI=5.37–48.74) but not with 3-month mRS. In the overall cohort, IPR was associated with greater 24-hour NIHSS improvement (beta-coeff=1.93, 95% CI=0.39–3.47), better 3-month mRS (pswOR=2.50, 95% CI=1.56–4.00), and higher rates of excellent outcome (mRS 0–1, pswOR=3.10, 95% CI=1.72–5.59). The same associations between IPR and clinical outcomes were observed in the cohort of IVT-treated patients

Conclusion: In MeVO-AIS treated with medical management, IPR is associated with improved functional outcome. While IVT increased the likelihood of IPR, persistent occlusion remained frequent, suggesting that more effective revascularisation strategies are needed to improve recanalisation rates and thus clinical outcomes.

Disclosure: François Fenter: nothing to disclose. Vincent Dunet: nothing to disclose. Patrik Michel: nothing to disclose. Davide Strambo: advisory board for Boehringer Ingelheim.

EPO-0353 | Neuroanatomical correlates in stroke-related ipsilateral hemiparesis: Single center experience and review of the literature

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Background and aims: Ipsilateral hemiparesis (ILH) in stroke is rare and challenges the conventional understanding of motor system organization. We aim to describe ILH strokes from our academic stroke center, complemented by a literature review.

Methods: All patients with hemiparesis and an acute ischemic lesion on neuroimaging were selected from 2003–2023 in our stroke registry. ILH cases were identified, and all their investigations were reviewed, including MRI-tractography, functional MRI, and TMS (if performed). We also performed a review of the literature of ILH cases in ischemic and hemorrhagic strokes. We compared all identified cases with all patients with contralateral hemiparesis in our local registry.

Results: We found 4 ILH cases among 2,426 consecutive hemiparetic stroke patients (0.16%). MRI-tractography showed physiological corticospinal tract decussation and fMRI found bilateral motor activation in all 4. The systematic literature review

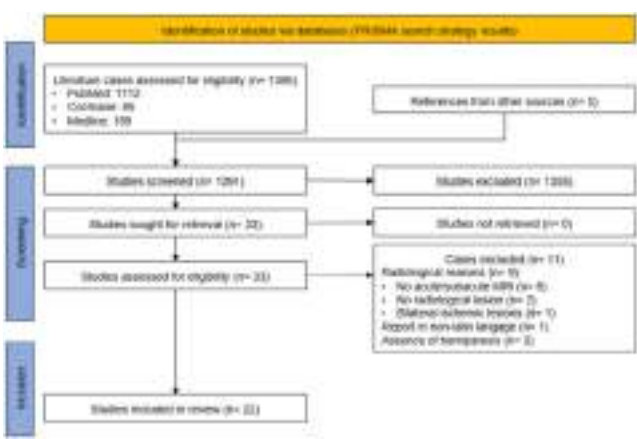


FIGURE 1 PRISMA search strategy results realized in December 2024.

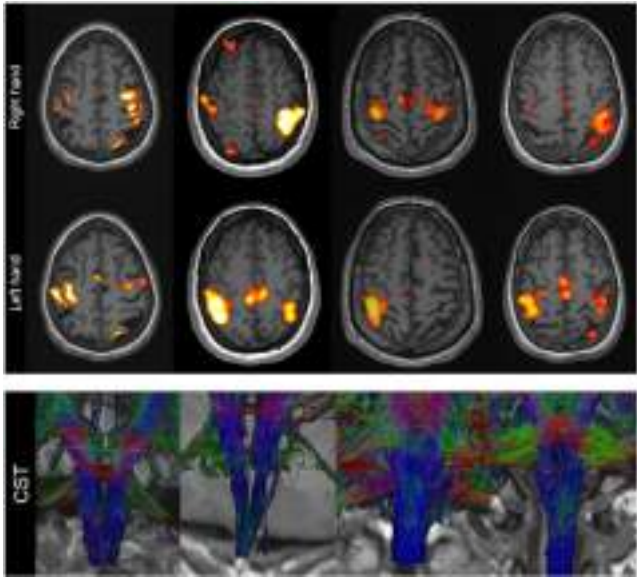


FIGURE 2 fMRI (upper rows), and MR-tractography (third row) of our 4 ILH patients who all had left MCA ischemic strokes.

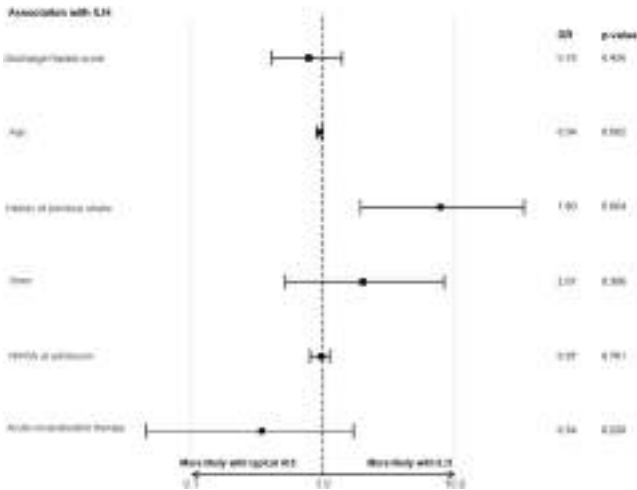


FIGURE 3 Multivariable binary regression model showing patients' ad stroke features independently associated with ILH. Results expressed as odds ratio (OR) and p-value. X-axis on logarithmic scale.

yielded 61 additional cases of ILH related to stroke, resulting in a total of 14 TMS, 18 fMRI, and 14 MRI-tractographic exams. In multivariable analysis, ILH was associated with younger age (beta=0.94; $p < 0.01$) and history of previous stroke (OR=7.99; $p < 0.01$). The additional investigations revealed evidence of corticospinal tract reorganization, aberrant interhemispheric connectivity, corticospinal tract anatomy variation, and involvement of secondary motor area as potential mechanisms.

Conclusion: ILH is a rare manifestation of acute stroke and seems to occur more frequently in younger patients with prior strokes. Our findings suggest that some ILH result from corticospinal reorganization and recruitment of secondary motor pathways, potentially highlighting the importance of neuroplasticity in post-stroke motor control.

Disclosure: Nothing to disclose.

EPO-0354 | **Clinical impact of systemic inflammatory response in ischaemic stroke due to atherosclerotic tandem lesions**

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Background and aims: Systemic inflammatory response (SIR) is frequently elevated in patients with atherosclerotic ischaemic stroke. This study aimed to assess the clinical impact of SIR in patients with tandem lesions treated with mechanical thrombectomy (MT) and emergent carotid artery stenting (eCAS).

Methods: We analysed data from the prospective ARTISTA registry, including patients with ischaemic stroke secondary to atherosclerotic tandem lesions treated with MT and eCAS between 2019 and 2023. Baseline SIR were assessed using the neutrophil-to-lymphocyte ratio (NLR) on admission. Associations between NLR and acute carotid stent thrombosis (< 24 hours), symptomatic intracranial haemorrhage (sICH), poor functional outcome (modified Rankin Scale [mRS] > 3), and 3-month mortality were evaluated using multivariable binary logistic regression models adjusted for age, sex, and predefined covariates.

Results: 149 patients were included; 78.5% were male, with a median age of 67years (IQR 60–77). The median NLR was 1.13 (IQR 0.67–2.16). Acute stent thrombosis occurred in 9 patients (6.7%), sICH in 14 (9.4%), poor functional outcome in 63 (42.3%), and 30 patients (20.1%) died within 3 months. After adjustment for age and sex, higher baseline NLR remained independently associated with acute carotid stent thrombosis (adjusted odds ratio [aOR] 1.08, 95% CI 1.01–1.16; $p=0.02$), but not with sICH, poor functional outcome, or mortality. This association persisted after further adjustment for antiplatelet therapy.

Conclusion: Patients with tandem lesions and elevated baseline SIR are at increased risk of acute carotid stent thrombosis. These findings may guide future studies to identify patients who could benefit from intensified periprocedural antithrombotic strategies.

Disclosure: Nothing to disclose.

TABLE 1 Association between baseline neutrophil-to-lymphocyte ratio and clinical outcomes (p value calculated using multivariable linear regression adjusted for age and sex).

Systemic inflammatory index		p ^a
Patient count	Stent thrombosis	
622 (422-1200)	1346 (258-1298)	0.02
No sICH	sICH	
812 (486-1250)	2035 (344-1890)	0.06
mRS 0-2	mRS 3-6	
523 (429-1105)	890 (1283-1280)	0.88
TICI 0-2a	TICI 2b-3	
924	674 (405-1497)	0.99
TICI 0-2b	TICI 2c-3	
735 (421-1863)	150 (170-1678)	0.57
No mortality	Mortality	
578 (487-1200)	890 (1237-1878)	0.83
NHSS ≤10	NHSS >10	
481 (232.8-905)	522 (1370-1360)	0.23

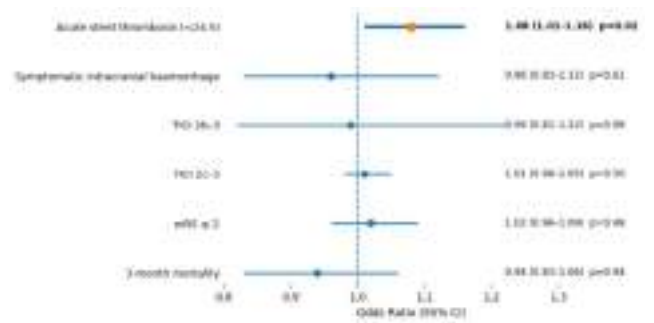


FIGURE 1: Association between baseline neutrophil-to-lymphocyte ratio and clinical outcomes (model adjusted for age, sex, and antiplatelet therapy).

EPO-0355 | **Impact of liver fibrosis on stroke outcomes in patients with intracranial atherosclerotic stenosis: A swiss cohort study**

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Background and aims: Chronic liver disease (CLD) is increasingly recognized as a contributor to cardiovascular disease, but its relationship with intracranial atherosclerotic stenosis (ICAS) and stroke outcomes remains underexplored. This study aimed to investigate the association between liver fibrosis, assessed by the FIB-4 score, and outcomes in acute ischemic stroke patients with/out ICAS.

Methods: We performed a retrospective observational cohort study using data from the Bernese Stroke Registry. A total of 500 patients with acute ischemic stroke (admitted 04/2022–12/2023) were categorized into four groups based on FIB-4 score (≤ 3.25 / > 3.25) and ICAS presence (yes/no). Clinical

characteristics, vascular risk factors, ICAS location, treatment, and 3-months outcomes were compared.

Results: Patients with high FIB-4 scores were significantly older (median 82.5 vs. 71 years, $p < 0.001$) and had a higher burden of atrial fibrillation and renal insufficiency (both $p < 0.001$). Arterial hypertension was more prevalent in ICAS groups (83.6/86.7% vs. 67.3/68.2%, $p < 0.001$), while hypercholesterolemia was less frequent in high-FIB-4 groups ($p = 0.028$). No significant differences were found in acute revascularization therapies. Patients with FIB-4 > 3.25 and ICAS had significantly poorer 3-months outcomes: only 10.3% achieved an excellent outcome ($mRS \leq 1$) compared to 36.3% in the low FIB-4/no ICAS group ($p = 0.030$), recurrent ischemic stroke was more frequent (7.1% vs. 1.6%; $p = 0.041$) and mortality was significantly elevated (43.3% vs. 13.3%; $p < 0.001$).

Conclusion: High liver fibrosis (FIB-4 > 3.25) is linked to worse outcomes in acute ischemic stroke, particularly with ICAS, including more recurrent stroke, and worse 3-months functional outcome, highlighting the value of assessing liver fibrosis in stroke prognosis.

Disclosure: None.

EPO-0356 | Stroke in people living with HIV and 6-month outcomes in northwestern Tanzania

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Background and aims: HIV patients face a higher risk of stroke, either ischemic or hemorrhagic, which is often associated with traditional risk factors. We aimed to evaluate the current trends, characteristics, and 6-month outcomes among people living with HIV (PLWH) compared to people without HIV (PWOH).

Methods: This prospective cohort study included 463 adults with stroke registered in the Lake Zone Stroke Registry at Bugando Medical Center between November 2023 and March 2025. Baseline characteristics, stroke severity, and outcomes were assessed using the National Institutes of Health Stroke Scale (NIHSS) and the modified Rankin Scale (mRS), respectively. The Kaplan-Meier analysis was used to describe survival, and the Cox regression model to determine predictors of mortality.

Results: We analyzed 295 adults, with an HIV proportion of 8.8% (95% CI: 5.9%–12.7%) (26/295). The mean age (SD) for the overall study cohort was 66.2 ± 37.4 years, and 57.2% (164/295) were males. Among PLWH, the mean age (SD) was 53.2 ± 14.5 years,

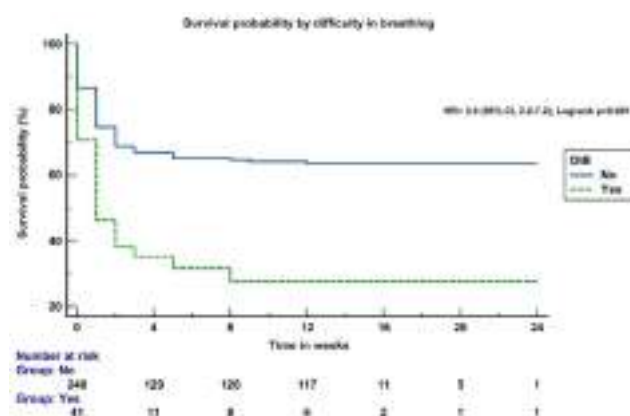


FIGURE 1 Kaplan Meier survival probability by difficulty in breathing.

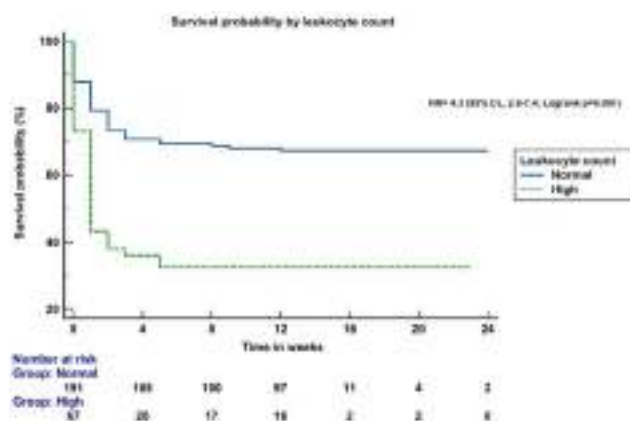


FIGURE 2 Kaplan Meier survival probability by leukocyte count.

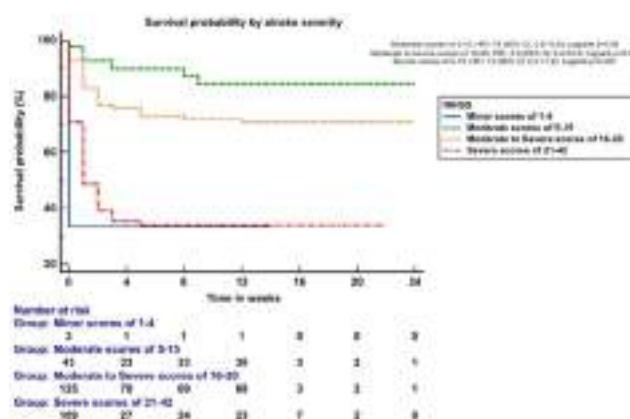


FIGURE 3 Kaplan Meier survival probability by stroke severity based on NIHSS scores.

and females were 61.5% (16/26). The median CD4 count was 518 IQR (151.5–571) cells/ μ L, and 84.6% (22/26) were naïve to antiretroviral therapy (ART). PLWH experienced longer hospital stay, 5 days (IQR 2–7.3), $p = 0.001$. Both groups experienced poor functional outcomes, mRS 3–6, 84.7% (250/295), and a mortality rate of 38.5% (114/295) at 6 months. In the multivariate analysis, difficulty in breathing, leukocytosis, and NIHSS scores of 21–42 were independent predictors of mortality.

Conclusion: Although 6-month outcomes were comparable between both groups, targeted strategies are needed to address healthcare disparities and reduce the additional burden of stroke in PLWH.

Disclosure: Nothing to disclose.

EPO-0357 | Validation of the DIAS³ score in cerebral venous thrombosis patients treated by decompressive surgery

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Background and aims: Cerebral venous thrombosis (CVT) is associated with a risk of late seizures, particularly in patients with severe disease requiring decompressive surgery. The DIAS³ score was developed to predict late seizures after CVT, but its performance in surgically treated patients is unknown. We aimed to validate the DIAS³ score in a high-risk cohort of CVT patients treated by decompressive surgery.

Methods: This was a substudy of the DECOMPRESS2 – a prospective, multicentre cohort, including patients with CVT treated with decompressive surgery. Late seizures were defined as those occurring more than 7 days after CVT diagnosis, up to

1 year follow-up. DIAS3 score was calculated for all included patients. Predictive performance was assessed only for the 1-year risk of late seizures. Discrimination was evaluated using receiver operating characteristic analysis, and calibration by comparing predicted and observed seizure risks. Diagnostic performance was assessed across multiple probability thresholds.

Results: Eighty-nine patients were included (mean age 37.3 years; 69.7% female). Late seizures occurred in 22 patients (24.7%). The mean predicted 1-year seizure risk according to the DIAS³ score was 28.4%. The DIAS³ score showed poor discrimination (area under the curve 0.44, 95% confidence interval

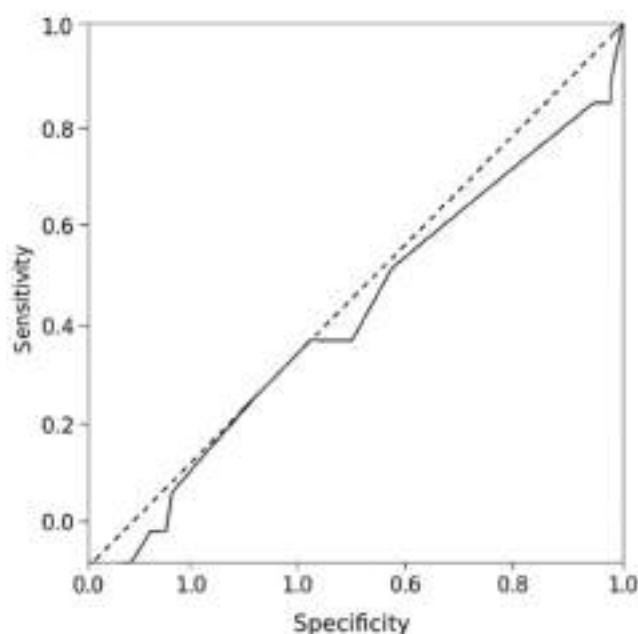


FIGURE 1 ROC curve for late seizure prediction using the DIAS3 score in the DECOMPRESS2 cohort (AUC=0.44, 95% CI 0.30–0.59). The dashed diagonal line represents the reference line of no discrimination.

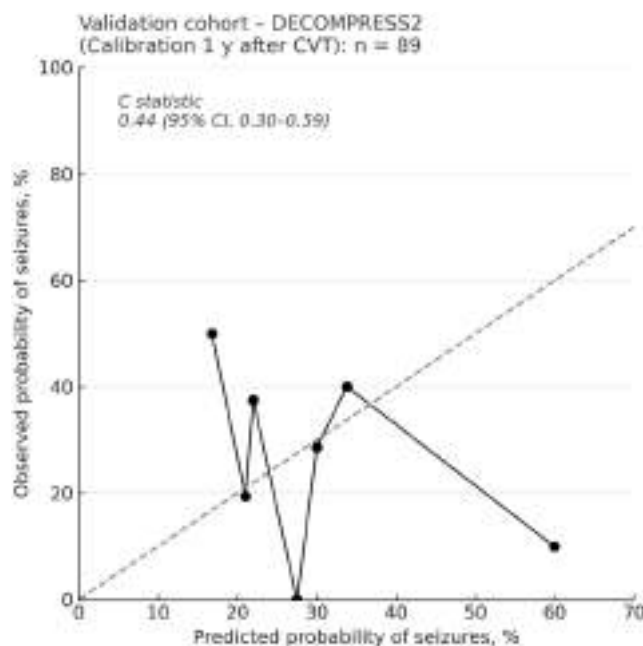


FIGURE 2 Calibration plot for late seizure prediction using the DIAS3 score in the DECOMPRESS2 validation cohort (n = 89).

0.30–0.59) and poor calibration. Across all tested thresholds, sensitivity and specificity showed an expected trade-off, but overall predictive accuracy was limited.

Conclusion: The DIAS³ score demonstrated poor discrimination and calibration in CVT patients treated by decompressive surgery. These findings suggest that the DIAS³ score should not be used to predict late seizures in this high-risk surgical subgroup.

Disclosure: Nothing to disclose.

EPO-0358 | Impact of ambient temperatures on ischemic stroke characteristics and in-hospital recurrence in a swiss cohort

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Background and aims: The association between ambient temperatures and ischemic stroke remains poorly characterized in Europe. This study aimed to examine the influence of acute heat and cold exposure on stroke characteristics.

Methods: We conducted a retrospective cohort study of 500 patients experiencing a first ischemic stroke, using data from the Bernese Stroke Registry (09/2018–12/2020). Patients were categorized based on the temperature on the day of stroke: normal (0–30°C; $n=365$), hot ($>30^{\circ}\text{C}$; $n=22$), and cold ($<0^{\circ}\text{C}$; $n=113$). Stroke etiology, vascular risk factors, and in-hospital stroke recurrence were compared across temperature groups.

Results: Cardioembolism was the most prevalent stroke etiology across all temperature groups, with the highest proportion observed in the cold group on the day prior to stroke (43.9%). Coronary heart disease prevalence was significantly higher in the hot group (41.7%) compared with the normal temperature group (20.1%; $p=0.039$). The incidence of new in-hospital ischemic strokes was markedly elevated in the extreme temperature groups: 36.4% in the hot group and 18.0% in the cold group, versus 8.9% in the normal temperature group ($p<0.001$). Temperature on the stroke day was also associated with significant differences in stroke etiology ($p=0.014$), with cardioembolism observed in 38.9% of patients in the cold group. No significant differences were noted for other vascular risk factors, occlusion location, or acute interventions.

Conclusion: Extreme temperatures influence ischemic stroke profiles and in-hospital recurrence. Heat is associated with increased cardiovascular comorbidity and recurrence risk, while cold exposure favors cardioembolic strokes, suggesting delayed cardiovascular effects. These findings underscore ambient temperature as a clinically relevant, modifiable stroke risk factor.

Disclosure: None.

EPO-0359 | Association of admission LDL cholesterol with clinical profile and 3 months outcomes after ischaemic stroke

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Background and aims: While low-density lipoprotein cholesterol (LDL-C) is a well-established modifiable risk factor for stroke, recent evidence suggests a “lipid paradox,” where low LDL-C levels may be associated with worse outcomes.

Methods: In this retrospective cohort study, we analyzed 1634 patients aged ≥ 18 years admitted to the Bernese Stroke Centre (02/2015–03/2023). We investigated the association between admission LDL-C levels and clinical characteristics after first-ever acute ischaemic stroke. Patients were stratified into five LDL-C groups: >3.0 mmol/L ($n=493$), 2.6–3.0 mmol/L ($n=225$), 1.8–2.6 mmol/L ($n=490$), 1.4–1.8 mmol/L ($n=254$), and <1.4 mmol/L ($n=172$). Functional outcome was assessed at 3 months.

Results: Patients with LDL-C <1.4 mmol/L were older (median 78 vs. 73 years; $p<0.001$), more often male (59% vs. 44%; $p<0.001$), and had a higher burden of comorbidities, including arterial hypertension, coronary heart disease, atrial fibrillation, heart failure, and renal impairment (all $p<0.001$). Over 70% of patients in the lowest LDL-C group were already receiving lipid-lowering therapy at admission, versus $<10\%$ in the highest group ($p<0.001$). Laboratory markers associated with frailty showed significant differences: haemoglobin levels were lower (124 vs. 139 g/L; $p<0.001$) and creatinine levels higher (92 vs. 79–83 $\mu\text{mol/L}$; $p<0.001$) in the lowest versus highest LDL-C group. Favourable (mRS 0–2) and excellent (mRS 0–1) functional outcomes at 3 months declined across groups (42% to 29% and 36% to 23%, respectively), as did survival (80% to 61%; all $p<0.05$).

Conclusion: Low LDL-C at stroke onset identifies older, multimorbid, and frail patients with poorer 3 months outcomes, likely reflecting underlying vulnerability and comorbidities rather than independently causing poor prognosis.

Disclosure: None.

EPO-0360 | CLadribine tablets level of respOnse preDICTors in cliNical practice: Interim analysis of CLODINA

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Background and aims: CLODINA is a multicenter, Phase IV ambispective, non-interventional study in patients diagnosed with RMS who have received cladribine tablets, aiming to identify baseline patient characteristics predicting the time to treatment discontinuation in real-world. Secondary objectives are to assess relapses, disability progression and improvement, MRI activity, and safety.

Methods: This interim analysis, conducted after 50% of patients completed the 2-year follow-up, reports Annualized Relapse Rate (ARR), disability progression and improvement, and MRI data descriptively. A predictive model for discontinuations was not created, as it would be superseded by the final analysis.

Results: The Full Analysis Set included 365 patients (33.7% prospective, 66.3% retrospective); median age was 38 years. 72.1% were female, and 73.4% (268) had received a DMT prior to cladribine tablets, with 79.4% (213) switching from platform therapies and 20.6% (55) from high-efficacy therapies. The main reason for switching was inadequate response to prior DMT; psychiatric disorders were the most common comorbidities (15.3%). MRI assessment within 12 months before the index date was available for 98.9% of patients. At year 2, relapse data were available for 87.1% of patients, and ARR was 0.08 (0.03, 0.08). 298 patients (81.6%) were relapse-free. 6-month confirmed disability progression/improvement was seen in 4.1% and 3.3%. 233 patients had available MRI data at year 2, of whom 189 (81%) were free from MRI activity. 19.7% reported adverse events.

Conclusion: Cladribine tablets are highly effective, showing low ARR over a 2-year follow-up, with a generally safe and well-tolerated profile.

Disclosure: RH received honoraria as consultant on scientific advisory boards from Merck, Biogen, Genzyme-Sanofi and Teva, research funding from Merck and Biogen, and speaker honoraria

from Sanofi-Genzyme and Novartis. AKL received honoraria or consultation fees from: Bayer, Biogen, Merck, Novartis, Sanofi-Genzyme, Bristol-Meyers Squibb, Janssen, Teva, Sandoz. TR has received financial travel support, educational support, consultancy fees and/or speaker fees from Roche, Biogen, Sanofi, Merck, Teva, Novartis and Janssen Pharmaceuticals. She is a scientific board member of the Sanofi MS Academy in Flanders, Belgium. JS has received personal compensation for participation in advisory boards from Janssen, Merck, Novartis, Roche and Sanofi; consulting fees from Novartis; speaking honoraria from Almirall, Merck, Novartis, Roche and Sanofi; and travel expenses for scientific meetings from Bayer, Biogen, Janssen, Merck, Novartis, Sanofi and Teva. MV has received personal compensation for participation in advisory boards from Biogen, BMS, Janssen, Merck, Novartis, Roche and Sanofi; consulting fees from Biogen, Roche and Novartis; speaking honoraria from Biogen, BMS, Merck, Janssen, Novartis, Roche and Sanofi; and travel expenses for scientific meetings from Bayer, Biogen, Janssen, Merck, Novartis, Sanofi and Teva. SD is an employee of Merck S.A, Greece, an affiliate of Merck KGaA AA is an employee of Merck S.A, Greece, an affiliate of Merck KGaA PV is an employee of Merck N.V.-S.A, Overijse, Belgium, an affiliate of Merck KGaA PR is an employee of Merck Specialties, India, an affiliate of Merck KGaA NG has received honoraria and travel support from Biogen Idec, Biologix, Novartis, TEVA, Bayer, Merck, Genesis Pharma, Sanofi Genzyme, Roche, has received consultancy fees from Biogen Idec, Novartis, Teva, Bayer, Merck, Genesis Pharma, Sanofi Genzyme, Celgene, ELPEN, Roche, has received lecture fees from Biogen Idec, Novartis, Teva, Bayer, Merck S.A, Greece, an affiliate of Merck KGaA, Genesis Pharma, Sanofi Genzyme, ROCHE and research grants from Biogen Idec, Novartis, Teva, Merck S.A, Genesis Pharma, Sanofi Genzyme, Roche.

EPO-0361 | Post-hoc analysis of the OPTIMUM long-term extension study: Preservation of independent ambulation in patients treated with Ponesimod

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Background and aims: Disability accumulation in relapsing multiple sclerosis (RMS) is commonly quantified using the Expanded Disability Status Scale (EDSS). Reaching EDSS 6 marks the need for an ambulatory aid, while EDSS 7 reflects loss of independent ambulation and wheelchair dependence, representing major milestones in long-term disease burden.

Methods: Following 108 weeks of double-blind treatment with ponesimod 20 mg or teriflunomide 14 mg in the core OPTIMUM study, participants could enter the long-term extension (LTE) and receive open-label ponesimod 20 mg. This post hoc analysis confirmed EDSS 6 and EDSS 7 over a follow-up of up to 7 years. Kaplan–Meier estimated the cumulative incidence of reaching these disability milestones.

Results: Overall, 1133 patients were randomized in the core study, and 877 entered the LTE. At core baseline, mean EDSS was 2.56 ± 1.20. Median cumulative exposure to ponesimod during the combined core plus LTE period was 6.3 years (IQR

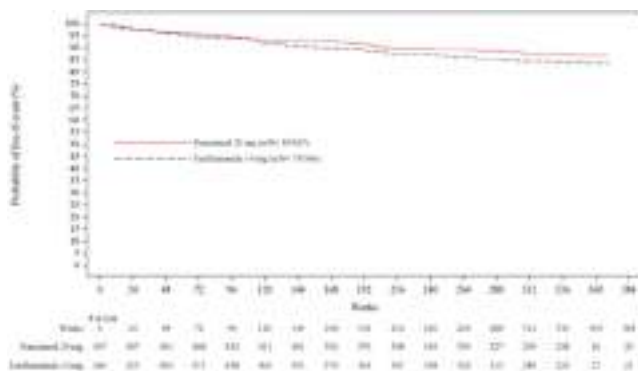


FIGURE 1 EDSS superior or equal to 6 free probability, Kaplan-Meier curve, combined analysis period.

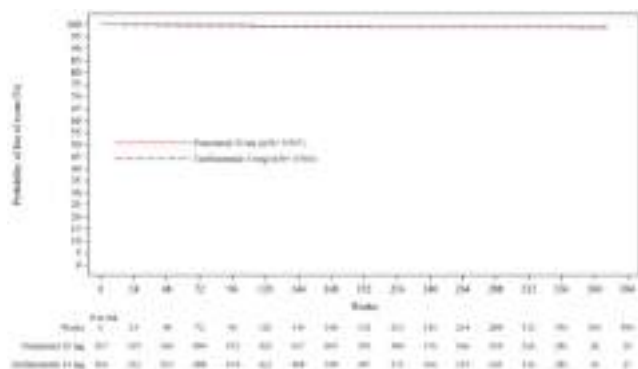


FIGURE 2 EDSS superior or equal to 7 free probability, Kaplan-Meier curve, combined analysis period.

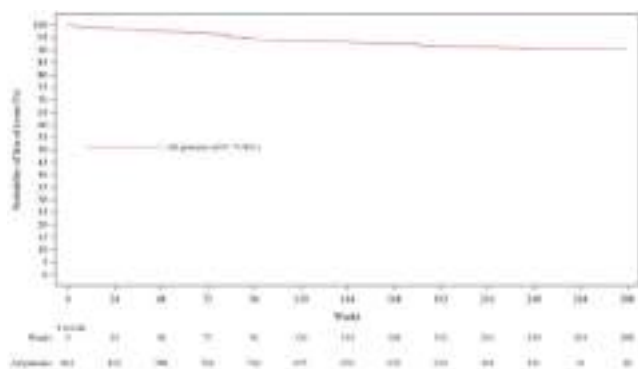


FIGURE 3 EDSS superior or equal to 6 free probability, Kaplan-Meier curve, extension period.

2.8–6.7). Among patients treated continuously with ponesimod ($n=567$), 86.8% had not reached EDSS 6 (Figure 1) and 98.7% had not reached EDSS 7 by week 360 (Figure 2). During the LTE period, 90.5% of patients remained free from EDSS 6 (Figure 3) (92.3% in those initially randomized to ponesimod and 88.8% in those initially randomized to teriflunomide), and 99.3% remained free from EDSS 7 (99.1% and 99.5%, respectively).

Conclusion: This post-hoc analysis of OPTIMUM-LTE shows a sustained preservation of mobility and functional independence with ponesimod over up to 7 years of follow-up, supporting its durable efficacy in patients with RMS.

Disclosure: MLMG declare fees for lectures, consultations, travel support, assistance to congresses, advisory meetings,

teaching or research from: Almirall, Amgen, AstraZeneca, Biogen, Bristol Myers Squibb, Janssen, Merck, Novartis, Roche, Sanofi, Teva, Neuraxpharm, Juvise. M.H, S.G, A.A and A.S are employees of Juvise Pharmaceuticals.

EPO-0362 | The effect of cladribine tablets on retinal microglial activity in relapsing multiple sclerosis: Optical coherence tomography findings from MAGNIFY-MS

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Background and aims: Hyperreflective foci (HRF) are retinal punctate structures, observable with optical coherence tomography (OCT), and are potential neuroinflammatory biomarkers. Pathological correlates of HRF may be microglial accumulation. We evaluated the presence and change of HRF in cladribine tablets (CladT)-treated people with highly-active relapsing multiple sclerosis (pwRMS) over 24 months and determined relationships with clinical and magnetic resonance imaging (MRI) outcomes. **Methods:** Overall, 36 pwRMS enrolled in the OCT sub-study in MAGNIFY-MS (NCT03364036). Clinical (progression independent of relapse activity [PIRA]) and MRI (lesions and brain volume) outcomes were analysed. Total HRF (sum of HRF in the ganglion cell inner plexiform layer [GCIPL] and inner nuclear layer [INL]), GCIPL-HRF, and INL-HRF were determined by OCT. All outcomes measured at baseline, month (M) 12, and M24. Linear regression models were used; data are presented as mean and standard deviation. HRF analysis was performed by an experienced rater (D.J.).

Results: From baseline to M24, CladT significantly decreased the total number of HRF ($p=0.007$) and GCIPL-HRF ($p=0.002$; Table 1). Compared to MRI-stable pwRMS, those with in-trial MRI activity had higher baseline GCIPL-HRF (5.6 vs. 3.0, $p=0.032$), and a larger decrease in GCIPL-HRF over 24 months (-2.6 vs. -0.6 , $p=0.013$; Table 2). PIRA activity was associated with baseline INL-HRF ($p=0.034$). Total baseline HRF was the only predictor of 12-month brain volume loss ($R^2=0.323$, $p=0.038$).

Conclusion: In our exploratory analysis, CladT treatment reduced overall HRF burden in pwRMS. Baseline GCIPL-HRF was associated with early inflammatory activity, whereas INL-HRF was associated with chronic neurodegenerative changes.

Disclosure: DJ has nothing to disclose JH reports a grant for OCT research from the Friedrich-Baur-Stiftung, Horizon/Amgen, Sanofi and Merck, personal fees and nonfinancial support from Alexion, Amgen, Bayer, Biogen, BMS, Merck, Novartis and Roche, and nonfinancial support of the Sumaira-Foundation and Guthy-Jackson Charitable Foundation, all outside the submitted work LL has received honoraria for consulting services or

TABLE 1 Results from a pairwise analysis of HRF in GCIPL and INL retinal layers.

Variable	Number of participants	Baseline Mean (SD)	M24 Mean (SD)	Within subject p value
Total HRF	28	15.6 (9.7)	12.5 (7.7)	0.007
GCIPL-HRF	28	4.2 (3.5)	2.57 (2.3)	0.002
INL-HRF	28	11.4 (6.9)	9.9 (6.4)	0.141

GCIPL, ganglion cell inner plexiform layer; HRF, hyperreflective foci; INL, inner nuclear layer; M, month; SD, standard deviation.

TABLE 2 Differences in HRF between groups with and without disease activity over the 24-month study period.

Variable	Disease activity	Number of participants	Mean (SD)	p value
MRI	No	18	3.0 (2.2)	0.002
	Yes	17	9.6 (4.3)	
GCIPL-HRF change 0–M24	No	14	−3.8 (2.2)	0.013
	Yes	18	8.9 (5.5)	
INL-HRF ΔL	No	17	−12.4 (7.0)	0.302
	Yes	14	−0.9 (5.7)	
PIRA change 0–M24	No	14	−1.1 (3.0)	0.511
	Yes	14	−0.1 (0.5)	
PIRA	No	30	50.1 (8.5)	0.004
	Yes	5	17.0 (6.0)	

ΔL, baseline; GCIPL, ganglion cell inner plexiform layer; HRF, hyperreflective foci; INL, inner nuclear layer; M, month; MRI, magnetic resonance imaging; PIRA, progression independent of relapse activity; SD, standard deviation.

speaking activities from Merck and Roche. MD is an employee of Merck Santé S.A.S, Lyon, France, an affiliate of Merck KGaA. LG is an employee of EMD Serono Research & Development Institute, Inc, Billerica, MA, USA, an affiliate of Merck KGaA. NDS has received honoraria from Biogen-Idec, Bristol Myers Squibb, Celgene, Genzyme, Immunic, Merck, Novartis, Roche, and Teva for consulting services, speaking, and travel support. He serves on advisory boards for Immunic, Merck, Novartis, Biogen-Idec, Roche, and Sanofi. He has received research support from the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. He is Associate Editor of Neurological Sciences and co-founder of Siena-Imaging s.r.l.

EPO-0363 | Clinical and MRI outcomes of Siponimod versus ocrelizumab in secondary progressive multiple sclerosis: A real-world study

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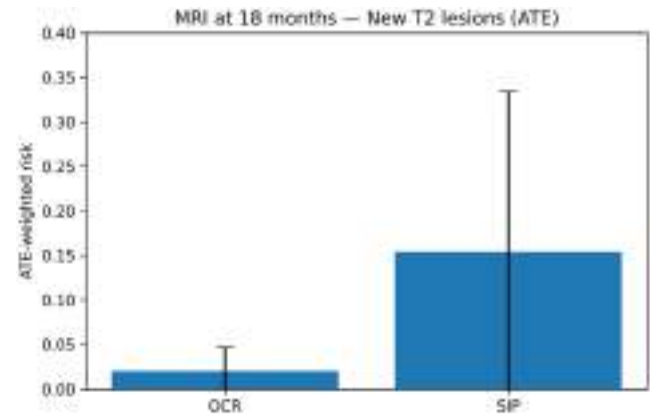


FIGURE 1 ATE-weighted risk of new T2 lesions at 18 months in patients treated with ocrelizumab (OCR) or siponimod (SIP). Error bars represent 95% confidence intervals derived from stabilized inverse probability weighting.

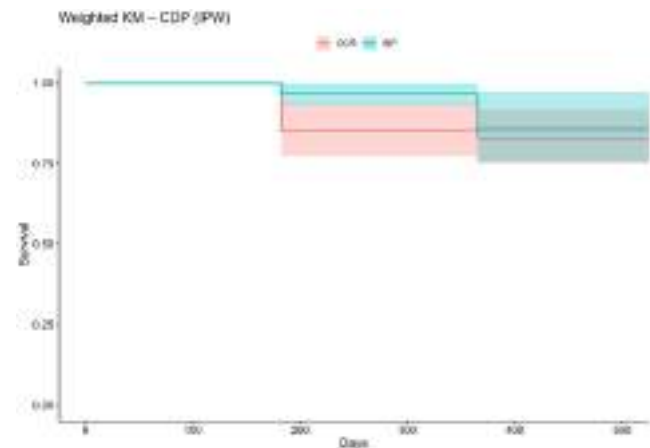


FIGURE 2 IPW-weighted Kaplan–Meier curves for confirmed disability progression over 18 months in patients treated with ocrelizumab or siponimod. Shaded areas indicate 95% confidence intervals.

Background and aims: We compared the effectiveness and safety of siponimod (SIP) versus ocrelizumab (OCR) in real-world cohort of patients with secondary progressive multiple sclerosis (SPMS).

Methods: We retrospectively included SPMS patients from five Italian centers initiating SIP or OCR and followed-up for 18 months. Outcomes included time to first relapse, annualized relapse rate (ARR), confirmed disability progression (CDP), progression independent of relapse activity (PIRA), MRI activity at 18 months (new T2 lesion or Gd+ lesion), and safety. Baseline differences were addressed using stabilized inverse probability of treatment weighting and overlap weighting. Weighted Cox and negative binomial models were applied.

Results: Among 182 patients (98 OCR; 84 SIP; mean age 50.9 years), clinical outcomes were similar: time to first relapse SIP vs OCR (HR 1.41; 95% CI 0.58–3.45; $p=0.451$); recurrent relapses (HR 1.20; 0.52–2.76; $p=0.667$); CDP (HR 0.83; 0.38–1.82; $p=0.643$); PIRA (HR 0.86; 0.38–1.94; $p=0.708$); ARR (IRR 1.78; 0.83–3.81; $p=0.136$). MRI activity at 18 months differed between groups, with new lesions occurring more frequently under SIP (OR 9.12; 1.26–66.34; $p=0.029$), confirmed in overlap-weighted analysis (OR 7.14; 1.09–46.63; $p=0.040$). SIP was associated

with lower white blood cells/lymphocytes/neutrophils (all $p < = 0.0037$) and higher liver enzymes ($p < = 0.049$); infection and adverse-event rates were similar.

Conclusion: In SPMS, short-term clinical outcomes were comparable between SIP and OCR, whereas MRI activity at 18 months differed, with higher odds of new lesions under SIP. When disability endpoints fail to discriminate over short follow-up, suppression of subclinical inflammatory MRI activity may represent the key differentiator guiding treatment choice, particularly in patients with residual inflammatory activity.

Disclosure: The authors report no relevant relationships/activities/interests related to this work.

EPO-0364 | Evaluating the value of serum neurofilament light chain after DMT initiation for monitoring early treatment response

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Background and aims: Serum neurofilament light chain (sNfL) is an established biomarker of axonal injury in multiple sclerosis (MS). While sNfL typically decreases with effective disease-modifying therapy (DMT), it remains unclear whether early post-initiation sNfL levels or their change predict treatment response. We evaluated sNfL as an early biomarker of DMT response in relapsing MS (RMS).

Methods: Within an ongoing prospective observational cohort at the Medical University of Vienna, we included patients with RMS newly initiating any DMT. Serum NfL was measured at treatment start (baseline) and at 6 months and expressed as age- and BMI-adjusted z-scores. Minimum follow-up was 24 months. Treatment failure (EDA-3) was defined by relapse, MRI activity, and/or confirmed disability progression.

Results: Ninety-two patients were analyzed (mean age 35 ± 8 years; 71% on high-efficacy DMT); 22 (24%) developed EDA-3 during follow-up. Baseline sNfL was not significantly associated with subsequent EDA-3 (aOR 1.36, 95% CI 0.90–1.99; $p = 0.20$). At 6 months, median sNfL z-scores were lower in those remaining relapse-free (0.03 in NEDA-3 vs 1.94 in EDA-3; $p < 0.001$; change of Δz -0.94 vs 0.01; $p = 0.014$). Discrimination for future EDA-3 was moderate for both absolute 6-month z-scores (AUC 0.752; optimal cut-off 0.935) and Δz -scores (AUC 0.733; cut-off -0.811), with high negative predictive values (95.6% and 97.8%). In multivariable analysis, 6-month absolute z-scores predicted future EDA-3 (aOR 3.40, 95% CI 1.91–7.07; $p = 0.017$).

Conclusion: Six-month sNfL z-scores, particularly their normalization after DMT initiation, may early identify patients with favorable treatment response in RMS.

Disclosure: No disclosures or relationships of interest relevant to the context of this study.

EPO-0365 | CAR T-cell therapy in neuroimmunological diseases – The vienna model (Neuro CAR-T Vienna)

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Background and aims: Chimeric antigen receptor (CAR) T-cell therapies have undergone rapid development in recent years, with increasing evidence supporting their efficacy and safety in severe, treatment-refractory autoimmune diseases of the nervous system. In response, the Medical University of Vienna has established Austria's first center dedicated to the application of CAR T-cell therapy in neuroimmunological disorders.

Methods: The Neuro CAR-T Vienna model is based on a structured, interdisciplinary collaboration between Neurology, Transfusion Medicine & Cell Therapy, and Hemato-Oncology, with additional involvement of Rheumatology, Neuroradiology, and Gynecology. Central to the model is an interdisciplinary CAR T-cell board, where patients are systematically evaluated and indications reviewed. Referrals are accepted for patients with neuroimmunological diseases that are insufficiently controlled by conventional therapies and carry a high risk of a deleterious disease course. Evaluation requires comprehensive documentation of disease and treatment history, current imaging and laboratory findings, and confirmation of the patient's willingness and psychological suitability for an invasive therapy.

Results: CAR T-cell therapy may be administered either as an individual compassionate-use treatment using in-house manufactured CAR T cells or within clinical trials. Patients receive comprehensive interdisciplinary counseling, including potential effects on fertility and family planning, with fertility preservation options discussed when appropriate. For pharmacovigilance purposes, treated patients commit to a structured

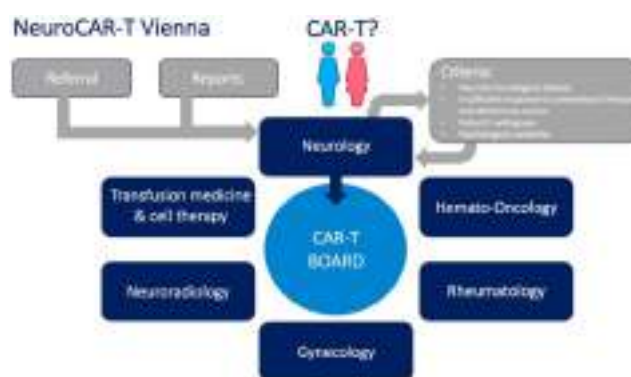


FIGURE 1 Neuro CAR-T Vienna.

long-term follow-up with at least annual assessments for a minimum of five years.

Conclusion: The Neuro CAR-T Vienna model represents Austria's first standardized interdisciplinary framework for CAR T-cell therapy in neuroimmunological diseases, enabling safe, transparent, and patient-centered implementation and serving as a reference model for future national expansion.

Disclosure: Nothing to disclose.

EPO-0366 | Efficacy and safety of fenebrutinib vs teriflunomide in relapsing multiple sclerosis: Results of the FENhance 1 and 2 studies

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Background and aims: Bruton's tyrosine kinase inhibitors (BTKis) may address both relapsing and progressive disease biologies in multiple sclerosis (MS) via a dual mechanism of action. Phase III trials in patients with relapsing MS (RMS) have not met their primary endpoint in showing that covalent, irreversible BTKis reduce annualized relapse rate (ARR) vs teriflunomide. The objective of the FENhance Phase III studies was to evaluate the efficacy and safety of fenebrutinib—a noncovalent, reversible, highly selective BTKi—vs teriflunomide in adults with RMS.

Methods: FENhance 1 (NCT04586010) and FENhance 2 (NCT04586023) are two identical Phase III, multicenter, randomized, double-blind, double-dummy, parallel-group studies enrolling adults aged 18–55 years with RMS (2017 revised McDonald criteria; Expanded Disability Status Scale [EDSS] score 0–5.5). Patients were randomized 1:1 to oral fenebrutinib 200 mg twice daily or teriflunomide 14 mg once daily for a minimum of 96 weeks. The primary endpoint was ARR.

Results: In total, 1497 patients were enrolled globally. The mean (SD) age at baseline was 35.8 (9.4) years, mean (SD) EDSS score was 2.5 (1.3) and 66.5% were female. The mean (SD) duration since MS symptom onset was 5.5 (5.9), and mean (SD) duration since diagnosis was 3.6 (5.1) years. Primary efficacy and safety data from both FENhance studies will be presented.

Conclusion: While other BTKis have not shown superiority to teriflunomide in suppressing disease activity, the FENhance trials will determine whether fenebrutinib's unique molecular

design, optimized CNS exposure and promising Phase II results translate into significant benefits in RMS.

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EPO-0367 | Anti-CD20 drugs and Natalizumab confer equal protection against progression independent of relapse activity (PIRA) in people with multiple sclerosis

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Background and aims: Highly effective (HE) disease-modifying therapies (DMTs) reduce relapse-associated worsening (RAW) and progression independent of relapse activity (PIRA). Natalizumab (NTZ) and anti-CD20 agents show similar efficacy on relapse suppression, but long-term comparative data on PIRA are limited. We aimed to compare PIRA accrual in relapsing-remitting MS (RRMS) patients treated with NTZ and anti-CD20 drugs over a long follow-up.

Methods: We included RRMS patients followed at our centre and recorded in the Italian MS and Related Disorders Register with at least 5 years of continuous treatment with NTZ or anti-CD20 agents (Ocrelizumab, Rituximab, Ofatumumab), at least 3 Expanded Disability Status Scale (EDSS) assessments, and complete demographic and clinical data. Patients were matched for sex, age at treatment initiation, and disease duration. Logistic regression and Cox proportional hazard models were applied.

Results: Of 267 eligible patients (178 NTZ, 89 anti-CD20), optimal matching yielded 89 balanced pairs. A first PIRA event was observed in 37.1% of NTZ-treated and 32.6% of anti-CD20-treated patients. Although the absolute number of PIRA events was higher in the NTZ group (47 vs 30; $p=0.015$), adjusted analyses revealed no significant differences in cumulative risk (RR: 1.15; 95% CI: 0.57–2.37; $p=0.692$) or time to first PIRA event (HR: 1.21; 95% CI 0.47–1.39; $p=0.446$).

Conclusion: In this real-world cohort with long-term follow-up, NTZ and anti-CD20 therapies conferred comparable protection in controlling disability progression beyond relapse, leaving space for further survey, desirably in a larger cohort, to confirm and refine these observations.

Disclosure: Nothing to disclose.

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Background and aims: Ocrelizumab has been approved for both relapsing and primary progressive multiple sclerosis (MS), but the impact of this treatment on brain innate immune cell neuro-inflammation remains unknown.

Methods: People with MS (PwMS) aged 18–60 years with relapsing–remitting (RRMS), active secondary progressive (SPMS), or primary progressive MS (PPMS) underwent neurological evaluation and a 90-min dynamic PET–MR using the [18F] DPA714 TSPO tracer at baseline and at months 6 and 24 after ocrelizumab initiation. Ocrelizumab was administered as 600-mg infusions every 24 weeks. Age-, sex-, and TSPO-genotype-matched healthy controls underwent a single PET–MR scan. The primary outcome was the change in active voxels in total white matter (TWM) between baseline and month 24, defined using [18F] DPA714 thresholds derived from comparisons with controls. Secondary analyses assessed longitudinal changes at months 6 and 24 and within MS subgroups.

Results: Thirty-seven PwMS completed all evaluations. The proportion of DPA-active voxels in TWM was not significantly reduced between month-24 and baseline (median relative change: +3.58% [–43.32%,+23.98%]; $p=0.86$), but decreased significantly between baseline and month 6 (–26.75% [–39.86%,–10.61%]; $p=0.017$), with a subsequent increase trend between month-6 and -24. Significant reductions between baseline and month 6 were observed in TWM and normal-appearing WM in RRMS patients ($p=0.027$, 0.037) and in lesions in RRMS and SPMS subjects ($p=0.005$, 0.035). No significant longitudinal changes were detected in PPMS subjects.

Conclusion: Ocrelizumab initiation was associated with reduced white matter neuroinflammation at month 6, an effect that was no longer detectable at month 24.

Disclosure: M Hamzaoui, T Soulier: Nothing to disclose V AG Ricigliano reports fees for travel, consulting/advisory board fees and speaker's honoraria from Novartis, Merck, Roche, Sanofi, Sandoz, Biogen, Janssen, M3 Global Research and Atheneum

Partners. D Hajage, J Dufour: Nothing to disclose A Lazzarotto reports fees for traveling and speaker's honoraria from Novartis, Roche, Merck and Sandoz. I Laurent, J Palmyre: Nothing to disclose C Louapre has received consulting or travel fees from Biogen, Merck, Novartis, Roche, Sanofi and Oculis, and research grant from Biogen. V Lebon, A Kas, M Bottlaender: Nothing to disclose B Bodini reports fees for traveling and speaker's honoraria from Novartis, Genzyme, Roche, and Merck Serono; B Stankoff has received research grants and personal fees for lectures from Roche, Novartis, and Merck-Serono, personal fees for lectures from Alexion, and Janssen. The study was funded by Roche and sponsored by APHP.

EPO-0369 | Characterizing patient profiles linked to treatment persistence in multiple sclerosis: Federated analysis of clinical real-world data in different healthcare systems

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Background and aims: The therapeutic landscape of Multiple Sclerosis (MS) has evolved significantly in recent years and the treatment strategy has become a contentious debate. It has been shown that persistence is critical to treatment effectiveness. However, more real-world evidence is needed to understand correlates of persistence among people with MS (pwMS). In this study, we applied Federated Analysis (FA) to simplify data analysis across three sites in two countries, investigating covariates of treatment persistence and its effects on clinical and socioeconomic outcomes in MS across different healthcare systems.

Methods: The study cohorts included people with MS (pwMS) initiating a Disease Modifying Treatment (DMT) between 2018 and 2021 with at least 2 years of follow-up data. Persisters were defined as pwMS having continuous records of a certain class of DMT within the 2-year follow-up period. Outcomes were collected retrospectively, harmonized, and made available for FA. This approach keeps data localized at the institutions and transmits only aggregated data.

Results: We analyzed data of 2528 pwMS across three clinical sites (Dresden University of Technology, Stanford University and Yale University). 76.5% of pwMS were persisters. The likelihood of persistence was positively associated with treatment efficacy, age, and conversely with a lower risk of hospitalization or need for walking aids or wheelchairs.

Conclusion: We explored covariates of persistence and their association with clinical outcomes in different healthcare systems. The results will foster an understanding of the influence of different treatment approaches in the real-world and the benefits of leveraging FA spanning multiple healthcare systems.

Disclosure: Funding statement: Sponsored by F. Hoffmann-La Roche Ltd Disclosures: V P Illiano, M C Elze, G Zilborri, B Tackenberg and M Antonin are employees of F. Hoffman-La Roche Ltd. A Morgan has been a consultant for Genentech. T

Görbing and N Orr have nothing to disclose. E Longbrake has received honoraria for consulting from Bristol Myers Squibb, Sanofi, Alexion, Novartis, TG Therapeutics, EMD Serono, Genentech. She has received research support from Biogen, Genentech. T Ziemssen reports scientific advisory board and/or consulting for Biogen, Roche, Novartis, Celgene, and Merck; compensation for speakers' bureaus for Roche, Novartis, Merck, Sanofi, Celgene, and Biogen; research support from Biogen, Novartis, Merck, and Sanofi. L Steinman has received research contracts from F.Hoffmann-La Roche Ltd.

EPO-0370 | Real-world short-term outcomes of ofatumumab and ocrelizumab in RR-MS

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Background and aims: Real-world comparative data about ofatumumab (OFA) and ocrelizumab (OCR) efficacy and safety in multiple sclerosis (MS) are still scarce.

Methods: We compared efficacy and safety of our 103 relapsing–remitting (RR) MS OFA-treated patients with those of the first consecutive 103 RR-MS OCR-treated patients. Only OFA-treated patients with at least 6 months FU were included.

Results: OFA and OCR patients exhibited similar demographic and clinical features at baseline [OFA: 64.1% females, mean (SD) age 40.6 (10.4) years, disease duration (DD) mean 10.9 (9.7) years, median EDSS 1.5, 30.8% naïve; OCR: 59.2% females, mean age 39.5 (11) years, DD mean 11.5 (11.3) years, median EDSS 2.5, 15% naïve]. No patients showed relapses at 1-year and 2-year FU. At 1-year, comparing OFA and OCR no significant differences in terms of MRI activity [12/85 (14.1%) vs 19/103 (18.4%)] and PIRA events [3/85 (3.5%) vs 3/103 (2.9%)] were observed. 1-year No Evidence of Disease Activity-3 (NEDA-3) was achieved by 70/85 (82.4%) and 84/103 (81.6%) respectively. At 2-year, no significant differences in terms of MRI activity [8/38 (21.1%) vs 18/99 (18.2%)] and PIRA events [3/38 (7.9%) vs 9/99 (9.1%)] were observed. MRI activity occurred within the first 6 months of treatment (median 2.8 and 4 months respectively). At 2-year FU only one new cortical lesion (CL) was observed in the OCR cohort. At 2-year FU, severe infections were reported in 1 and 4 patients respectively (OFA: 1 urosepsis; OCR: 4 pneumonias, 2 during COVID-19 pandemic).

Conclusion: OFA and OCR showed similar short-term efficacy and safety. Our data demonstrated the efficacy of both therapies also in preventing CLs development.

Disclosure: GB received personal fees from Novartis and Roche. AL received fees for consultation, public speaking or both from Roche, Genzyme, Merck, Biogen, Novartis and Bristol-Myers Squibb. MI received grants from NIH, NMSS, FISM, and Ministero dell'Università e ricerca (MUR) and received fees for consultation from BMS, Janssen, Roche, Genzyme, Merck, Biogen and Novartis. CL has received honoraria for speaking, travel grants and participating in advisory boards from Merck, Sanofi, Novartis, Roche and Alexion. NC, EC, SAQ, FB and EC have nothing to disclose.

EPO-0371 | Real-world effectiveness of ocrelizumab: Altering the six-year disease trajectory in multiple sclerosis

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Background and aims: Ocrelizumab is highly effective for relapsing and progressive multiple sclerosis (MS). We evaluated clinical trajectories during the three years preceding and six years following treatment initiation.

Methods: This retrospective study analyzed 268 patients (158 RRMS, 110 SPMS). Clinical outcomes were compared across three-year intervals: pre-treatment, Years 0–3, and Years 3–6. Effectiveness was assessed using annualized relapse rates (ARR), EDSS progression, and MRI activity. NEDA-3 (no relapses, no disability progression, and no new MRI lesions) was the primary endpoint. Regression models identified predictors for long-term NEDA-3 achievement.

Results: Patients showed dramatic improvement, particularly in Years 3–6 versus Years 0–3. Mean ARR fell from 1.00 to 0.03, MRI activity dropped from 31.5% to 1.5%, and disability progression slowed from 42.9% to 13.4%. By the second period, 84% of patients achieved NEDA-3. Notably, 76.5% of patients who lacked NEDA-3 in the first three years reached stability during the second period. For SPMS, early NEDA-3 achievement was the strongest predictor of long-term success (OR = 4.30, $p = 0.019$).

Conclusion: Ocrelizumab almost completely stops MS activity over a six-year period. The treatment's benefits increase over time, with the best results seen after the third year. Achieving stability early in treatment is key to long-term success, particularly for those with progressive MS.

Disclosure: Nothing to disclose.

Neuroimaging 1

EPO-0372 | Bayesian predictive validation of a multimodal Alzheimer disease modeling framework

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Background and aims: Disease-progression models inform cost-effectiveness analyses, payer decisions, and simulation studies of Alzheimer therapies. Multimodal latent models may improve biological fidelity, but their value for health-economic modeling depends on predictive validation. We evaluated calibration, accuracy, and decision relevance of a Bayesian latent trajectory model.

Methods: We analyzed 932 β -amyloid-positive participants from the Alzheimer's Disease Neuroimaging Initiative (ADNI) with repeated multimodal measures spanning neurodegeneration, amyloid/tau pathology, metabolism, neuroaxonal injury, and cognition. Probabilistic principal component analysis generated a harmonized latent severity score (PPC1). Hierarchical Bayesian regression estimated participant-specific trajectories.

Predictive validity was assessed using posterior predictive checks, cross-validated predictive error, predictive-interval coverage, widely applicable information criterion (WAIC), expected log predictive density, and Pareto-smoothed importance sampling leave-one-out cross-validation (PSIS-LOO).

Results: Posterior predictive distributions closely matched observed latent trajectories. Adding apolipoprotein E (APOE) genotype improved predictive accuracy while preserving calibrated uncertainty. Predictive-interval coverage was ~98%, supporting probabilistic sensitivity analysis. WAIC and expected log predictive density favored the genotype-augmented model by 16–22 points. PSIS diagnostics were stable with fewer influential observations, and conclusions were consistent across sensitivity analyses.

Conclusion: This validated multimodal Bayesian framework provides accurate, well-calibrated forward projections of Alzheimer progression with uncertainty suitable for decision modeling, supporting cost-effectiveness analyses, payer simulations, and trial-enrichment forecasting.

Disclosure: All authors are employees of Eisai Inc.

EPO-0373 | A multimodal latent severity axis for Alzheimer disease progression modeling

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Background and aims: Reliable quantitative severity scales are essential for disease-progression, value-based modeling, and simulation studies. Single-modality measures can misrepresent the interconnected biology of Alzheimer disease. We developed a multimodal latent disease-severity metric using probabilistic principal component analysis (PPCA) to integrate structural, molecular, metabolic, and cognitive signals into a biologically anchored index related to—but distinct from—clinical and biological staging.

Methods: Thirteen biomarkers from an amyloid-positive baseline cohort ($N=1,058$)—MRI atrophy, CSF tau and A β species, plasma neurofilament light, amyloid PET, FDG-PET, and cognitive scores—were harmonized, sign-aligned, and z-scaled after age and intracranial-volume adjustment where appropriate. PPCA was fit to the baseline matrix to define a latent coordinate system. Follow-up visits were projected using the PPCA conditional distribution to accommodate missing modalities, yielding a continuous severity axis (PPC1) robust to missingness. Structural stability was assessed by bootstrap resampling (100 replicates).

Results: PPC1 explained 38.7% of multimodal variance and was highly stable (loading correlations ≈ 0.997 ; score correlations ≈ 0.999). Loadings reflected coherent neurobiology spanning neurodegeneration, tau/amyloid dysregulation, metabolic decline, neuroaxonal injury, and cognitive impairment. Diagnostic groups aligned along an ordered continuum. PPC1 captures latent continuous severity inferred from multimodal patterns rather than a clinical stage, although its distribution tracked standard staging categories. Residual age effects were minimal after adjustment, and the multimodal score reduced noise relative to individual biomarkers.

Conclusion: PPCA yields a robust, biologically grounded severity index enabling cleaner longitudinal modeling, uncertainty propagation, and simulation inputs for value assessment.

Disclosure: All authors are employees of Eisai Inc.

EPO-0374 | Abstract withdrawn

EPO-0375 | Magnetic resonance neurography in neuralgic amyotrophy: Clinical and electrodiagnostic correlations

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Background and aims: Neuralgic amyotrophy (NA) is a non-traumatic peripheral nervous system disorder predominantly affecting the brachial plexus, typically presenting with unilateral involvement of the upper and middle trunks. Diagnosis is primarily clinical, while complementary studies mainly exclude alternative diagnoses. Advances in magnetic resonance neurography (MRN) allow detailed assessment of roots, trunks, and denervated muscles, revealing anatomical involvement in NA beyond clinical and electrodiagnostic evaluation. The objective is to describe MRN findings in patients with clinically diagnosed NA and assess concordance with clinical and electrodiagnostic features.

Methods: We retrospectively reviewed adult patients with NA undergoing MRN 2012–2025. Demographic and clinical variables, including symptom laterality and topography, were collected. Electrodiagnostic (EMG/NCS) and MRN findings were analyzed, focusing on root, trunk, and muscle involvement.

Results: Twenty-four patients were included (79% male; mean age 45.7 years). Mean time from symptom onset to evaluation was 2.3 months. Pain and weakness were present in 95.8%, and muscle atrophy in 75%. Proximal upper limb involvement in 87.5%. Electrodiagnostic studies ($n=21$) were abnormal in 85.7%, mainly involving the upper trunk (55.6%). MRN showed abnormal findings in all patients, predominantly affecting roots and trunks of the upper trunk territory (91.7%). Muscle denervation most commonly involved the supra- and infraspinatus muscles (41.7%). Concordance between clinical and electrodiagnostic topography was high, but MRN identified additional nerve and muscle involvement, including subtle or subclinical abnormalities.

Conclusion: MRN identified a broader extent of nerve and muscle involvement than clinical examination and electrodiagnostic studies. These findings support that NA may represent a more extensive, potentially bilateral and asymmetric radiculoplexopathy, usually unilateral clinically.

Disclosure: Nothing to disclose.

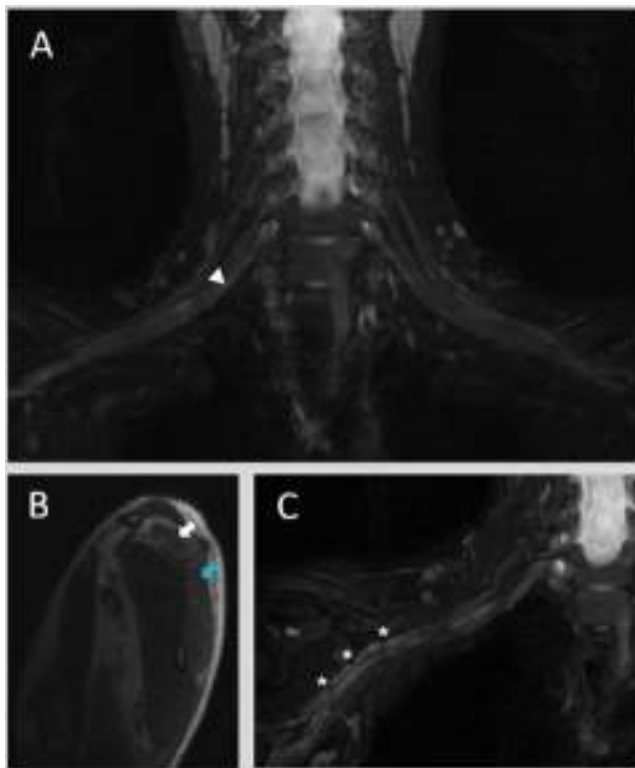


FIGURE 1 (A) MR neurography showing C5–C6 nerve enlargement and hyperintensity (arrow). (B) Sagittal T2 images revealing hyperintensity in the supraspinatus (white arrow) and infraspinatus (blue arrow). (C) Focal constrictions of upper trunk nerves (ast).

TABLE 1 Concordance between clinical presentation, electrodiagnostic (EDX), and MR neurography (MRN) topography in patients with neuralgic amyotrophy.

	Clinical (N=23)	EDX (N=23)	MRN (N=23)
			Neurography
Upper trunk (C5–C6)	23 (100%)	10 (50%)	21 (91.3%)
Middle trunk (C7)	12 (52.2%)	4 (25%)	10 (43.5%)
Lower trunk (L5–S1)	10 (43.5%)	3 (25%)	7 (30.4%)

EPO-0376 | Spatial covariance of cortical metabolism with multiple metrics of nigrostriatal degeneration in Dementia with Lewy bodies (DLB)

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Background and aims: From a biomarker perspective, Dementia with Lewy bodies (DLB) is characterised by striatal dopamine transporter (DaT) deficits and by specific patterns of cortical hypometabolism, assessable through DaT-SPECT and FDG-PET, respectively. However, the relationship between dopaminergic and metabolic imaging features remains unclear. This study aimed to

investigate the association between dopaminergic deafferentation in different striatal regions (caudate, anterior and posterior putamen) and cortical metabolic patterns in DLB.

Methods: 76 DLB patients (27 females, 49 males) who underwent both DaT-SPECT and FDG-PET were included. DaT-SPECT scans were analysed using DaTQUANT 2.0 to obtain striatal binding ratios (SBR). FDG-PET images were flipped to align the most affected hemisphere to the right. Correlations between SBR values and FDG-PET metabolic patterns were assessed using SPM12, correcting for age, sex and MMSE. Family-wise error-corrected p values <0.05 at the cluster level were considered significant.

Results: Caudate SBR reduction correlated with hypometabolism in parietal, occipital and lateral temporal cortices, with the most significant cluster in the lateral temporal cortex (p[FWE] <0.001). Anterior putamen SBR reduction was associated with a smaller hypometabolic cluster in similar regions, mainly in the angular gyrus (p[FWE] <0.005). Posterior putamen SBR reduction correlated predominantly with occipital hypometabolism, particularly in the inferior occipital gyrus (p[FWE] <0.001).

Conclusion: In DLB, progressive dopaminergic deafferentation from the posterior putamen to the caudate is associated with increasingly widespread cortical hypometabolism, with region-specific striatal deficits linked to distinct cortical metabolic patterns.

Disclosure: Nothing to disclose.

EPO-0377 | Intranasal esketamine expands key frontal and hippocampal regions and reduces serum neurofilament levels in patients with major depressive disorder

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Background and aims: We investigated the effects of intranasal esketamine (ESK-IN) on brain structural volumes and serum neurofilament light chain (sNfL) levels over a six-month period in patients with treatment-resistant depression (TRD).

Methods: 22 patients with TRD received ESK-IN in addition to ongoing oral antidepressant therapy. Clinical assessments were performed at baseline and after 1, 3, and 6 months of treatment. Structural brain MRI and blood sampling were conducted at baseline and at the six-month follow-up. MRI data from 44 matched healthy controls were also analyzed to enable comparisons of basal brain volume measures. Brain volumetric analyses were performed using VolBrain2 and DSI Studio software.

Results: At baseline, TRD patients showed significantly reduced volumes compared to healthy controls in the insula, frontal cortex, superior parietal cortex, and both superior and inferior temporal cortices. Reduced volumes were also observed in subcortical structures, such as the nucleus accumbens, putamen,

thalamus, and hippocampus. Following six months of ESK-IN treatment, significant volumetric increases were detected in the frontal cortex, superior frontal gyrus, frontal operculum, and anterior cingulate cortex, as well as in the hippocampus. Additionally, increases were observed in the volume and integrity of white-matter tracts involved in emotional regulation and cognitive control, including the frontoparahippocampal, frontoparietal, and frontal aslant tracts. Concomitantly, sNfL concentrations decreased after treatment, suggesting a reduction in ongoing neuronal damage.

Conclusion: Altogether, these findings indicate that ESK-IN may induce neuroplastic structural changes in brain regions critical for mood regulation while exerting potential neuroprotective effects in patients with TRD.

Disclosure: Nothing to disclose.

EPO-0378 | Molecular changes induced by anti-CGRP monoclonal antibodies for chronic migraine prophylaxis: A brain magnetic resonance spectroscopy study

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Background and aims: Calcitonin gene-related peptide monoclonal antibodies (CGRP-mAb) are effective treatments for preventing chronic migraine (CM). However, the molecular basis of indirect effects on the central nervous system (CNS) is still to be explored. We applied Brain Magnetic Resonance Spectroscopy (MRS) to investigate the neurochemical changes in the CNS induced by CGRP-mAb in CM.

Methods: This longitudinal study included 9 patients with CM without aura, evaluated before and six months after CGRP-mAb treatment initiation. At baseline, subjects underwent brain MRS (3T scanner, voxel 8 cm³, TE 135 ms) and comprehensive clinical evaluation including headache frequency, headache intensity (HIT-6), disability (MIDAS), cognitive assessment (MOCA) and mood (BDI). At 6-month follow-up, patients were re-evaluated with clinical assessment and MRS. All patients were scanned interictally. Linear mixed models were applied to evaluate changes in metabolite concentrations before and after treatment.

Results: After treatment neurochemical changes were observed: phosphocreatine (PCr) increased ($p=0.02$) and glutathione

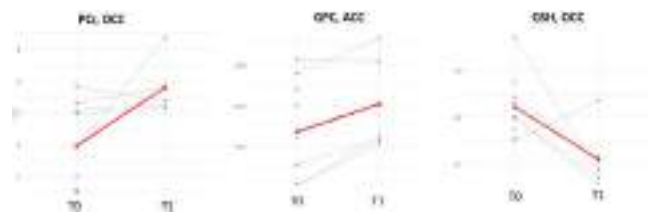


FIGURE 1

(GSH) decreased ($p=0.02$) in occipital cortex (OCC), whereas glycerophosphocholine (GPC) increased ($p=0.03$) in anterior cingulate cortex (ACC). Trends suggested positive correlations between baseline migraine intensity and changes in PCr and GPC concentration in OCC and ACC, respectively.

Conclusion: The main finding of this study is the increase of PCr concentration in OCC, in line with previous evidence of the key role of brain energetics in CM. Whether this represents a central correlate of treatment or an epiphenomenon related to the reduction in migraine frequency requires further investigation.

Disclosure: Nothing to disclose.

EPO-0379 | Multispiral computed tomographic angiography in diagnosing neurovascular conflict in classical trigeminal neuralgia: A 15-year study

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Background and aims: The aim of the study was to compare the accuracy of magnetic resonance imaging (MRI) and 3D multispiral computed tomographic angiography (MSCTA) in differentiating neurovascular conflict in patients with classical trigeminal neuralgia (CTN).

Methods: The study included 500 patients diagnosed with CTN based on the actual ICHD and the ICOP classification. Each patient underwent MRI scans and MSCTA in 3D mode with bolus contrast. The study focused on identifying neurovascular conflict (NVC) by examining the superior cerebellar artery loop's apex as it transitions from the anterior pontomesencephalic segment to the lateral segment, relative to the Meckel cave and the trigeminal nerve root. Microvascular decompression (MVD) was performed on patients who did not respond to conservative treatment. Intraoperative assessments were conducted to verify the MRI and MSCTA findings.

Results: The MRI performed on patients effectively excluded secondary causes of CTN, but it could not determine the type of NVC or distinguish between NVC and contact. MSCTA revealed NVC type 1 in 320 patients (64%), type 2 in 180 patients (36%). Intraoperative assessments in 412 patients indicated that MRI misdiagnosed neurovascular conflict in 64 cases (15.5%). Notably, 78 patients (18.9%) with MRI-confirmed neurovascular conflict who underwent microvascular decompression did not experience pain relief.

Conclusion: MRI has limitations in differentiating between NVC and contact, leading to concerns about overdiagnosis and misinterpretation in CTN patients and healthy individuals. This reliance on MRI may reduce favorable surgical outcomes.

MSCTA demonstrates higher accuracy in identifying the neuro-vascular relationship and indicating the need for MVD.

Disclosure: Nothing to disclose.

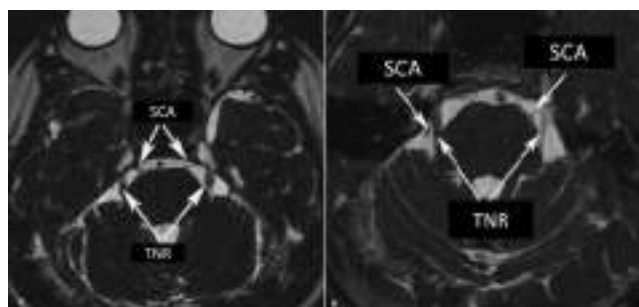


FIGURE 1 Magnetic resonance imaging: a) there is no conflict between the trigeminal nerve root (TNR) and the superior cerebellar artery (SCA); b) neurovascular conflict between the trigeminal nerve root and the superior cerebellar nerve on the left.

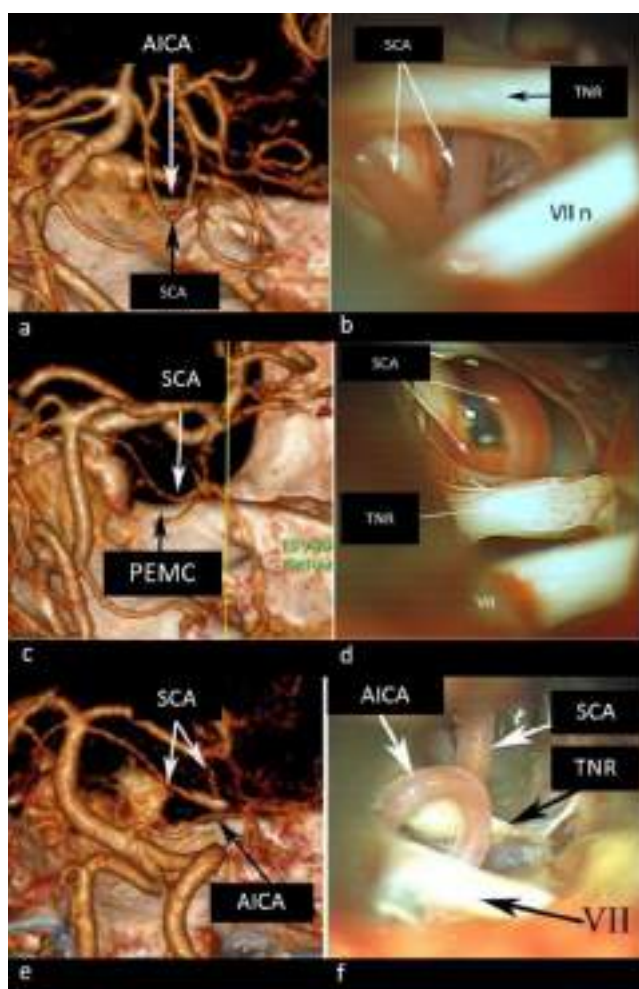


FIGURE 2 Results of multispiral computed tomographic angiography (MSCTA) in 3D mode with bolus contrast and intraoperative pictures during microvascular decompression: a) Neurovascular conflict type 1 – MSCTA; b) Neurovascular conflict type 1.

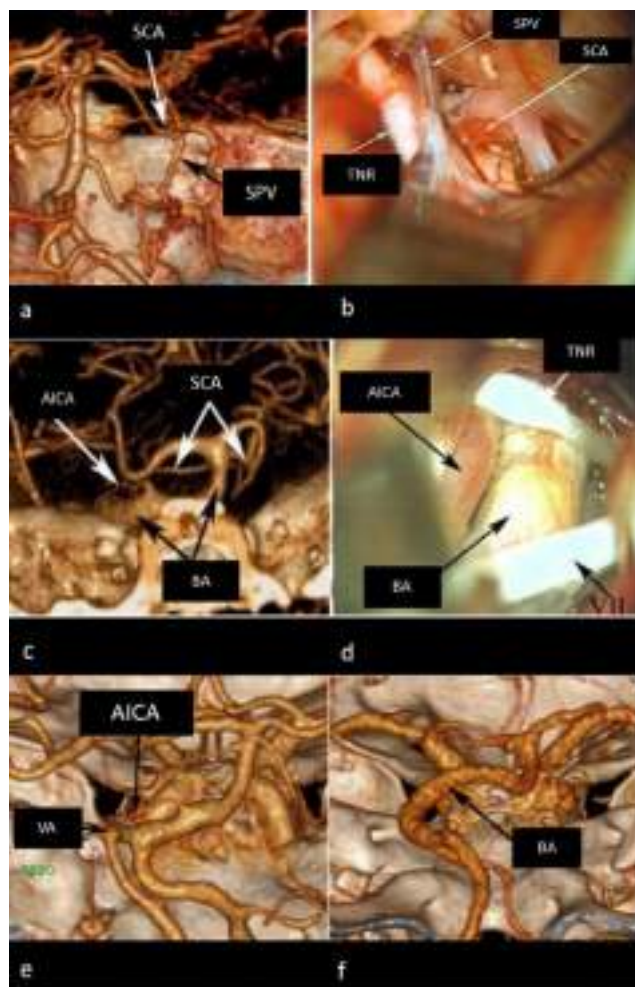


FIGURE 3 Results of multispiral computed tomographic angiography (MSCTA) in 3D mode with bolus contrast and intraoperative pictures during microvascular decompression: a) Arterio-venous sandwich (trigeminal nerve root between an artery and a vein).

EPO-0380 | A progressive lesional mantle adjacent to the frontal cortices disclosing a CSF1R leukoencephalopathy in presymptomatic stage

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Background and aims: Colony-stimulating factor-1 receptor (CSF1R) related leukoencephalopathy usually presents around the 4th decade of life and frequently progresses to death in a few years. Transmission is autosomal dominant but de novo mutations and incomplete penetrance may account for a negative family history. Allogenic bone marrow transplantation has been reported to stabilize patients and a therapeutic trial with a receptor agonist is ongoing. Early diagnosis will be essential to allow treatment before neurologic damage installs.

Methods: We report the evolution of a presymptomatic patient and describe a particular MRI pattern that could help to identify other patients.

Results: A 35-year-old woman with no relevant personal medical history, underwent a brain MRI for tension-type headaches. Besides a few unspecific deep white matter point lesions, a

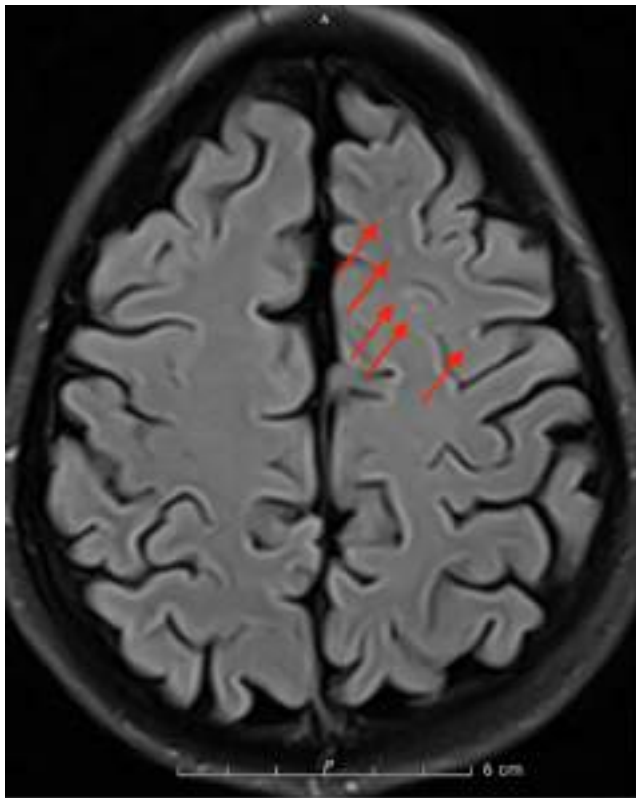


FIGURE 1 30years-old. FLAIR. Small lesions adjacent to the left frontal cortex.

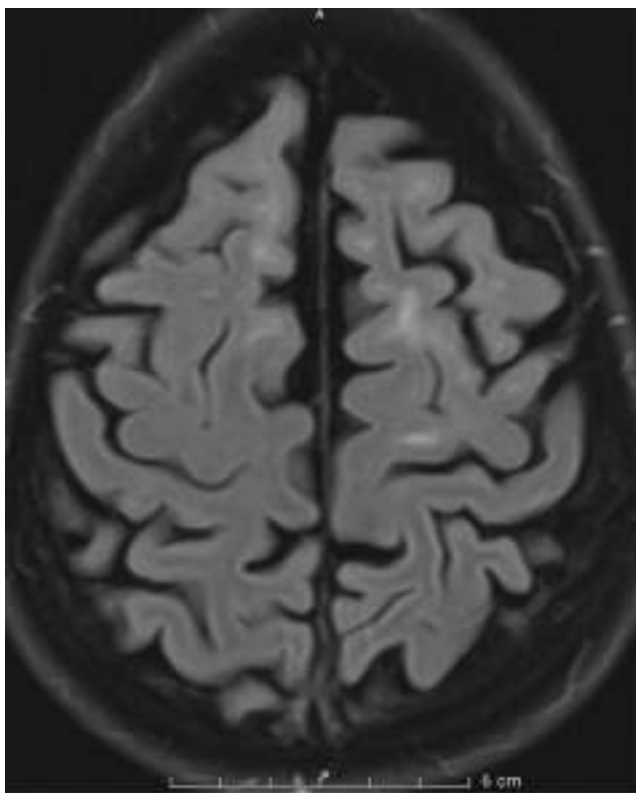


FIGURE 2 40 years-old. FLAIR. This unusual pattern raised the hypothesis of a leukodystrophy.

few unusual small lesions adjacent to both prefrontal cortices (but sparing the U-fibers) were present. A subsequent MRI at 39 years-old showed a degree of coalescence of the frontal lesions

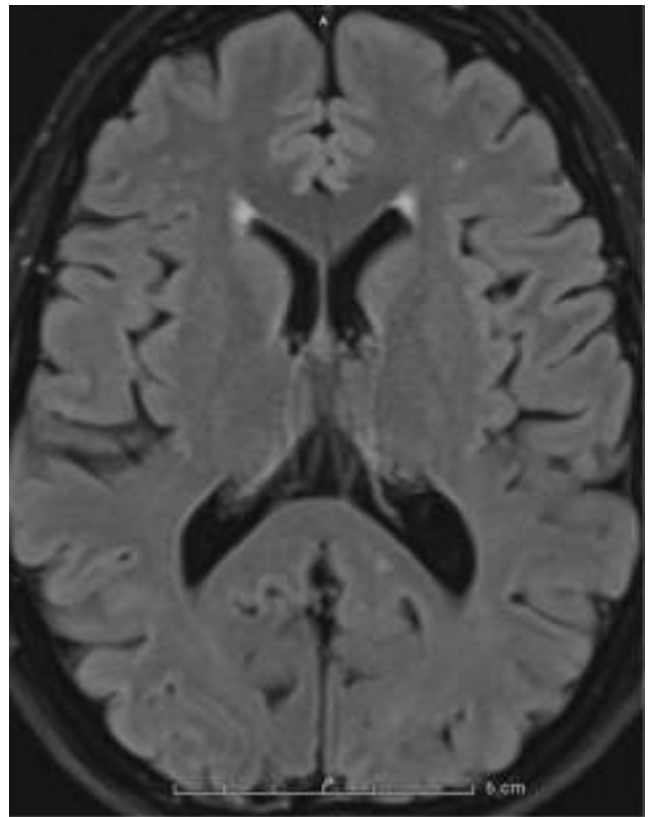


FIGURE 3 40years-old. Lesions elsewhere in the brain are sparse and unspecific and wouldn't disclose the diagnosis by themselves.

adjacent to the cortex, starting to form a “mantle”. One year later this mantle started to appear also in the right temporal lobe and 2 small lesions showed restriction on diffusion. She remained asymptomatic but a first neurological examination by that time noted some mild attention deficits and a bilateral ankle clonus. She reported that her mother had died of an obscure neurological disease by the age of 44. Genetic testing disclosed a c.2625G > A mutation. At 41 years-old she remains stable.

Conclusion: A patchy mantle of small frontal juxtacortical lesions can disclose this disease in a very early phase.

Disclosure: Nothing to disclose.

EPO-0381 | Substantia nigra neuromelanin magnetic resonance imaging in LRRK2 asymptomatic carriers

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Background and aims: Parkinson's disease (PD) is a progressive neurodegenerative disorder, with long pre-clinical and prodromal stages. The development of non-invasive biomarkers for pre-diagnostic stages is of utmost importance. Neuromelanin (NM)-sensitive magnetic resonance imaging (MRI) of the substantia nigra (SN) is an established biomarker of neurodegeneration in PD, which is expected to begin years before motor symptoms emerge, and for which LRRK2 mutation asymptomatic carriers are at higher risk. We aim to investigate NM MRI in asymptomatic LRRK2 carriers

Methods: Asymptomatic at-risk first-degree relatives of LRRK2-PD patients were sequentially recruited. An extensive clinical evaluation, including motor and non-motor symptoms, as well as a study of neuromelanin, using a specific T1-weighted MRI sequence, was performed. Targeted genetic testing for LRRK2 variants was performed after clinical evaluation and imaging studies.

Results: Nineteen asymptomatic participants were included, of which 12 LRRK2 non-carriers (NC) and 7 carriers (AC). Baseline demographics did not differ between groups (median age AC 46years (IQR 35–53) vs NC 51.5 (39–59) $p=0.379$). Motor scores did not differ between groups (MDS-UPDRS III median 2 (2–4) in AC vs 2.5 (1.5–5.4) in NC, $p=0.863$). NM area was significantly smaller in AC when compared to NC, (16 mm^2 (14.9–19.1) vs 20.6 mm^2 (19.3–24.0), $p=0.025$).

Conclusion: NM-MRI can be significantly decreased in LRRK2-G2019S AC. This technique may be a promising biomarker of neurodegeneration in pre-diagnostic stages of PD.

Disclosure: Nothing to disclose.

EPO-0382 | Association of glymphatic imaging markers with functional independence and white matter hyperintensity burden after intracerebral hemorrhage

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Background and aims: Glymphatic system plays a critical role in cerebral metabolite clearance and white matter injury. However, its clinical relevance and underlying mechanisms in intracerebral hemorrhage (ICH) remain poorly understood. This study aimed to investigate imaging-based glymphatic markers in relation to functional outcomes and white matter hyperintensity (WMH) burden in patients with ICH.

Methods: We included 70 patients with acute supratentorial ICH who underwent diffusion tensor imaging (DTI). Glymphatic-related imaging parameters were derived using DTI and T1-weighted MRI, including the contralateral DTI-ALPS index, free water (FW) ratio in contralateral deep white matter (WM), and relative perivascular space (PVS) volume. Relative WMH volume was defined as absolute WMH normalized to intracranial volume and log-transformed. Functional independence was defined as a 3-month modified Rankin Scale score of 0–2. Associations were examined using multivariable logistic and linear regression models.

Results: In fully adjusted logistic regression models, higher FW in deep WM was independently associated with a lower likelihood of functional independence at 3 months (OR=0.59; 95% CI, 0.36–0.98; $P=0.041$), with consistent findings across sequential

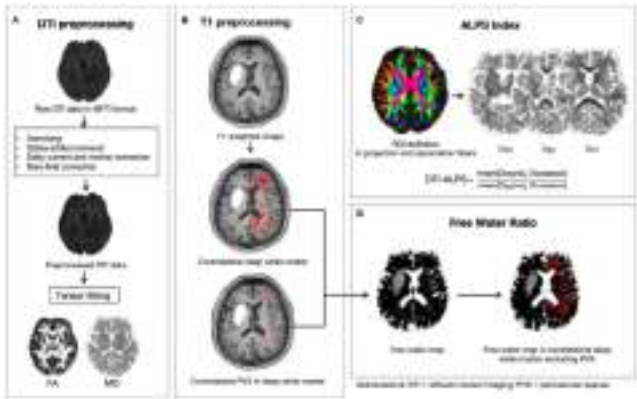


FIGURE 1 Flowchart of imaging processing. (A) Diffusion tensor imaging preprocessing. (B) Generation of contralateral deep white matter (WM) and PVS masks. (C) Calculation of the DTI-ALPS. (D) Free water ratio in contralateral deep WM excluding PVS.

TABLE 1 Associations between glymphatic imaging parameters and functional independence.

OR (95% CI)			P-value	OR (95% CI)			P-value	OR (95% CI)			P-value	
Model 1				Model 2				Model 3				
FW in deep WM	0.48 (0.28 to 0.81)	0.001		Contralateral ALPS	1.01 (0.68 to 1.50)	0.95		Relative PVS volume	1.11 (0.59 to 1.95)	0.73		
Age	0.99 (0.98 to 1.01)	0.195		Contralateral ALPS	1.01 (0.68 to 1.50)	0.95		Age	0.99 (0.98 to 1.01)	0.295		
NIHSS	0.99 (0.98 to 1.01)	0.195		Age	0.99 (0.98 to 1.01)	0.295		NIHSS	0.99 (0.98 to 1.01)	0.295		
Baseline hematoma volume	0.99 (0.98 to 1.00)	0.001		Baseline hematoma volume	0.99 (0.98 to 1.01)	0.001		Baseline hematoma volume	0.99 (0.98 to 1.01)	0.001		
Scanner	0.97 (0.71 to 1.34)	0.533		Scanner	0.98 (0.71 to 1.25)	0.543		Scanner	0.98 (0.71 to 1.35)	0.533		
Model 2				Model 3				Model 4				
FW in deep WM	0.53 (0.32 to 0.88)	0.011		Contralateral ALPS	1.01 (0.67 to 1.50)	0.95		Relative PVS volume	1.11 (0.59 to 1.95)	0.73		
Age	0.99 (0.98 to 1.01)	0.195		Age	0.99 (0.98 to 1.01)	0.295		Age	0.99 (0.98 to 1.01)	0.195		
NIHSS	0.99 (0.98 to 1.01)	0.001		Baseline hematoma volume	0.99 (0.98 to 1.01)	0.001		Baseline hematoma volume	0.99 (0.98 to 1.01)	0.001		
Scanner	0.98 (0.71 to 1.34)	0.195		Scanner	0.98 (0.71 to 1.34)	0.195		Scanner	0.98 (0.71 to 1.34)	0.195		
Model 3				Model 4				Model 5				
FW in deep WM	0.48 (0.28 to 0.81)	0.001		FW in deep WM	0.48 (0.28 to 0.81)	0.001		FW in deep WM	0.48 (0.28 to 0.81)	0.001		
Age	0.99 (0.98 to 1.01)	0.195		Age	0.99 (0.98 to 1.01)	0.195		Age	0.99 (0.98 to 1.01)	0.195		
NIHSS	0.99 (0.98 to 1.01)	0.195		NIHSS	0.99 (0.98 to 1.01)	0.195		NIHSS	0.99 (0.98 to 1.01)	0.195		
Baseline hematoma volume	0.99 (0.98 to 1.00)	0.001		Baseline hematoma volume	0.99 (0.98 to 1.01)	0.001		Baseline hematoma volume	0.99 (0.98 to 1.01)	0.001		
Scanner	0.97 (0.71 to 1.34)	0.195		Scanner	0.98 (0.71 to 1.34)	0.195		Scanner	0.98 (0.71 to 1.34)	0.195		

TABLE 2 Linear regression analyses between relative WMH volume and glymphatic parameters. Model 1: Unadjusted. Model 2: Adjusted for age, NIHSS, and baseline hematoma volume. Model 3: Adjusted for variables in model 2, mean diffusivity and scanner.

	FW in deep WM	Contralateral ALPS	Relative PVS volume
Model 1	0.002 (0.001 to 0.003)	-0.001 (-0.002 to 0.001)	-0.002 (-0.003 to -0.001)
Model 2	0.001 (0.000 to 0.002)	-0.001 (-0.002 to 0.001)	-0.001 (-0.002 to 0.001)
Model 3	0.143 (0.008 to 0.277)	0.008 (0.000 to 0.016)	-0.001 (-0.002 to 0.001)

models. Linear regression analyses further demonstrated that greater relative WMH burden was significantly associated with increased FW in deep WM ($\beta=0.143$; 95% CI, 0.008–0.277; $P=0.038$) and with smaller relative PVS volume in fully adjusted models.

Conclusion: These findings suggest that white matter injury may influence functional recovery after ICH through glymphatic system dysfunction, with FW emerging as the most sensitive imaging marker linking WMH burden to adverse functional outcomes.

Disclosure: We declare no conflict of interests.

EPO-0383 | Peri-nidal hypoperfusion on multi-delay ASL is associated with epilepsy in unruptured brain arteriovenous malformation

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Background and aims: The hemodynamics of epilepsy in unruptured brain AVM remains unclear. This study aims to investigate the perfusion characteristics of the whole brain and peri-nidal tissues and explore their relationships with AVM-related epilepsy.

Methods: This study enrolled patients with unruptured brain AVMs. Time-encoding multi-delay ASL was performed to obtain corrected cerebral blood flow (cCBF), arterial transit time (ATT), and arterial cerebral blood volume (aCBV). After excluding draining veins, five concentric perinidal zones were delineated, each 2mm thick. Within each zone, perfusion parameters were specifically quantified for the entire brain tissue, gray matter, and white matter separately. Independent samples t-tests were applied to compare these parameters between AVM patients with and without epilepsy.

Results: A total of 40 AVM patients (20 with epilepsy, 20 without) were included. While no global perfusion differences were found, patients with epilepsy showed significantly lower cCBF and aCBV in the tissue closest to the nidus (≤ 2 mm), particularly in gray matter, compared to non-epilepsy patients. This difference diminished with increasing distance from the nidus, with the most severe impairment found in the peri-nidal gray matter.

Conclusion: The findings of a pronounced and distance-dependent steal effect in epilepsy patients support the hemodynamic steal hypothesis. This suggests that an AVM's high-flow shunt critically diverts blood, causing chronic ischemia in the adjacent cortex. Such ischemia may contribute to neuronal

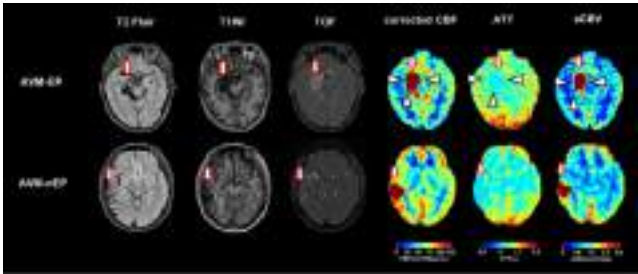


FIGURE 3 Imaging comparison and the peri-nidal steal phenomenon.

dysfunction, selective loss, and gliosis, which collectively lower the seizure threshold and create an epileptogenic focus, revealing a potential hemodynamic mechanism for epilepsy.

Disclosure: Nothing to disclose.

Cognitive Neurology/Neuropsychology 3

EPO-0384 | Augmenting clinical reasoning: Integrating artificial intelligence into neuropsychological and mental health assessment

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Background and aims: Artificial intelligence (AI) is increasingly entering neuropsychological and mental health practice, yet its adoption remains limited by ethical concerns, professional responsibility, and clinician acceptability. Beyond technical performance, effective integration of AI in neurology and neuropsychology depends on alignment with clinical reasoning, ethical standards, and professional identities. This presentation reflects on these challenges through the design of Flow, an AI-enabled clinical platform developed in collaboration with mental health professionals.

Methods: This presentation adopts a conceptual, practice-informed approach grounded in participatory and ethical-by-design principles. Flow was developed through iterative exchanges with neuropsychologists and clinicians, focusing on real-world workflows. The platform functions as a supervisory tool that supports clinical reasoning without generating autonomous diagnoses. Transparency, clinician control, and clear boundaries of responsibility guide its design.

Results: Drawing on clinical feedback and field experience, this presentation synthesizes key themes related to clinician expectations, perceived risks, and conditions for acceptability of AI tools. Tensions between efficiency and clinical nuance, automation and professional judgment, and innovation and regulation are highlighted.

Conclusion: Overall, the future of AI in neuropsychology and mental health depends less on algorithmic sophistication than on ethical design, clinician trust, and alignment with professional values.

Disclosure: Annie Desmarais is the founder and CEO of Flow Factor, the company developing the Flow clinical AI platform. Her postdoctoral research is conducted on and in relation to the Flow platform.

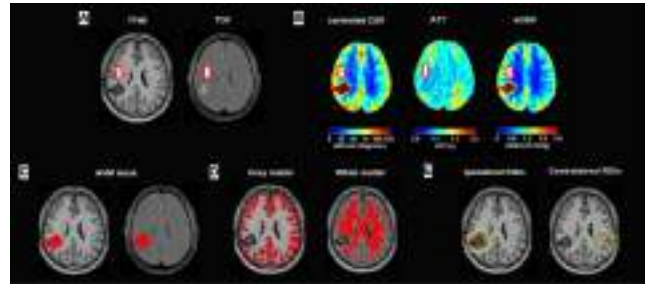


FIGURE 1 Anatomical axial images, perfusion maps, and tissue segmentations.

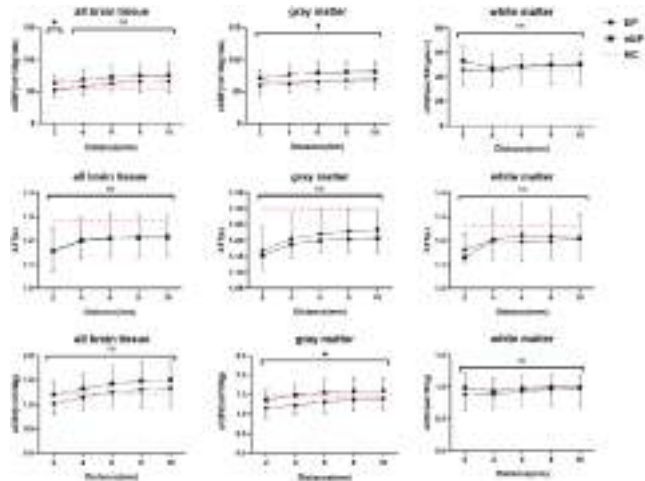


FIGURE 2 Comparison of perinidal cCBF, ATT, and aCBV between AVM patients with and without epilepsy.

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Background and aims: Growing evidence reported social cognition impairments in patients following stroke, with converging findings highlighting a higher prevalence in individuals with right-hemisphere lesions. Despite increasing recognition of socio-cognitive deficits after stroke, substantial variability remains across studies with respect to prevalence rates, affected sub-domains, and underlying neural correlates. This systematic review therefore aimed to synthesize current evidence on post-stroke social cognition impairments, with a specific focus on domain-specific performance and hemispheric lesion patterns, to define the post-stroke socio-cognitive profile.

Methods: A systematic literature search was conducted in PubMed, Scopus, and Web of Science databases following PRISMA guidelines. The risk of bias assessment was assessed with the AXIS and the NIH Quality Assessment tools.

Results: Among the 1293 papers initially retrieved, 33 studies met eligibility criteria. Quality assessment showed a middle-high overall quality of evidence. Emotion recognition is the most frequently investigated socio-cognitive sub-domain, with impairments reported in about 25–35% of post-stroke patients without any consistent emotion-specific pattern. Theory of Mind deficits are reported in both cognitive and affective components, especially following right hemisphere strokes. Findings on empathy are more heterogenous, with studies reporting reduced cognitive empathy alongside relatively preserved affective empathy.

Conclusion: Despite consistent evidence across studies, heterogeneity in assessment methods, timing and samples strongly prevents a clear profiling of social cognition impairments in post-stroke patients. Standardization of cognitive assessment protocols and harmonization of neuropsychological procedures is needed to overcome current literature gaps and improve diagnostic precision, support targeted rehabilitation, and enhance functional and interpersonal outcomes in stroke survivors.

Disclosure: None.

EPO-0386 | Early-life multidomain engagement relates to semantic fluency and episodic learning in aging and mild cognitive impairment

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Background and aims: Engagement in cognitively, physically, creatively, and socially stimulating activities is recognized as a modifiable protective factor against cognitive decline and dementia. However, whether early-life periods represent sensitive windows for later-life neuropsychological resilience remains insufficiently characterized, particularly across the continuum

TABLE 1 Significant HMR results — dNA → Cognitive.

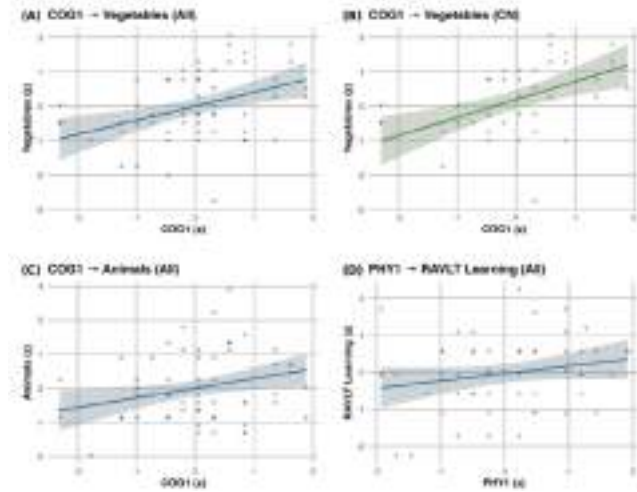
dNA	It	Cog Test	N	β	95%-CI	p	FDR p	AK Block	p (F-change)
COG1	All	Vegetables	58	0.410	[0.239, 0.543]	.001	.003	0.154	0.000
COG1	CN	Vegetables	19	0.521	[0.239, 0.811]	.001	.003	0.245	0.000
COG1	All	Animals	58	0.487	[0.283, 0.771]	.002	.005	0.162	0.011
PHY1	All	RAVLT— Learn	55	0.417	[0.154, 0.681]	.001	.006	0.233	0.007

Note: Block 1 = age, sex, edu, diagnosis; Block 2 = dNA domains from the same time windows (TW). dNA = dynamic Neurocognitive Adaptation; CN = cognitively normal; MCI = mild cognitive impairment; FDR = False Discovery Rate adjusted p-value (Benjamini-Hochberg procedure). Time windows: TW1 = 0–10 years, TW2 = 11–20 years, TW3 = 21–30 years, TW4 = 31–40 years, TW5 = 41–50 years, TW6 = 51–60 years.

dNA- Cognitive RESULTS

Fig. 1. dNA COG1 and PHY1 associations with cognitive scores
Panels show partial regressions with adjusted fits (solid line) and 95% confidence intervals (shaded bands); axes are z-scores. Covariates: age, sex, education, and diagnosis. The CN-only panel excludes participants with MCI.

Notes: Models are adjusted as above; no influential outliers; quadratic terms non-significant unless indicated in the text.



from cognitively unimpaired (CU) aging to mild cognitive impairment (MCI).

Methods: Fifty-eight participants, including CU older adults and individuals with MCI, completed the dynamic Neurocognitive Adaptation (dNA) scale, assessing engagement across four domains (cognitive, physical, creative, social) over seven life-course time windows. Neuropsychological outcomes included semantic fluency (vegetables, animals) and episodic learning (RAVLT). Participants also underwent amyloid PET assessment. Hierarchical regression analyses examined associations between domain- and time-specific dNA scores and cognitive performance, controlling for age, sex, education, and amyloid status.

Results: Childhood emerged as a sensitive period for neuropsychological outcomes. Higher childhood cognitive engagement (COG1) was associated with better semantic fluency (vegetables: $\beta=0.410$, $p=0.003$; animals: $\beta=0.487$, $p=0.005$), while higher childhood physical engagement (PHY1) was associated with stronger episodic learning ($\beta=0.417$, $p=0.006$). No dNA domain predicted delayed recall. All associations survived false discovery rate correction and were not moderated by sex, education, or diagnosis.

Conclusion: Early-life multidomain engagement, particularly cognitive and physical activity during childhood, is associated with preserved semantic and episodic memory performance in CU and MCI. These findings support early-life engagement as a protective factor for neuropsychological resilience and highlight the value of time-resolved tools such as the dNA for prevention research.

Disclosure: Nothing to disclose.

EPO-0387 | Alzheimer's disease in stroke: Prevalence in 3 cohorts and links with clinical, imaging, and cognitive status

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Background and aims: Amyloidopathy is reported in stroke populations with varying prevalence and cognitive impact. To address this limitation, the present study pooled data from the 3 largest stroke cohorts assessing cognitive status and amyloid burden using PET (IDEA3, ref 1, St Louis, 2 and STRIDE, 3). Our primary objective was to estimate the prevalence of amyloidopathy in stroke. Secondary objectives included associations between amyloidopathy and clinical characteristics, Mini-Mental State Examination (MMSE) score, cognitive status and brain imaging markers.

Methods: A cross-sectional analysis was performed on 218 patients (age 70.8 ± 10.8 years; 59% male; education 9.5 ± 4.4 years), including infarct, TIA, and hemorrhage. Cognitive status was categorized as dementia, mild cognitive impairment, or normal. Data were analyzed using mixed models adjusted for age, education, stroke subtype, stroke severity, and prestroke dementia.

Results: Amyloidopathy prevalence was 19.7% and increased with age ($p=0.001$), with no effect of stroke-PET time interval ($p=0.1$). Amyloidopathy was associated ($p=0.012$) with lower MMSE score but not with cognitive status. Regarding imaging amyloidopathy was associated with lobar microbleeds ($p=0.04$) but not with deep microbleeds, white matter hyperintensities and hippocampal volume.

Conclusion: Amyloidopathy affects about one fifth of stroke patients, increasing with age. It worsens cognitive performance as measured by MMSE but does not influence overall cognitive status, highlighting the role of vascular lesions in post-stroke impairment. The presence of lobar microbleeds should prompt the search for amyloidopathy, even in ischemic stroke. References 1. Godefroy O et al. Stroke 2025;56:74–83 2. Koenig L.N et al. NeuroImage: Clinical 2021, 102553 3. Yang J et al. Alzheimers&dementia 2015, Vol: 11, Pages: 16–23.

Disclosure: Marissa Streitz and John C. Morris are funded by National Institutes of Health grants (P30AG066444, P01AG003991, P01AG026276). Tammie Benzinger and Brian Gordon report no conflicts of interest other than support from the main Alzheimer's Disease Research Center grants. Sandrine Canaple reports having received travel funding from Medtronic. Olivier Godefroy has served, within the last two years, on scientific advisory boards and as a speaker for GE Healthcare, ESAI SAS, and Novo Nordisk; he did not receive funding for travel or meetings from pharmaceutical companies. Jeremy Neel, Vincent C.T. Mok, Pierre-A. Cervellera, Martine Roussel, Bonnie Y.K. Lam, Chantal Lamy, Betsy Grant, Audrey Courselle-Arnoux, Adrian Wong, and Claire Leclercq declare no conflicts of interest.

EPO-0388 | Sex-specific cognitive divergence in Parkinson's disease

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Background and aims: Sex differences in cognitive impairment in Parkinson's disease (PD) have been reported, with several studies suggesting greater cognitive decline in males. However, evidence on long-term trajectories across specific cognitive domains is limited, as most prior work is cross-sectional. Thus, we aim to investigate sex-specific longitudinal changes across multiple cognitive domains over 9 years in idiopathic PD, compared to demographically matched controls.

Methods: We analyzed data from the prospective ParkWest study, including $n=190$ persons with PD (PwPs) and $n=202$ age- and sex-matched controls, followed for 9 years. Regularly assessed cognitive domains included semantic fluency, processing speed, cognitive control, memory, and visuospatial ability. Linear mixed-effect models were used, including a three-way interaction between time, disease status and sex, to test whether sex-specific cognitive change in PD differed from that observed in controls. Models were adjusted for age, years of education, baseline depression, and UPDRS part II.

Results: Male PwPs showed faster decline than female PwPs across most cognitive measures, resulting in gradually increasing sex-differences over 9 years. However, compared with controls, the differential sex-specific decline was statistically significant only for semantic fluency ($p=0.013$) and processing speed ($p=0.027$ and $p=0.035$). These findings remained significant after covariate adjustment.

Conclusion: Over 9 years, PD was associated with increasing sex-differences in semantic fluency and processing speed, driven by faster decline in males. These findings confirm the male susceptibility to cognitive decline in PD and suggest that sex-specific vulnerabilities in cognitive trajectories in PD are domain-specific.

Disclosure: Nothing to disclose.

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Background and aims: Early identification of mild cognitive impairment due to Alzheimer's disease (MCI) is essential for timely intervention. Although episodic memory deficits are central to MCI, language performance may also be affected early. This study examined cognitive and language performance differences between individuals with MCI and cognitively healthy older adults using the Bulgarian version of the Scales of Cognitive and Communicative Ability for Neurorehabilitation (SCCAN).

Methods: Thirty individuals with MCI and 30 cognitively healthy controls were assessed with the Bulgarian SCCAN. Group differences were examined using independent samples t-tests for the total SCCAN score and selected domains, including episodic memory, attention, problem solving, and oral expression.

Results: Participants with MCI showed significantly lower total SCCAN scores than healthy controls (mean 75.7, SD 8.0 vs. mean 85.2, SD 8.8; $p < 0.001$), with a large effect size (Cohen's $d = 1.13$). Episodic memory demonstrated the largest group difference ($p < 0.001$, Cohen's $d = 1.75$). Oral expression was also significantly reduced in the MCI group ($p = 0.006$, Cohen's $d = 0.73$). Differences in attention and problem solving were smaller and did not reach statistical significance, although moderate effect sizes were observed.

Conclusion: Beyond memory impairment, individuals with MCI exhibit clinically meaningful deficits in oral language performance. These findings highlight the importance of incorporating language performance measures into early cognitive assessment and support the clinical utility of the Bulgarian SCCAN for identifying cognitive-communicative profiles in MCI.

Disclosure: The authors report no conflicts of interest.

EPO-0390 | Sustained cognitive and mood benefits of a prismatic adaptation-based digital therapy in stroke rehabilitation

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Background and aims: Stroke survivors often experience persistent cognitive deficits and mood disturbances, which can hinder functional recovery. Non-invasive neuromodulation combined with game-based digital therapies has been proposed as a novel approach to enhance cognitive and emotional rehabilitation. This study investigated whether a prismatic adaptation-based digital therapy could provide sustained cognitive and mood benefits compared with standard pen-and-paper rehabilitation.

Methods: Patients with a first-ever ischemic or hemorrhagic stroke affecting either hemisphere were recruited at the Santa

Table 1: Demographic and baseline characteristics of study participants. Continuous variables are reported as mean ± standard deviation (SD), and categorical variables as number and percentage (%). VIS = visual perception; HHG = spatial attention/hemineglect; LAN = language; MEM = memory; EXF = executive functions.

	Experimental (N=28)	Control (N=29)	Group differences (p-value)
Age, mean (SD)	60.86 (14.67)	61.85 (11.48)	$p=0.628$
Sex, Female, No (%)	14 (50%)	17 (58.6%)	$p=0.514$
Education, years, mean (SD)	13.39 (4.46)	13.51 (3.97)	$p=0.918^*$
Stroke location, Left, No (%)	13 (46.4%)	10 (34.5%)	$p=0.389$
VIS, mean (SD)	201.96 (54.81)	176.12 (73.31)	$p=0.367$
HHG, mean (SD)	32.11 (15.83)	32.33 (13.94)	$p=0.962$
LAN, mean (SD)	86.07 (29.24)	86.83 (25.48)	$p=0.941$
MEM, mean (SD)	88.11 (28.13)	88.89 (29.24)	$p=0.978$
EXF, mean (SD)	87.14 (26.11)	81.84 (27.13)	$p=0.488$

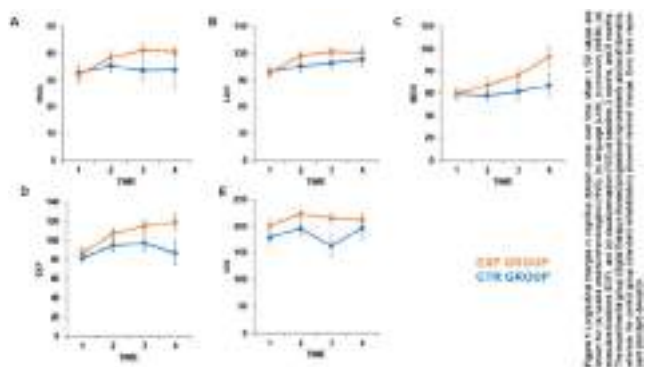


FIGURE 1 Longitudinal changes in cognitive domain scores over time.

Lucia Foundation IRCCS, Rome, Italy. Participants were randomly assigned to an experimental group ($n = 28$) receiving the digital therapy or a control group ($n = 29$) receiving standard cognitive rehabilitation. Cognitive outcomes were measured across five domains: visual perception, spatial attention/hemineglect, language, memory, and executive functions. Depressive symptoms were assessed using the Hamilton Depression Rating Scale (HAM-D) at baseline, 3 months, and 6 months.

Results: Significant Group $\times$ Time interactions were observed for all cognitive domains. Participants in the experimental group demonstrated progressive improvements over time, whereas the control group showed no significant changes. Cognitive gains were most pronounced at the 3- and 6-month follow-ups. Additionally, depressive symptoms were significantly reduced in the experimental group at 3 months compared with baseline.

Conclusion: Prismatic adaptation-based digital therapy resulted in sustained cognitive improvements and reduced depressive symptoms post-stroke. Combining non-invasive neuromodulation with game-based cognitive training may enhance rehabilitation outcomes by addressing both cognitive and emotional recovery in stroke survivors.

Disclosure: None.

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Background and aims: The Dual Loop Model with hubs in inferior frontal and parieto-temporal cortex, combining the dorsal pathway for processing sequences and the ventral pathway for hierarchical-structural categorisation, was suggested as the anatomical substrate for syntax in all modalities. The caudal pathway, from multimodal associations in the angular gyrus to abstract concept formation in anterior temporal lobe (ATL), was suggested as essential for semantics. In stroke patients, we searched for the common anatomical substrate for syntactic and semantic deficits in apraxia, aphasia and neglect.

Methods: 165 patients with a single left medial cerebral artery infarction were tested for apraxia, the Token Test and spatial attention. We used the new method of streamline-based lesion-symptom mapping (SLSM, FDR 0.001) to detect white matter disconnections related to common behavioural deficits.

Results: Two Principal components (PC) explained 60% of variance across domains. PC1 (50% of variance) was associated with progressive loading for more complex apraxia tests, the Token Test and neglect. PC2 (an additional 10%) related to easier apraxia tests and extinction (Figure 1). Affected streamlines associated with PC1 included the dorsal and ventral pathway of the dual loop, additionally the Middle longitudinal Fascicle (MdIF) as the caudal pathway. PC2 was only associated with the dorsal pathway and MdIF. The interaction analysis showed that the ventral pathway, predominantly connecting inferior frontal cortex with ATL, was selectively involved in PC1 (Figure 2).

Conclusion: A lesion of the ventral pathway leads to multimodal deficits when syntax and semantics have to be combined.

Disclosure: MR, EvdH, and CW are supported by EBRAINS2.0 AD is supported by the Berta-Ottenstein Advanced Clinical Scientists program.

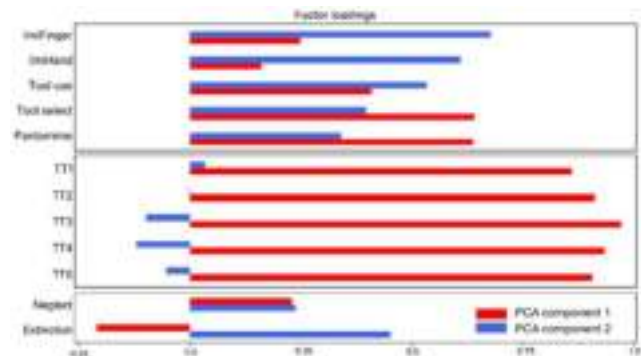


FIGURE 1

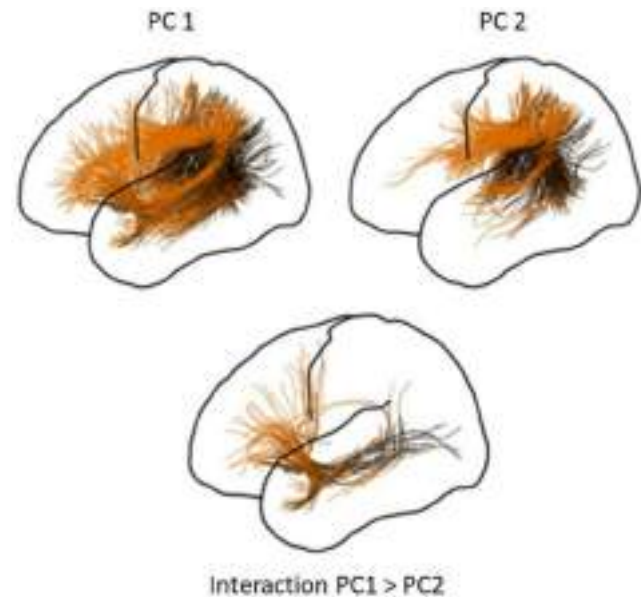


FIGURE 2

EPO-0392 | From eye clinic to neurology: Diagnostic clues in Heidenhain-variant Creutzfeldt–Jakob disease

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Background and aims: Creutzfeldt–Jakob disease (CJD) is a rare and invariably fatal prion-related neurodegenerative disorder, typically presenting with rapidly progressive dementia. The Heidenhain variant (HvCJD), however, manifests predominantly with isolated visual symptoms at onset, frequently leading to misdiagnosis and delayed neurological assessment. This report describes two cases of HvCJD initially attributed to ophthalmological pathology, highlighting key diagnostic challenges.

Methods: We retrospectively analyzed two patients who presented with isolated visual disturbances and underwent cataract surgery before a neurological diagnosis was established. Clinical course, neuroimaging findings, cerebrospinal fluid (CSF) biomarkers (14-3-3 protein and RT-QUIC), and electroencephalographic (EEG) data were reviewed. Findings were contextualized through a focused review of the literature on HvCJD to identify common diagnostic pitfalls.

Results: An 80-year-old man presented with persistent visual impairment and hemianopia refractory to cataract surgery; brain MRI revealed posterior cortical hyperintensities consistent with prion disease. A second patient, a 60-year-old man, developed progressive visual symptoms followed by gait instability after cataract surgery, with MRI and EEG findings suggestive of CJD. CSF analysis was positive for both 14-3-3 protein and RT-QUIC in both cases. Published series confirm that isolated visual symptoms frequently result in misdiagnosis, delayed referral to neurology, and exposure to unnecessary invasive procedures.

Conclusion: HvCJD should be considered in patients with unexplained or rapidly progressive visual disturbances, particularly when symptoms are inconsistent with primary ocular disease.

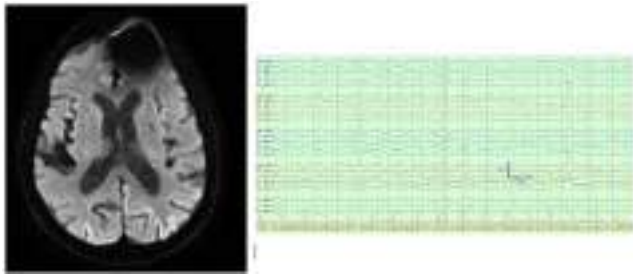


FIGURE 1 MRI and EEG of the first patient explained.

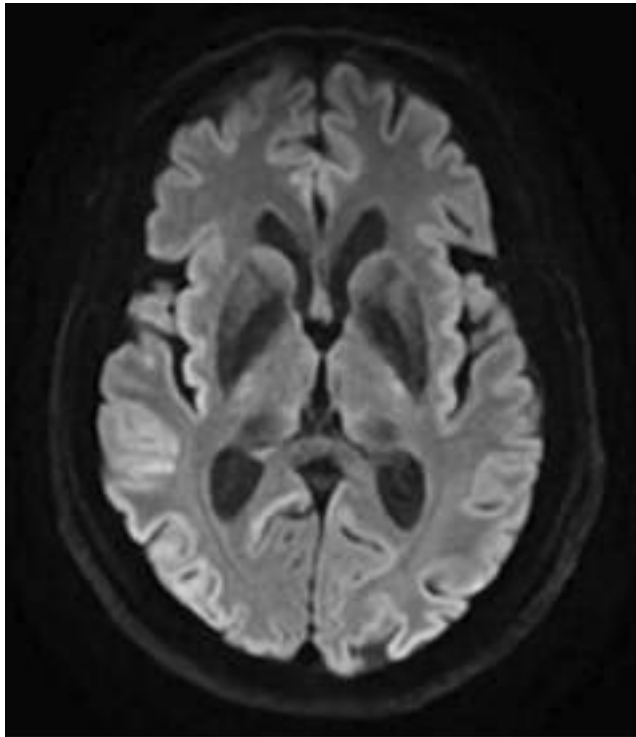


FIGURE 2 MRI with restriction on diffusion (second patient).

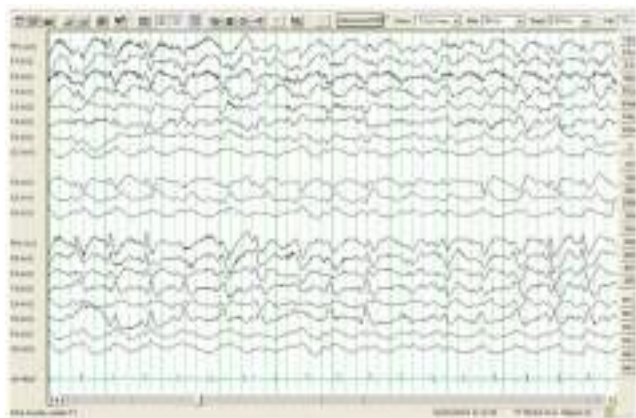


FIGURE 3 EEG showing triphasic waves (second patient).

Visual manifestations reflect cortical involvement rather than ophthalmological pathology. Increased awareness among ophthalmologists and neurologists, together with early neuroimaging and CSF biomarker assessment, is essential to reduce diagnostic delay and avoid iatrogenic risks.

Disclosure: Nothing to disclose.

EPO-0393 | Impact of a structured neurocognitive and psychosocial intervention on quality of life and psychosocial morbidity in drug-resistant epilepsy: An RCT

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Background and aims: Patients with drug-resistant epilepsy (DRE) experience substantial psychosocial morbidity, including depression, anxiety, stigma, and functional disability, which contribute significantly to reduced quality of life (QOL). Despite optimal medical management, these challenges remain under-addressed in routine clinical practice. We assessed the effectiveness of a structured neuro-cognitive and psychosocial intervention module in improving psychosocial outcomes and QOL in individuals with drug-resistant epilepsy.

Methods: A randomized controlled trial was conducted on 60 adults with DRE, allocated to an intervention group ($n=30$) and a control group ($n=30$). The intervention consisted of psycho-education, cognitive-behavioral strategies, cognitive retraining, and family counseling, delivered over 8 weeks, in addition to standard medical treatment. Psychosocial outcomes were evaluated using QOLIE-31, BDI, HARS, Stigma Scale for Epilepsy (SSE), WHODAS, and Dysfunctional Analysis Questionnaire (DAQ). Per-protocol and intention-to-treat analyses were performed.

Results: Patients receiving the intervention showed a clinically meaningful improvement in quality of life, with QOLIE-31 scores improving from 52.4 ± 8.5 at baseline to 67.8 ± 7.2 post-intervention ($p < 0.001$). Significant reductions were observed in anxiety (HARS: 21.3 ± 5.8 to 12.1 ± 4.7 , $p < 0.001$), depressive symptoms, perceived stigma ($p=0.03$), psychosocial dysfunction, and disability scores. The control group showed no statistically significant changes over the same period. Seizure frequency remained stable, indicating that improvements were independent of seizure control.

Conclusion: The integrated neuro-cognitive and psychosocial intervention is effective in reducing psychosocial morbidity and improving quality of life in patients with drug-resistant epilepsy. Incorporating such structured interventions into routine epilepsy care may substantially enhance holistic patient outcomes.

Disclosure: Nothing to disclose.

EPO-0394 | Wernicke encephalopathy with normal imaging and labs in a severely malnourished elderly patient: A case report

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Background and aims: Wernicke's encephalopathy is a neurological emergency secondary to thiamine deficiency, with a frequently atypical clinical presentation. The absence of the classic triad and normal complementary investigations may delay

diagnosis. We report a case of probable Wernicke's encephalopathy revealed by a progressive neuropsychiatric syndrome.

Methods: We report the case of a 65-year-old female patient with a history of recent untreated hyperthyroidism, laryngeal carcinoma treated with radiotherapy, and hypertension. She was admitted for behavioral disturbances including psychomotor agitation, opposition to care, aggressiveness, and irritability, associated with memory impairment and visual hallucinations, with progression over 20 days. Investigations included blood tests, thyroid function assessment, brain imaging, electroencephalography, and lumbar puncture with cerebrospinal fluid analysis.

Results: On admission, the patient was fully conscious with a Glasgow Coma Scale score of 15/15, but severely malnourished with a low body mass index, dehydrated, and uncooperative, with no focal neurological deficits. Biological investigations were unremarkable. Brain computed tomography showed mild ventricular enlargement, while brain magnetic resonance imaging and electroencephalography were normal. Cerebrospinal fluid analysis revealed no abnormalities. Given the strong clinical suspicion of thiamine deficiency, vitamin B1 therapy was initiated, along with nutritional and hydration support. This resulted in a marked and rapid improvement in behavioral symptoms, cognitive functions, and general condition. Visual hallucinations partially persisted. The patient was discharged on chlorpromazine 25 mg nightly, with follow-up scheduled one month later.

Conclusion: This case highlights the importance of considering Wernicke's encephalopathy in malnourished elderly patients with acute or progressive neuropsychiatric syndromes. Early thiamine administration should not be delayed when suspected.

Disclosure: Nothing to disclose.

EPO-0395 | When the past catches up: Iatrogenic Creutzfeldt–Jakob Disease with a 39-year incubation period

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Background and aims: Creutzfeldt–Jakob disease (CJD) is a rare, fatal prion disease, most commonly occurring sporadically but occasionally transmitted through medical procedures, resulting in iatrogenic CJD (iCJD).

Methods: A 39-year-old man presented to Queen's Medical Centre, Nottingham, UK with a four-week history of rapidly progressive cognitive and behavioural decline. His family reported impaired executive function, including leaving dirty cookware in cupboards, difficulty following instructions at work, misplacing personal items, and gait disturbance with leftward leaning. At six weeks of age, he developed meningitis complicated by seizures and bilateral subdural empyema. On 7 January 1986, he underwent craniotomy with dural repair using a cadaveric dura mater graft (Lyodura). He had lifelong learning difficulties, dyslexia, and communication and social difficulties.

Results: During admission, he deteriorated rapidly, becoming bedbound, non-verbal, and doubly incontinent. He developed

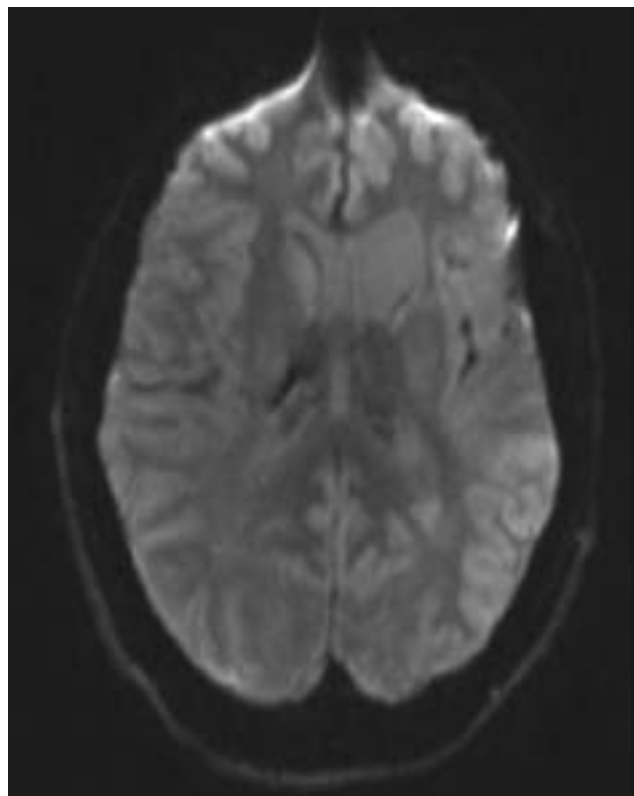


FIGURE 1 MRI brain demonstrated diffusion restriction in the bilateral basal ganglia with multifocal cortical ribboning, more prominent on the left.

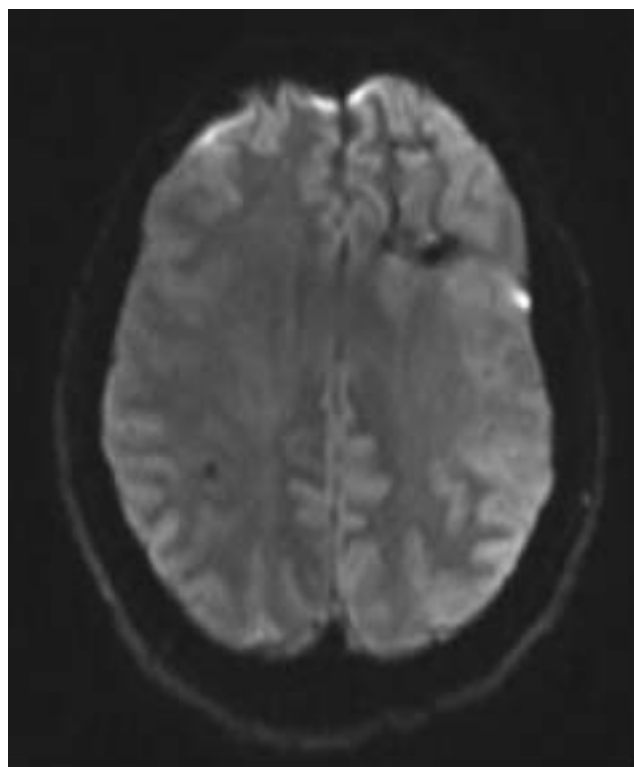


FIGURE 2 MRI brain demonstrated diffusion restriction in the bilateral basal ganglia with multifocal cortical ribboning, more prominent on the left.

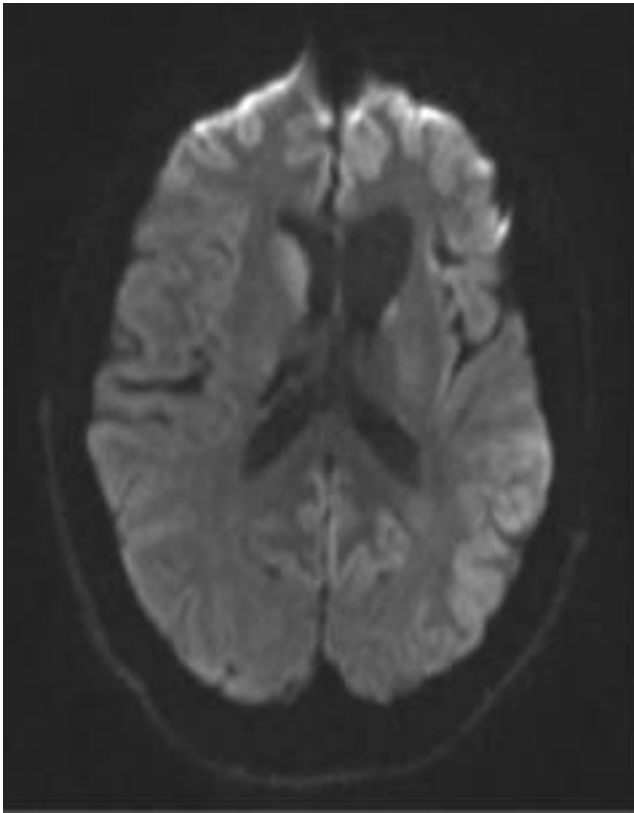


FIGURE 3 MRI brain demonstrated diffusion restriction in the bilateral basal ganglia with multifocal cortical ribboning, more prominent on the left.

stimulus-induced myoclonus, initially treated with levetiracetam but switched to clonazepam due to rash. MRI brain demonstrated diffusion restriction in the bilateral basal ganglia with multifocal cortical ribboning, more prominent on the left. EEG showed non-specific encephalopathy. Cerebrospinal fluid was acellular with normal protein, and RT-QuIC was positive. He was referred to palliative care and died in a hospice within two weeks of discharge.

Conclusion: The case highlights the diagnostic value of a detailed lifetime surgical history, particularly childhood neurosurgical interventions that may otherwise be overlooked. Incubation periods in iCJD can exceed 30–40 years, making careful lifetime surgical history essential. Continued vigilance, prompt recognition, and systematic reporting are essential to support surveillance efforts and improve understanding of iatrogenic prion disease in neurological practice.

Disclosure: Nothing to disclose.

EPO-0396 | Transient epileptic amnesia as an underdiagnosed form of late-onset temporal lobe epilepsy

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Background and aims: Late-onset epilepsy is defined as epilepsy with first seizure onset from the age of 50 years onward. Within this spectrum, temporal lobe epilepsy in older adults (TLE-OA) is increasingly recognized. Among its clinical phenotypes, transient epileptic amnesia (TEA) represents a distinctive but frequently underdiagnosed form.

Methods: We conducted a retrospective analysis of patients followed for TLE-OA at the Neurology Department of Razi Hospital. Clinical features, interictal electroencephalography (EEG) and brain magnetic resonance imaging (MRI) were reviewed. Diagnostic criteria included the presence of recurrent episodes of transient amnesia with preservation of other cognitive functions, associated with ictal features, EEG abnormalities, or a clear response to antiseizure medication.

Results: Ten out of fourteen patients fulfilled diagnostic criteria for TEA. The mean age was 65.2 years (range 51–74), with equal sex distribution. The median time to diagnosis was 16.5 months. Two patients reported persistent cognitive/behavioral symptoms. EEG revealed epileptiform abnormalities involving the frontal and/or temporal regions in 50% of cases. MRI revealed focal abnormalities in one case. Anti-seizure therapy, most often with a single agent, resulted in improvement in all eight cases with available follow-up.

Conclusion: TEA represents a frequent yet underrecognized presentation of TLE-OA rather than a separate diagnostic entity. Because routine EEG and MRI findings may be normal, TEA is particularly prone to diagnostic delay and misclassification. Improved awareness of TEA as a form of epilepsy in older adults is essential, as early clinical recognition enables appropriate anti-seizure treatment and may limit long-term cognitive impact.

Disclosure: Nothing to disclose.

EPO-0397 | Long-term outcomes and predictors of response to vagus nerve stimulation in drug-resistant epilepsy

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Background and aims: Vagus nerve stimulation (VNS) is an established neuromodulatory treatment for drug-resistant epilepsy. Long-term studies suggest that its efficacy may increase over time. This study evaluate long-term clinical outcomes and potential predictors of treatment response in patients who underwent VNS implantation at a tertiary epilepsy center.

Methods: We retrospectively reviewed patients who received VNS therapy between 2005 and 2025. Potential predictors of VNS response included epilepsy classification (focal vs. generalized), underlying etiology (structural vs. non-structural), age at seizure onset, duration of epilepsy prior to VNS therapy, previous epilepsy surgery, maximum stimulation voltage, and duration of VNS therapy.

Results: A total of 122 patients were included. At the last follow-up, 30% of patients achieved a $\geq 50\%$ reduction in seizure frequency and were classified as responders. Patients with structural epilepsy ($n=64$) demonstrated a significantly higher responder rate compared to those with non-structural etiologies ($p=0.006$). The duration of VNS therapy was significantly longer in responders compared to non-responders (8.02 ± 4.73 years vs. 4.89 ± 4.02 years, $p=0.001$), supporting a time-dependent treatment effect. No significant associations were observed between treatment response and age at seizure onset ($p=0.373$), age at VNS implantation ($p=0.195$), duration of epilepsy prior to VNS therapy ($p=0.592$), or maximum stimulation voltage ($p=0.202$). Furthermore, maximum stimulation voltage was not associated with the occurrence of adverse effects.

Conclusion: VNS therapy demonstrates greater efficacy in patients with structural epilepsy and in those with longer treatment duration, supporting a cumulative and time-dependent therapeutic effect.

Disclosure: Nothing to disclose.

EPO-0398 | Clinical and demographic correlates of multifocal epileptiform activity in hospitalized adults

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Background and aims: Multifocal epileptiform activity, defined as the presence of at least three independent epileptiform foci involving both cerebral hemispheres on EEG, is poorly characterized clinically. Clinical observations suggest an association with advanced age, comorbidities, and systemic illness; however, these relationships have not been studied. We aimed to evaluate the clinical and demographic correlates of multifocal epileptiform activity in hospitalized adults.

Methods: Methods: We conducted a retrospective observational study including 80 hospitalized patients selected in reverse

chronological order from our institutional EEG database (accessed 30 November 2024): 40 with multifocal epileptiform activity and 40 with non-multifocal activity. Demographic data, medical history, EEG indication, metabolic disturbances defined by the ILAE as causes of acute symptomatic seizures, and neuroimaging findings were collected. Statistical significance was set at $p < 0.05$.

Results: Patients with multifocal epileptiform activity were older than those with non-multifocal activity ($p=0.003$). EEG indications differed between groups ($p=0.002$), with altered consciousness more frequent in the multifocal group and convulsive seizures in the non-multifocal group. Cognitive decline (OR 2.9; 95% CI 1.1–7.2; $p=0.024$) and sepsis (OR 9.8; 95% CI 1.2–82.1; $p=0.029$) were more frequent in the multifocal group. In a multivariable logistic regression, explaining approximately 25% of the variability ($p < 0.001$), only age remained independently associated with multifocal activity (OR 1.04; 95% CI 1.01–1.08; $p=0.020$).

Conclusion: Multifocal epileptiform activity in hospitalized adults occurs predominantly in older patients and is associated with altered consciousness and sepsis, suggesting that it reflects global disease severity rather than a specific epileptic etiology.

Disclosure: Nothing to disclose.

EPO-0399 | Efficacy and adverse event profile of pharmacological and non-pharmacological therapies in Dravet Syndrome: A systematic review

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Background and aims: Dravet Syndrome (DS) is a severe epileptic encephalopathy with onset in early infancy that requires long-term pharmacological management [1]. Increasing evidence has highlighted significant treatment-related adverse effects, including cognitive, behavioral, and systemic complications [2]. This study aims to analyze and summarize treatment-associated complications in patients with Dravet Syndrome.

Methods: We conducted a systematic literature review following PRISMA guidelines. Of 74 studies, 7 were included. PubMed was searched using the keywords Dravet syndrome, treatment regimens, adverse effects, and treatment complications. Treatment regimens, outcomes, and adverse effects were evaluated.

Results: We analyzed 275 patients with Dravet syndrome. The mean age was 14.3 years. The most frequent treatments were fenfluramine, valproate, clobazam, and stiripentol, see Figure 1 Adjuvant therapies included cannabidiol/THC (19 patients) and a ketogenic diet (60 patients). A $\geq 50\%$ reduction in seizures was achieved in 58–83% of patients, with seizure freedom in 7.6–25%. Adverse effects were common (up to 59.1% somnolence, 31.5% total events) but generally manageable, leading to discontinuation in $< 21\%$ of cases.

Conclusion: Available evidence suggests that fenfluramine and ketogenic diet can reduce crisis in Dravet, with a minority achieving freedom from disease, while cannabinoids show variable answers. However, design heterogeneity, co-interventions and incomplete security report limit comparisons and robust

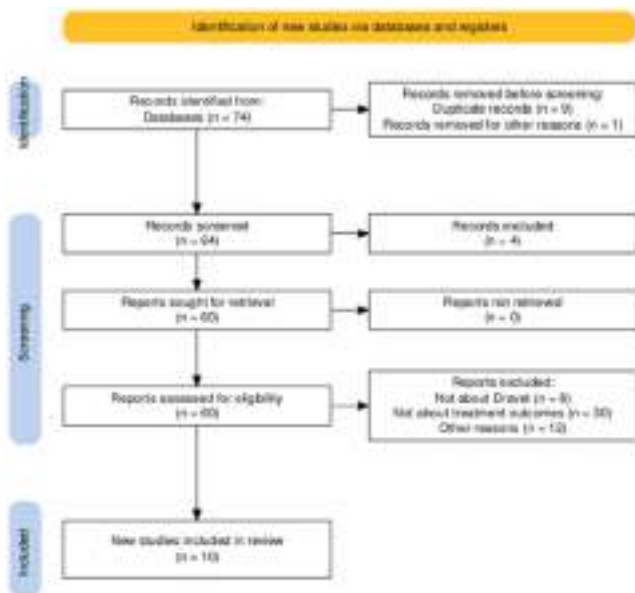


FIGURE 1 Prisma-P flowchart of the process of inclusion of studies in the systematic review.

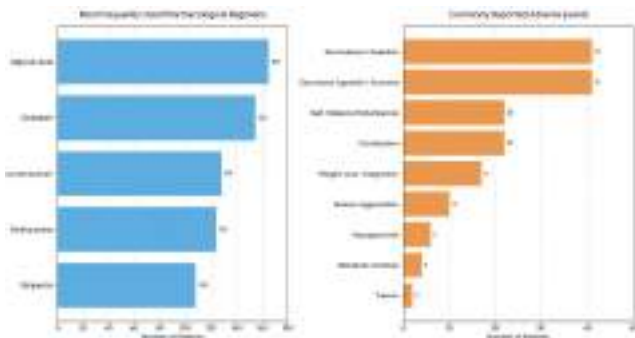


FIGURE 2 Frequency of Pharmacological Regimens and Their Adverse Events of Dravet Syndrome.

meta-analysis. Studies with standardized structures are required and systematic follow-up of adverse events to better define risk-benefits.

Disclosure: Nothing to disclose.

EPO-0400 | Abstract withdrawn

EPO-0401 | Structures associated with ictal alpha and theta activity in temporal lobe epilepsy – A volumetric study

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TABLE 1

Male (%)	25 (41.7)	
Age, M (interval)	46 (22-71)	
Febrile seizures (%)	34 (56.7)	
Age at epilepsy onset, M (interval)	11.5 (1.0-44.0)	
Seizure days per month, M (interval)	5.0 (0.15-90.0)	
Left MTLE (%)	38 (63.3)	
Epilepsy duration at time of surgery, M (interval)	23 (2-55)	
Number of ASM, M (interval)	5 (2-8)	
Histopathology, N (%)	HS ILAE type I	39 (65.0)
	HS ILAE type II	12 (20.0)
	HS ILAE type III	1 (1.7)
	HS	6 (10.0)
	Inadequate sample	2 (3.3)

M – median, MTLE – mesial temporal lobe epilepsy, ASM – antiseizure medication, N – number, HS – hippocampal sclerosis, ILAE – International League Against Epilepsy

Background and aims: Ictal rhythms ≥ 5 Hz are well described as important predictors of favourable outcome after mesial temporal lobe epilepsy (MTLE) surgery. However, the precise anatomical structures involved in generation and propagation of these activities have not been fully understood. Aim of our study was to analyze structural brain alterations of anatomical regions typically involved in seizures in MTLE to determine associations with these rhythms.

Methods: This retrospective study included MRIs of 60 patients who underwent successful MTLE surgery and had at least one seizure documented during video-EEG monitoring (Table 1). Patients were classified based on observed rhythmic ictal activity ≥ 5 Hz in alpha and fast theta frequency. Volumes of 25 brain structures were analyzed. Univariate and multivariate logistic regression models were used to determine structures associated with temporal alpha (TA) and fast temporal theta (FTT).

Results: We observed TA in 20 seizures in seven patients and FTT in 219 seizures in 39 patients. Demographic and clinical variables (age, sex, and age at epilepsy onset) did not differ significantly between patients groups based on presence of TA and FTT. Univariate logistic regression analysis identified significant association between TT and three corpus callosum regions: mid-posterior, central and mid-anterior, while TA showed an association with ipsilateral thalamus and isthmus gyri cinguli. Multivariate analysis revealed that only the mid-anterior corpus callosum was an independent predictor of TT, while ipsilateral thalamus and isthmus gyri cinguli were both independent predictors of TA.

Conclusion: Mid-anterior corpus callosum, thalamus, and isthmus gyri cinguli are significantly associated with TT and TA in MTLE.

Disclosure: Nothing to disclose.

EPO-0402 | Cenobamate for refractory epilepsy secondary to primary CNS tumours: Real-world outcomes across WHO classifications

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Background and aims: Epilepsy is the commonest chronic and life-limiting neurological co-morbidity associated with primary central nervous system (CNS) tumours. Cenobamate

(CNB), a third generation antiseizure medication (ASM), has demonstrated high efficacy in focal epilepsy. Evidence for its efficacy in brain tumour-related epilepsy (BTRE) remains limited. We evaluated the real-world tolerability and effectiveness of CNB in BTRE.

Methods: Patients with BTRE attending the Queen Elizabeth hospital Birmingham who were prescribed CNB between 2022 and 2025 were observed. Tumours were classified according to the 2021 WHO CNS tumour classification. Data including tumour histology and grade, baseline seizure frequency and adverse events were recorded. The primary outcome was significant ($\geq 50\%$) seizure reduction. Additional outcomes included $\geq 90\%$ seizure reduction, change in ASM burden and tolerability.

Results: 25 Patients representing a broad spectrum of WHO tumour entities (grades I–IV) were included. Median duration of follow up was 19 months. Mean number of concomitant ASMs decreased from 2.75 at baseline to 1.79 following CNB initiation. 11/25 patients (44%) achieved a $\geq 50\%$ reduction in seizure frequency. 6 patients (24%) achieved $\geq 90\%$ seizure reduction. Response was observed across multiple tumour subtypes and WHO grades. CNB was generally well tolerated. Treatment discontinuation due to adverse effects (commonly somnolence and sedation) occurred in 3/25 patients (12%).

Conclusion: This is the largest real-world cohort of patients with refractory BTRE treated with CNB. CNB was associated with significant seizure reduction in over 40%. These findings support Cenobamate as a valuable therapeutic option in BTRE, irrespective of tumour type.

Disclosure: Dr Shanika Samarasekera has received speaker honoraria from Angelini Pharma. QEHB has been supported by Angelini Pharma with a grant for an epilepsy pharmacist.

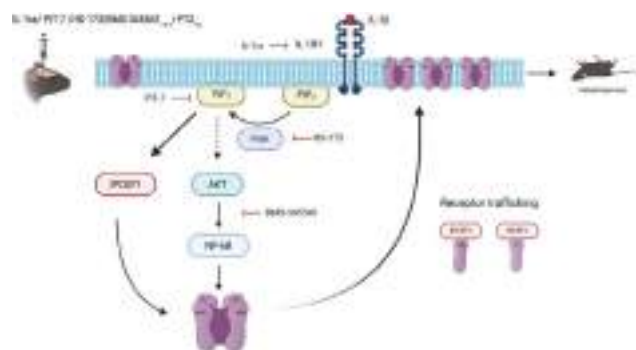
EPO-0403 | IPCEF1-mediated NMDA receptor trafficking: A novel IL-1 β -driven mechanism linking inflammation to epileptogenesis and NREM sleep desynchrony

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Background and aims: Epilepsy often co-occurs with sleep disturbances, but molecular links between inflammation, excitatory plasticity, and these comorbidities are unclear. We hypothesised that IL-1 β signalling activates the PIP3–AKT–NF- κ B cascade to upregulate IPCEF1, promoting NMDA receptor (NMDAR) trafficking and hippocampal hyperexcitability, thereby driving seizures and non-rapid eye movement (NREM) sleep desynchrony.

Methods: Pentylentetrazol (PTZ) kindling (35 mg/kg i.p., every 48 h for 14 days) was induced in wild-type and IL-1R1 knockout mice. Interventions included IL-1 receptor antagonist (IL-1ra) and inhibitors of PIP3–AKT–NF- κ B signalling (PIT-7, HS-173, BMS-345541). Continuous EEG/EMG monitored seizures and sleep architecture. Hippocampal inflammation (IL-1 β , microglia, astrocytes) and NMDAR subunit expression/trafficking were assessed by Western blotting and immunofluorescence. IPCEF1 was specifically silenced to isolate its role.



IL-1 β -driven IPCEF1-mediated NMDAR trafficking links inflammation to epileptogenesis and cortical desynchrony in NREM sleep.

Results: PTZ kindling elevated hippocampal IL-1 β , glial activation, synaptic NMDAR enrichment, seizure frequency, and disrupted NREM slow-wave synchrony. Blocking IL-1R1 or downstream cascade prevented inflammation, reduced NMDAR trafficking, suppressed seizures, and normalised sleep. Notably, IPCEF1 silencing selectively reduced seizures and restored NREM synchrony without affecting total sleep–wake distribution.

Conclusion: IL-1 β -driven IPCEF1-mediated NMDAR trafficking links inflammation to epileptogenesis and cortical desynchrony in NREM sleep. The IL-1-IPCEF1-NMDAR axis represents a precise therapeutic target for controlling seizures and sleep comorbidities in epilepsy.

Disclosure: Nothing to disclose.

EPO-0404 | From seizures to vascular risk: Carotid ultrasound markers in epilepsy

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Background and aims: Cardiovascular risk may be increased in epilepsy, potentially influenced by chronic anti-seizure medication (ASM) exposure. Carotid intima-media thickness (IMT) is a validated surrogate marker of subclinical atherosclerosis. We investigated whether carotid IMT differs according to drug-resistant epilepsy (DRE) and exposure to enzyme-inducing ASMs.

Methods: We consecutively enrolled patients attending a tertiary epilepsy center between January 2025 and January 2026. Seventy-one subjects underwent carotid ultrasound assessment. Right, left, mean, maximal IMT and side-to-side IMT difference were recorded, along with carotid stenosis. Participants were stratified by DRE status and by enzyme-inducing ASM exposure.

Results: No demographic or lifestyle differences were observed between DRE and non-DRE groups, nor between participants with vs without exposure to enzyme-inducing ASMs. Carotid IMT measures were not significantly different between DRE vs non-DRE (right IMT $p=0.673$; left IMT $p=0.173$; mean IMT $p=0.223$; maximal IMT $p=0.143$). A non-significant trend toward higher left and maximal IMT was observed in DRE. No significant differences were found according to enzyme-inducing ASM exposure (right IMT $p=0.813$; left IMT $p=0.503$; mean

IMT $p=0.673$; maximal IMT $p=0.513$). Carotid stenosis prevalence was comparable between exposure groups ($p=0.972$).

Conclusion: In this cohort, DRE and enzyme-inducing ASM exposure were not associated with increased carotid IMT. Larger samples may clarify whether the observed DRE trend reflects a true biological effect.

Disclosure: Nothing to disclose.

EPO-0405 | Plasma NfL and phosphorylated tau as emerging biomarkers of neuronal injury in pediatric drug-resistant epilepsy: A preliminary study

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Background and aims: Pediatric drug-resistant epilepsies represent a significant clinical challenge, profoundly affecting neurocognitive development, quality of life, and long-term prognosis. The identification of novel neurodegenerative biomarkers could improve patient stratification, clinical management, and support the development of therapies.

Methods: A prospective study was conducted analyzing blood samples from patients with drug-resistant epilepsy followed at the Pediatric Neuropsychiatry Unit of Messina. Biomarker levels were measured. Descriptive analyses were performed, while Spearman's rank correlation analyses were conducted to evaluate the relationships between biomarker levels and clinical variables.

Results: The study included 14 pediatric patients with drug-resistant epilepsy (mean age 8.5 years), and 8 (57.1%) were male. Plasma biomarker showed a mean neurofilament light chain (NfL) level of 31.997 pg/mL, pTau181 mean value of 1.172 pg/mL, pTau217 showed a mean of 0.179 pg/mL. Compared with age matched reference values, 10 patients (71.4%) exhibited elevated NfL levels, and 7 (50%) showed increased pTau217. The most frequent diagnosis was epileptic encephalopathy (42.9%), including Lennox-Gastaut (21.4%) and Dravet syndrome (7.1%). EEG abnormalities were mainly diffuse fronto-centro-temporal (28.6%), parieto-temporal/post-central (28.6%), or non-localized (21.4%). Overall, patients with early onset complex encephalopathies showed the most severe biochemical profiles, whereas those with Lennox-Gastaut syndrome exhibited consistently lower NfL levels.

Conclusion: Correlation analyses showed NfL positively associated with pTau217 ($p=0.55$, $p=0.041$) and pTau181 with pTau217 ($p=0.70$, $p=0.005$). These results suggest NfL and pTau217 are sensitive, complementary biomarkers of neuronal injury in children with drug-resistant epilepsy, with potential for disease stratification and clinical decision.

Disclosure: Nothing to disclose.

EPO-0406 | Evaluating gender related outcomes with adjunctive cenobamate in focal-onset seizures

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Background and aims: Sex differences are observed in neurological disorders and may be apparent in prevalence, clinical presentation, and treatment outcomes. In epilepsy, sex related factors can influence the selection and dosing of antiseizure medications (ASMs), yet little is known about variations in treatment response. This analysis explored potential differences in the efficacy and safety of adjunctive cenobamate (CNB).

Methods: This post-hoc analysis of the C017 placebo-controlled double-blind trial evaluated efficacy and safety of CNB in adults with uncontrolled focal-onset seizures despite 1–3 prior ASMs at stable doses. Outcomes were assessed by sex across CNB dose groups (100, 200, and 400 mg) and placebo.

Results: Females represented 49.5% of the randomized population. They had a median age of 40 years, 25 years since epilepsy diagnosis, and a baseline seizure frequency of 10 seizures/28 days. Males had a median age of 37 years, 21.4 years since diagnosis, and a baseline seizure frequency of 8.65 seizures/28 days. Seizure freedom rates during maintenance-phase in males were 3.6%, 10.2%, and 25.0% for CNB 100, 200, and 400 mg, respectively, versus 1.8% for placebo. In females, rates were 4.3%, 12.2%, and 17.6% for the same CNB doses, with 0% for placebo. The incidence and nature of adverse events were similar between sexes across CNB groups.

Conclusion: Cenobamate demonstrated consistent efficacy and a similar tolerability profile in both females and males. Although small numerical differences were observed, no clear meaningful sex related differences emerged. These findings suggest that treatment-benefit from CNB is not sex dependent.

Disclosure: SL: Speaker/consultant: Angelini Pharma, Eisai, GW Pharmaceuticals, Medscape, NewBridge Pharmaceuticals, and UCB Pharma Advisor: Angelini Pharma, Arvelle Therapeutics, BIAL, EISAI, GW Pharmaceuticals, Rapport Therapeutics, and UCB Pharma VV: Consultant/advisor: Angelini Pharma, BIAL, Biocodex, Eisai, Esteve, GlaxoSmithKline, Jazz, Novartis, Sandoz, Takeda, UCB Pharma, Xenon; Speaker: Angelini Pharma, BIAL, Cevomed, Eisai, Esteve, Jazz, Newbridge, Paladin, UCB Pharma; Research support: Angelini Pharma, BIAL, Eisai, Jazz, UCB Pharma. BS: Consultant/advisor: Angelini, B. Braun Melsungen, Desitin, Eisai, GW Pharmaceuticals, UCB Pharma, Zogenix; Research support: Eisai, GW Pharmaceuticals, SK Life Science, UCB Pharma. JPL, PPD, EAB: Employees, Angelini Pharma. KT: Consultant: Angelini Pharma.



FIGURE 1 Seizure Freedom Rates during the Maintenance Phase.

EPO-0407 | Time to pre-randomization monthly seizure frequency for patients with uncontrolled focal onset seizures treated with cenobamate

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Background and aims: The updated EMA guideline on clinical investigation in epileptic disorders (September 2025) recognizes time to event endpoints as a potential approach for primary analyses, with the aim of reducing exposure to placebo. This post hoc analysis evaluated the time required to reach pre randomization monthly seizure frequency as a measure of cenobamate (CNB) efficacy.

Methods: Data were derived from the C017 randomized, placebo-controlled trial in adults with uncontrolled focal onset seizures receiving 1–3 concomitant ASMs at stable doses. Time to achieving the individual 28-day baseline seizure rate was compared between placebo and CNB treatment groups (100, 200, and 400 mg/day). Analyses included the proportion of patients reaching baseline seizure frequency and the median time to event during the maintenance phase.

Results: The proportion of patients reaching baseline seizure rate was 89.1% with placebo versus 76.4% ($p=0.0161$), 66.9% ($p<0.001$), and 51.7% ($p<0.001$) with CNB 100, 200, and 400 mg, respectively. Median (95% CI) time to baseline seizure frequency was 5.34 (4.71–6.29) weeks for placebo, compared with 6.00 (5.57–8.14) weeks for CNB 100 mg, 8.71 (7.43–11.57) weeks for CNB 200 mg, and 10.86 (8.43–NA) weeks for CNB 400 mg.

Conclusion: Cenobamate significantly prolonged the time to pre randomization monthly seizure rate and reduced the proportion of patients reaching it compared with placebo in a dose-dependent manner. Consistent with previously reported outcomes, higher CNB doses resulted in greater effects. These findings support time to baseline seizure rate as a promising endpoint for future epilepsy trials.

Disclosure: SL: Speaker/consultant: Angelini Pharma, Eisai, GW Pharmaceuticals, Medscape, NewBridge Pharmaceuticals, and UCB Pharma Advisor: Angelini Pharma, Arvelle

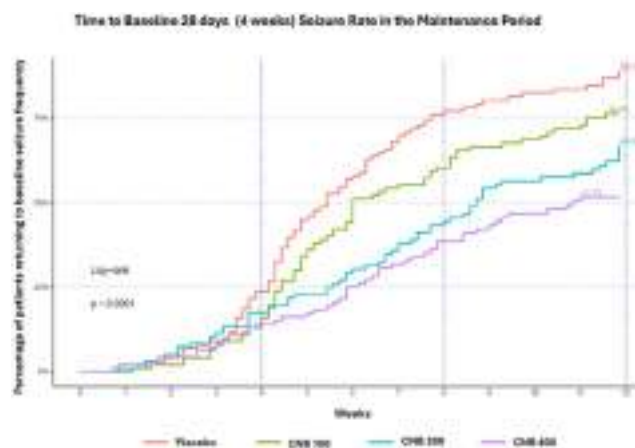


FIGURE 1 Time to Baseline 28 days (4 weeks) Seizure Rate in the Maintenance Period.

Therapeutics, BIAL, EISAI, GW Pharmaceuticals, Rapport Therapeutics, and UCB Pharm VV: Consultant/advisor: Angelini Pharma, BIAL, Biocodex, Eisai, Esteve, GlaxoSmithKline, Jazz, Novartis, Sandoz, Takeda, UCB Pharma, Xenon; Speaker: Angelini Pharma, BIAL, Cevomed, Eisai, Esteve, Jazz, Newbridge, Paladin, UCB Pharma; Research support: Angelini Pharma, BIAL, Eisai, Jazz, UCB Pharma. BS: Consultant/advisor: Angelini, B. Braun Melsungen, Desitin, Eisai, GW Pharmaceuticals, UCB Pharma, Zogenix; Research support: Eisai, GW Pharmaceuticals, SK Life Science, UCB Pharma. JPL, PPD, EAB: Employees, Angelini Pharma. KT: Consultant: Angelini Pharma.

Headache 3

EPO-0408 | Migraine without vascular risk factors versus controls: White matter hyperintensities align with systemic inflammation and atherogenic lipid ratios

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Background and aims: To compare white matter hyperintensities (WMH) between a control group and migraine patients classified according to the Fazekas score, and to evaluate their relationships with disease burden, inflammatory and lipid indices.

Methods: Retrospective cross-sectional analysis of 320 adults: controls ($n=90$), migraine with Fazekas-0 ($n=112$), and migraine with Fazekas 1–3 ($n=118$). Participants had no vascular comorbidities. WMH were graded on brain magnetic resonance imaging (MRI). We analyzed C-reactive protein (CRP), systemic immune-inflammation index (SII), total-cholesterol to high-density lipoprotein ratio (TC/HDL) and atherogenic index of plasma (AIP). Group comparisons used Welch or Kruskal-Wallis tests with Holm correction. In migraine, Spearman correlations and logistic regression assessed associations with WMH.

Results: CRP increased across groups (median 1.22 in controls, 1.12 in Fazekas-0, 2.19 in Fazekas 1–3; $p=1.1e-8$). TC/HDL also

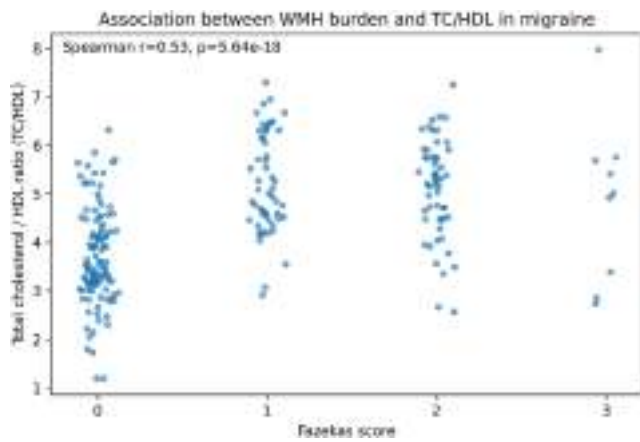


FIGURE 1 Relationship between Fazekas score and TC/HDL in migraine group.

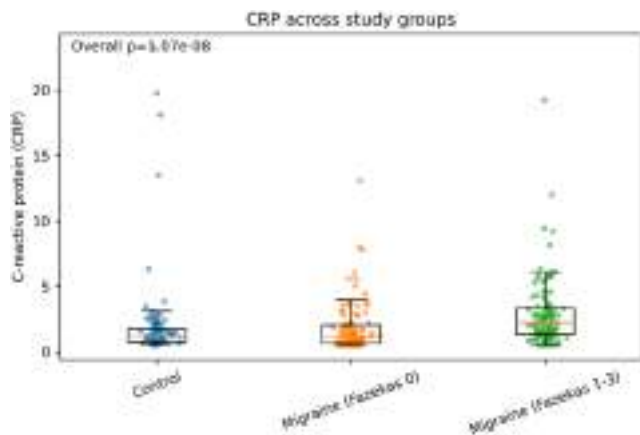


FIGURE 2 CRP – Comparison of 3 groups; found to be statistically significant.

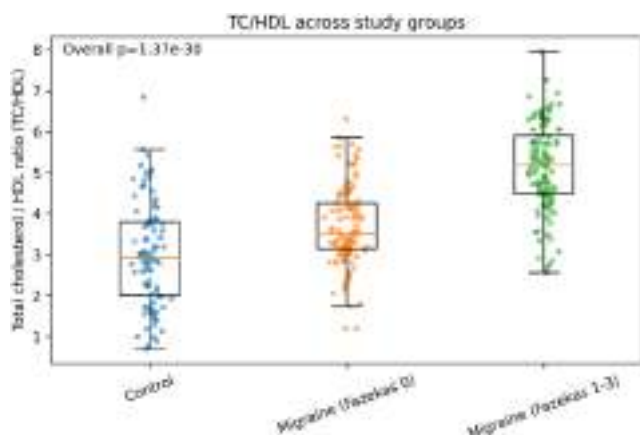


FIGURE 3 TC/HDL – Comparison of 3 groups; found to be statistically significant.

increased (mean 3.00, 3.70, 5.15; $p=1.4e-30$), and AIP was higher in Fazekas 1–3 (mean 0.14, 0.15, 0.17; $p=0.0011$). SII did not differ ($p=0.256$). In migraine, Fazekas score correlated with TC/HDL (Spearman $r=0.53$, $p=5.6e-18$) and CRP ($r=0.30$, $p=4.3e-6$). After adjustment for age, migraine duration, and monthly attack frequency, TC/HDL (odds ratio (OR) 3.84 per-unit, $p=5.1e-8$) and CRP (OR 1.47 per-unit, $p=0.0019$) remained associated with

WMH. In the Fazekas 1–3 migraine group, disease duration and monthly attack frequency were significantly higher than in the Fazekas-0 group ($p<0.001$). Among aura patients, right-to-left shunt rates were higher in Fazekas 0 than Fazekas 1–3, but not statistically significant (38.7% vs 33.3%).

Conclusion: In migraine without vascular comorbidities, higher WMH burden is linked to higher CRP and more atherogenic lipid profile (TC/HDL and AIP), while SII was not discriminative. Longitudinal studies are needed.

Disclosure: Nothing to disclose.

EPO-0409 | Most bothersome non-pain symptoms in migraine and their response to CGRP monoclonal antibodies: A service evaluation

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Background and aims: Monoclonal antibodies targeting calcitonin gene-related peptide (CGRP-mAbs) have been in wide use as migraine preventives for five years. They have potential efficacy in improving the most bothersome non-pain symptoms (MBS).

Methods: Patients currently on mAbs at King's College Hospital, London completed a questionnaire, and clinical and efficacy data were collected from patient records as part of a service evaluation. Associations between categorical variables were assessed using Pearson Chi-Square tests.

Results: A total of 223 patients responded (median age 48 years; 82% female): chronic migraine (62%), episodic migraine (29%), and new daily persistent headache with migraine phenotype (9%). Fatigue was the most frequent MBS ($n=44$, 20%), followed by brain fog ($n=38$, 17%), neck stiffness ($n=31$, 14%), nausea ($n=26$, 12%) and photophobia ($n=21$, 9%). Overall, 191 (88%) reported >30% improvement in headache with their current CGRP-mAb; however, only 107 (56%) of responders reported improvement in their MBS. Improvement of the MBS had a statistically significant positive effect on headache improvement ($\chi^2=7.77$, $P=0.005$). Of those who reported headache improvement with mAbs, majority of those who identified fatigue (56%), brain fog (58%), photophobia (67%) and nausea (77%) as the MBS reported improvement and only 46% of those who identified neck stiffness as the MBS reported improvement.

Conclusion: Fatigue, brain fog and neck stiffness are the most common non-pain symptoms among migraine patients. Despite headache improvement with CGRP-mAbs, many patients continue to experience their most bothersome non-pain symptoms, particularly neck stiffness.

Disclosure: PA, AG, FG, SM, UA, MV and NK have nothing to declare. PJG reports, over the last 36 months, personal fees from Aeon Biopharma, Abbvie, Aurene, CoolTech LLC, Dr Reddy's, Eli-Lilly and Company, Kallyope, Linpharma, Lundbeck, Pfizer, PureTech Health LLC, Satsuma, Seaport Therapeutics, Shiratronics, and Teva Pharmaceuticals.

EPO-0410 | CGRP monoclonal antibodies in migraine: Persistence of premonitory symptoms in the absence of headache — A service evaluation

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Background and aims: Monoclonal antibodies targeting calcitonin gene-related peptide (CGRP-mAbs) have been in wide use for migraine prevention for five years. They have efficacy on headache and potential effects on premonitory symptoms (PreS).

Methods: Patients currently receiving CGRP-mAbs at King's College Hospital, London completed a questionnaire, including data on PreS as part of a service evaluation. Clinical data were extracted from patient records. Associations between categorical variables were assessed using McNemar's tests.

Results: A total of 223 patients responded (median age 48 years; 82% female); chronic migraine (62%), episodic migraine (29%), and new daily persistent headache with migraine phenotype (9%). Overall, 188 patients (84%) recognised PreS, with a median of 6 symptoms (IQR 2–9). Most common PreS were fatigue (67%), neck stiffness (58%) and brain fog (58%). Yawning (McNemar's $\chi^2 = 4.985$, $P = 0.026$) and euphoria (exact McNemar test, $P < 0.001$) were more likely to occur in the premonitory phase than the headache phase. Food cravings were more common in the premonitory phase without statistical significance. Build-up of PreS without headache was reported by 114 (53%), which included 54% ($n = 104$) of patients with $> 30\%$ improvement in headache and 38% ($n = 10$) of those with $< 30\%$ improvement. Median number of PreS in this group was 5 (IQR 3–7). Brain fog (60%), fatigue (54%), and neck stiffness (51%) were most common and the duration of PreS was 1–24 hours in most (67%).

Conclusion: PreS are recognised by a significant majority of migraine patients. Over half of those responding to mAbs continue to experience PreS without headache.

Disclosure: PA, AG, FG, SM, UA, MV and NK have nothing to declare. PJG reports, over the last 36 months, personal fees from Aeon Biopharma, Abbvie, Aurene, CoolTech LLC, Dr Reddy's, Eli-Lilly and Company, Kallyope, Linpharma, Lundbeck, Pfizer, PureTech Health LLC, Satsuma, Seaport Therapeutics, Shiratronics, and Teva Pharmaceuticals.

EPO-0411 | Persistent headache attributed to ischemic stroke: Impact on health-related quality of life

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Background and aims: Persistent headache attributed to ischemic stroke (PHAIS) is a chronic headache disorder developing

after ischemic stroke. While demographic and clinical characteristics have already been described, impairment of the health-related quality of life (QoL) in patients suffering from PHAIS has not yet been demonstrated. This study aims to investigate whether PHAIS impairs the QoL at 12 months after stroke using the Stroke Impact Scale (SIS).

Methods: Data was derived from the Prospective Stroke Cohort Augsburg (SCHANA I & II). After baseline assessment, patients completed a 3- and 12-month follow-up regarding headache characteristics (Rostock Headache Questionnaire) and QoL (SIS). PHAIS was defined as new onset headache since stroke persisting at 12 months. Statistical analyses comprised Kruskal-Wallis-Tests including post-hoc tests and median regression adjusted for age, sex and history of migraine. Bonferroni-correction was applied to account for multiple testing.

Results: Of 973 valid responses at 12-month follow-up, 35 (3.6%) met PHAIS criteria. Patients classified with PHAIS had significantly reduced scores in SIS Domain Memory compared to patients without headaches ($p = 0.015$). In multivariable median regression, SIS Domain Memory remained impaired (estimate: -5.03 , 95% confidence interval: -7.02 to -3.05 , $p < 0.001$). Phenotype of PHAIS was heterogeneous with 15% of patients having migraine-like, 12% tension-like and 15% unspecific symptoms.

Conclusion: PHAIS significantly impairs SIS Domain Memory at 12 months after stabilisation of stroke, independently of age, sex and history of migraine. Our findings support the consideration of routine headache assessment in poststroke follow-ups and may indicate the necessity of future adaptations of PHAIS in poststroke guidelines.

Disclosure: Nothing to disclose.

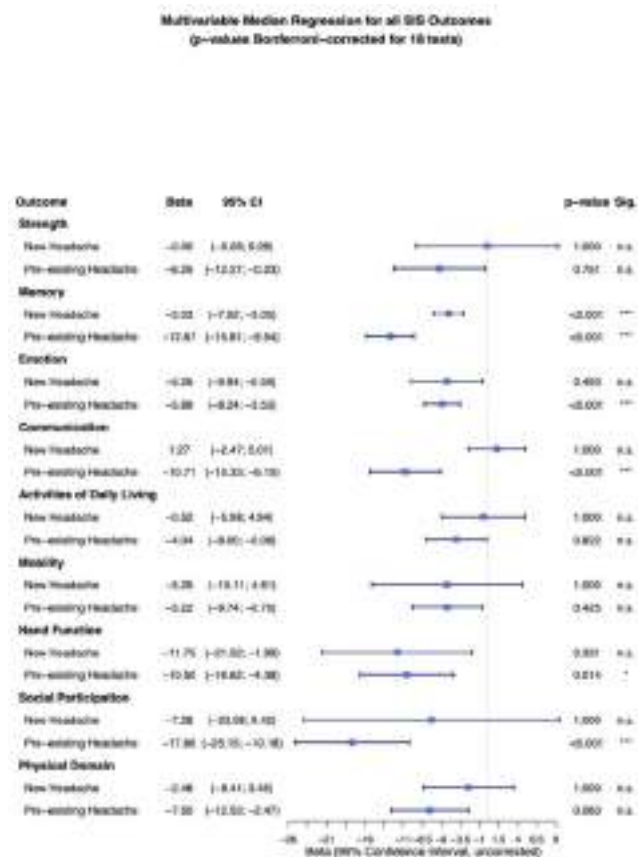


FIGURE 1: Multivariable median regression for all SIS outcomes.

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Background and aims: Individuals with migraine may experience suboptimal efficacy or adverse events with triptans, potentially contributing further to disease burden, particularly among working populations. CaMEQ-I, a multi-country, cross-sectional, observational survey study, assessed the burden of migraine internationally. This analysis evaluates the impact of the number of triptans trialed on work productivity loss and annual indirect costs, among employed individuals with migraine.

Methods: This post hoc analysis included employed adults with complete Work Productivity and Activity Impairment (WPAI) data from CaMEQ-I, stratified by number of triptans trialed: current user (0), 1, 2, and ≥ 3 . Median work impairment and indirect costs were measured via WPAI. Indirect costs were estimated in respondents' local currency, scaled using country-specific working weeks, and adjusted for inflation to October 2025 values.

Results: Here, data from US ($n=565$), UK ($n=506$), and Germany ($n=571$) are reported; results for all six countries are in the complete analysis. Median WPAI: overall work productivity impairment ranged from 50.0–85.8%, increasing with the number of triptans trialed (Figure 1). Parallel trends were observed in median annual indirect costs. Those who trialed ≥ 3 triptans generally demonstrated the highest indirect costs: \$32,927 (US), £18,964 (UK), and €24,716 (Germany). Across all countries, indirect cost generally increased across response strata, with most countries showing the greatest cost burden among individuals who trialed ≥ 3 triptans (Figure 2).

Conclusion: Insights from the CaMEQ-I study suggest that among employed individuals with migraine, trialing multiple triptans is associated with increases in both work productivity loss and indirect costs, across multiple countries.

Disclosure: Jan Versijpt received personal fees and nonfinancial support from Teva, Pfizer and Medtronic, personal fees from Novartis and Lundbeck, and grants and nonfinancial support from Allergan/AbbVie. Kristina Fanning is the Managing Director of MIST Research, which has received research funding from AbbVie, AESARA, Aptar, CoolTech, Migraine Canada, NYC Langone Health, Pfizer, UC Irvine, Ipsen. Elizabeth Leroux has received consultancy fees and/or speaker fees from AbbVie, Eli Lilly, Miravo, Lin Pharmaceutical, Lundbeck, Novartis, Paladin, Pfizer, and Teva. Dawn Buse has received research funding from the US Food and Drug Administration, the National Headache Foundation and Amgen and received consulting fees from Amgen, Allergan/AbbVie, Eli Lilly, Ipsen, Lundbeck, Pfizer, Theranica, and Teva. Raquel Gil-Gouveia reports honoraria for consultant and/or advisory boards from Allergan/AbbVie, Astra Zeneca, Almirall, Bial, Biogen,

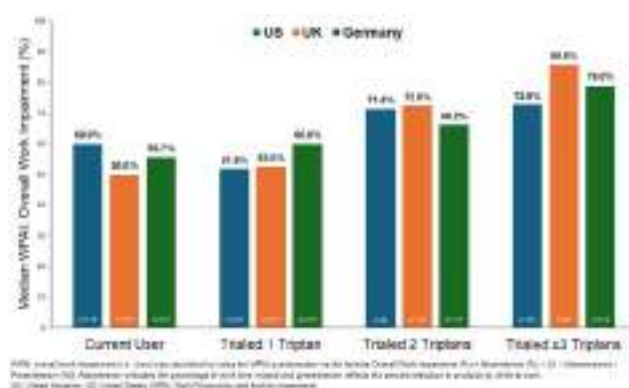


FIGURE 1 Median WPAI: Overall Work Productivity Loss by Number of Triptans Trialed in the US, UK, and Germany.

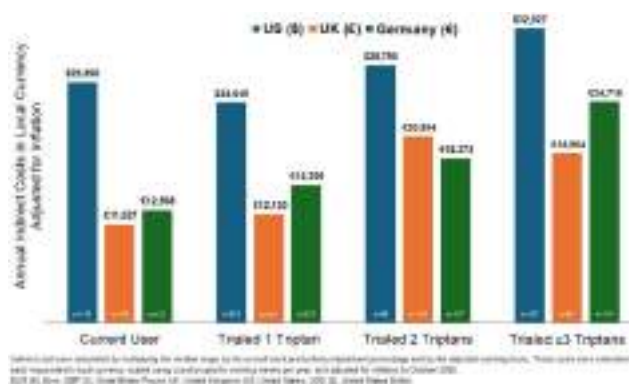


FIGURE 2 Median Annual Indirect Costs by Number of Triptans Trialed in the US, UK, and Germany Adjusted for Inflation (USD/GBP/EUR).

Bristol-Myers Squibb, CMBE, FLOAT, Lilly, Lundbeck, Merk, Novartis, Novo Nordisk, Organon, Pfizer, Roche, Sanofi, Tecnifar and Teva; research funding from Fundação Ciência Tecnologia (Project 29675, MigN2Treat, 02/SAICT/2017); Learning-Health, Luz Saúde (LiON, Luz Innovation on Neurosciences); Novartis-Sociedade Portuguesa de Cefaleias; Bolsa -Projecto Centro de Investigação Interdisciplinar em Saúde and UCP.

EPO-0413 | Familial hemiplegic migraine and treatment with CGRP-mabs – A swiss cohort study

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Background and aims: Familial Hemiplegic Migraine (FHM) is a rare hereditary migraine subtype characterized by motor aura with potentially severe neurological manifestations. Treatment is mostly based on standard migraine therapies, as FHM is usually excluded from clinical trials and existing evidence mainly consists of case reports and series. We present a single-center cohort study to evaluate disease course and treatment, focusing on calcitonin gene-related peptide (CGRP)-based therapies.

Methods: We retrospectively analyzed a cohort of 13 patients with FHM ($N=11$) or probable FHM ($N=2$) treated at

Inselspital Bern, based on medical records and headache diaries. Two patients received the anti-CGRP monoclonal antibody (mab) erenumab.

Results: Eight women and five men were followed for a median of 2, 35 years (range 0–11). Three patients had genetically confirmed FHM type 1 and two had type 2. Clinical course varied, ranging from infrequent mild attacks to severe migraines with prolonged neurological symptoms. Patients requiring prophylaxis, showed favorable responses to lamotrigine, flunarizine, or propranolol. Several patients used triptans acutely without reported adverse events. Two patients (one with FHM1, one with FHM2) with frequent, severe attacks refractory to multiple prophylactic therapies were treated with erenumab. In combination with other medications, antibody therapy reduced attack frequency and severity, although efficacy declined over time in one patient.

Conclusion: This case series highlights the heterogeneity of FHM and the need for individualized treatment strategies. While evidence remains limited, mabs, and combination therapies may represent effective and well-tolerated prophylactic options in selected patients. Further data collection on CGRP mabs in FHM is warranted.

Disclosure: FR: Speaker and consultancy honoraria from Lundbeck, Teva, Abbvie, Merz The authors declare no potential conflicts of interest.

EPO-0414 | Lasmiditan efficacy and cardiovascular safety in high-risk patients: A systematic review and meta-analysis

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Background and aims: Approximately 20% of migraine patients have cardiovascular contraindications to triptans. Lasmiditan, a selective 5-HT_{1F} receptor agonist without vasoconstrictive properties, may offer a safer alternative, though systematic evidence in cardiovascular-risk populations remains limited.

Methods: We systematically searched MEDLINE, Embase, Scopus, and Cochrane CENTRAL from inception to June 2025. Primary outcomes were pain freedom at 2 hours and major adverse cardiovascular events (MACE). We performed random-effects meta-analyses and assessed bias using Cochrane RoB 2 and ROBINS-I tools.

Results: Sixteen studies were included (5 lasmiditan efficacy trials with 5,555 participants, 10 triptan safety studies). Lasmiditan significantly increased pain freedom versus placebo (OR 2.28, 95% CI 1.94–2.67, $p < 0.001$; NNT 4.2–15.4) with clear dose-response relationships (50 mg: OR 1.29; 100 mg: OR 1.72; 200 mg: OR 2.10). Treatment effects remained consistent

across cardiovascular risk categories ($p = 0.56$ for interaction). Cardiovascular adverse events occurred in $< 1\%$ of patients with no increase in highest-risk patients (OR 1.48, $p = 0.56$). Zero vasoconstriction events occurred across $> 19,000$ treatment occasions. Central nervous system effects were common (dizziness 8.6–18.1% vs 2.5% placebo; NNH 6.4–16.4) but predominantly mild-to-moderate. Observational triptan data showed apparent MACE protection (HR 0.82, 95% CI 0.73–0.92), likely reflecting confounding.

Conclusion: Lasmiditan demonstrates robust efficacy and favorable cardiovascular safety with complete absence of vasoconstriction events. Given tolerability profiles, gepants may represent preferred first-line cardiovascular-safe alternatives, with lasmiditan reserved for gepant non-responders.

Disclosure: Nothing to disclose.

Figure 3a. Forest plot of 2-hour MBS freedom with lasmiditan versus placebo

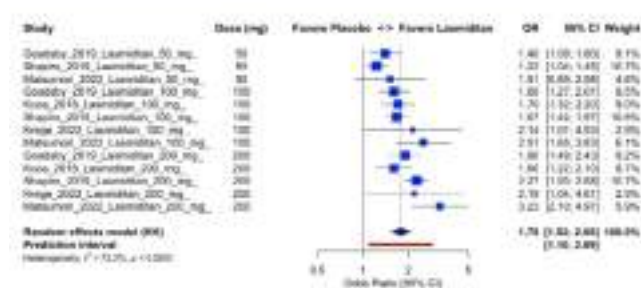


Figure 3b. Forest plot of 2-hour MBS freedom stratified by lasmiditan dose

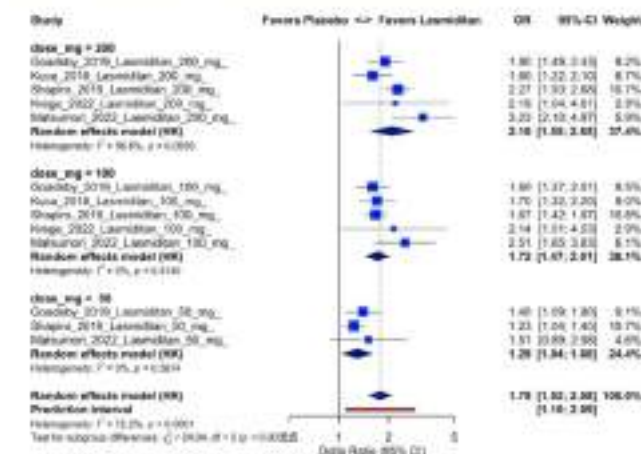


FIGURE 1

Figure 4a. Forest plot of composite major adverse cardiovascular events with triptan use versus non-triptan comparison

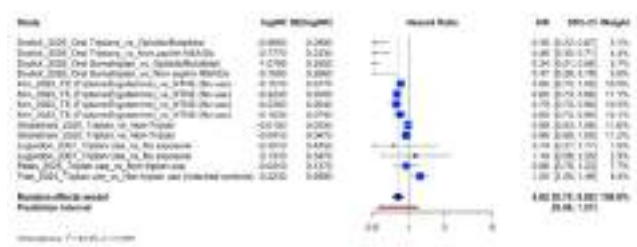


Figure 4b. Forest plot of nonfatal myocardial infarction with triptan use



Figure 4c. Forest plot of nonfatal stroke with triptan use



FIGURE 2

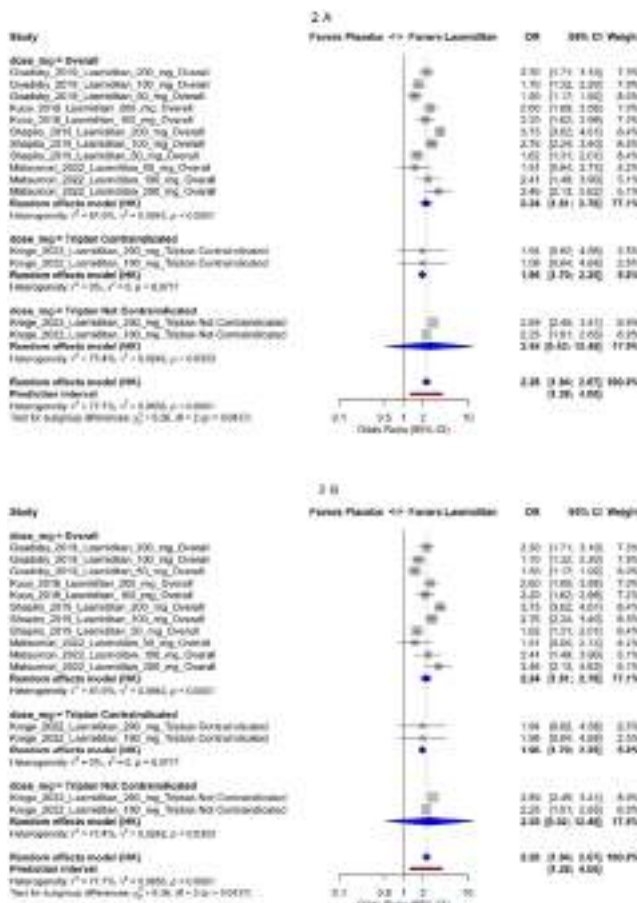


FIGURE 3

EPO-0415 | Impact of atogepant on functional outcomes for the preventive treatment of migraine: A Phase 3, open-label, 156-week, long-term safety extension study

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Background and aims: Evaluate the long-term impact of atogepant on functional outcomes for the preventive treatment of migraine.

Methods: An open-label, 156-week, long-term safety extension study enrolled adult participants with chronic or episodic migraine who had an inadequate response to 2–4 classes of conventional oral preventive treatment, and completed the lead-in studies PROGRESS or ELEVATE, respectively. The primary outcome was safety. Clinical efficacy and functional outcomes were prespecified exploratory analyses. Functional outcomes included change from baseline (CFB) in performance of daily activities (PDA) and physical impairment (PI) domain scores of the Activity Impairment in Migraine-Diary (AIM-D) and Migraine-Specific Quality of Life Questionnaire v2.1 (MSQv2.1) Role-Function Restrictive (RFR) domain score during 156 weeks.

Results: A total of 529 participants were included in the modified intent-to-treat population (PROGRESS, $n=285$; ELEVATE, $n=244$). Overall safety results were consistent with the known safety profile of atogepant. The mean (SD) change from lead-in study baseline in PDA domain score at weeks 13–16 was -16.0 (14.1), and this reduction (improvement) was consistent at 153–156 weeks [CFB = -16.0 (14.1)] (Figure 1a). The mean (SD) change from lead-in study baseline in PI domain score at weeks 13–16 was -13.2 (13.1), and this reduction was consistent through 153–156 weeks [CFB = -13.4 (13.6)] (Figure 1b). An improvement in MSQv2.1 RFR scores was demonstrated at week 12 and was consistent at week 156 (Figure 2).

Conclusion: Atogepant demonstrated improvements in functional outcomes at Week 12 and was consistent at 156 (MSQv2.1 RFR), and at Weeks 13–16 and was consistent at 153–156 (PDA and PI).

Disclosure: Peter J. Goadsby over the last 36 months reports personal fees from CoolTech LLC, Dr Reddy's, Epalex, Ipsen, Kallyope, Linpharma, Lundbeck, Orion Pharma, Pfizer, PureTech Health LLC, Satsuma, Scilex Pharmaceuticals, Seaport Therapeutics, Septurna and Teva Pharmaceuticals, and personal fees for advice through Gerson Lehrman Group, Guidepoint, SAI Med Partners, and fees for educational materials from CME Outfitters and WebMD, and publishing royalties or fees from Massachusetts Medical Society, Oxford University Press, UpToDate and Wolters Kluwer Messoud Ashina has served as an advisory board member, consultant, and or speaker for AbbVie, AstraZeneca, Eli Lilly, GlaxoSmithKline, Incyte, Lundbeck, Pfizer, Teva, and Vedana Therapeutics Inc. He has received

institutional research grants from the Danish National Research Foundation, Lundbeck Foundation, Novo Nordisk Foundation, and Novartis. He serves on the editorial board of *Neurotorium* and has received honoraria. Sait Ashina received honoraria for consulting from AbbVie/Allergan, Amgen, Biohaven, Eli Lilly, Impel NeuroPharma, Linpharma, Lundbeck, Novartis, Pfizer, Percept, Satsuma, Supernus, Teva, and Theranica. Cristina Tassorelli has participated in advisory boards for AbbVie, Dompé, Eli Lilly, Lundbeck, Pfizer and Teva, and has lectured at symposia sponsored by AbbVie, Eli Lilly, Lundbeck, Pfizer and Teva. She is principal investigator or collaborator in clinical trials sponsored by AbbVie, Eli Lilly, Lundbeck, Pfizer and Teva. She has received research grants from the European Commission, the Italian Ministry of Health, the Italian Ministry of University, the Migraine Research Foundation, and the Italian Multiple Sclerosis Foundation. Soo-Jin Cho has received honoraria for speaking or consulting from AbbVie, Allergan, Eli Lilly and Company, Lundbeck, Handok-Teva, Pfizer, and SK chemicals. Pranav Gandhi, Jonathan Stokes, Jonathan H. Smith, Krisztian Nagy, and Rebeka Man are employees of AbbVie and may hold AbbVie stock.

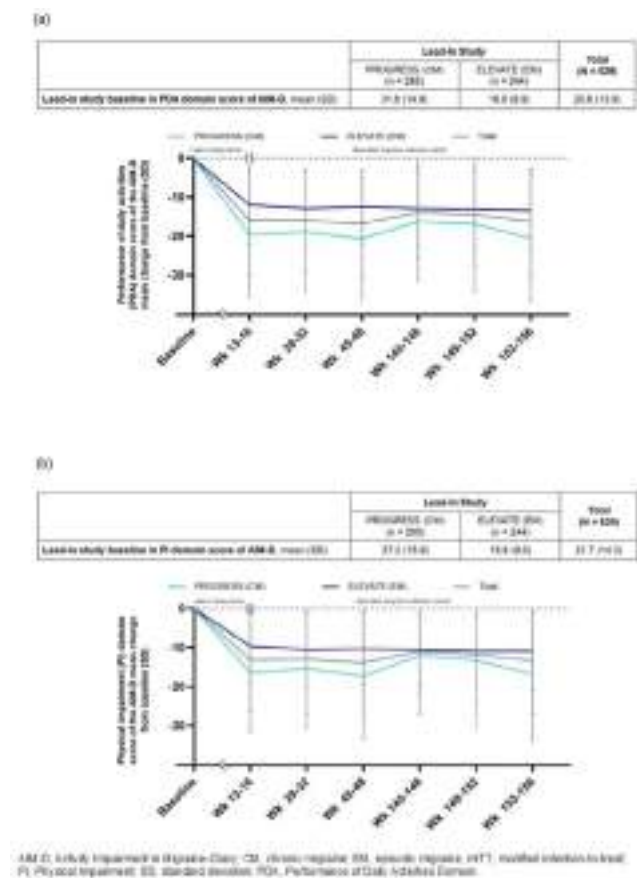


FIGURE 1 Changes from lead-in study baseline in mean Activity Impairment in Migraine-Diary (AIM-D) domains: (a) PDA and (b) PI at weeks 13–16, 29–32, 45–48, 145–148, 149–152, and 153–156 (mITT population).

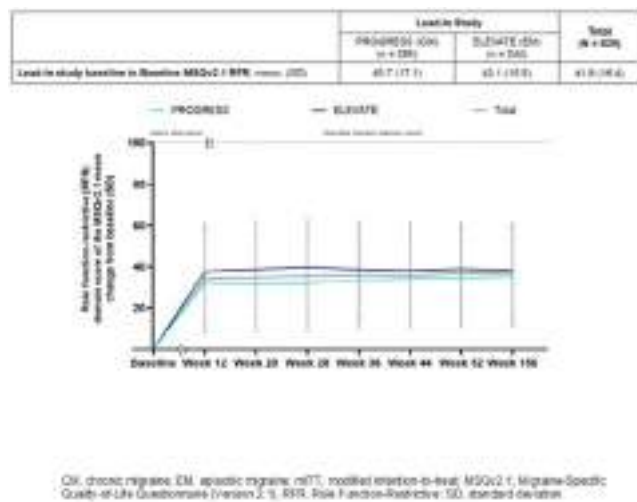


FIGURE 2: Migraine-Specific Quality of Life Questionnaire v2.1 (MSQv2.1): Mean changes from lead-in study baseline in MSQv2.1 (A) role function-restrictive (RFR), at Weeks 12, 20, 28, 36, 44, 52, and 156 (in mITT population).

EPO-0416 | Workplace productivity improvements following preventive treatment with eptinezumab: Region-based analyses from the phase 3 SUNRISE trial

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Background and aims: To evaluate migraine-related impairments in productivity and impact of eptinezumab versus placebo in the SUNRISE trial in chronic migraine, and to explore regional differences.

Methods: SUNRISE (NCT04921384) confirmed the migraine-preventive efficacy and safety of eptinezumab in participants with chronic migraine, predominantly from Asian countries. Adults (18–75y) diagnosed with chronic migraine were randomized 1:1:1 to 12-week double-blind treatment with IV eptinezumab 100mg, 300mg, or placebo. The patient-reported Migraine-specific Work Productivity and Activity Impairment questionnaire (WPAI:M), captured at baseline and at Weeks 4, 8, and 12, includes sub-scores for absenteeism, presenteeism, work productivity loss, and activity impairment. Region-based subgroup analyses were conducted post hoc for Asia and Europe. *p*-values are descriptive.

Results: Of 978 participants treated, 63%/37% were from Asia/Europe. Mean WPAI:M baseline sub-scores (total population) were absenteeism: 7–10%; presenteeism: 53–57%; work productivity loss: 55–60%; and activity impairment: 58–60% (Europe > Asia across sub-scores). In the full population, both eptinezumab

doses were associated with greater improvements from baseline versus placebo ($p < 0.05$) in presenteeism, productivity loss, and activity impairment at Weeks 4, 8, and 12. Smaller changes from baseline in absenteeism may reflect lower baseline scores, which could suggest participants working despite migraine burden.

Conclusion: In a predominantly Asian population with chronic migraine, eptinezumab provided greater improvements than placebo in workplace productivity and activity from Week 4 through Week 12. Participants from Asia reported less absenteeism (at baseline and improvement) than participants from Europe, reflecting a more pronounced pattern of work attendance despite a similar high level of migraine burden.

Disclosure: SY declares no conflicts of interest. YM reports personal consultancy fees from Amgen Astellas BioPharma K.K., Daiichi Sankyo Company, Limited, Eli Lilly Japan K.K., and Otsuka Pharmaceutical Co, Ltd. B-KK has received honoraria as a consultant and speaker from AbbVie, Teva Korea, Lundbeck Korea, Organon Korea, Pfizer Korea, and SK Pharmaceuticals. His research group has received research grants from Pfizer Korea and funding for clinical trials from AbbVie, Lundbeck, Novartis, Pfizer, and Teva Pharmaceuticals. He is on editorial boards of Headache and Pain Research and Journal of Clinical Neurology. AG-D has received fees from AbbVie, Amgen, Lundbeck, Pfizer, and Teva while serving as a principal investigator in clinical trials, as well as for speaking and training. GG received fees from Lundbeck while serving as a principal investigator in clinical trials. PP-R has received honoraria, in the last 36 months, as a consultant and speaker from AbbVie, Almirall, Dr. Reddy's, Eli Lilly, Lundbeck, Medscape, Novartis, Organon Pfizer, and Teva Pharmaceuticals. Her research group has received research grants from AbbVie, AGAUR, EraNet Neuron, FEDER RIS3CAT, Instituto Investigación Carlos III, MICINN, Novartis, and Teva Pharmaceuticals, and has received funding for clinical trials from AbbVie, Amgen, Biohaven, Eli Lilly, Lundbeck, Novartis, Pfizer, and Teva Pharmaceuticals. She is the Honorary Secretary of the International Headache Society, is on the editorial board of Revista de Neurologia, and is an associate editor for Cephalalgia and Neurologia. She is a member of the Clinical Trials Guidelines Committee of the International Headache Society. She has edited the Guidelines for the Diagnosis and Treatment of Headache of the Spanish Neurological Society. She is the founder of www.midolordecabeza.org, a platform to give information and tools to physicians and people who suffer from migraine and other headaches. MKJ and BS are full-time employees of H. Lundbeck A/S and own stock or stock options in H. Lundbeck A/S. KR, AE, and AM are all full-time employees of H. Lundbeck A/S. TT is an advisor to Hedgehog MedTech, Inc. and Sawai Pharmaceutical Co, Ltd.

EPO-0417 | Hormonal migraine across the life course: European migraine in women survey results (EMHA), H2 2025

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Background and aims: Migraine attack improvement or worsening has been attributed to hormonal life stages, yet prospective, longitudinal data is limited and tailored management remains inconsistent. EMHA, with oversight from an interdisciplinary Scientific Committee, launched the Migraine in Women Survey to generate patient-reported evidence to inform education and care pathway improvements.

Methods: A cross-sectional online survey was conducted across 13 European countries. Recruitment used GAD3 and dissemination via EMHA national patient associations. Women (18–70) were screened using the validated 3-item ID Migraine tool (positive screen $\geq 2/3$), yielding 5,410 screened-positive respondents for analysis. Questions assessed headache patterns and care experiences across menstruation/perimenopause, pregnancy/postpartum, and menopause, with a separate path for transgender participants using hormone therapy.

Results: Overall, 42% reported no formal migraine diagnosis and 76% reported having discussed their attacks with a healthcare professional. Among menstruating/perimenopausal respondents ($n = 3,783$), 66% linked attacks to their period (38% most/every period; 28% some periods) and 47% reported attacks a few days before bleeding. Period-related attacks were more painful (36%), longer-lasting (28%), and more medication-resistant (20%) than non-menstrual attacks, yet only 23% reported being offered specific treatment options. 16% rated current treatments “very effective,” and 72% wanted more education on hormone-headache links.

Conclusion: Hormonal influences on migraine are commonly reported across Europe, but substantial gaps persist in diagnosis, healthcare engagement, and tailored management, supporting targeted clinician/patient education and improved hormonal migraine pathways.

Disclosure: ¹ Nothing to declare*; ² Nothing to declare*; ³ PJG reports, over the last 36 months, personal fees for consulting from Abbvie, CoolTech LLC, Dr Reddy's, Epalex, Ipsen, Kallyope,

Linpharma, Lundbeck, Orion Pharma, Pfizer, PureTech Health LLC, Satsuma, Scilex Pharmaceuticals, Seaport Therapeutics, Septurna and Teva Pharmaceuticals.; ⁴ Nothing to declare; ⁵ Nothing to declare; ⁶ Nothing to declare; ⁷ REN has on-going relationship with Abbott, Astellas, Bayer HealthCare AG, Besins Healthcare, Biocodex, Exeltis, Fidia, Gedeon Richter, Merck & Co, Novo Nordisk, Organon & Co, Shionogi Limited, Theramex, Viartis, and Vichy Laboratories. REN is the current president of the International Menopause Society (IMS). REN declares that none of these could be perceived as prejudicing the impartiality of the research reported; ⁸ reports fees for advisory board and presentations for/from Novartis, Eli Lilly, Almirall, and Lundbeck; ⁹ Nothing to declare; ¹⁰ Nothing to declare

EPO-0418 | Rimegepant for the prevention of migraine in adults with inadequate response to 3 or 4 categories of non-migraine-specific oral preventive medication

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Background and aims: A previous phase 4 trial (NCT05518123) conducted in 14 countries evaluated the efficacy and tolerability of rimegepant for the prevention of episodic migraine in participants with a documented history of inadequate response to 2 to 4 categories of non-migraine-specific oral preventive medications (OPMs). This exploratory analysis evaluated rimegepant efficacy in participants with inadequate response to 3 or 4 prior non-migraine-specific OPM categories.

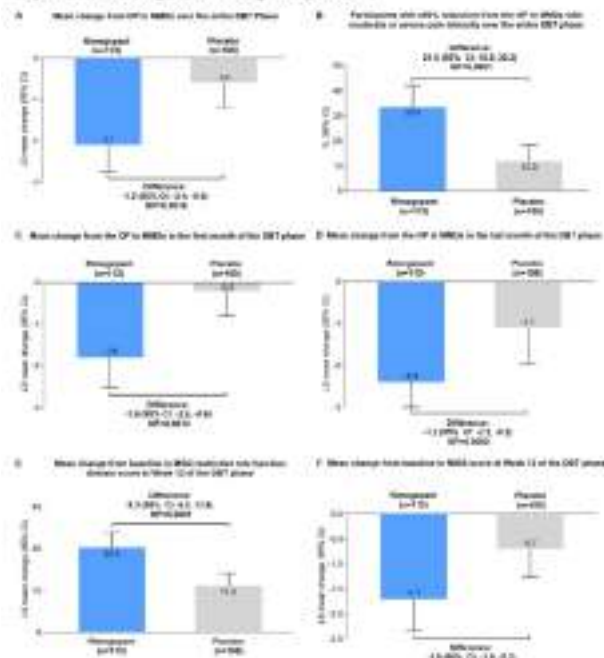
Methods: Following a 28-day observation phase (OP), adults not using preventive therapy with 4–14 monthly migraine days (MMDs) and <15 monthly headache days were stratified by OPMs with prior inadequate response and migraine days in the OP then randomized to double-blind treatment (DBT) with rimegepant 75 mg orally disintegrating tablet (ODT) or placebo every other day (EOD) for 12 weeks.

Results: 114 participants with documented prior inadequate response to 3 or 4 OPM categories were enrolled into the rimegepant group and 109 into the placebo group. In both DBT groups, most participants had 3 prior OPMs (81.6% and 84.4%) and ≥8 migraine days in the OP (64.9% and 66.1%). The primary endpoint analysis favored rimegepant vs. placebo: mean change from the OP in MMDs over the entire DBT phase was -2.1 vs. -0.6 (difference = -1.5; 95% CI = -2.4, -0.6; nominal $p = 0.0016$; Figure). Key secondary endpoint analyses also favored rimegepant (all nominal $p < 0.05$; Figure).

Conclusion: Rimegepant 75 mg ODT EOD demonstrated efficacy for the prevention of episodic migraine in participants with a documented history of inadequate response to 3 or 4 categories of non-migraine-specific OPM.

Disclosure: This study was sponsored by Pfizer. In the past 24 months, Paul G Mathew has served as a consultant for Pfizer, Abbvie, Axsome, Lilly, Theranica, and Tonix. For this abstract, he served as an uncompensated consultant and not in his capacity as a member of the HMS/MGB Faculty. Annelies Van Dycke has served on advisory/scientific boards and/or given lectures

Figure. Efficacy endpoint analyses in participants with a documented history of inadequate response to 3 or 4 categories of non-migraine-specific OPM



A shows the primary endpoint analysis in the subgroup of participants with a documented history of inadequate response to 3 or 4 categories of non-migraine-specific OPMs. B-F show key secondary endpoint analyses in the subgroup of participants with a documented history of inadequate response to 3 or 4 categories of non-migraine-specific OPMs. In these analyses, participants who had a ≥8% reduction from OP to MMDs over the entire DBT phase (A, C, D, and F) were analyzed using a modified effect size of 0.5 (0.5 = 0.50). The modified effect size was used for the primary endpoint analysis (A) and for the secondary endpoint analyses (B, C, D, E, and F) because the effect size of 0.5 is a more conservative estimate of the effect size. The modified effect size was used for the primary endpoint analysis (A) and for the secondary endpoint analyses (B, C, D, E, and F) because the effect size of 0.5 is a more conservative estimate of the effect size. The modified effect size was used for the primary endpoint analysis (A) and for the secondary endpoint analyses (B, C, D, E, and F) because the effect size of 0.5 is a more conservative estimate of the effect size.

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EPO-0419 | Effectiveness And Safety of Monthly Versus Quarterly Fremanezumab for migraine prevention: A 12-Month Italian multicenter real-world study

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Background and aims: Fremanezumab, an anti-calcitonin gene-related peptide (CGRP) monoclonal antibody approved for migraine prevention, can be administered monthly (225 mg) or quarterly (675 mg). In clinical practice, regimen selection is mainly based on patient preference, while real-world evidence comparing the effectiveness of the two dosing strategies remains limited. We aimed to compare monthly versus quarterly fremanezumab in a real-life setting over 12 months.

Methods: This prospective, multicenter, real-world study involved four Italian headache centers. Patients received monthly or quarterly fremanezumab according to preference. Primary endpoints were changes in monthly migraine days (MMD) and monthly headache days (MHD) at 3, 6, and 12 months, and the occurrence of adverse events (AEs). Secondary endpoints included $\geq 30\%$, $\geq 50\%$, and $\geq 75\%$ responder rates in migraine frequency, reductions in monthly acute medication days (AMD) and pills (AMP), changes in disability and clinical scores (MIDAS, HIT-6, NRS, ASC-12), and medication-overuse headache (MOH) cessation.

Results: A total of 294 patients were enrolled (152 monthly; 142 quarterly). At 6 months, 138 and 133 patients, and at 12 months, 98 and 102 patients completed follow-up in the monthly and quarterly groups, respectively. Baseline characteristics were comparable. Both regimens showed similar reductions in MMD and MHD across all time points, with no significant differences in AEs. Improvements in AMP, AMD, MIDAS, HIT-6, NRS, and ASC-12 scores were comparable, as were responder rates and MOH cessation.

Conclusion: Monthly and quarterly fremanezumab demonstrated comparable effectiveness and safety for migraine prevention over 12 months in a real-world setting.

Disclosure: Nothing to disclose.

Movement disorders 3

EPO-0420 | [18F]- FDG-PET metabolic patterns in idiopathic normal pressure hydrocephalus and neurodegenerative disorders: A voxel-wise analysis

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Background and aims: Idiopathic normal pressure hydrocephalus (iNPH) is a potentially treatable cause of gait impairment that frequently overlaps clinically and radiologically with neurodegenerative disorders (ND). We aimed to identify [18F]-FDG-PET metabolic patterns associated with iNPH in comparison with ND in patients presenting with ventriculomegaly.

Methods: We retrospectively analysed 50 adults (mean age 72.0 $\pm$ 7.5 years) with gait impairment and ventriculomegaly, characterised by disproportionately enlarged subarachnoid-space hydrocephalus, with supportive features including Evans Index >0.30 and callosal angle $<90^\circ$. Diagnostic classification integrated neurological and cognitive assessments, brain MRI, CSF biomarkers when available, and dopaminergic imaging when required. Final diagnoses and key characteristics are summarised in Tables 1 and 2. FDG-PET scans underwent blinded visual interpretation at baseline by an experienced nuclear medicine specialist. Voxel-wise analyses were performed in SPM on spatially and intensity-normalised images. Group comparisons used

TABLE 1 Demographic and clinical characteristics of patients with ventriculomegaly classified by predominant clinical phenotype.

Characteristic	Predominant ND phenotype (n= 24)	Predominant iNPH phenotype (n=26)
Sex M/F ^a	9/15	10/16
Age years ^b	73.1 $\pm$ 7.2	70.8 $\pm$ 7.9
Symptom duration at imaging (FDG-PET/MRI), years	1.4 $\pm$ 0.4	1.5 $\pm$ 0.3
Disease follow up at the last consultation	4.8 $\pm$ 0.5	5.0 $\pm$ 0.4
Parkinsonism n (%)	19 (79%)	16 (62%)
Oculomotor abnormalities (supranuclear vertical gaze palsy/impairment in vertical saccades) n (%)	14 (58%)	0 (0%)
Hypophonia n (%)	12 (50%)	0 (0%)
Falls n (%)	15 (63%)	0 (0%)
Dysphagia n (%)	8 (33%)	0 (0%)
MMSE	26,3 $\pm$ 2.1	27,1 $\pm$ 2.4
MCI amnesic	5 (21%)	2 (8%)
MCI non-amnesic	18 (75%)	14 (54%)
Normal cognition	1 (4%)	10 (39%)

TABLE 2 Final clinical diagnosis in patients with ventriculomegaly.

PSP n (%)	14 (44%)
AD n (%)	5 (14%)
DLB n (%)	3 (4%)
PD n (%)	1 (2%)
FTD n (%)	1 (2%)
iNPH n (%)	26 (36%)
Total	50 (100%)

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Background and aims: PRKRA-related dystonia-parkinsonism (DYT-PRKRA) is a rare autosomal recessive disorder typically presenting with childhood-onset progressive generalised dystonia. We report a case with an acute encephalopathic presentation triggered by febrile illness, a phenotype that remains poorly characterised.

Methods: We describe the longitudinal clinical, neuroimaging, and genetic findings of a patient with an acute encephalopathic onset followed by progressive dystonia. Clinical data were obtained from serial neurological assessments. Brain MRI studies were reviewed longitudinally, with particular focus on basal ganglia involvement. Genetic investigations included metabolic testing, mitochondrial studies, and whole-exome sequencing.

Results: A previously healthy child developed an acute encephalopathy at 3 years of age following a viral febrile illness, characterised by somnolence, loss of ambulation, axial hypotonia, ataxia, dysarthria, and pyramidal signs. Cerebrospinal fluid, metabolic, and infectious investigations were unremarkable. Brain MRI demonstrated bilateral putaminal T2 hyperintensities, subsequently evolving into progressive putaminal atrophy (Figure 1). Mitochondrial genetic testing and muscle biopsy were normal. Partial neurological recovery occurred, followed by progressive generalised dystonia with prominent oromandibular involvement during adolescence. Treatment with levodopa, biotin, and thiamine was ineffective. Whole-exome sequencing identified a homozygous likely pathogenic variant in PRKRA (c.665C>T; p.Pro222Leu), confirming the diagnosis of DYT-PRKRA.

Conclusion: This case demonstrates that DYT-PRKRA may present with an acute encephalopathic phenotype triggered by febrile illness, mimicking ATP1A3-related rapid-onset dystonia-parkinsonism, but distinguished by striatal necrosis on MRI. These findings support a shared clinicopathological spectrum among PRKRA-, ATP1A3-, and SLC19A3-related

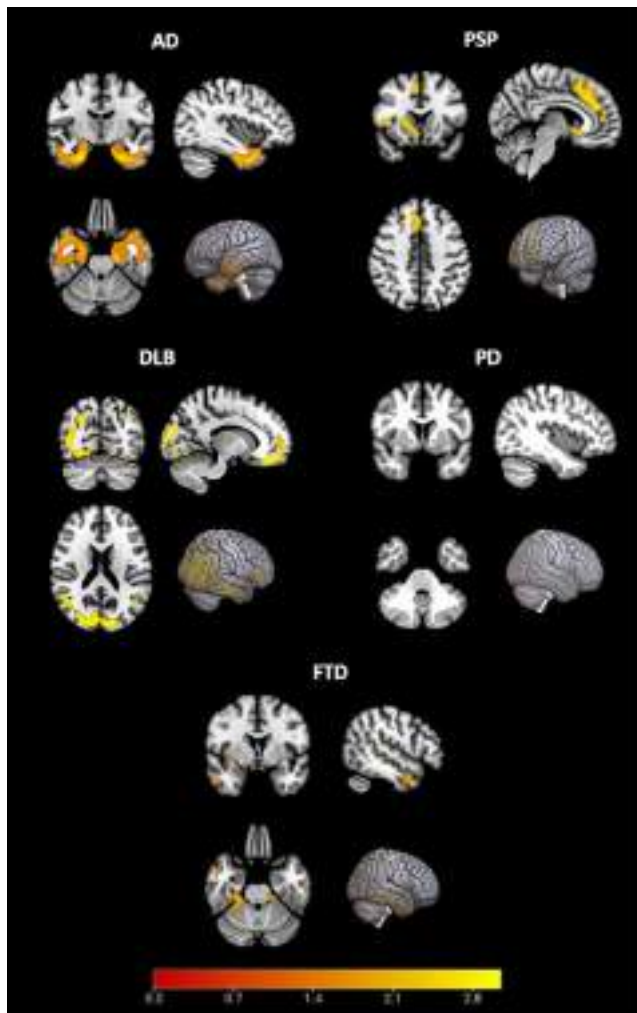


FIGURE 1 Voxel-wise FDG-PET metabolic patterns across major neurodegenerative disorders in patients with ventriculomegaly.

independent-sample t-tests and ANOVA at a voxel-level threshold of $p < 0.005$. Statistical parametric maps were projected onto a T1-weighted MRI template.

Results: Voxel-wise comparisons demonstrated metabolic differences between iNPH and ND subgroups. Relative to progressive supranuclear palsy, iNPH showed higher metabolism in ventromedial frontal regions and basal ganglia. Compared with Alzheimer's disease, iNPH displayed increased metabolism in bilateral medial temporal cortices. Compared with Lewy body disease, iNPH demonstrated greater metabolism in bilateral posterior cortical regions. Relative to frontotemporal dementia and Parkinson's disease, iNPH showed higher metabolism in the cerebellar vermis ($p < 0.005$) (Figure 1).

Conclusion: Voxel-wise FDG-PET reveals regionally distributed metabolic differences in iNPH, supporting its role in early clinical characterisation within a spectrum of neurodegenerative disorders.

Disclosure: Nothing to disclose.

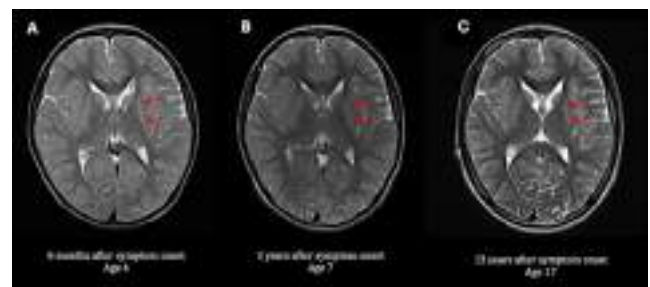


FIGURE 1 Longitudinal MRI evolution of bilateral putaminal involvement in DYT-PRKRA.

disorders and highlight the importance of considering PRKRA in acute dystonia presentations with basal ganglia involvement. **Disclosure:** Nothing to disclose.

EPO-0422 | Lower urinary tract symptoms in functional neurological disorder: A prospective cohort study

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Background and aims: Lower urinary tract symptoms (LUTS) are common in neurological disorders but unexpected in functional neurological disorder (FND) in which neural pathways are structurally intact. Evidence on this association is scarce. The primary objective was to describe the prevalence and pattern of LUTS in FND, and the secondary objective was to identify associated factors.

Methods: In a two-year prospective cohort study, we enrolled consecutive patients assessed for FND, with a diagnosis confirmed by positive clinical signs. Conditions causing LUTS were excluded. LUTS, pelvic symptoms, psychiatric comorbidity, pain, and quality of life were assessed using validated questionnaires; anticholinergic load was quantified by the Anticholinergic Drug Scale. The presence of LUTS was defined by cutoffs on the Urinary Symptom Profile or on a visual analog scale of pelvi-perineal discomfort. Factors independently associated with LUTS were identified using stepwise multivariable logistic regression.

Results: Of 145 participants (mean [SD] age 43 [13.7] years; 81% women), 64 (44%) reported LUTS, predominantly storage-type (30/64, 47%), frequently associated with coexisting bowel and sexual dysfunction (46/61, 72%). Independent factors associated with LUTS were (OR [95% CI] per 1-SD increase): higher symptom count (1.98 [1.31–3.12]; $p=0.002$), anticholinergic load (1.86 [1.23–2.93]; $p=0.005$), pain intensity (1.81 [1.19–2.90]; $p=0.008$), and childhood adversity (1.61 [1.09–2.43]; $p=0.019$).

Conclusion: Prospective systematic assessment revealed frequent LUTS in FND (44%), mainly storage symptoms, exceeding previous prevalence estimates. The combination of multiple associated factors suggests multifactorial mechanisms. These findings support systematic screening and targeted management, including medication review and multimodal pain management.

Disclosure: Nothing to disclose.

EPO-0423 | Unique challenges and barriers to increasing equitable access to neuropalliative care in Parkinson's disease: A scoping review

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Background and aims: Persons with Parkinson's disease (PD) have significant palliative care needs that can be addressed through Neuropalliative care (NPC). NPC aims to improve quality of life through non-motor symptom and pain management, caregiver support, emotional/spiritual support, advanced care planning and specialist/hospice referral. However, NPC is underutilized by individuals with PD from underserved communities and the reasons for this inequity are not fully understood.

Methods: We conducted a scoping review of the literature with the primary research question, "What are the barriers to equitable access to neuropalliative care in Parkinson's disease?" A literature search was completed on PubMed and Covidence was utilized to review the search results.

Results: Of the 383 articles identified and 27 were included in the final review. Four Themes were identified: a) Lack of knowledge of characteristics of normal aging as distinct from PD symptoms; b) Systemic barriers such as skewed geographic distribution of neurologists, lack of inclusive research, insurance coverage for specialist care etc. can impede access to NPC; c) Sociocultural misunderstanding and resistance to palliative care; d) Underutilization of innovative and culturally responsive models such as telehealth, and partnerships with community partners.

Conclusion: Multifactorial barriers impede underserved persons with PD from accessing NPC. Future research is needed to further understand and address the barriers identified. Future directions for improving access to NPC among those from underserved communities could include increasing community outreach, improving awareness about resources provided by NPC, demystifying the term "palliative care" through community based programs, and increasing appropriate referrals from primary care to neurology.

Disclosure: Nothing to disclose.

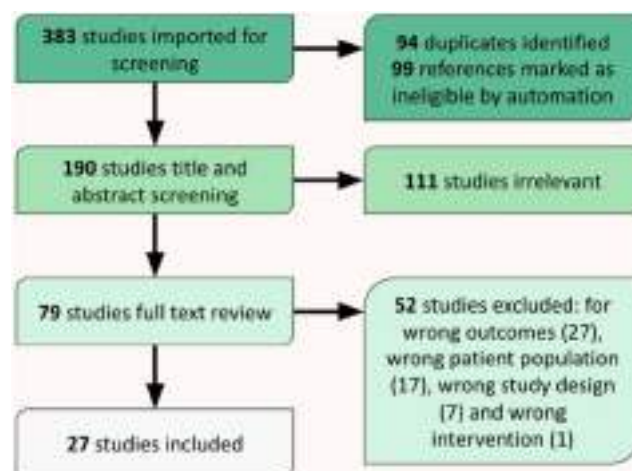


FIGURE 1 Covidence Results.

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Background and aims: Huntington's disease (HD) is an incurable neurodegenerative disorder. Therefore, identifying lifestyle factors that influence its progression is of particular interest. This study aimed to evaluate the impact of smoking tobacco on the onset and progression of HD symptoms.

Methods: The study is based on the Enroll-HD database. A cohort of 2,438 HD subjects (799 of whom were presymptomatic) who had four consecutive annual visits was extracted. Logistic regression models were used to evaluate the impact of lifetime smoking on progression to the symptomatic phase. For the premanifest group, a Cox proportional hazards model was applied to estimate the hazard ratio of HD diagnosis for smokers versus nonsmokers during the three-year observation period. Multivariate linear regression models were used to investigate the relationship between smoking and the progression of HD clinical measures.

Results: Among presymptomatic HD females, but not males, we found that lifetime smoking was associated with a higher risk of transitioning to the symptomatic phase (odds ratio: 1.26, $p=0.026$). Current female smokers were at a higher risk of progressing to the symptomatic phase than nonsmokers (hazard ratio: 1.35, $p=0.023$). For the symptomatic HD cohort, regardless of sex, smoking was associated with faster progression of motor symptoms (Total Motor Score, $p=0.038$; 95% confidence interval [CI], 0.03–0.9) and cognitive impairment (Stroop Word Reading Test, $p=0.04$; 95% CI, 0.1–0.9).

Conclusion: Lifetime and current smoking may be associated with an earlier age of onset and faster progression of HD.

Disclosure: Nothing to disclose.

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Background and aims: Sex differences in the treatment of people with Parkinson's disease (PwP) with a device-aided therapy (DAT) have been poorly investigated. Our aim was to analyze sex differences in the management and response to a DAT in PwP in daily clinical practice (DCP).

Methods: Data collected in the DATs-PD GETM Spanish Registry until October 30, 2025, were used. This is a descriptive, observational, prospective, multicenter clinical registry with progressive inclusion of PwP treated with a DAT in DCP conditions in more than 30 centers from Spain (Santos-García. PLoS One 2025). Sex differences in the DAT received and changes in quality of life (QoL), motor symptoms (MS), non-motor symptoms (NMS), and autonomy for activities of daily living (AADL) after 6 months of treatment were analyzed.

Results: A total of 618 PD patients (66.9 ± 9.5 years old; 57.6% males) were treated with a DAT. A significant difference was observed in the DAT type according to sex ($p=0.006$), with 73.1% of PwP who were treated with deep brain stimulation being males (Figure-1). At the time of DAT indication, females were older, received a higher levodopa equivalent daily dose, and had a worse health-related QoL and AADL (Table-1). OFF time decreased, whereas MS and NMS burden and health-related QoL improved at 6 months follow-up in both groups, males and females (Table-2). AADL improved only in males during the OFF state (Table-2).

Conclusion: Sex differences were detected in the use of DATs in PwP. Improvement in OFF time, MS, NMS, and QoL were detected in both groups.

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Background and aims: Friedreich's Ataxia (FRDA) is a rare neurodegenerative disorder characterized by progressive ataxia. Clinical assessment relies on observer-based scales such as the Scale for the Assessment and Rating of Ataxia (SARA) and the modified Friedreich's Ataxia Rating Scale (mFARS), which may be insensitive to subtle motor changes. This study applies video-based analysis and unsupervised anomaly detection to quantify upper-limb coordination.

Methods: 778 video recordings of the finger-nose (SARA, mFARS), finger-tapping (mFARS) and pronation-supination tasks (SARA, mFARS) of 64 individuals with FRDA (48% female), and 58 controls (47% female) acquired during routine clinical visits were analyzed. Features describing movement speed, frequency, smoothness, entropy, and variability were extracted. Group- and severity-related differences were assessed using Mann-Whitney U Test. To capture multivariate deviations in movement patterns not detectable by univariate testing, unsupervised anomaly detection models trained exclusively on controls were used. Here, subject-specific anomaly scores and assess their relevance to disease status were extracted.

Results: Most features differed significantly between patients and controls and across increasing SARA and mFARS severity. Greater severity was associated with prolonged task execution, slower and less frequent movements, increased variability, and higher spectral entropy. Anomaly scores increased with severity, showing strong associations in finger fapping (Spearman's $\rho \approx 0.64$ – 0.65) and pronation–supination ($\rho \approx 0.71$ – 0.72), with moderate associations in nose–finger task ($\rho \approx 0.33$ – 0.36).

Conclusion: Video-based upper-limb kinematic analysis combined with anomaly scoring provides an objective, feature-driven measure of motor impairment with potential utility for quantifying disease severity and tracking progression in FRDA.

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FIGURE 1 Workflow for Feature Extraction and Anomaly Scoring in Upper-Limb Task.



FIGURE 2 Distributions of some features across clinical severity levels for all tasks.

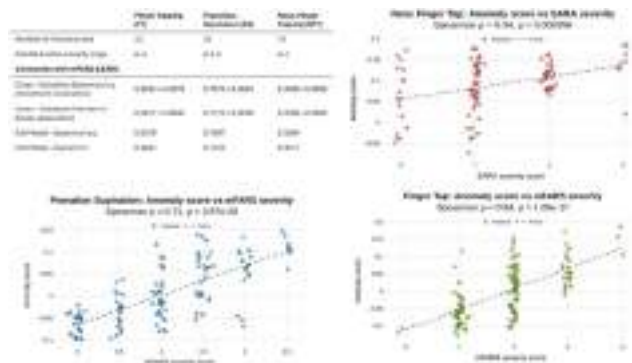


FIGURE 3 Anomaly detection model performance across tasks and clinical severity levels.

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Background and aims: Spinal cord stimulation (SCS) has recently been suggested to improve gait impairment in Parkinson's disease (PD). However, it remains unclear whether any specific stimulation paradigm is superior. Here, we aimed to compare different paradigms of SCS in PD patients with gait impairment.

Methods: Eight gait-impaired PD patients who had previously received SCS were included. Four stimulation paradigms (Fast Acting Sub-perception Therapy [FAST], MicroBurst, low frequency/high pulse width, and high frequency/low pulse width) were administered in a randomised order, each for two months. The Postural Instability and Gait Disorder (PIGD) score was assessed before initiation of SCS (approximately two years prior) and during each stimulation paradigm. A linear mixed model was used to compare the stimulation paradigms. In addition, the individually optimal paradigm was compared with a natural cohort of gait-matched PD patients ($n=8$) without SCS.

Results: Seven participants completed follow-up for at least two settings (four completed all settings, one completed three, and two completed two). One participant was excluded due to non-compliance. No stimulation paradigm was found to be overall superior to the others. After two years, participants receiving SCS demonstrated an improvement in PIGD when using their individually optimal paradigm, which was significantly better compared with the two-year PIGD deterioration observed in a natural cohort of PD patients without SCS ($p=0.001$).

Conclusion: Our findings suggest that the SCS paradigm should be individualised rather than based on a “one-size-fits-all” approach. Furthermore, long-term SCS may mitigate the natural progression of gait deterioration in PD.

Disclosure: This work was funded by the Independent Research Fund Denmark and the Danish Parkinson's Association. Funders had not impact on trial design, execution or analysis.

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Background and aims: Parkinson's disease psychosis (PDP) is a disabling non-motor feature of PD. Treatment options are limited, with clozapine constrained by safety concerns, quetiapine demonstrating variable efficacy, and pimavanserin approved specifically for PDP but unavailable outside of the U.S. Long-term real-world data on treatment effects are scarce. This study examined whether initiation of PDP pharmacotherapy alters the longitudinal trajectory of psychosis and subjective cognitive symptoms.

Methods: We performed a retrospective longitudinal observational study using electronic medical records from University of California San Diego movement disorders clinics from 2015–2025. Eligible patients had PD and a Movement Disorder Society–Unified Parkinson's Disease Rating Scale Part I item 1.2 score of at least one. Patients were categorized by treatment (pimavanserin, quetiapine, combination (pimavanserin and quetiapine), or no treatment). Ordinal psychosis and cognitive symptom scores were analyzed using cumulative link mixed-effects models. Treated groups were modeled with pre- and post-treatment trajectories.

Results: 100 patients were included (pimavanserin $n=14$; quetiapine $n=12$; combination $n=16$; untreated $n=58$), followed for a mean of 4.3 years (SD 2.7; range 0–12.8). After adjustment for demographics and disease duration, only pimavanserin was associated with a statistically significant attenuation of psychosis progression after treatment initiation (Δ slope = -0.498 ; $p=0.045$). Quetiapine and combination therapy showed non-significant reductions. Cognitive symptoms worsened prior to treatment in all groups, with significant post-treatment attenuation observed for pimavanserin and combination therapy, but not quetiapine.

Conclusion: Pimavanserin was associated with long-term attenuation of psychosis and cognitive symptom progression in this real-world cohort, exceeding effects observed with other treatments.

Disclosure: This research was funded by Acadia Pharmaceuticals. The authors report no other disclosures.

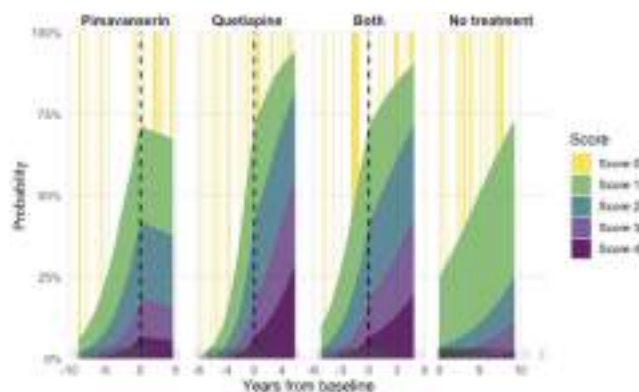


FIGURE 1 Predicted distribution of Parkinson's disease psychosis symptoms (MDS-UPDRS 1.2 score) by treatment group over time.

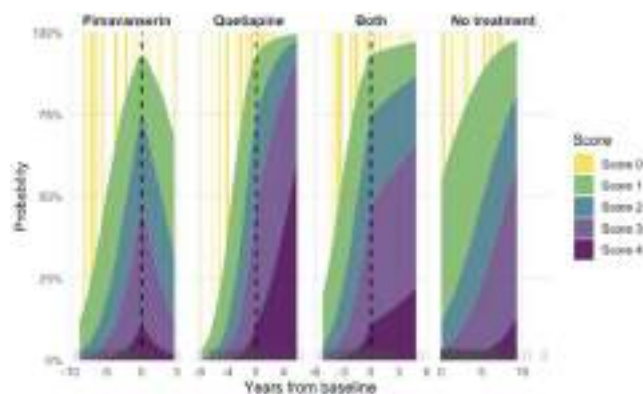


FIGURE 2 Predicted distribution of Parkinson's disease cognitive symptoms (MDS-UPDRS 1.1 score) by treatment group over time.

EPO-0429 | Effects of proprioceptive–visual and vestibular–visual integration training on balance and gait in Parkinson's disease: A randomized controlled trial

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Background and aims: Postural instability and impaired sensory integration are major contributors to falls and disability in Parkinson's disease (PD), yet the optimal sensory integration training strategy remains unclear.

Methods: 184 individuals with PD were allocated randomly to the control group, proprioceptive–visual integration group A and vestibular–visual integration group B. All participants received 4 weeks of conventional rehabilitation, while group A and B additionally performed 30-minute sensory integration training with center-of-mass visual feedback, with or without

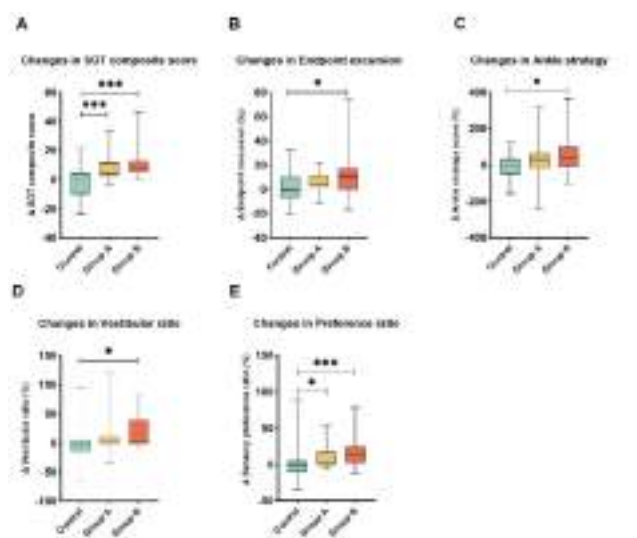


Figure 2. Changes in balance and sensory integration outcomes after intervention. A–E: Changes in SOT composite score, endpoint excursion, ankle strategy, vestibular ratio, and sensory preference ratio after intervention. Group B showed greater improvements than the control group across all outcomes.

FIGURE 2 Results.

an unstable surface. The primary outcome was the Sensory Organization Test (SOT) composite score. Secondary outcomes included clinical balance, motor symptoms, sensory integration parameters, gait measures, and quality of life.

Results: Vestibular–visual integration training combined with conventional rehabilitation can better improve balance control, motor symptoms and gait symmetry in patients with PD.

Conclusion: Vestibular–visual integration training combined with conventional rehabilitation can better improve balance control, motor symptoms, and gait symmetry in patients with PD. This approach may be particularly beneficial for patients with prominent postural instability and elevated fall risk.

Disclosure: Nothing to disclose

EPO-0430 | Echinacoside Attenuates Pathogenic α -Synuclein Aggregation by Inhibiting the PERK–eIF2 α –ATF3/CHOP Pathway in the PFFs Models of Parkinson's Disease

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Background and aims: Abnormal aggregation of α -synuclein (α -Syn) is a core pathological hallmark of Parkinson's disease (PD), and the α -Syn pre-formed fibrils (PFFs) model closely mimics this process. Our previous work showed that echinacoside (ECH) protects against MPTP-induced injury by suppressing endoplasmic reticulum (ER) stress. Here, we asked whether ECH ameliorates α -Syn mediated dopaminergic damage in PFFs-based models and whether this effect involves ER-stress related ATF3 signaling.

Methods: In vivo, mice received unilateral striatal PFFs injections followed by intragastric ECH (40 or 20 mg/kg) for 1 month. Motor function was assessed by standard behavioral tests, and substantia nigra pathology was evaluated by

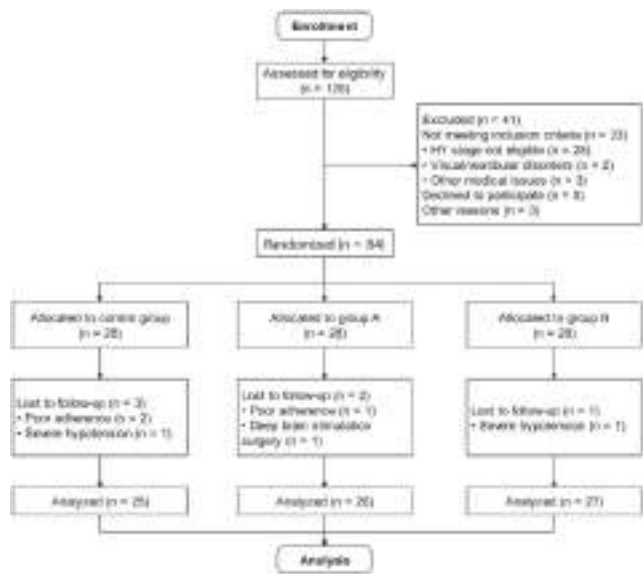


Figure 1. CONSORT flow diagram of participant enrollment, randomization, and analysis. This figure illustrates participant flow through the study.

FIGURE 1 CONSORT flow diagram.

tyrosine hydroxylase (TH) immunofluorescence and Western blot of phosphorylated α -syn (p- α -syn), GRP78 and PERK-eIF2 α -ATF3-CHOP proteins. In vitro, PFFs-treated SH-SY5Y cells were exposed to the ER-stress inducer thapsigargin (Tg) or to ECH. Cell viability, TH, p- α -syn and the same ER-stress markers were quantified.

Results: PFFs induced motor deficits, TH loss, p- α -syn accumulation and up-regulation of GRP78 and PERK-eIF2 α -ATF3-CHOP in mouse substantia nigra. ECH improved motor behavior, increased TH expression and significantly decreased p- α -syn and ER-stress marker levels ($p < 0.05$; Figure 1). In SH-SY5Y cells, Tg further enhanced PFFs-induced GRP78, p-PERK, p-eIF2 α , ATF3, CHOP and p- α -syn, whereas ECH reduced p- α -syn and down-regulated these ER-stress proteins ($p < 0.05$; Figure 2).

Conclusion: ECH protects nigrostriatal dopaminergic neurons and reduces pathological α -Syn aggregation in PFFs-induced cellular and mouse models of PD, an effect closely associated with inhibition of PERK-eIF2 α -ATF3-CHOP-mediated ER stress.

Disclosure: Nothing to disclose.

EPO-0431 | Significant elevation of serum pro-inflammatory cytokines in GBA1-associated Parkinson's disease: Implications for immune-mediated pathogenesis

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Background and aims: Parkinson's disease (PD) associated with GBA1 mutations (GBA-PD) is increasingly recognized as a clinically and biologically heterogeneous subtype[1]. This study aimed to investigate differences in serum inflammatory and neurodegeneration markers between Parkinson's disease (PD) patients carrying GBA1 variants (mPD) and patients with idiopathic sporadic PD (sPD), focusing on the potential role of interferon-gamma (IFN- γ), a key Th1 cytokine[2].

Methods: We enrolled 12 patients with GBA1-associated PD and 12 with sporadic PD, matched for age, sex, and disease duration. Clinical data and serum samples were collected. PD diagnosis followed Movement Disorder Society criteria. Serum cytokines (IL-1 β , IL-17A, IFN- γ , IL-4, TNF- α , IL-10, IL-6) were measured by ELISA. Neurodegeneration markers included neurofilament light chain (NfL) and brain-derived neurotrophic

TABLE 1 Demographics and clinical characteristics of mPD and sPD patients. Continuous variables are mean $\pm$ SD, categorical as counts. Groups were matched; no statistically significant differences were observed.

Mean	mPD(n=12)	sPD(n=12)	P value
Age (years)	62.3 $\pm$ 7.5	63.4 $\pm$ 8.0	0.732
Disease Duration	4.2 $\pm$ 2.5 years	4.4 $\pm$ 2.1 years	0.834
Men/women	10/2	10/2	1.0

factor (BDNF). Group differences were analyzed with Mann-Whitney U test ($p < 0.05$).

Results: IFN- γ levels were significantly elevated in GBA-PD compared to sPD ($p = 0.0078$), along with IL-1 β ($p < 0.05$), while other cytokines, NfL, and BDNF showed no differences. Comorbidities in GBA-PD included depression (66%) and melanoma (25%). The selective IFN- γ elevation suggests a Th1-skewed systemic immune activation in GBA1 mutation carriers.

Conclusion: GBA-PD patients exhibit a distinct peripheral immune-inflammatory profile characterized by Th1-associated IFN- γ and IL-1 β elevation, supporting the concept of a biologically unique PD subgroup. This Th1-skewed signature may contribute to both neurodegeneration and comorbid autoimmune or psychiatric manifestations[3]. IFN- γ represents a potential biomarker and therapeutic target, and modulation of Th1 pathways could offer novel strategies to personalize treatment in this PD subgroup. Validation in larger cohorts is warranted.

Disclosure: Nothing to disclose

Neurorehabilitation 1

EPO-0432 | Temporal precision requirements for neuroplasticity-based neurorehabilitation: A meta-regression analysis

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Background and aims: Brain-computer interface (BCI) systems combined with functional electrical stimulation (FES) demonstrate efficacy in research settings (pooled effect size $d = 0.72$), yet clinical adoption remains limited. This translation gap suggests that available systems fail to meet neurophysiological requirements, particularly temporal precision within the neuroplasticity window required for Hebbian learning.

Methods: Following PRISMA guidelines, a systematic review examined randomized controlled trials evaluating timing-sensitive neurorehabilitation interventions for post-stroke upper limb recovery. Interventions were classified as research prototypes or commercial systems. The primary outcome was change in the Fugl-Meyer Upper Extremity score. Meta-regression assessed associations between neural-to-stimulation latency and efficacy while adjusting for intervention duration, stroke chronicity, and study quality. Segmented regression identified latency thresholds, and mediation analysis evaluated whether latency explained performance differences.

Results: Across 28 studies including 1,247 participants, a critical latency threshold of 300 ms (95% CI: 250–350 ms) was identified. Systems operating below this threshold achieved larger effects ($d = 0.89$) than those exceeding it ($d = 0.38$; $p < 0.001$). Research prototypes demonstrated lower latencies (median 220 ms) and greater efficacy ($d = 0.85$) than commercial systems (median 1,150 ms; $d = 0.34$). Mediation analysis indicated that 73% of the efficacy gap was attributable to latency, with each additional 100 ms associated with a 0.042 reduction in effect size ($p < 0.001$).

Conclusion: Clinical translation failure is therefore attributable to latency-mediated loss of efficacy operating beyond the

TABLE 1 Systems with latency below 300ms achieve 3.6x better outcomes ($d = 1.02$ vs 0.28) than high-latency systems, demonstrating temporal precision as the primary driver of neuroplasticity-based recovery in stroke rehabilitation.

Table 1. Clinical Outcomes Stratified by System Latency Categories				
Latency Category	Studies (n)	Effect Size SMD (95% CI)	FIM-UE Points (95% CI)	p-value
Ultra-low (<200ms)	5	0.82 (0.61, 1.23)	10.2 (8.5, 12.5)	<0.001
Low (200-300ms)	7	0.81 (0.63, 0.99)	8.1 (6.3, 9.9)	<0.001
Medium (300-500ms)	9	0.52 (0.34, 0.70)	5.2 (3.4, 7.0)	<0.001
High (>500ms)	7	0.28 (0.09, 0.47)	2.8 (0.9, 4.7)	0.004

TABLE 2 Commercial systems operate 5x slower than research prototypes (1150ms vs 220ms) which explains why marketed BCI devices fail clinically despite promising research trials.

Table 2. Comparative Performance: Research Prototypes vs. Commercial Systems			
Metric	Research Prototypes (n=15)	Commercial Systems (n=10)	p-value
Median latency (FIM), ms	220 (180-250)	1150 (880-1600)	<0.001
Pooled effect size, SMD (95% CI)	0.85 (0.68-1.01)	0.35 (0.17-0.51)	<0.001
FIM-UE improvement, points (95% CI)	9.2 (7.4-10.0)	1.6 (1.3-5.4)	<0.001
Heterogeneity (I ²)	58%	31%	—
Studies achieving <300ms threshold, %	80% (12/15)	0% (0/10)	<0.001

TABLE 3 Low-latency systems are cost-effective (\$38K/QALY) despite higher costs, while cheaper high-latency systems exceed thresholds (\$94K/QALY) due to poor outcomes.

Table 3. Cost-Effectiveness Analysis by System Latency Category					
System Type	Total Cost (£)	FIM-UE Gain	QALY Gain	ICER (£/QALY)	CEA
Low latency (<300ms)	15,000	9.1	0.10	38,400	Yes
Medium latency (300-500ms)	11,000	8.2	0.18	54,300	No
High latency (>500ms)	12,000	3.6	0.13	94,200	No
Commercial therapy (comparison)	1,200	0	0	—	—

identified 300 ms threshold. Enforcing engineering standards based on segmented and mediation analyses, including sub-300 ms latency targets linked to superior cost-effectiveness, is essential for improving therapeutic and economic outcomes.

Disclosure: Nothing to disclose.

EPO-0433 | **Evaluation of reticulospinal plasticity using a novel wearable (PowerBead) for post-stroke finger extension recovery**

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Background and aims: Loss of finger extension excludes ~50% stroke survivors from rehabilitation. Strengthening the reticulospinal tract (RST) may aid recovery when the corticospinal tract (CST) is damaged. The RST, activated via muscle spindle afferents (MSA), can be stimulated by mechanical ‘taps’. Pairing these with timed auditory ‘clicks’ may induce neuroplastic



FIGURE 1 Depicts the device ‘The PowerBead’.

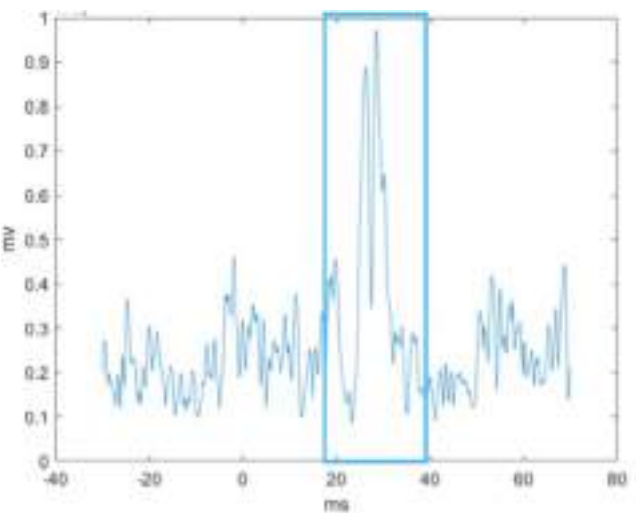


FIGURE 2 The blue box in the graph denotes a sample MSA response generated by the PowerBead.

changes through convergent input [Salisbury, Wang & Baker, n.d.]. The objective of this study is to evaluate the feasibility and physiological efficacy of a wearable tap-click neuromodulation approach in inducing reticulospinal plasticity relevant to post-stroke finger extension.

Methods: The PowerBead is a wearable delivering a ‘tap’ to the affected forearm and an earpiece providing a synchronised contralateral ‘click’. Co-designed with clinicians and therapists, it provides 4-hour treatment cycles to 20 healthy participants and 20 stroke survivors, guided by PPI feedback. Efficacy is assessed pre- and post-cycle using TMS-evoked EMG at the extensor digitorum, the Modified Ashworth Scale, grip/pinch strength, and Box & Block tests.

Results: Previous studies confirmed PowerBead induced MSA activation [Salisbury, Wang & Baker, n.d.]. Building on work by Baker et al (2021), which showed MSA-auditory pairing supports recovery, our device integrates this ‘tap-click’ loop to promote passive finger extension. Early data demonstrated RST plasticity after 4 hours of treatment, with increased cortical excitability and suppressed brainstem activity.

Conclusion: Early PowerBead findings indicate promising engagement of the RST through the ‘tap-click’ loop, highlighting its novelty in translating lab-based neuroplasticity into accessible home-based therapy. Data collection is ongoing, with plans for clinical trials and public availability.

Disclosure: This study is funded by NIHR [National Institute for Health and Care Research] i4i award and led by Dr Laura Salisbury [CEO of KnitRegen] in collaboration with UCL [University College London].

EPO-0434 | Evolving use of botulinum toxin in neurological practice: A national audit from Malta (2013–2025)

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Background and aims: Botulinum toxin (BoNT) is an established treatment for neurological conditions associated with spasticity and dystonia. Since the establishment of a dedicated national BoNT service in Malta, clinical demand and service complexity have increased. This audit aimed to evaluate patient characteristics, indications, treatment patterns, clinical outcomes, and documentation practices in order to identify areas for service development and standardization.

Methods: A retrospective audit was conducted of electronic and paper records for patients attending the Neurology and Spasticity Clinic between 2013 and 2025. Data collected included demographics, neurological diagnosis, treatment status, BoNT dosing patterns, follow-up intervals, documented symptomatic and functional outcomes, adjunct pharmacological therapy, and recording of multidisciplinary team (MDT) involvement and consent. Descriptive analyses were performed.

Results: A total of 187 patients were identified, of whom 146 received BoNT therapy and 134 remained in active follow-up. Mean age was 56 years, with balanced sex distribution. Indications included post-ischemic stroke spasticity, cerebral palsy, dystonia, and traumatic brain injury. Most patients were reviewed at clinically appropriate intervals, typically every 4–6 months. Mean BoNT dose per session was 79.4 units, with higher doses used for spasticity. Symptomatic and functional improvements were commonly documented.

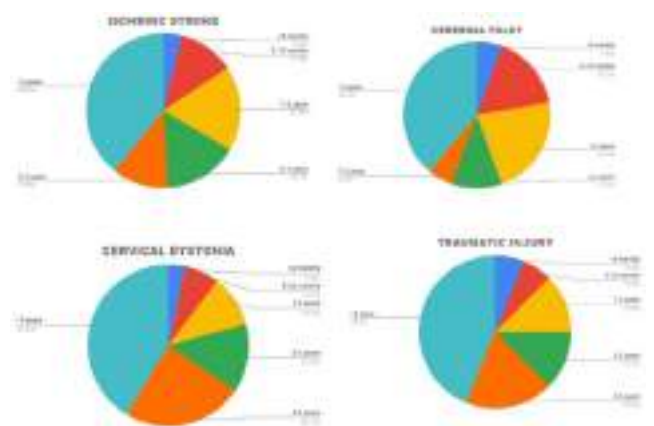


FIGURE 1 Results - Use of botulinum therapy in different neurological conditions.

Diagnosis	Average Dose per Session (U)	Average Time on BoTx (Years)	Average Number of Visits	Typical Follow-Up Interval
Cerebral Palsy	150	4.1	19.6	7–8 months
Ischemic Stroke	150	1.1	2.4	6–7 months
Spastic Paraparesis	180	4.9	25.3	5 months
Traumatic Brain Injury	180	6.5	19.5	4–5 months
Cervical Dystonia	75	1.8	6.1	8 months
Hemifacial Spasm	20	6.4	1.7	10 months

FIGURE 2 Results - Average doses of botulinum therapy used in different neurological conditions with follow up periods.

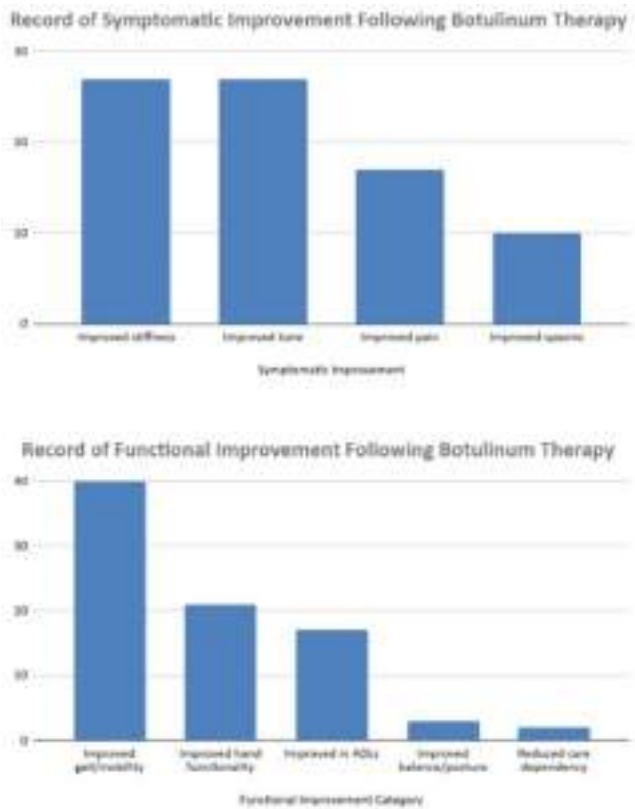


FIGURE 3 Results – Symptomatic and functional improvement following botulinum therapy.

Conclusion: This national audit demonstrates effective real-world use of BoNT across a heterogeneous neurological population. Variability in documentation of MDT input, dose titration, and follow-up highlights opportunities for standardization. Findings will inform the development of structured documentation tools and a local protocol, with re-audit planned to assess service improvement.

Disclosure: Nothing to disclose.

EPO-0435 | Long-term follow-up of patients with disorders of consciousness: Prognostic value of early CRS-R Improvement

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Background and aims: The Coma Recovery Scale–Revised (CRS-R) is the gold standard for assessing consciousness in prolonged Disorders of Consciousness (pDoC). We previously^{1,2} showed that baseline CRS-R and its 4-week increase iCRS-R predicted consciousness improvement at discharge from rehabilitation setting. This longitudinal follow-up evaluates the predictive value of baseline CRS-R and its early change for long-term survival and functional autonomy.

Methods: Using a cross-sectional approach, all patients enrolled in the previous study² were contacted. A structured telephone interview, including the Glasgow Outcome Scale–Extended (GOSE), was administered. Mortality rate, time to death were also recorded.

Results: Of 110 patients, 96 (87.3%) completed the interview and 69 patients (71.8%) died [median time from event 15 months (IQR: 52.5; 3–124)]. Lower iCRS-R (OR: 0.027; $p < 0.001$), non-traumatic aetiology (OR: 0.125; $p = 0.003$), and older age (OR: 1.049; $p = 0.020$) were significantly associated with death in the multivariate analysis. Younger age (HR: 1.038; $p = 0.001$) and greater iCRS-R (HR: 0.0284; $p = 0.007$) were associated with longer survival in the Cox-regression analysis

Conclusion: The early improvement in CRS-R during intensive rehabilitation represents a key prognostic marker of long-term survival. Longitudinal CRS-R assessment may help identify patients at higher risk of mortality and support clinical decision-making. References: 1. Portaccio et al. doi:10.1080/02699052.2018.1440420 2. Portaccio et al. doi:10.1016/j.apmr.2018.01.015

Disclosure: Nothing to disclose.

EPO-0436 | Single-Session Artificial Intelligence-Assisted Gait Training Improves Spatiotemporal Parameters in Dementia and Parkinson's Disease

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Background and aims: Gait impairment in dementia and Parkinson's disease (PD) drives falls and loss of independence, yet therapist-intensive rehabilitation limits scalability. Artificial intelligence (AI)-assisted gait training may standardize delivery, reduce manpower, and provide real-time feedback. The present study aims to test whether a single session of AI-assisted gait training improves spatiotemporal gait parameters in dementia and PD.

Methods: Adults aged 60–85 years with dementia or PD who could ambulate completed one training session ($\sim 23.2 \pm 9.6$ min) comprising standardized walking tasks with real-time feedback, delivered under “Support” or “Escort” mode walking with the

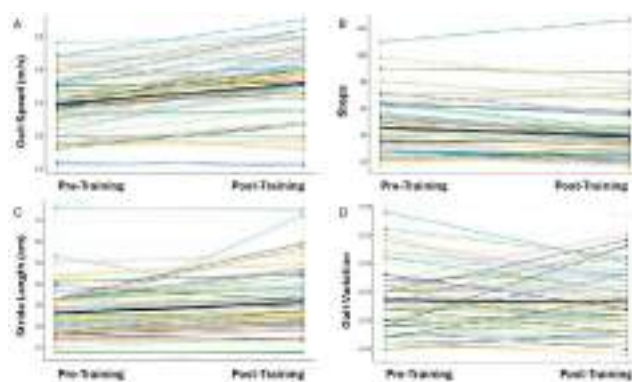


FIGURE 1 Individual changes in gait parameters following the training intervention in dementia subjects.

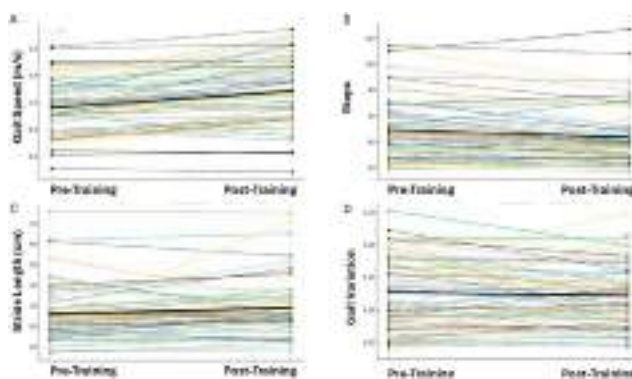


FIGURE 2 Individual changes in gait parameters following the training intervention in Parkinson's disease subjects.

AI-assisted gait training device as needed. Primary outcomes were pre–post changes in gait speed (m/s) and the secondary outcomes included step length (cm), step count, and gait variability obtained by the device. Two-sided paired t tests were performed within four strata: Dementia–Support ($n = 67$), Dementia–Escort ($n = 43$), PD–Support ($n = 64$), and PD–Escort ($n = 43$).

Results: Gait speed increased within session across all strata. Step length increased and step count decreased in Support walking for both diagnoses, whereas Escort walking showed non-significant trends for these spatial parameters. Gait variability showed no significant changes in any stratum. Cognitive status did not affect the changing of gait parameter, supporting feasibility for targeting elders.

Conclusion: A single AI-assisted gait training produced short-term functional gains in spatiotemporal gait parameters in dementia and PD, demonstrating a therapist-sparing, scalable training model. Longer-term trials should evaluate durability, dose–response, and transfer to everyday gait and falls, and refine automation for home or remote deployment.

Disclosure: Nothing to disclose.

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Background and aims: Post-stroke dysphagia is associated with aspiration, prolonged hospitalization, and poor recovery. Pharyngeal electrical stimulation (PES) is an emerging neuro-stimulation technique designed to enhance swallowing function through modulation of pharyngeal sensory pathways. This study aimed to systematically review and meta-analyze the effects of PES on swallowing outcomes and safety in patients with acute stroke.

Methods: We performed a systematic review and meta-analysis of randomized controlled trials evaluating PES in patients with post-stroke dysphagia. PubMed, Scopus, and Web of Science were searched from inception to December 2025. Outcomes included dysphagia severity, swallowing function, functional oral intake, feeding tube withdrawal, length of stay, and safety.

Results: Nine randomized controlled trials involving 448 patients were included. PES significantly improved overall swallowing function compared with control (SMD = -0.30, 95% CI: -0.50 to -0.10), with dysphagia severity scores also showing greater improvement (SMD = -0.35, 95% CI: -0.55 to -0.15). Patients receiving PES had higher rates of nasogastric tube withdrawal (RR = 2.5, 95% CI: 1.8–3.5) and improved functional oral intake. The analysis demonstrated a significant reduction in hospital length of stay, driven largely by post-extubation stroke populations (SMD = -0.5, 95% CI: -0.80 to -0.20). PES was well tolerated, with no increase in serious adverse events.

Conclusion: PES improves swallowing function and reduces dysphagia severity in patients with acute stroke. It increases the likelihood of nasogastric tube removal, improves oral intake, and shortens hospital stay, while remaining safe and well tolerated, supporting its use as an effective adjunct in stroke rehabilitation.

Disclosure: Nothing to disclose.

EPO-0438 | Abstract withdrawn

EPO-0439 | Upper-extremity functional outcomes following bilateral rTMS after stroke: Systematic review and meta-analysis

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Background and aims: The literature largely examines unilateral high-frequency stimulation of the lesioned hemisphere or low-frequency inhibition of intact hemisphere using repetitive Transcranial Magnetic Stimulation (rTMS) on stroke patients. In contrast, bilateral protocols combining facilitative and inhibitory effects remain underexplored. This review evaluates the effects of bilateral rTMS on upper extremity function.

Methods: A systematic search was conducted in PubMed, Web of Science, and Scopus up to November 22, 2025 (Figure-1). Randomized controlled trials (RCTs) were included if stroke patients were assessed with Fugl-Meyer Assessment (FMA) and received bilateral rTMS. Bilateral rTMS and sham groups were compared. The meta-analysis was conducted in RStudio (PROSPERO: CRD420251230168).

Results: Three RCTs were included in the study and demonstrated low risk and good methodological quality (Figure-2a,2b). The bilateral rTMS intervention demonstrated a significant advantage over sham in improving upper-extremity functional outcomes. Based on the common-effect model, the post-treatment effect was substantial (SMD = 1.54, 95% CI: 1.10–1.99), while the random-effects model yielded a wider and more uncertain estimate (SMD = 3.30, 95% CI: -1.06–7.66) due to heterogeneity ($I^2 = 96.6\%$, $p < 0.0001$). At the 3-month follow-up, the improvement was sustained and even greater under the common-effect model (SMD = 2.09, 95% CI: 1.60–2.58), whereas the random-effects model again showed uncertainty (SMD = 4.23, 95% CI: -0.67–9.13) due to heterogeneity ($I^2 = 96.8\%$, $p < 0.0001$). Although the overall direction of effect consistently favors active rTMS, the heterogeneity warrants cautious interpretation of the pooled estimates (Figures-3a,3b).

Conclusion: Bilateral rTMS may provide benefits for upper-extremity functional recovery after stroke, although the limited number of included studies contributes to substantial heterogeneity. These findings indicate the need for more well-designed RCTs.

Disclosure: Nothing to disclose.

PRISMA 2022 flow diagram for new systematic reviews which included searching of databases and registers only

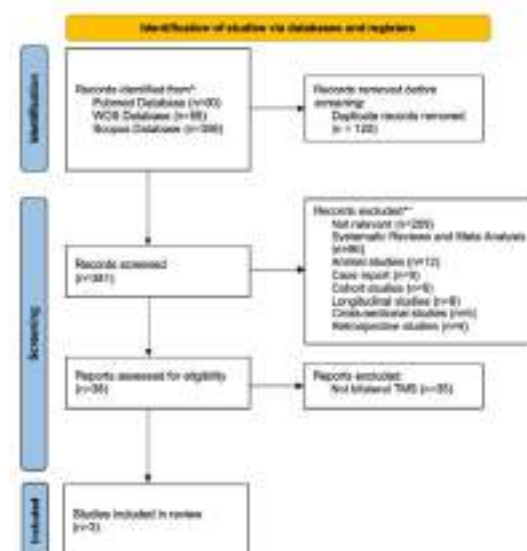


FIGURE 1 Flow Diagram.

Figure 2a: Risk of bias assessment (intention-to-treat analysis)



Figure 2b: Risk of bias summary for individual studies using the Cochrane Risk of Bias 2 tool



FIGURE 2a, 2b. RoB2 Assessment.

Figure 3a: Baseline and Post-Intervention Comparisons

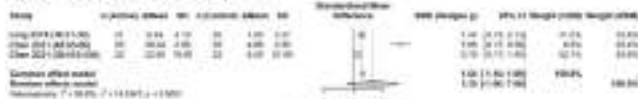


Figure 3b: Baseline and Follow-up Comparisons

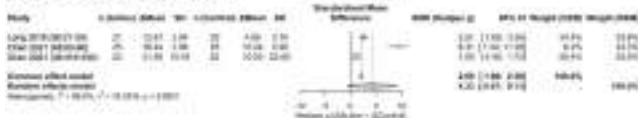


FIGURE 3a, 3b. Baseline and Post Intervention Comparisons.

EPO-0440 | Observational gait analysis tools for neurological populations: A COSMIN meta-analysis

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Background and aims: Observational gait analysis (OGA) tools are widely used due to their simplicity, low cost, and feasibility. However, the psychometric properties of OGA tools remain unclear. The aim was to conduct a systematic review and meta-analysis investigating psychometric properties of OGA tools among neurological populations.

Methods: Consensus-based Standards for the selection of health Measurement Instruments (COSMIN) guidelines were followed. COSMIN psychometric properties involve Validity (Content, Structural, Criterion, cross-cultural and Construct), Reliability (Internal Consistency, reliability and Measurement Error), and Responsiveness. The measurement Property for the will be rated as sufficient (+), insufficient (-), or indeterminate (?) using COSMIN criteria for good measurement properties. The recommendations for scales were mainly based on content validity and internal consistency.

Results: There were 26 included articles involving 8 OGA tools; Wisconsin Gait Scale (WGS), Gait Assessment and Intervention tool, Tinetti Gait Scale, Rivermead Visual Gait Assessment, Gait Abnormality Rating Scale, Malta Gait scale, Hemiplegic Gait Analysis Form, and New York Medical School Orthotic Gait Analysis. Included articles assessed through 64 studies all psychometric proprieties except structural validity. The 64 results were synthesized into 48 results in stroke, multiple sclerosis and Parkinson's disease. Thirty-one results rated as (+), 10 (-), and 8 (?). No OGA tool is currently recommended for use. All included OGA have potential for recommendation. WGS has the best potential.

Conclusion: No OGA tool is currently recommended due to absence of evidence for unidimensionality. Structural validity studies are highly required and should be prioritized to confirm unidimensionality and level of recommendations for OGA tools.

Disclosure: Nothing to disclose.

EPO-0441 | Putting family before patient: Physicians' reasons for prolonging life-sustaining treatment in chronic disorders of consciousness

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Background and aims: The Netherlands report the lowest prevalence of prolonged disorders of consciousness (PDOC), often related to an ethical-legal context allowing the discontinuation of life-sustaining treatment (LST) when recovery is unlikely or presumed patient consent is absent. Despite this, some PDOC patients receive LST for decades. This study explores physicians' reasons for doing this.

Methods: Semi-structured interviews were conducted with physicians responsible for patients diagnosed with unresponsive wakefulness syndrome, based on repeated Coma Recovery Scale-Revised assessments. Interviews, conducted at a mean of 14 years post-injury, began with the question: "Which factors influenced your treatment decision?" Transcripts were analysed using qualitative thematic analysis supported by ATLAS.ti software.

Results: Ten elderly care physicians in nursing homes, and responsible for nine patients were interviewed. In all cases, family requests to continue treatment overruled diagnosis, prognosis, and the patient's presumed perspective. Physicians prioritized maintaining family relationships and avoided conflict, without consulting external experts. Advance care planning was often delayed, even though physicians rated the patients' quality of life as very poor (mean 1.6/10). None of the physicians would want similar treatment for themselves. When asked about their preferred treatment for the patient if they had no family, all stated they would discontinue life-sustaining treatment.

Conclusion: Even in the unique Dutch medico-ethical context, life-sustaining treatment in PDOC can continue for decades. Family preferences largely dictate these decisions, while patient

interests are often overlooked. This highlights an ethical tension in PDOC care, emphasizing the need for clear role definitions, advance care planning, and better support for physicians facing moral distress.

Disclosure: Nothing to disclose.

EPO-0442 | Introducing Disorders of Consciousness Program Accreditation Standards

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Background and aims: Despite evidence and published guidelines demonstrating the need for specialized DoC rehabilitation, barriers to accessing these skilled programs remain. Recent evidence has provided guidance on the care for those with DoC, minimal competency recommendations for interdisciplinary teams providing care for persons with DoC, timing of withdrawal of life saving treatment, recovery and functional outcomes, and presence of consciousness in those with covert awareness. CARF International has worked with the field to create standards for transdisciplinary DoC rehabilitation programs which will focus on quality and performance improvement, advocate for individuals with DoC their family members and demonstrate the value of rehabilitation to all stakeholders including external payers and referrers.

Methods: referrers. CARF International conducted interviews with individuals with lived experience, family members, researchers, providers and payers to determine the need to create DoC Program standards. An International Standards Advisory Committee used a consensus-driven process to create draft DoC program standards, which were shared in a public field review where 133 individuals and organizations provided feedback which was then incorporated into the final DoC program standards.

Results: In July, 2026, these 24 DoC program standards will be used to provide the first DoC program accreditation, using peer-surveyors and an on-site survey process.

Conclusion: DoC Program accreditation standards will be used to establish a minimum standard for DoC programs which will allow easier identification of DoC programs for family members seeking DoC rehabilitation and create an enduring process to assess the quality of DoC rehabilitation programs.

Disclosure: I am a consultant with CARF International.

EPO-0443 | Overground gait training with body-weight support in individuals with neurological disorders: A scoping review

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Background and aims: Neurological disorders are a major cause of disability and are frequently associated with gait impairments that reduce functional independence and quality of life. Overground body-weight supported gait training has emerged as a potential alternative to treadmill-based approaches, but its clinical application remains insufficiently explored.

Methods: A scoping review was conducted following Joanna Briggs Institute methodology and PRISMA-ScR guidelines. Searches were performed in PubMed, PEDro, CENTRAL, EBSCO, and Web of Science. Studies investigating overground gait training with body-weight support in neurological populations were included and critically appraised using JBI tools.

Results: Seven studies were included, involving individuals with stroke, cerebral palsy, spinal cord injury, and Parkinson's disease. Overground body-weight supported gait training was associated with improvements in gait independence, walking speed, endurance, gait symmetry, motor function, fall risk, and freezing of gait. Outcomes were comparable or superior to treadmill-based or conventional gait training.

Conclusion: Evidence on overground body-weight supported gait training in neurological populations is limited and heterogeneous. Although promising gait-related outcomes have been reported, methodological limitations prevent strong conclusions, highlighting the need for high-quality randomised controlled trials with standardised protocols.

Disclosure: Nothing to disclose

Movement disorders 4

EPO-0444 | Abstract withdrawn

EPO-0445 | Greater clinical benefit with tiomolibdate choline versus standard-of-care in neurologic WD patients in the Phase 3 FoCUS Trial

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Background and aims: Wilson disease (WD) is a rare genetic disorder where excess copper (Cu) accumulation causes progressive neurologic disability. Tiomolibdate choline (TMC; ALXN1840) is an oral, once-daily, high affinity Cu-binding agent. This analysis evaluates outcomes in neurologic WD patients, a population at greater risk of symptomatic worsening on current WD therapy than asymptomatic patients.

Methods: In the open-label Phase 3 FoCUS trial (NCT03403205), 207 WD patients were randomized 2:1 to TMC or SoC for

48 weeks (W), with an optional TMC extension phase. Neurologic and clinical outcomes were analysed at 48W (primary) and 96W (extension) for patients with neurologic symptoms at baseline, defined as Unified WD Rating Scale [UWDRS] Part III score exceeding the minimum clinically important difference [MCID] of 4.668.

Results: Neurologic WD patients treated with TMC ($n=77$) demonstrated increasing clinical benefit over time, with statistically significant improvements in UWDRS Part III observed at 48W and continuing through 96W (mean change: -7.2 ; $p<0.001$). Benefit of TMC over SoC was evident by 48W, with significant UWDRS Part III improvement in the TMC arm ($p=0.002$) but not the SoC arm ($p=0.235$). Additionally, fewer TMC patients worsened by $\geq$ MCID at 48W vs. SoC (9% vs 25%). Disease improvement per Clinical Global Impression-Improvement (CGI-I) scale was statistically significantly greater for TMC vs. SoC at 48W ($p=0.003$).

Conclusion: In neurologic WD patients, 48 weeks of TMC treatment led to greater clinical benefit than SoC, with continued improvement through 96 weeks. These findings support the potential for TMC to improve neurologic outcomes in this at-risk population.

Disclosure: ML has received a travel grant from Monopar Therapeutics. CR, AC, and DT are employees of Monopar Therapeutics. AP, JB, DB, PD, IM, and TL have nothing to disclose.

EPO-0446 | Abstract withdrawn

EPO-0447 | Clinical and kinematic assessment of the “Head snap” sign in patients with essential and dystonic tremor

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Background and aims: The head snap sign (HSS) is a brief, involuntary jerk of the head observed during the finger-to-nose maneuver in patients with tremor [1], [2]. Originally described in essential tremor (ET), its prevalence and clinical significance across different tremor syndromes, particularly dystonic tremor (DT), remain unclear. We aimed to assess the prevalence and reliability of HSS in ET and DT and to explore its clinical and kinematic correlates.

Methods: Seventy-five patients (55 ET, 20 DT) underwent standardized video examination and optoelectronic kinematic recordings. Five blinded raters evaluated the presence of HSS and dystonic features. Inter-rater reliability was assessed using Krippendorff's alpha. Tremor severity and cognition were assessed with standardized scales. Associations with diagnosis and dystonia were tested using Fisher's exact test. Clinical and kinematic variables were then compared between HSS-positive (HSS+) and HSS-negative (HSS-) patients.

Results: HSS was identified in 20/75 (20.0% ET, 45.0% DT). HSS was more frequent in DT than in ET (OR = 3.21, $p=0.04$)

and was significantly associated with head dystonia (OR = 3.91, $p=0.03$). HSS+ patients exhibited lower cognitive performance ($p=0.01$). Kinematic analysis revealed a more severe tremor phenotype in HSS+ patients, with higher postural, kinetic, and head tremor amplitude (all $p<0.02$).

Conclusion: HSS is more prevalent in dystonic tremor and strongly associated with head dystonia, supporting a dystonia-related origin. Although also observed in ET, HSS emerges mainly in more advanced and severe tremor phenotypes, representing a potential clinical marker of dystonic involvement within the tremor spectrum.

Disclosure: Nothing to disclose.

EPO-0448 | Exploring the phenotypic features of a large hereditary ataxia cohort: Insights from a single-center experience

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Background and aims: Hereditary cerebellar ataxias (HCAs) are rare, genetically heterogeneous neurodegenerative disorders causing progressive cerebellar and multisystem involvement. Their diagnosis is challenging, particularly in populations with high consanguinity, such as Türkiye, where autosomal recessive (AR) forms are more prevalent. The aim of this study was to investigate the phenotypic features of hereditary ataxias in a large Turkish cohort.

Methods: A total of 481 individuals (207 index cases and 274 unaffected family members) were recruited from a tertiary referral center over 15 years. Clinical evaluation, pedigree analysis, and targeted genetic testing or whole-exome sequencing were performed. Variants were classified according to ACMG guidelines, and segregation analyses were completed when possible.

Results: Pathogenic variants were identified in 50 patients (24%). Of these, 83% displayed AR inheritance, reflecting the high prevalence of consanguinity. The most frequent disorder was autosomal recessive spastic ataxia of Charlevoix-Saguenay (ARSACS; 14%), followed by Friedreich's ataxia (6%), ANO10-related SCAR10 (8%), and CANVAS due to RFC1 expansions (8%). Less frequent variants included mutations in ATM, APTX, SETX, SYNE1, NPC1, and others. Overall, 14% of patients had late-onset disease, and several novel variants were documented.

Conclusion: This single-center study represents one of the largest hereditary ataxia cohorts reported in Türkiye. The predominance of AR inheritance highlights the impact of consanguinity on the genetic landscape. Recognition of specific phenotypic patterns, particularly treatable forms like CTX, is critical for timely diagnosis and therapeutic intervention. Advances in sequencing and emerging gene-targeted therapies emphasize the

TABLE 1 Molecular analysis results of the patients.

Gene	Inheritance pattern	Disease	Number of patients (%)
SACS	AR	AARSACS	7 (14%)
ATM	AR	Ataxia telangiectasia	4 (8%)
ANO8	AR	SCAR10	4 (8%)
BIC1	AR	CANVAS	4 (8%)
APEX	AR	Ataxia with oculomotor apraxia type 1	2 (4%)
BETX	AR	Ataxia with oculomotor apraxia type 2	2 (4%)
FXN	AR	Friedreich ataxia	2 (4%)
SYNE1	AR	SCAR8	2 (4%)
RPE1	AR	Pisumum Pisk type C	2 (4%)
ATP10	X linked	SCA3	2 (4%)
CLCN2	AR	Leukoencephalopathy with ataxia	2 (4%)
COX6A	AR	Coxsack Q10 deficiency with ataxia	2 (4%)
KCNK3	AD	SCA13	2 (4%)
SPTBN2	AR	SCAR14	2 (4%)
CYP27A1	AR	Cerebrotendinous xanthomatosis	1 (2%)
FGF14	AD	SCA23B, late onset	1 (2%)
AP5B2	AD	SCA28	1 (2%)
PREGG	AD	SCA14	1 (2%)
MMB	AD	SCA43	1 (2%)
WDR56	AD	SCA36	1 (2%)

importance of accurate molecular diagnosis to improve patient outcomes.

Disclosure: Nothing to disclose.

EPO-0449 | Sex differences in non-motor symptoms and quality of life in Parkinson's disease: A systematic review and meta-analysis

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Background and aims: Observational studies suggests that there are sex-specific symptom profiles in Parkinson's Disease (PD), with potential differences in perceived quality of life (QoL). **Methods:** PubMed, Embase, and Cochrane were systematically searched to identify sex-related differences in the Non-Motor Symptoms Scale (NMSS) and Parkinson's Disease Questionnaire-39 (PDQ-39). Statistical heterogeneity was evaluated with I2 statistics. A random-effects model was employed for outcomes with substantial heterogeneity. **Results:** Seven reports were included (n=2,968; 1,733 men, 1,235 women). The total NMSS Mean Difference (MD) 5.00 (95% CI 2.26–7.73), p=0.0003, I2=19% shows a greater overall non-motor symptom burden in women. Similarly, the PDQ-39

Sex Differences in Non-Motor Symptoms and Quality of Life in Parkinson's Disease: A Systematic Review and Meta-Analysis

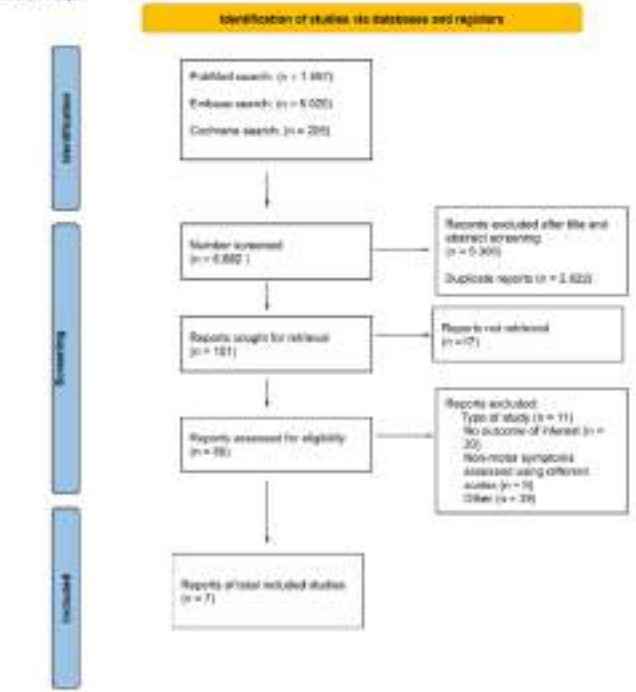


Figure 1. PRISMA flow diagram of study screening and selection

FIGURE 1 PRISMA - "Sex Differences in Non-Motor Symptoms and Quality of Life in Parkinson's Disease: A Systematic Review and Meta-Analysis".

TABLE 1 NMSS total forest plot.

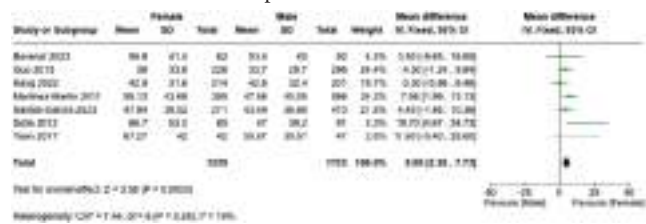
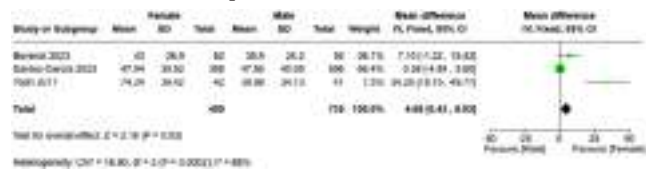


TABLE 2 PDQ 39 forest plot.



MD 4.68 (95% CI 0.43 to 8.93), p=0.03, I2=88% shows a worse QoL in women. Regarding the NMSS domains, women have worse scores in the urinary domain MD 0.55 (95% CI 0.17–0.92), p=0.004, I2=65%, attention MD 0.27 (95% CI 0.02–0.51), p=0.03, I2=15%, sleep/fatigue MD 1.38 (95% CI 0.70–2.07), p<0.00001, I2=93%. **Conclusion:** Women with PD exhibit a higher non-motor symptom burden and worse QoL, particularly in sleep/fatigue and mood/cognition domains. In contrast, men tend to have higher scores in the cardiovascular domain. However, substantial

heterogeneity was observed in some analyses, indicating that the strength of these findings varies across domains.

Disclosure: Nothing to disclose.

EPO-0450 | Dynamic STN beta oscillations and fronto-subcortical connectivity track state anxiety in Parkinson's disease

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Background and aims: Anxiety in Parkinson's disease (PD) involves altered autonomic regulation and cortico-subcortical network dynamics. Mechanisms underlying state anxiety remain unclear.

Methods: We examined the effects of experimentally induced state anxiety and recovery via mindfulness/deep breathing on autonomic function, STN and cortical oscillations, and STN-cortical connectivity in PD, and their interaction with DBS. Thirteen PD patients with STN-DBS completed a protocol including stress induction and deep breathing/mindfulness. STN local field potentials, EEG, and ECG were recorded with DBS ON and OFF (ON medication). Oscillatory power (theta, alpha, beta), STN-cortical connectivity, and heart rate variability (HRV) were analysed using repeated-measures ANOVAs.

Results: Stress increased state anxiety ($F(1.22)=22.24$, $p<0.001$), which decreased after deep breathing ($p<0.001$), with no stimulation effect. State anxiety correlated with trait anxiety ON ($\rho=0.678$, $p=0.016$) and OFF DBS ($\rho=0.820$, $p=0.001$). STN beta power showed a stimulation $\times$ condition interaction ($F(2)=5.0$, $p=0.04$): reduced beta during deep breathing vs rest ($p=0.02$) and stress ($p=0.02$) with DBS ON, and DBS-related beta suppression during rest ($p=0.03$) and deep breathing ($p=0.01$). No theta or alpha effects were found. Root Mean Square of Successive Differences (RMSSD), a key HRV metric, showed a condition effect ($F(2)=5.5$, $p=0.01$). Stress-related anxiety changes correlated negatively with RMSSD ($\rho=-0.69$, $p=0.02$) and STN beta power ($\rho=-0.91$, $p<0.001$) with DBS ON. Recovery was associated with increased right STN-frontal beta connectivity ($\rho=-0.681$, $p=0.03$).

Conclusion: State anxiety in PD is associated with dynamic modulation of STN beta oscillations and STN-frontal connectivity. DBS enhances coupling between subjective anxiety, autonomic regulation, and STN beta activity.

Disclosure: Nothing to disclose.

EPO-0451 | Multimodal mixed-reality screening for Parkinson's disease: Accuracy and key features

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Background and aims: Clinical evaluation of PD is often time-consuming and partially subjective. Objective, sensor-based

screening tools may enable scalable triage and longitudinal monitoring. The objective was to benchmark multimodal machine-learning models for Parkinson's disease (PD) detection during a mixed-reality assessment and to identify the tasks and sensor modalities most critical to diagnostic performance.

Methods: We analyzed 137 participants (66 with PD, 71 healthy controls) who performed 17 guided tasks with synchronized speech and motion capture. A total of 4,850 engineered features per participant were extracted. Data were split into training (60%) and testing (40%) sets. Missingness was substantial and group-dependent (51.8% controls; 27.6% PD). Imputation methods were compared using masked-value reconstruction RMSE; AttentionVAE achieved the lowest error (0.9481), outperforming MICE (0.9540) and mean/median imputation (1.0172/1.0424).

Results: Representation-learning approaches achieved strong classification performance. CrossModalAAVE combined with random forest reached a mean accuracy of 0.915 ± 0.060 (AUC 0.985 ± 0.019 ; F1 0.917 ± 0.059), while CrossTaskAAVE with gradient boosting achieved 0.901 ± 0.084 (AUC 0.982 ± 0.018 ; F1 0.904 ± 0.074). Speech features dominated (87.2% of informative variables). Removing head-movement features reduced accuracy from 0.873 to 0.691 and excluding both acoustic and head-movement modalities reduced accuracy to ~58%. Task-level ablations identified picture/story description and sustained vowel/a/ as most informative.

Conclusion: Mixed-reality multimodal assessment enables accurate PD screening and informs task prioritization to reduce patient burden. Larger external validation is required to establish generalizability and clinical utility.

Disclosure: Supported by the National Centre for Research and Development, Poland (LIDER/6/0049/L-12/20/NCBIR/2021).

EPO-0452 | When personality and polygenic risk scores interact to predict impulse control disorders in Parkinson's disease

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Background and aims: In Parkinson's disease (PD), patients may develop impulse control disorders (ICDs), as adverse effect of dopamine agonists. Some factors have already been associated with ICDs such as genetic polymorphisms (Cormier-Dequaire et al. 2018), as well as personality (Novelty Seeking/Self-Directedness dimensions) related to impulsivity in PD (Boussac et al. 2022; Meira et al. 2022). We aim to evaluate interactions effects between personality and polygenetic risk scores (PRS) on the risk of ICDs.

Methods: PD patients with ($n=139$) and without ($n=111$) ICDs were included in the BADGE-PD study (NCT02319395).

Personality dimensions were assessed using the Temperament and Character Inventory-Revised (TCI-R) (Cloninger et al. 1994), while 18 PRS associated with psychiatric or behavioral phenotypes were calculated. Logistic regression models were done to evaluate main and interaction effects between TCI-R dimensions and PRS on ICD risk by calculating odds ratios (OR).

Results: Main significant effects were found for the Self-Directedness personality dimension, for the attention deficit hyperactivity disorder (ADHD, PGS003753) and the major depressive disorder (MDD, PGS000907) PRS: higher scores being associated with higher risk to develop ICDs (ORs between 1.31 and 1.54). Moreover, some personality*PRS interactions significantly modulated this risk either amplifying or attenuating it. Highest ICD risk was found from the interaction between Self-Directedness and MDD PRS (global OR of 2.78) corresponding to a 278% increased risk of ICD for each increase of one standard deviation in both variables.

Conclusion: This study highlights some important interactions between genetic and personality to consider in personalized risk assessment for ICDs in PD.

Disclosure: I declare no conflict of interest for this study. This study was funded by a grant of the French Ministry of Health (PHRC AOM 1008), with the support of the Mutuelles AXA health sponsorship program for the ancillary part of this study.

EPO-0453 | A Bayesian reappraisal of disease-modifying drug candidates for Parkinson's disease

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Background and aims: Parkinson's disease (PD) remains without a confirmed disease-modifying therapy (DMT). Numerous clinical trials have investigated candidate compounds, yet conclusions often rely on frequentist analyses that may obscure the strength or weakness of the evidence.

Methods: We performed a Bayesian reappraisal of major DMT trials—covering cinpanemab, deferiprone, CoQ10, amroxol, NLY01, liraglutide, lixisenatide, and exenatide—focusing on motor and non-motor outcomes, with standardized effect sizes, Bayes Factors (BF₁₀), and posterior probabilities.

Results: Deferiprone (FAIRPARK-II) showed extreme evidence for a detrimental effect on 36-week MDS-UPDRS total scores (BF₁₀ = 5.03 × 10⁶; P(H₁) = 0.993). Liraglutide demonstrated strong evidence for improvement in Non-Motor Symptoms Scale scores (BF₁₀ = 41.7; P(H₁) = 0.977). Lixisenatide showed moderate evidence for a small motor improvement on MDS-UPDRS-III at 12 months (BF₁₀ = 4.75; P(H₁) = 0.826). CoQ10, cinpanemab, amroxol, NLY01, and exenatide (2025) all showed BF₁₀ < 1, indicating no support for efficacy.

Conclusion: Bayesian analysis aligns with the strongest frequentist signals while clarifying ambiguous or inconsistent results, underscoring that the magnitude of the effect and

evidential strength vary substantially across compounds. GLP-1R agonists show promising efficacy as DMT candidates for PD treatment. Pharmacokinetic and pharmacodynamic differences among the compounds could underlie the different efficacy profiles concerning the disease stage and different symptoms (i.e., motor or non-motor).

Disclosure: Nothing to disclose.

EPO-0454 | The longitudinal Progression of Gait Symptoms in Early Parkinson's Disease: A 7-year study from the PPMI dataset

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Background and aims: Gait disturbances are cardinal symptoms of Parkinson's disease (PD). Existing studies in early PD focused on aggregate scores, lacking sensitivity to sub-item level changes. Correlations between gait and cognitive impairment were demonstrated but findings remain inconsistent. This study longitudinally tracked the progression of early gait disturbances along with cognitive impairment to understand their impact on Activities of Daily Living (ADL) over 7-years.

Methods: Data was obtained from 150 PD participants in the Parkinson's Progression Markers Initiative (PPMI) database. Motor symptoms, cognition and ADL were measured using the Movement Disorder Society- Unified Parkinson's Disease Rating Scale (MDS-UPDRS), the Montreal Cognitive Assessment (MoCA) and the Modified-Schwab England (ADL- SE) scale respectively from PPMI annual visits.

Results: Patient reported outcomes (PROs) identified walking and balance dysfunction in 32% of the cohort at baseline, 53% by year 3 and 69% at year 7. Whereas, clinicians identified gait disturbances in 55% at baseline, 60% at year 3 and 74% at year 7 with a significant impact on ADLs. Freezing of gait (FOG) presented between year 4 (3%) and 5 (5%) in clinician scores, but 5% of patients already reported FOG at baseline that increased to 17% by year 5 and 35% at year 7 on PROs. At year 7, FOG, gait, and postural stability scores correlated negatively with MoCA scores ($p < 0.012$).

Conclusion: This study shows that gait disturbances are widely prevalent in early PD with a fast rate of progression, highlighting the importance of appropriate physiotherapy and management early in the disease course.

Disclosure: Nothing to disclose.

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Background and aims: Adult-onset genetic movement disorders can present with autonomic failure alongside parkinsonism, ataxia, and other movement disorders. Autonomic dysfunction, a frequent cause of misdiagnosis of multiple system atrophy (MSA), has not been systematically evaluated across these disorders. This study aimed to comprehensively characterize dysautonomia in adult-onset genetic movement disorders.

Methods: A systematic PubMed search using predefined terms identified original English-language research articles. Of 313 papers retrieved on September 8, 2025, 82 were included (61 original studies, 5 case series, and 16 case reports). Exclusions comprised 7 review articles, 12 chapters, 206 unrelated publications, and 6 non-English papers. Extracted data included study population, age at onset, inheritance pattern and genetic mutation, presence of control groups, clinical phenotype, autonomic assessment methodology, and key findings related to autonomic dysfunction. Results were summarized in a table of evidence.

Results: Thirty-four adult-onset genetic movement disorders caused by mutations in 52 genes were identified as manifesting autonomic failure in association with ataxia, parkinsonism, dystonia, spastic paraplegia, chorea, or complex phenotypes. Several conditions—including spinocerebellar ataxias, hereditary spastic paraplegias, certain genetic parkinsonian syndromes, fragile X tremor–ataxia syndrome, LMNB1-related autosomal dominant leukodystrophy, and RFC1-related disease—emerge as MSA mimics. The phenotype and severity of dysautonomia varied considerably across disorders.

Conclusion: Many adult-onset genetic movement disorders can closely resemble MSA, creating significant diagnostic challenges. Autonomic dysfunction is an important but often overlooked feature in these conditions and should be considered in the differential diagnosis of MSA. Comprehensive clinical history, autonomic testing, and targeted genetic testing in selected cases may improve diagnostic accuracy and reduce misdiagnosis.

Disclosure: Nothing to disclose.

EPO-0456 | Epigenetic modifications of cerebrospinal fluid cell-free DNA as a marker of neuronal damage

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Background and aims: Degenerative processes during Multiple Sclerosis (MS) progression are scarcely captured by currently available biomarkers. Cell-free DNA (cfDNA) are fragments of DNA shed into biological fluids by dying cells and retain the cell-of-origin DNA methylation. Our aim is to utilize DNA methylation signatures in cfDNA from cerebrospinal fluid (CSF) to detect ongoing neuronal death in MS and other neurological diseases.

Methods: We profiled bulk methylation (mC) using enzymatic methylation sequencing (EMSeq) and/or true methylation (5mC) and hydroxymethylation (5hmC) using 6-base sequencing in cfDNA extracted from CSF of healthy controls and people with MS, amyotrophic lateral sclerosis (ALS), narcolepsy type 1, movement disorders, or traumatic brain injury (TBI).

Results: While cfDNA extraction from CSF yielded a low amount of DNA for all but TBI samples, EMSeq and 6-base sequencing successfully generated sequencing libraries, although with low genome coverage. To overcome the latter, we aggregated EMSeq reads and calculated methylation scores for neuronal or glial damage. TBI samples showed significantly higher scores than HC, followed by increased scores in ALS, suggesting more extensive neural cell death in these conditions. To increase sensitivity for neuronal damage, we quantified the neuron-enriched hmC with 6-base sequencing, and we confirmed higher neuronal DNA content in CSF cfDNA from TBI. Clustering of samples based on regions enriched for hmC successfully separated HC from neurological conditions, including MS.

Conclusion: Analysis of DNA methylation modifications of cfDNA has the capacity of capturing a neuronal signature in cell-free CSF. Further studies are needed to implement cfDNA epigenetic analysis as real-time biomarker of neurodegeneration.

Disclosure: CN, AS, LS, TC and FPuddu are current employees of biomodal Limited and hold share options. KF has received research grants from UCB, Merck and AMICUS, all outside the submitted work. PS has received lecture honoraria grants from AbbVie, MERZ, AstraZeneca, and fees for serving on DMC in clinical trials with Lundbeck. CI has consulted for Cytokinetics, Pfizer, BioArctic, Novartis, Tikomed, Ferrer, Amylyx, Prilenia,

Mitsubishi and Abbvie. She is also a board member of Tobii Dynavox and of the Stichting TRICALS Foundation; all outside the submitted work. F Piehl has received research grants from Denka, Janssen, Merck KGaA, Novartis, Pfizer and UCB, and fees for serving on DMC in clinical trials with Lundbeck and Roche, and preparation of expert witness statement for Novartis. CSC, CRP, YH, MK, BE, CS, FF, EPT, LK, MN and MJ have nothing to disclose.

EPO-0457 | Smelling and tasting neurodegeneration: Chemosensory dysfunction and retinal changes in multiple sclerosis: A pilot study

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Background and aims: To characterize gustatory and olfactory function in relapsing-remitting multiple sclerosis (RRMS) and explore associations with clinical variables and optical coherence tomography (OCT) retinal measures.

Methods: Single-centre cross-sectional pilot study (Cagliari, Italy; February to April 2025), 78 adults with RRMS completed Taste Strips (Total Taste score 0–16; hypogeusia defined as less than 9). A subgroup ($n=60$) completed Sniffin Sticks battery (Threshold, Discrimination, Identification; TDI score 1–48). Spectral-domain OCT measured peripapillary retinal nerve fibre layer (pRNFL) and ganglion cell-inner plexiform layer (GCIPL) thickness in eyes without prior optic neuritis. Correlations analyses and multivariable linear regression assessed associations between chemosensory measures, clinical variables and pRNFL thickness.

Results: Mean Total Taste score was 11.5 (standard deviation 2.85) and 13 (16.7%) had hypogeusia; sour required higher concentrations for correct identification than sweet, salty and bitter ($p<0.05$). Higher body mass index was associated with lower taste scores ($r=-0.30$; $p=0.033$) and smoking with hypogeusia ($p=0.023$); taste performance was not associated with Expanded Disability Status Scale (EDSS), disease duration, pRNFL or GCIPL. Mean TDI was 29.5 (3.38) and 23 (38%) were hyposmic. TDI correlated with disease duration ($r=-0.56$; $p<0.001$) and pRNFL thickness ($r=0.28$; $p=0.038$). In multivariable models for pRNFL thickness, only disease duration remained independently associated ($p=0.029$).

Conclusion: Chemosensory dysfunction is common in RRMS and should be recognized clinically. Taste impairment appears mainly linked to modifiable factors (BMI, smoking), whereas olfactory performance reflects cumulative disease burden; the olfaction-pRNFL association was not independent of disease duration. Larger longitudinal, multimodal studies including magnetic resonance imaging and cognition are needed.

Disclosure: Nothing to disclose.

EPO-0458 | Enhancing clinical assessment in multiple sclerosis: The role of patient-reported outcomes and the PDDS scale

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Background and aims: The multiple sclerosis (MS) management increasingly integrates traditional clinical measures with Patient-Reported Outcomes (PROs) to capture dimensions like fatigue and psychological well-being often missed by physician-led scales. Aim of the Study: To evaluate the efficacy of the “Patient Determined Disease Steps” PDDS as a simplified self-assessment tool, equivalent to the Expanded Disability Status Scale (EDSS), for monitoring functional status.

Methods: MS patients were assessed using PDDS and various PROs. Clinical and demographic data have been collected for each participant. Agreement between PDDS and EDSS was evaluated via quadratically weighted Cohen's kappa (κ) and Spearman's rho. Potential effect modifiers (demographics, clinical data, and PROs) were analyzed to assess stability of the agreement.

Results: 221 participants (71.9% female; mean age 48.4) were enrolled, predominantly RRMS (94.6%). Median EDSS and PDDS were 2.0 and 1.0, respectively. High prevalence of depression (38.5%), fatigue (37.6%), and anxiety (26.7%) was observed. A substantial agreement was found between EDSS and PDDS ($\kappa=0.84$, 95% CI:0.783–0.879; $\rho=0.73$; $p<0.001$). Subgroup analysis revealed higher concordance in RRMS ($\kappa=0.831$) than in progressive form ($\kappa=0.600$; $\Delta\kappa=0.231$, $p<0.05$). No other factors significantly influenced the agreement.

Conclusion: The robust agreement between EDSS and PDDS confirms its validity as an accessible tool for disability monitoring. Its stability across most variables supports PDDS use in remote monitoring and research. The lower concordance in progressive forms suggests differing disability perceptions in advanced stages. Integrating PDDS into clinical follow-up promotes a more personalized, patient-centered approach to MS care.

Disclosure: Nothing to disclose.

EPO-0459 | Sensor-based gait and balance testing can detect subclinical sensorimotor dysfunction in radiologically isolated syndrome

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Background and aims: Radiologically isolated syndrome (RIS) presents with MRI features suggestive of multiple sclerosis (MS) without overt clinical signs or symptoms. We hypothesized

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Background and aims: Multiple Sclerosis (MS) is an autoimmune demyelinating disease of the CNS for which current high-efficacy disease-modifying therapies often cause severe immunosuppression. Dysregulated Ca^{2+} -calcineurin-NFAT signalling, driven by overactivated Store-Operated Ca^{2+} entry (SOCE), promotes immune activation and pro-inflammatory cytokine release (Fig1). This study evaluates SOCE as a therapeutic target using CIC-39, a novel SOCE inhibitor in preclinical development, to restore immune homeostasis. Targeting SOCE may provide a safer, immune-preserving strategy for MS, reducing immunosuppressive side effects (Fig2).

Methods: Peripheral blood mononuclear cells from MS patients and healthy controls were collected (Ethical Committee CE152/2024). CD4^{+} T cells were isolated, activated, and differentiated. CIC-39 (1–3 μM) was tested in vitro for SOCE, proliferation, and cytokine release. In vivo, EAE mice were treated with CIC-39 60 mg/kg via minipumps for 15 days (prot. 503/2024-PR).

Results: SOCE was overactivated in PBMCs from MS patients, and CIC-39 effectively restored Ca^{2+} homeostasis. Unlike the immunosuppressant teriflunomide, CIC-39 did not impair CD4^{+} T-cell proliferation but significantly reduced Th1/Th17-associated cytokines (IL-17A, TNF- α , IFN- γ , GM-CSF) while preserving iTreg differentiation and regulatory function. In vitro and preliminary EAE studies demonstrated that CIC-39 attenuated pro-inflammatory responses, decreased circulating cytokines, and

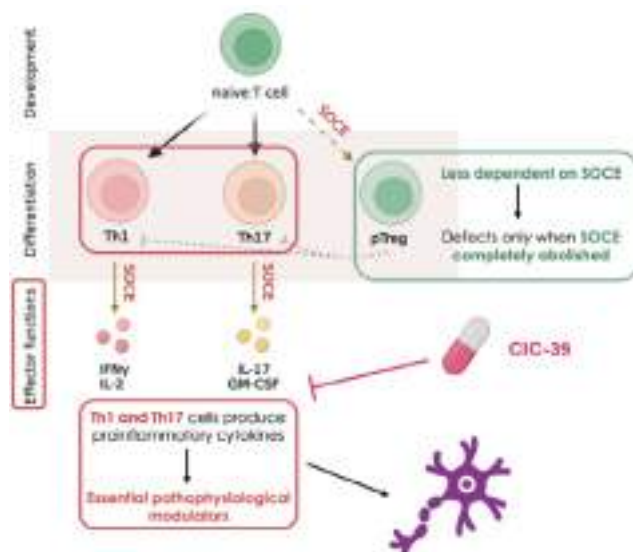


FIGURE 2 Role of SOCE in Th1 and Th17 pro-inflammatory cytokine release.

effectively arrested disease progression, highlighting its potential as an immune-preserving therapeutic strategy for MS.

Conclusion: Our findings demonstrate that CIC-39 selectively modulates overactive SOCE in MS, restoring physiological Ca^{2+} levels and preserving lymphocyte proliferation. It reduces Th1/Th17 cytokines, and preliminary studies in EAE mice confirmed rapid disease arrest with decreased IFN- γ and IL-17A, supporting CIC-39 as a promising safer and immune-preserving disease-modifying therapy for MS.

Disclosure: Beatrice Riva owns stocks of ChemiCare srl. Luigi Azzarone and Carlotta Muschitiello are at present employees of ChemiCare srl.

EPO-0462 | Preliminary results from the brain-age MS study: NAWM CEST and T1 show no association with paramagnetic rim lesion count

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Background and aims: Paramagnetic rim lesions (PRLs) are established imaging markers of chronic active multiple sclerosis (MS), reflecting focal inflammatory pathology. While PRLs are well characterised at the lesion level, it remains unclear whether PRL-count is associated with diffuse alterations in normal-appearing white matter (NAWM). Chemical exchange saturation transfer (CEST) imaging is sensitive to metabolic tissue properties beyond conventional MRI. In this preliminary

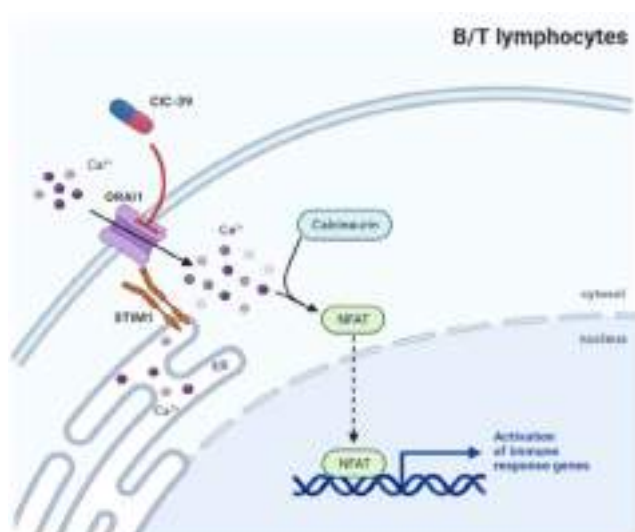


FIGURE 1 SOCE-Calcineurin-NFAT pathway in B and T lymphocytes.

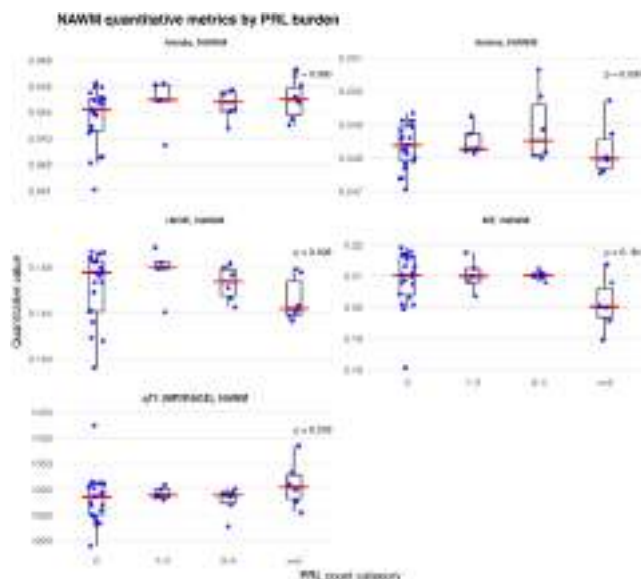


FIGURE 1 NAWM CEST metrics and quantitative T1 stratified by PRL count. Boxplots show distributions across PRL categories with overlaid individual values; red lines indicate medians. *p*-values derive from linear regression trend tests between PRL count and NAWM met.

analysis of the brain-age MS study (NCT06221631), we investigated whether PRL-count is associated with CEST-metrics and T1 relaxation in clinically stable MS.

Methods: Clinically stable MS patients underwent 7T MRI including CEST imaging (amide, amine, rNOE, MT) and MP2RAGE-based T1 mapping. PRLs were identified on susceptibility-weighted imaging, and PRL-count was determined per subject. NAWM regions of interest were conservatively defined by excluding T2-FLAIR-visible lesions. Linear associations between PRL-count and median NAWM quantitative metrics were assessed.

Results: No significant linear associations were observed between PRL-count and NAWM CEST or T1. Substantial inter-individual variability was present, but no consistent monotonic trends were observed with increasing PRL-count.

Conclusion: In this preliminary analysis of clinically stable MS patients from the brain-age MS study, PRL count was not associated with metabolically sensitive NAWM CEST metrics or quantitative T1. These findings suggest that PRL-related pathology remains spatially focal and is not reflected by diffuse NAWM alterations at this stage. Larger longitudinal studies are needed to determine whether NAWM metabolic changes emerge with disease progression.

Disclosure: Nothing to disclose.

EPO-0463 | OCT and OCT angiography in differential diagnosis of MS: The retina as a window on the CNS

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Background and aims: MS is an inflammatory demyelinating disease of the CNS characterized by immune-mediated perivenular inflammation, reflected by the perivenular distribution of white matter lesions (PVL) on MRI by the central vein sign (CVS). In contrast, several MS-like and atypical MS disorders share overlapping clinical and radiological features but show a lower prevalence of PVL, suggesting alternative pathogenic mechanisms in which vascular dysfunction may play a prominent role. Optical coherence tomography (OCT) and OCTangiography (OCTA) enable non-invasive assessment of retinal neuroaxonal integrity and microvascular perfusion, providing insights into CNS vascular involvement.

Methods: 56 patients underwent OCT-OCTA and 19 received 3T MRI with CVS assessment (CVS+ if PVL>50%). OCT measured retinal nerve fiber layer (RNFL) and ganglion cell complex (GCC) thickness, while OCTA assessed optic nerve head vessel density and superficial (SCP) and deep (DCP) capillary plexus density.

Results: Patients were classified as MS (*n*=34), atypical-MS (*n*=8), and not-MS (nMS;*n*=15), including other inflammatory, vascular, and neoplastic CNS disorders. Previous optic neuritis was associated with RNFL and GCC thinning, without significant changes in vessel density. MS showed significant temporal RNFL thinning compared with atypical-MS and nMS (*p*<0.001). Conversely, atypical-MS and not-MS exhibited a marked reduction in SCP and DCP density, in the absence of major structural retinal damage. In the CVS subgroup, CVS-eyes demonstrated significantly lower vessel density than CVS+eyes, with high concordance between CVS+ and SCP status.

Conclusion: Atypical-MS and not-MS are characterized by predominant retinal microvascular impairment with minimal neuroaxonal loss, supporting a primary vascular pathogenic

TABLE 1 Basal characteristics of the whole population and subgroups.

	Whole population (<i>n</i> =59)	MS (<i>n</i> =34)	Atypical-MS (<i>n</i> =8)	nMS (<i>n</i> =15)
Sex	44 (74.6%) F	28 (82.4%) F	5 (62.5%) F	11 (73.3%) F
Mean age at clinical onset (years, SD)	38.8 (11.4)	38.8 (10.8)	42 (10)	42.8 (10.8)
Mean disease duration (months, SD)	118 (157)	138 (167)	84.8 (35.4)	148 (175)
Previous ON	22 (37.3%)	17 (50%)	2 (25%)	3 (20%)

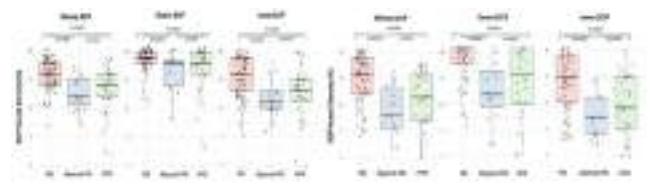


FIGURE 1 Superficial (SCP) and Deep (DCP) capillary plexus vessel density of different zones in subgroups MS, atypical-MS and not-MS.

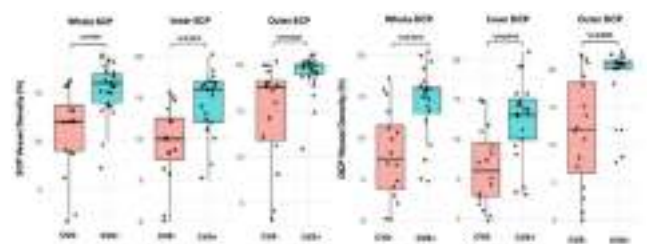


FIGURE 2 Superficial (SCP) and Deep (DCP) capillary plexus vessel density of different zones in CVS+ and CVS-.

contribution. OCT-OCTA may assist early differential diagnosis of MS-like syndromes and help identify non-demyelinating disease mechanisms.

Disclosure: Nothing to disclose.

EPO-0464 | Exploring the impact of multiple sclerosis on social determinants of health: Results from the SocialMS study

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Background and aims: Multiple Sclerosis (MS) can affect several social determinants of health (SDH), but research remains limited and fragmented, with no studies exploring multiple SDH simultaneously. We examined four SDH (education, work, financial resources and social life) to assess the impact of MS and interrelationships, to identify risk factors, and to study associations with social support.

Methods: SocialMS is a cross-sectional Italian questionnaire-based study including adults with MS. The number of SDH affected was the primary endpoint, while secondary outcomes included impact on at least one domain and on individual domains. Risk factors were identified using Poisson and logistic regression.

Results: We included 1,039 participants followed at 68 MS centers (mean age: 43.82years (standard deviation: 11.53); 68.82% females; 86.53% relapsing remitting, median Patient Determined Disease Steps (PDDS): 1.00 (interquartile range (IQR): 0.00–3.00)). Median number of SDH affected was 1 (IQR: 0–3), with social life (51%) and work (48%) most impacted; high interrelationships among domains were observed (Figure 1). Relevant risk factors included socioeconomic factors (education incomplete at diagnosis: incidence-rate ratio (IRR)=1.19, $p=0.004$; non-working or early retired: IRR=1.18, $p=0.007$; hardly making ends meet: IRR=1.47, $p<0.001$); comorbidities (IRR=1.23, $p<0.001$) and PDDS (IRR=1.17, $p<0.001$) (Table 1). Economic strain and disability were associated with all secondary outcomes ($p<0.05$). Almost 90% received tangible (64.29%) or intangible (85.18%) social support, and number of impacted SDH was positively associated with both (odds ratios=1.88 and 1.25, $p<0.05$, Figure 2).

Conclusion: MS substantially affects multiple SDH, with greater impact in individuals with socioeconomic and MS-related vulnerabilities.

Disclosure: MP has nothing to disclose, AS received speaker's honoraria from Chiesi, Novartis, and Horizon and grant from

MSBase outside from this work, DL received travel funding from Biogen, Merck Serono, Sanofi, Teva, Bristol Myers Squibb, Mylan, Neuraxapharm, speaking or consultation fees from Sanofi, Merck Serono, Teva, Biogen, Roche, Novartis, Bristol Myers Squibb, Janssen, Alexion, Amgen, AB reports consulting fees (Arboretum LifeSciences, Inc), payment or honoraria for lectures, presentations, speakers bureaus, manuscript writing or educational events (Columbia University and BiostatEpi) and participation on a data safety monitoring board or advisory board (MarvelBiome, Inc), IS has received personal compensation for speaking and consulting from Roche, Biogen, Hoya, Hippocrates Research, D.M.G. Italia, EyePharma, and DreamsLab. None of these relationships are related to the submitted work, LL received consultancy and speaker fees from Biogen, Novartis, Roche, Idorsia, Bristol, Lilly, Roche, Amgen, Alexion, Merck, and Sanofi for advisory services and participation in conferences, ADS reports grants or contracts from any entity (Roche, Sanofi), payment or honoraria for lectures, presentations, speakers bureaus, manuscript writing or educational

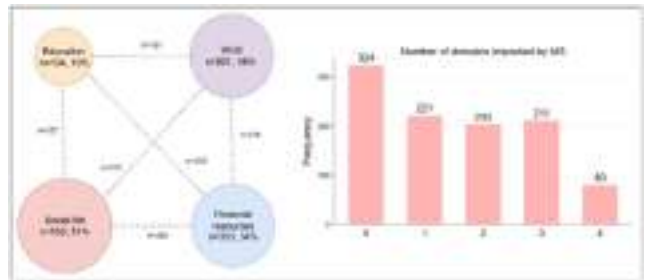


FIGURE 1 Impact of multiple sclerosis (MS) on social determinants of health (SDH). Left: Frequency of participants reporting impact of MS on the four domains. Right: Distribution of participants according to the number of domains impacted by MS (0–4).

TABLE 1 Poisson regression models examining the association of sociodemographic and multiple sclerosis (MS) characteristics with the number of SDH impacted by MS. Variables with $p<0.10$ in univariable models were included in the multivariable analysis.

	Univariable analysis		Multivariable analysis	
	IRR (95% CI)	p-value	IRR (95% CI)	p-value
Sociodemographic characteristics				
Age				
<35	1.00 (ref)	—	1.00 (ref)	—
[36; 45]	1.05 (0.82; 1.21)	0.673	1.01 (0.87; 1.18)	0.877
[46; 55]	1.10 (1.05; 1.16)	0.009	0.96 (0.91; 1.04)	0.675
≥56	1.16 (1.08; 1.24)	0.008	0.88 (0.72; 1.07)	0.187
Geographical area				
North	1.00 (ref)	—	1.00 (ref)	—
Center	1.17 (1.03; 1.31)	0.014	1.01 (0.89; 1.14)	0.921
South and islands	1.14 (1.01; 1.28)	0.028	1.02 (0.90; 1.15)	0.798
Female sex	1.04 (0.89; 1.15)	0.535	—	—
Non-foreign citizen	1.02 (0.79; 1.31)	0.885	—	—
Not Italian citizen since birth	1.17 (0.85; 1.70)	0.112	—	—
Level of education				
High school or lower	1.00 (ref)	—	1.00 (ref)	—
Bachelor's degree	0.99 (0.81; 1.11)	0.493	1.07 (0.91; 1.24)	0.457
Master's degree or higher	0.88 (0.75; 0.96)	0.012	1.07 (0.94; 1.22)	0.291
Education not completed at MS diagnosis	1.13 (1.07; 1.19)	0.016	1.29 (1.06; 1.57)	0.008
Occupational status				
Working or retired due to age	1.00 (ref)	—	1.00 (ref)	—
Unemployed, unemployed, or early retired	1.17 (1.15; 1.21)	<0.001	1.38 (1.09; 1.74)	0.007
Student	1.17 (1.01; 1.35)	0.038	1.10 (0.96; 1.41)	0.463
Making ends meet, considering total household income				
Easily	1.00 (ref)	—	1.00 (ref)	—
Hardly	1.06 (1.50; 1.88)	<0.001	1.47 (1.31; 1.64)	<0.001
Comorbidities	1.18 (1.13; 1.23)	<0.001	1.23 (1.11; 1.37)	<0.001
MS				
Phenotype, % (%)				
Relapsing remitting	1.00 (ref)	—	1.00 (ref)	—
Primary or secondary progressive	1.65 (1.46; 1.86)	<0.001	0.97 (0.83; 1.13)	0.663
Disease duration >35 years	1.12 (1.10; 1.14)	<0.001	0.99 (0.88; 1.11)	0.800
PDDS	1.18 (1.16; 1.20)	<0.001	1.17 (1.13; 1.20)	<0.001



FIGURE 2 Frequency of tangible and intangible social support according to the number of domains impacted by MS (0–4).

events (Novartis, Alnylam, Roche, Merck, Janssen, Bristol Myers Squibb, Horizon, Alexion), support for attending meetings and/or travel (Novartis, Merck, Roche, Sanofi, Biogen) and participation on a data safety monitoring board or advisory board (Sanofi, Merck, Roche, Alexion, Sandoz), RF received travel support, consultations fees and honoraria for advisory boards from Novartis, Merck-Serono, Biogen, Roche, Alexion, Neuraxpharm, FM has received personal compensation for consulting services and speaker activities from Merck Serono, Novartis, Roche, Biogen, BMS, MPS received consulting fees from Roche, Biogen, Merck, Novartis, Sanofi, Celgene, Immunic, Geneuro, GSK, Medday; received payment or honoraria for lectures, presentations, speakers' bureaus, manuscript writing or educational events from Roche, Biogen Merck, Novartis, Sanofi, Celgene; participated on a Data Safety Monitoring Board or Advisory Board for Roche, Sanofi, Novartis, Merck.

EPO-0465 | Microglial activation in MS brain contributes significantly to brain age gap between MS patients and controls

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Background and aims: Disease- and aging-related factors impair brain health in people with MS. Aging MS patients typically experience gradual disease progression without relapses due to glial activation causing neuroaxonal damage. Aging-, MS-CNS pathology- and autoimmunity-related factors may exacerbate detrimental glial activation and neuroaxonal damage in MS brain. We sought to investigate how aging associates with microglial activation and white matter damage and evaluate differences in brain age gap among healthy controls (HCs) and people with MS. **Methods:** 128 MS patients and 69 age- and sex-matched HCs underwent 18-kDa translocator protein (TSPO)-binding radioligand 11C-PK11195 positron emission tomography for assessment of glial activation using distribution volume ratio (DVR) and concomitant magnetic resonance imaging (MRI) including diffusion tensor imaging (DTI). Brain age gap was calculated as difference between chronological and biological age estimated using data on 1) MRI-measured atrophy, 2) atrophy and DTI and 3) the machine-learning tool Braineager.

Results: Normal-appearing white matter (NAWM) DVR values increased ($p=0.001$) and DTI metrics worsened ($p<0.01$ for all

measures) with increasing age among MS patients but not HCs. Brain age gap was significantly elevated in MS patients vs. HCs and this gap increased further when NAWM DVR was included in the model.

Conclusion: Higher age associated with increased glial activation among MS patients but not among HCs and co-occurred with loss of white matter structural integrity in the NAWM and increases in biological brain age in MS. Increased TSPO signal in aging MS patients likely represents increasing compartmentalized inflammation, which can be accelerated by general inflammation leading to premature brain neurodegeneration.

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TABLE 1 Clinical characteristics, MRI volumes, and TSPO Distribution Volume Ratios (DVR) for MS patients and healthy controls. Expressed as median (interquartile range) unless otherwise stated.

	MS (n=128)	HC (n=69)	p-value
Female, n (%)	81 (75.1)	43 (62.3)	0.06
Age (years)	41.6 (39.2–54.5)	44.7 (33.9–53.8)	0.71
Disease duration (years)	12.6 (7.2–19.8)		
EDSS (avg/med)	25.7 (22.3–29.8)	24.5 (23.2–26.3)	0.004
EDSS	3 (3–4)		
DTI use, n (%)	70 (54)		
Whole brain volume PF, %	81.4 (80.5–86.1)	86.0 (83.7–87.8)	<0.001
NAWM volume PF, %	23.4 (21.2–33.1)	34.8 (33.9–36.4)	<0.001
Central WM volume PF, %	19.8 (18.9–23.0)	31.3 (32.3–34.7)	<0.001
Thalamus volume PF, %	1.0 (0.9–1.1)	1.1 (1.0–1.1)	<0.001
Whole brain DVR	1.00 (1.18–1.22)	1.00 (1.18–1.29)	0.004
NAWM DVR	1.21 (1.17–1.25)	1.11 (1.14–1.28)	<0.001
Thalamus DVR	1.32 (1.29–1.37)	1.31 (1.27–1.35)	0.049

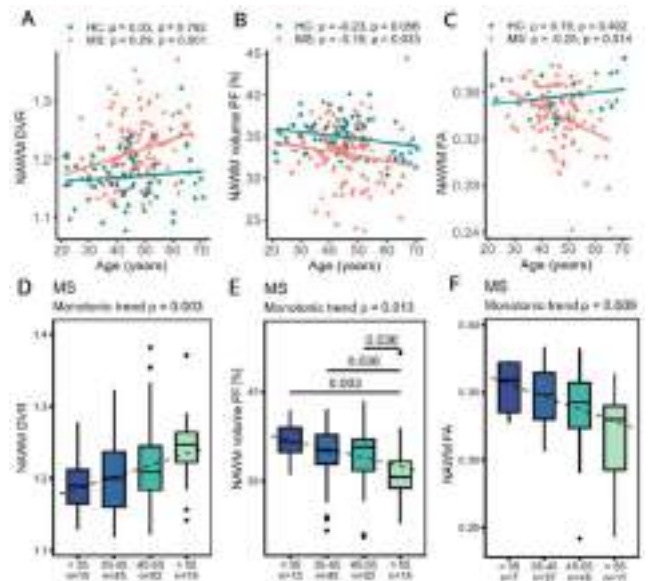


FIGURE 1 Age and NAWM DVR (A) correlate positively, age and volume PF% (B) and FA (C) negatively in MS but not in HCs. NAWM DVR (D), volume PF% (E), and FA (F) by age group in MS. Correlation coefficients, trend lines, p -values reported above panels

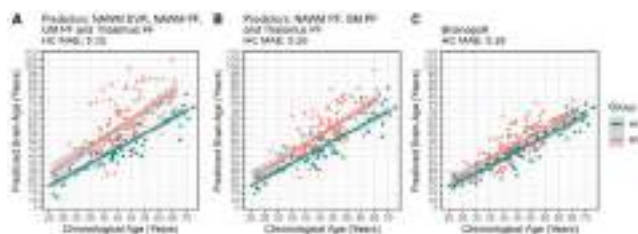


FIGURE 2 Biological brain age is highest in MS patients compared to controls in model using brain parenchymal fractions and DVR (A) compared to models using PFs alone (B) or BraineageR (C). Mean absolute error displayed.

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EPO-0466 | Serum neurofilament light chain predicts early subsequent disability progression in progressive MS

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Background and aims: Disability progression in multiple sclerosis (MS) is dominantly driven by chronic compartmentalized rather than acute focal inflammation, emphasizing the need for biomarkers to capture this biology. Evidence supporting the prognostic value of common genetic variants, serum neurofilament light chain (sNfL) and glial fibrillary acidic protein (sGFAP) in progressive MS is limited. We assessed whether sNfL and sGFAP predict disability progression in a cohort of people diagnosed with primary progressive MS (pwPPMS) and evaluated the interaction with relevant common genetic variants.

Methods: Baseline sNfL and sGFAP Z-scores were acquired. Risk-carriership of rs10191329 and HLA-DRB1*15:01 were collected from SNP-array data and MS severity and susceptibility weighted genomic risk scores were calculated. Logistic regression models including age and immunomodulatory treatment (IMT) assessed prediction of disability progression at 1, 2, and 3 years.

Results: 173 pwPPMS (median age 60 years, 51% female) were included. sNfL Z-scores correlated positively with disability severity, as measured by Expanded Disability Status Scale (EDSS) ($p=0.19$, $p=0.014$) and Age-Related Multiple Sclerosis Severity Score (ARMSS) ($p=0.25$, $p<0.001$). Higher sNfL Z-scores predicted EDSS progression at 1 year (OR 1.83 [95% CI 1.03–3.26], $p=0.04$) and 2 years (OR 1.67 [95% CI 1.02–2.67], $p=0.04$), but not at 3 years. Results of GFAP and genetic analyses will be presented.

Conclusion: Elevated sNfL predicted early disability progression. These findings support sNfL as a clinically relevant biomarker in progressive MS, with potential implications for prognostication and personalised treatment.

Disclosure: This work was funded by Stichting BeterKeten, the Dutch National MS Foundation (OZ2021-019 and 2019-019), and Siemens Healthineers. Joost S received research support from Biogen, Roche, Siemens Healthineers and Hansa Biopharma, and lecture and/or consultancy fee from Biogen, Merck, Novartis, Roche, and Sanofi. The remaining authors have nothing to disclose.

Muscle and neuromuscular junction disorder 3

EPO-0467 | Risk of myasthenia gravis exacerbation during pregnancy and postpartum: A nationwide cohort study

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Background and aims: Pregnancy and postpartum represent unique physiological states that may influence autoimmune diseases such as myasthenia gravis (MG). Evidence on MG exacerbations during these periods is scarce and often based on small case series. We aimed to assess the risk of MG exacerbation during pregnancy and up to one year postpartum using nationwide longitudinal register data.

Methods: We conducted a population-based cohort study including women with MG and their pregnancies in Sweden

between 1987 and 2019. Data were obtained through linkage of the Swedish MG Register with national health and sociodemographic registers. The primary outcome was hospital admissions for MG during pregnancy and up to one year postpartum, compared with the year preceding pregnancy. Cox regression models estimated hazard ratios (HR), adjusting for disease duration and thymectomy. Secondary outcomes included changes in immunosuppressive MG medications and whether women conceived during periods of relative disease stability.

Results: Our findings indicate that pregnancy is generally associated with clinical stability, whereas the postpartum period may carry an increased risk of exacerbation in a subset of women. Patterns of immunosuppressive therapy appear to reflect these trends. These results provide population-level evidence on MG risk dynamics surrounding pregnancy.

Conclusion: This nationwide cohort study offers novel insights into MG risk dynamics during pregnancy and postpartum. These insights will inform clinical counselling and management strategies for women with MG planning pregnancy.

Disclosure: Nothing to disclose.

EPO-0468 | Effect of exercise on functional decline in Inclusion body myositis based on the Yale IBM registry

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Background and aims: Inclusion body myositis (IBM) is a rare, progressive muscle disease with preferential involvement of quadriceps and long finger flexors, leading to muscle weakness and atrophy, gait impairment, and loss of hand function. The potential beneficial role of exercise has been implicated, but the impact of exercise in IBM, including the safety of exercise in IBM, is not well established.

Methods: In this study, we leveraged IBM functional rating scale (IBM-FRS) scores, longitudinally collected by the Yale IBM registry (IBMR). A linear mixed-effects model was used to examine the role of exercise on functional decline in IBM.

Results: We included 401 patients (age 68.4 ± 8.4 years), of whom 139 were women (34.7%). The baseline mean IBM-FRS score was 26.9 ± 6.8 . All patients showed a decline in IBM-FRS over time. We did not notice any major differences in disease progression by gender or age. The habit of exercise was associated with a slower rate of decline (p -value < 0.01), with a graded benefit for more active patients (p -value < 0.001). Patients actively engaged in physical therapy and walking showed slower deterioration than those with no exercise (p -values of 0.003 and 0.04, respectively). Analyses of within-person changes emphasized the role of exercise continuity.

Conclusion: These findings, based on patients' self-reports, suggest safety and the potential benefit of remaining physically active in IBM. Large and long-duration clinical trial with a structured exercise regimen to delineate the true impact of exercise on IBM disease progression is warranted.

Disclosure: AO, GC, EC, KP have nothing to disclose. GC reports to be in the Data and Safety Monitoring Boards of Applied

Therapeutics, AI therapeutics, Amgen-NMO peds, AMO Pharma, Argencx, Astra-Zeneca, Bristol Meyers Squibb, CSL Behring, DiamedicaTherapeutics, Horizon Pharmaceuticals, Immunic, Inhrbx-sanofi, Karuna Therapeutics, Kezar Life Sciences, Medtronic, Merck, Meiji Seika Pharma, Mitsubishi Tanabe Pharma Holdings, Prothena Biosciences, Novartis, Pipeline Therapeutics (Contineum), Regeneron, Sanofi-Aventis, Teva Pharmaceuticals, United BioSource LLC, University of Texas Southwestern, Zenus Biopharmaceuticals. He is also in the Consulting or Advisory Boards: Alexion, Antisense Therapeutics/Percheron, Avotres, Biogen, Clene Nanomedicine, Clinical Trial Solutions LLC, Endra Life Sciences, Genzyme, Genentech, Immunic, Klein-Buendel Incorporated, Kyverna Therapeutics, Inc, Linical, Merck/Serono, Noema, Neurogenesis, Perception Neurosciences, Protalix Biotherapeutics, Regeneron, Revelstone Consulting, Roche, Sapience Therapeutics, Tenmile. He is employed by the University of Alabama at Birmingham and President of Pythagoras, Inc. a private consulting company located in Birmingham AL. BR has served as a consultant/advisor for Alexion (now part of AstraZeneca), Takeda, Sanofi, and argencx. Additionally, BR has received research support from the Martin Shubik Fund for IBM at Yale University, NIH, MDA, Abcuro Pharmaceuticals, argencx, Immunovant, and Takeda. BR has small stocks in Cabaletta Bio, CAVA, and Pfizer.

EPO-0469 | Continuing medical education improves competence in implementing new therapies for myasthenia gravis

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Background and aims: Treatments for myasthenia gravis (MG) are evolving, requiring physicians to consider new mechanisms including FcRN or complement inhibition. We sought to improve knowledge of these new therapies and their application in today's clinical practice.

Methods: Physicians participated in a 3-part video-based, online CME activity (available at www.medscape.org/view-article/1002773). The education effects were assessed using a 3-question, repeated pairs, pre-assessment/post-assessment study design. One question assessed confidence. Pre- to post-assessment differences were evaluated using the McNemar's test. $p \leq 0.05$ is the significance level. The activity launched in 08-2025 and data were collected through 10-2025.

Results: 53 neurologists and 80 primary care physicians (PCPs) completed all pre- and post-assessment questions. Overall significant improvements were seen after participation for neurologists (correct pre-assessment response rate of 47% vs 73% at post-assessment; $p < 0.001$, Cohen's $d = 0.9$), and PCPs (correct pre-assessment response rate of 31% vs 54% at post-assessment; $p < 0.001$, Cohen's $d = 0.98$). Improvements were observed in clinicians' understanding of the rationale for FcRN inhibition in MG, and how best to tailor new therapies in different clinical scenarios (Figure 1). After participating, 38% of neurologists and 26% of PCPs had measurable improved confidence in determining which patients with MG may benefit from a targeted therapy.



FIGURE 1 Changes in Knowledge and Competence Pre- vs Post-Education.



FIGURE 2 Intended practice changes post-education.

Post-education, 52% of neurologists and 42% of PCPs said they intend to consider targeted therapies earlier for patients with severe or rapidly progressive MG (other responses in Figure 2).

Conclusion: This study demonstrates the success of online, multi-component, video-based education in improving competence in implementing new targeted therapies for MG in practice.

Disclosure: Nothing to disclose.

EPO-0470 | Effect of subcutaneous self-administered Gefurulimab on ocular muscle function in myasthenia gravis in the PREVAIL Trial

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Background and aims: Gefurulimab, a novel dual-binding nanobody, blocks complement component 5 activation. The phase 3 PREVAIL trial, assessing the efficacy and safety of gefurulimab in adults with anti-acetylcholine receptor antibody-positive (AChR-Ab+) generalised myasthenia gravis (gMG) (NCT05556096), met all primary and secondary endpoints and demonstrated clinically meaningful improvement from baseline in Myasthenia Gravis Activities of Daily Living (MG-ADL) total score at week 26 compared with placebo. Here, we present the effect of gefurulimab on ocular symptoms.

Methods: Adults with AChR-Ab+ gMG were randomised 1:1 to weekly subcutaneous self-administration of gefurulimab or placebo. The primary endpoint was change from baseline

in MG-ADL total score at wk26. MG-ADL and Quantitative Myasthenia Gravis (QMG) subdomain scores were also assessed. **Results:** 260 patients were randomized (gefurulimab: $n=131$; placebo: $n=129$). Mean $\pm$ SD baseline MG-ADL and QMG total scores were 9.0 ± 2.16 and 14.8 ± 4.37 , respectively. Improvements in ocular subdomain scores were observed at first assessment after treatment initiation (MG-ADL, wk1; QMG, wk4) and sustained through wk26. Least squares mean $\pm$ SEM change from baseline at wk26 in MG-ADL and QMG ocular subdomain scores for gefurulimab versus placebo were -1.2 ± 0.14 versus -0.6 ± 0.13 (treatment difference, -0.6 ± 0.19 ; 95% CI: $-1.0, -0.2$; $p=0.0016$) and -1.7 ± 0.14 versus -0.9 ± 0.14 (treatment difference, -0.7 ± 0.19 ; 95% CI: $-1.1, -0.4$, $p=0.0002$), respectively. Improvements were also observed in other MG-ADL and QMG subdomains. Adverse events were similar between groups; no meningococcal infections were reported.

Conclusion: Gefurulimab, self-administered once weekly via subcutaneous injection, showed early and sustained improvements in MG-ADL and QMG ocular subdomain scores, demonstrating benefits for patients with gMG experiencing ocular symptoms.

Disclosure: FS has received public speaking honoraria from Alexion, argenx, Biogen, Genpharm, Medpharma, Medison Pharma, Mylan, Neopharm Israel, Novartis, Roche, Sanofi, UCB, and Zai Lab; compensation for advisory boards from Alexion, Almirall, Amgen, argenx, AstraZeneca, Avexis, Biogen, Dianthus, Johnson & Johnson, Lexeo, Novartis GmBh, Reata, Sandoz, UCB, and Zai Lab; consultation fees from Alexion, argenx, Dianthus, EPG Communication Holdings Ltd, Johnson&Johnson, Medscape, PeerView, Vitaccess; clinical trial support from Almirall; funding from Agenzia Italiana del Farmaco (AIFA), Alexion, Associazione Italiana per la lotta alle Sindromi Atassiche (AISA), Freidreich Ataxia Research Alliance (FARA), Roche SpA, Tristan Allamby Research Fund (TARFfa); and was a clinical trial principal investigator from Alexion, argenx, Dianthus, Immunovant, Lediand, Lexeo, Novartis, Prilenia, Remgen, and Sanofi. DA has received study funding from Alexion, AstraZeneca Rare Disease; and serves on the CHAMPION-MG study steering committee. MM has received compensation for lectures, consulting and travel expenses from Alexion, AstraZeneca Rare Disease, Almirall, argenx, Biogen, BMS, CSL Behring, Hexal, Genzyme, GSK, Merck, Novartis, Roche, Sanofi, Teva, and UCB. MT has received consulting fees from Alexion Pharma GK, Alexion AstraZeneca Rare Disease, argenx, Hanall Biopharma, and UCB Pharma; research support from the Japan Blood Products Organization; and speaker honoraria from Alexion Pharma GK, AstraZeneca Rare Disease, argenx, and UCB. SR, JS, and SS are employees of Alexion, AstraZeneca Rare Disease, and hold stock or stock options in AstraZeneca. TV has received research support from Alexion/AstraZeneca Rare Disease, Amgen, Amylyx, argenx, Cartesian, CSL Behring, Dianthus, Healy Platform Trials, Immunovant, Ipsen, Johnson & Johnson, PTC Therapeutics, Regeneron, Sanofi, UCB, and Woolsey Pharma; served on speakers bureaus for Alexion, AstraZeneca Rare Disease, argenx, and CSL Behring; and served on ad boards or as consultant for Alexion/AstraZeneca Rare Disease, Amgen, argenx, ImmunAbs, Johnson & Johnson, and UCB.

EPO-0471 | Expanded phenotypic and ultrasound characterization of the p.Ser55Phe myotilinopathy variant: The MYOT MUR study

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Background and aims: Myotilinopathies are the most frequent myofibrillar myopathy in Spain, mainly the p.Ser55Phe MYOT variant. We characterized the phenotype and disease progression in the largest known cohort of this variant from Murcia, Spain.

Methods: We conducted an ambispective observational study (2022–2025) on 46 p.Ser55Phe carriers. Annual evaluations included history, pedigree analysis, neuromuscular ultrasound (NMUS) and neurological examination including North Star Ambulatory Assessment (NSAA), Medical Research Council (MRC) scale, and 6-minute walk test (6mWT). Sex differences were analyzed using non-parametric tests.

Results: The cohort ($n=46$; 44% female) had a mean onset age of 47.4 years. 71.7% were symptomatic. Weakness was asymmetric (89.7%). Lower limb weakness (67.4%) typically had distal onset ($n=15$) with frequent foot drop (60.6%), progressing proximally. Upper limb weakness (54.3%) was mostly proximal at onset (68.8%), progressing distally. Pain (60.9%) and dyspnea (37%) were frequent. Males were more symptomatic (84% vs 55%, $p=0.049$) with earlier onset than females (45.3 vs 51.5 years, $p=0.013$). Males exhibited lower limb predominance ($p=0.006$), more foot drop ($p<0.001$), atrophy ($p<0.001$), and lower NSAA, MRC, and 6mWT scores across most visits. Females showed axial predominance. NMUS revealed increased echogenicity initially in the tibialis anterior and extensor digitorum brevis, progressing to the vastus intermedius and brachialis.

Conclusion: We describe the largest p.Ser55Phe myotilinopathy cohort to date, an expanded clinical phenotype, and a characteristic ultrasound pattern. Weakness is asymmetric and distal-predominant. We highlight a significant sex difference, with males experiencing a more severe course and worse functional prognosis.

Disclosure: Nothing to disclose.

EPO-0472 | Immediate FcRn inhibitor initiation following fast-acting treatment in generalised myasthenia gravis: A single-centre retrospective cohort study

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Background and aims: Early initiation of fast-acting treatment (FT; intravenous immunoglobulin, plasma exchange, or intravenous methylprednisolone) improves long-term outcomes

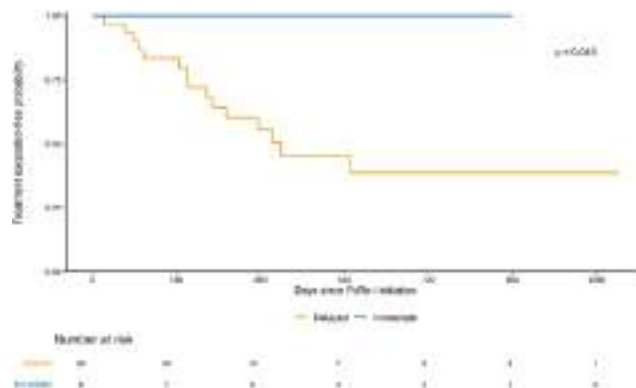


FIGURE 1 Kaplan–Meier curves of treatment escalation–free probability since FcRn-i initiation in gMG (immediate $n=8$ vs delayed $n=30$). Tick marks indicate censoring; numbers at risk are shown below. Log-rank $p=0.019$.

in patients with generalised myasthenia gravis (gMG). However, the optimal approach to achieve durable disease control after FT remains unclear. We hypothesised that the timely initiation of an FcRn inhibitor (FcRn-i) as a "consolidation therapy" following FT may promote sustained benefit, particularly in patients with shorter disease duration.

Methods: This retrospective single-centre cohort study included adults with gMG treated with FcRn-i between January 2022 and September 2025. Immediate initiation was defined as FcRn-i administration within 60 days after the last FT and disease duration ≤ 2 years; all others were classified as deferred. The primary outcome was the time to treatment escalation to additional FT or C5 inhibitor therapy. Group differences were analysed using Kaplan–Meier curves with log-rank tests and Firth's penalised Cox models adjusted for age and MGFA classification.

Results: Thirty-eight patients were included (immediate, $n=8$; deferred, $n=30$). Median follow-up was 355 days (IQR 190–572). Treatment escalation occurred in 0/8 immediate patients and 15/30 deferred patients. Kaplan–Meier analysis showed a significant difference (log-rank $p=0.02$). In Firth's Cox model, immediate initiation was associated with a markedly lower hazard of treatment escalation (HR 0.09, 95% CI 0.001–0.70).

Conclusion: Immediate post-FT initiation of FcRn-i was associated with a reduced hazard of treatment escalation to additional FT or C5 inhibitor therapy. This consolidation-type strategy may support more stable disease control in patients early in the disease course and will be further evaluated in larger cohorts.

Disclosure: Nothing to disclose.

EPO-0473 | Measuring daily living ambulation of patients with high accuracy to derive meaningful clinical conclusions

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TABLE 1 Stride detection results by pathology. Precision is defined as the ratio of well-detected strides among all strides detected by the wDHT. Recall is defined as the ratio of well-detected strides among all existing strides.

Pathology	Number of participants	Precision	Recall
Amyotrophic lateral sclerosis	10	99.4%	97.8%
Charcot-Marie-Tooth disease	10	99.9%	99.8%
Myotonic dystrophy type 1	9	99.8%	99.6%
Limb girdle muscular dystrophy	8	99.9%	97.7%
Pompe disease	3	99.8%	99.7%
Myasthenia gravis	3	99.7%	99.1%
Huntington disease	4	99.7%	99.5%
Parkinson's disease	3	100%	97.7%
Chronic inflammatory demyelinating polyneuropathy	5	99.8%	99.7%
Spinal muscular atrophy	9	98.0%	88.9%
Duchenne muscular dystrophy	16	99.3%	97.7%
Healthy volunteers	20	98.7%	98.6%

Background and aims: Wearable devices are increasingly used in clinical trials in neurology. However, their actual capacity to precisely identify and measure movements in different conditions is never reported. The qualification of a digital measure, stride velocity 95th centile, as a primary endpoint in Duchenne muscular dystrophy (DMD) showed that suitable wearable digital health technologies (wDHT) can be reliable tools to measure disease progression and efficacy of therapeutic interventions in diseases impacting ambulation. It is crucial to establish in various populations the wDHT accuracy to detect strides and to precisely measure gait features.

Methods: This study enrolled 100 participants, including patients with amyotrophic lateral sclerosis, Charcot-Marie-Tooth disease, myotonic dystrophy type 1, limb girdle muscular dystrophy, Pompe disease, myasthenia gravis, spinal muscular atrophy, Huntington disease, Parkinson's disease, chronic inflammatory demyelinating polyneuropathy, DMD and healthy volunteers. Participants wore Syde ankle sensors and walked at different paces on path with turns, in a room equipped with a motion capture system (reference).

Results: Individual strides were correctly detected with an average precision of 99.5% and average recall of 97.8% (Table 1). The mean difference between the wDHT and the reference on stride length and stride velocity were −0.8 to 4.8 cm and −0.9 to 4.3 cm/s, respectively. Standard deviations of the absolute errors were between 1.5–4.1 cm for stride length and 1.5–3.5 cm/s for stride velocity.

Conclusion: We demonstrated Syde excellent precision to detect individual strides and similar performance in measuring gait parameters in several pathologies affecting ambulation, supporting its application to develop digital endpoints.

Disclosure: This study was funded by Sysnav. NN, OP, AT and DE are employees of Sysnav. LS has given consultancy for Sysnav.

EPO-0474 | Anti-Titin antibodies and thymoma in myasthenia gravis: A 10-year age-adjusted portuguese cohort study

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Background and aims: Anti-titin antibodies are detected in approximately one-third of patients with myasthenia gravis (MG) and are considered a sensitive biomarker for thymoma in patients aged ≤50years. However, their high prevalence in late-onset MG reduces specificity, limiting their clinical utility in older patients. We aimed to assess the predictive value of anti-titin antibodies for thymoma in a Portuguese MG cohort.

Methods: We conducted a retrospective study of all patients newly diagnosed with MG between January 1, 2015, and August 1, 2025. Anti-titin antibody status was assessed by immunoblotting, and thymoma was evaluated using chest CT, PET-CT, or histopathology. Associations between anti-titin positivity, age, and thymoma were analyzed.

Results: Among 114 MG patients (mean age 66.6years), 34 had thymoma and 81 were anti-titin–positive. In patients <50years, anti-titin positivity showed a non-significant trend toward association with thymoma (OR 4.44, *p*=0.181). In patients ≥50years, anti-titin positivity was not predictive of thymoma (OR 0.44, *p*=0.171) and was inversely associated with thymoma in those aged 50–75 years (OR 0.23, *p*=0.042). Age significantly modified the predictive value of anti-titin antibodies (*p*=0.039). Anti-titin positivity was markedly more frequent in older non-thymoma patients (≥50: 81.2% vs <50: 27.3%, *p*<0.001), while sensitivity among thymoma patients was similar across age groups.

Conclusion: In MG patients over 50years, anti-titin antibodies show reduced specificity for thymoma due to high positivity among non-thymoma cases. In patients aged 50–75years, anti-titin positivity may favor the absence of thymoma. These findings highlight the age-dependent limitations of anti-titin antibodies in thymoma prediction.

Disclosure: The authors have nothing to disclosure.

EPO-0475 | Clinical and immunological features of thymoma patients with and without myasthenia gravis: A pilot study

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Background and aims: Thymoma-associated myasthenia gravis (TAMG) is nearly invariably associated with acetylcholine receptor antibodies (AChRAb). However, approximately 15% of thymoma patients have AChRAb in the absence of MG and rarely may develop post-thymectomy MG (PTMG). This study evaluates the clinical and immunological features of thymoma

patients with and without MG to identify predictive biomarkers of PTMG.

Methods: Patients with thymoma treated in our Centre between 2011 and 2025 were included in the study. Demographic and clinical data were collected, and available serum samples were evaluated for in vitro complement activation using a flow cytometry live cell-based assay.

Results: Fifty thymoma patients were included in the study: 17/50 (34%) TAMG, 27/50 (54%) thymoma patients with no MG and AChRab negative (AChRab-MG-), 6/50 (12%) AChRab positive thymoma patients without MG (AChRab+MG-), of whom 2/6 (33%) developed PTMG. Types AB/B thymomas predominated across groups ($n=40/50,80\%$) without statistically significant difference between MG and non-MG patients (with/without AChRab). Most had a Masaoka stage I–II ($n=43/50,86\%$). The majority of patients had maximum MGFA>II (13/19, 68%). Sixteen serum samples were available for further analyses (10 TAMG, 2 PTMG, 4 AChRab+MG-). Complement activation was significantly lower in AChRab+MG- thymoma patients compared to TAMG and PTMG patients ($p=0.0332$, Mann-Whitney U test). In one PTMG patient with available serial serum samples, level of complement activation increased significantly from thymoma diagnosis to MG onset.

Conclusion: In vitro assessment of complement activation could represent a novel biomarker of PTMG. Future studies are warranted to investigate in-depth antibody as well as thymic and B-cells properties in PTMG patients.

Disclosure: The authors report no disclosures or conflicts of interest relevant to this work.

EPO-0476 | Omaveloxolone for Friedreich ataxia: What does 'Cochrane' have to say about it?

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Background and aims: Omaveloxolone (RTA 408) is thought to activate the nuclear factor erythroid-derived 2-related factor (Nrf2) signalling which is grossly impaired in Friedreich ataxia (FRDA) which is a rare autosomal recessive, progressive disease, typically presents around puberty. Approximately one third of people with FRDA are diagnosed after the age of 20. It usually presents with unsteadiness, followed by progressive limb and gait ataxia. It can be associated with scoliosis, pes cavus, cardiomyopathy and diabetes mellitus. Cochrane Reviews are systematic reviews and preform meta-analyses, where possible. They are a key resource in evidence-based medicine.

Methods: The aim of the review was to assess the effects of pharmacological treatments in Friedreich ataxia after one year's treatment. We found 7 randomised controlled trials of 1 year duration. We synthesised results for each outcome using meta-analysis, used GRADE to assess the certainty of evidence.

Results: Pharmacological treatments probably make little or no difference to change in ataxia rating scale (ARS) but improved upper limb dexterity (ULD) There was variation in that omaveloxolone helped ARS and leriglitazone helped ULD. The evidence for both outcomes was moderate and was an unexpected finding given that both outcomes share similar competencies.

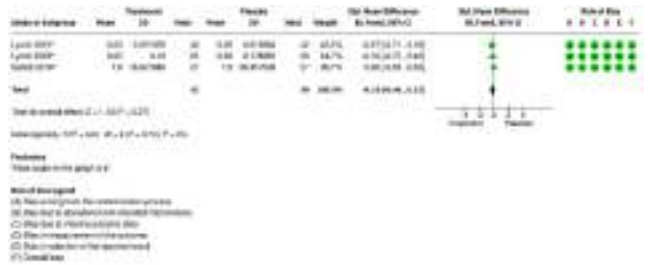


FIGURE 1 Forest plot- change in ataxia rating score after 12 months treatment.

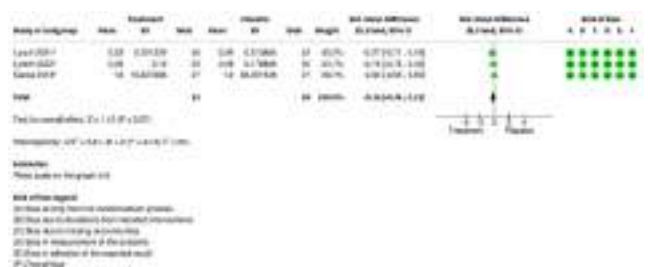


FIGURE 2 Forest plot - change in upper limb dexterity after 12 months treatment.

Summary of findings table									
Background: Omaveloxolone (RTA 408) is thought to activate the nuclear factor erythroid-derived 2-related factor (Nrf2) signalling which is grossly impaired in Friedreich ataxia (FRDA) which is a rare autosomal recessive, progressive disease, typically presents around puberty. Approximately one third of people with FRDA are diagnosed after the age of 20. It usually presents with unsteadiness, followed by progressive limb and gait ataxia. It can be associated with scoliosis, pes cavus, cardiomyopathy and diabetes mellitus. Cochrane Reviews are systematic reviews and preform meta-analyses, where possible. They are a key resource in evidence-based medicine.									
Comparison: Omaveloxolone (RTA 408) vs. Placebo									
Outcome: Change in ataxia rating score (ARS) after 12 months									
Study	Participants	Events	Events	Events	Events	Events	Events	Events	Events
Study 1	100	10	10	10	10	10	10	10	10
Study 2	100	10	10	10	10	10	10	10	10
Study 3	100	10	10	10	10	10	10	10	10
Study 4	100	10	10	10	10	10	10	10	10
Study 5	100	10	10	10	10	10	10	10	10
Study 6	100	10	10	10	10	10	10	10	10
Study 7	100	10	10	10	10	10	10	10	10
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S. Gokhale⁵; C. Briggs⁵; U. Siddiqui⁵; S. Randhawa⁵;

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Background and aims: The classical complement pathway plays a significant role in the pathogenesis of generalized myasthenia gravis (gMG). Claseprubart is a monoclonal antibody that targets the classical pathway by inhibiting active C1s. The MaGic trial assessed the safety and efficacy of claseprubart in adults with acetylcholine receptor antibody positive (AChR+) gMG.

Methods: MaGic (NCT06282159) is a randomized, double-blind, placebo-controlled Phase 2 study. 65 participants with AChR+ gMG were randomized 1:1:1 to receive: claseprubart 300 mg (Q2W), claseprubart 600 mg (Q2W), or placebo for 13 weeks, followed by a 52-week open-label extension and 40-week safety follow-up. Endpoints at 13 weeks included safety, tolerability, efficacy (MG activities of daily living (MG-ADL), Quantitative MG score (QMG), Minimal Symptom Expression (MSE) and MG Composite scale (MGC).

Results: Claseprubart was well tolerated with no serious adverse events, no serious bacterial infections, and no autoimmune activation. Injection site reactions were infrequent and mild to moderate. At Week 13, claseprubart improved MG-ADL by 4.6 ($p=0.0113$) for 300 mg, 5.4 ($p=0.0006$) for 600 mg, and 2.8 for placebo (no significant difference between active treatment arms). Significant improvement in MG-ADL was observed as early as Week 1. At Week 13, claseprubart 300 mg improved QMG by 4.4 ($p=0.0144$), claseprubart 600 mg by 4.5 ($p=0.0111$), and 2.0 for placebo. MSE was achieved by 37% (300 mg) and 27% (600 mg) versus 14% for placebo. MGC for 300 mg was 8.7 ($p=0.0008$), 8.6 ($p=0.0008$) for 600 mg versus 3.1 for placebo.

Conclusion: With a favorable safety profile, claseprubart demonstrated rapid, statistically significant, and clinically meaningful improvements in MG-ADL, QMG, MGC.

Disclosure: SP grants or honoraria from Dianthus, Amgen, Avidity, Amicus Swixx, Argencx, AstraZeneca, Immunabs, Octapharma, Roche, Viatrix, Zentiva. PN grants or honoraria from AHRQ, PCORI, Argencx, Cabaletta Bio, AstraZeneca, Amgen, Cartesian, CVS, Dianthus, Serono, Immuneabs, Immunovant, J&J, UCB, Viridian, Vor-Biol; Sanofi, Argencx, NMD. MS nothing to declare. AS nothing to declare. SG nothing to declare. CB employee of Dianthus Therapeutics US employee of Dianthus Therapeutics SR employee of Dianthus Therapeutics MT employee of Dianthus Therapeutics LH employee of Dianthus Therapeutics SS grants or honoraria from Dianthus therapeutics, J&J, Argencx, Astrazenica, Alexion, Mediaon, Neopharm, Novartis and Sanofi. TV grants or honoraria from Alexion/AstraZeneca, Amgen, Argencx, Cartesian, COUR, Dianthus, EMD Serono, ImmunAbs, Immunovant, J&J, NMD, Regeneron, VOR, UCB, CSL Behring, and VOR. JV grants

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EPO-0478 | Interim analysis of an observational, prospective, multicenter study of efgartigimod as consolidation therapy after acute exacerbation in MG

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Background and aims: Patients with myasthenia gravis (MG) still require consolidation therapy after acute exacerbation to control symptoms and prevent relapse. This interim analysis aims to explore whether efgartigimod in addition to standard of care (SOC) provides added benefit as consolidation therapy.

Methods: Adult patients with Acetylcholine Receptor (AChR) antibody-positive generalized MG who achieved clinically meaningful improvement after one treatment cycle of efgartigimod for acute exacerbation were enrolled in the study (NCT07079020). Patients were allocated to either efgartigimod plus SOC (EFG) group or SOC alone (SOCA) group based on real-world clinical

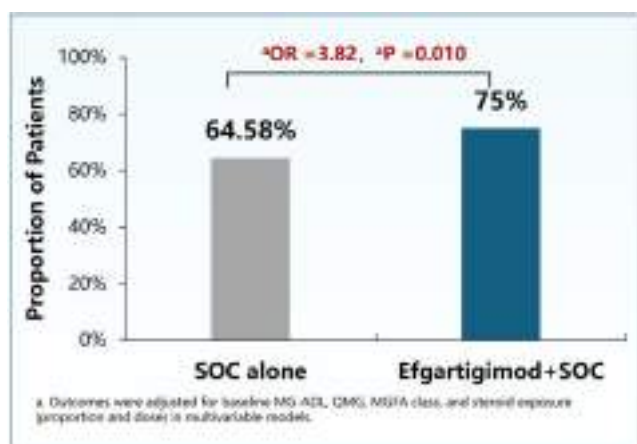


FIGURE 1 Proportion of patients achieving MSE at the interim analysis.

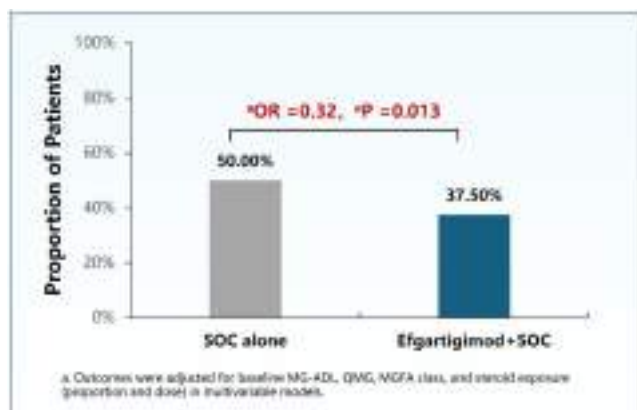


FIGURE 2 Proportion of patients with MG worsening compared with the previous visit.

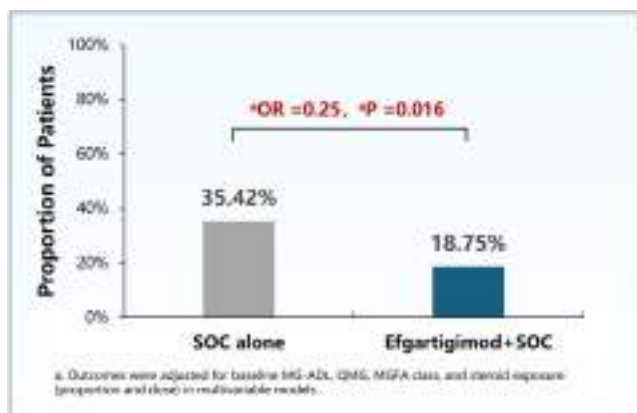


FIGURE 3 Proportion of patients with MG worsening compared with the baseline.

decision-making, with a 6-month follow-up period. The primary endpoint was achievement of minimal symptom expression (MSE) within the second month of the consolidation phase and sustained MSE for more than four consecutive months.

Results: A total of 112 patients with ≥ 2 months of follow-up by October 2025 were included (48 in SOCA group, 64 in EFG group). MSE was achieved in 64.58% of patients in the SOCA group and in 75% of patients in the EFG group ($p=0.010$)

(Figure 1). MG worsening was less frequently observed in the EFG group compared with the SOCA group, either relative to the previous visit (37.50% vs 50.00%, $p=0.013$) or to the baseline (18.75% vs 35.42%, $p=0.016$) (Figures 2 and 3).

Conclusion: In this interim analysis, efgartigimod was associated with higher rates of achieving MSE, and reduced risk of MG worsening in consolidation therapy. These findings support the role of efgartigimod as a therapeutic strategy in post-exacerbation MG management.

Disclosure: This study was supported by the China Alliance for Rare Diseases (non-profit organization).

Neurogenetics 3

EPO-0479 | Intrathecal AAV-based gene therapy for metachromatic leukodystrophy: Preclinical efficacy and safety in a large animal model

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Background and aims: Metachromatic leukodystrophy (MLD) is an autosomal recessive neurodegenerative lysosomal storage disorder caused by deficiency of arylsulfatase A (ARSA), leading to progressive motor dysfunction, spastic tetraparesis, ataxia, seizures, optic atrophy, and cognitive decline. The aim of this study was to develop and evaluate a gene therapy product based on recombinant adeno-associated virus serotype Olig001 (AAVOlig001) carrying a codon-optimized human ARSA gene. **Methods:** AAVOlig001-ARSA containing a unique codon-optimized ARSA sequence was generated. Following intravenous administration of mannitol (1 g/kg), pigs received an intrathecal injection of AAVOlig001-ARSA at a dose of 2×10^{13} gc/kg. ARSA enzymatic activity was dynamically assessed in cerebrospinal fluid (CSF) and plasma using pNCS. One month after administration, central nervous system (CNS) tissues were collected for analysis of ARSA activity, gene expression (qPCR), and protein expression by immunohistochemistry (IHC). Safety was evaluated using biochemical and hematological blood analyses.

Results: No significant differences in plasma ARSA activity were observed between experimental and control animals, while ARSA activity in CSF was detectable on day 28. In contrast, CNS tissue homogenates showed a marked increase in ARSA activity, most prominently in the cerebellum, cervical, thoracic, and lumbar spinal cord, and brainstem. Overexpression of the codon-optimized ARSA gene and ARSA protein expression in the CNS were confirmed by qPCR and IHC. Blood biochemical parameters remained within physiological ranges; however, hematological changes observed on day 28 suggested systemic exposure to the vector and a potential immune response (Figure 1).

Conclusion: In summary, intrathecal administration of AAVOlig001-ARSA enabled efficient CNS delivery and induced ARSA overexpression.

Disclosure: This work was supported by the Kazan Federal University Strategic Academic Leadership Program (PRIORITY-2030).

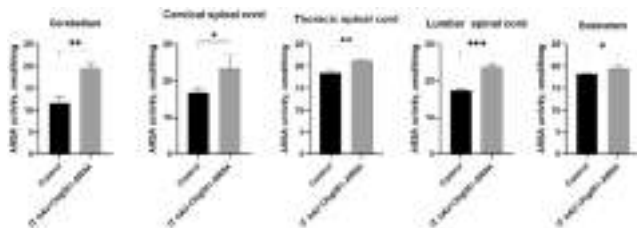


FIGURE 1 ARSA enzymatic activity analysis in homogenates of various CNS regions on day 28 after AAV-Olig001-ARSA administration. Data represent the mean of five biological replicates $\pm$ SD. * — $p < 0.001$.

EPO-0480 | Giant axonal neuropathy: A homogeneous phenotype, a diverse genetic landscape, and the emergence of gene therapy

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Background and aims: Giant Axonal Neuropathy (GAN) is a genetic autosomal disorder caused by mutations in the GAN gene, patients usually develop a progressive degeneration of the central and peripheral nervous systems [1]. This study aims to investigate the correlation between specific genetic variants and clinical profiles to better define the disease spectrum.
Methods: We conducted a systematic literature review following PRISMA protocols, emphasizing abnormal presentations. PubMed and Google Scholar databases were searched using keywords "Giant Axonal Neuropathy". We evaluated age, genotype, CNS signs, cranial nerve involvement and physical signs.
Results: We included 29 studies, reporting 128 patients with Giant Axonal Neuropathy (GAN) revealed a uniform and severe

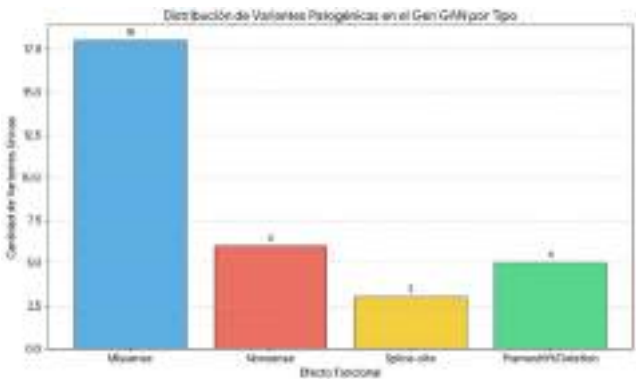


FIGURE 2 Distribution of Pathogenic Giant Axonal Neuropathy Gene Variants by Type.

clinical presentation. The mean age of onset was 2.8 years, with diagnosis at 9.3 years. Motor (100%) and sensory (99%) neuropathy were nearly universal, accompanied by cerebellar ataxia (93%) and curly hair (93%). Scoliosis was present in (52%) of patients. Autonomic dysfunction was less common (<10%). More than 20 pathogenic variants were identified in the GAN gene, see Figure 1 Intrathecal gene therapy (NCT02362438) in 14 patients showed stabilization of motor function (MFM-32) and neurophysiological improvements, offering the first disease-modifying intervention.
Conclusion: Despite the various genetic variants, GAN presents a remarkably uniform clinical profile, dominated by early-onset neuropathy, ataxia, and characteristic capillary changes. This phenotypic consistency, along with new gene therapy data, provides a clear framework for diagnosis and highlights a promising intervention for disease modification.
Disclosure: Nothing to disclose

EPO-0481 | Multiple systemic lipomatosis associated with the 8344A>G tRNA^{Lys} mtDNA variant in a large Italian family: Searching for a threshold effect

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Background and aims: Myoclonic epilepsy with ragged red fibers (MERRF) is a rare mitochondrial DNA (mtDNA) disorder commonly caused by the 8344A>G mutation in the MT-TK gene encoding tRNA^{Lys}. Clinical expression is highly variable due to tissue-specific heteroplasmy and threshold effects. In addition to the classic neurological and systemic features, multiple systemic lipomatosis (MSL) has been described. The aim of this study was to investigate a large Italian family carrying the 8344A>G variant and to explore a possible threshold effect for the development of MSL.
Methods: The proband was a 28-year-old woman presenting with myoclonic epilepsy, cerebellar ataxia, peripheral neuropathy, and a large cervical lipoma. Following molecular confirmation of the 8344A>G variant, 14 maternally related family members were evaluated. Clinical assessment focused on

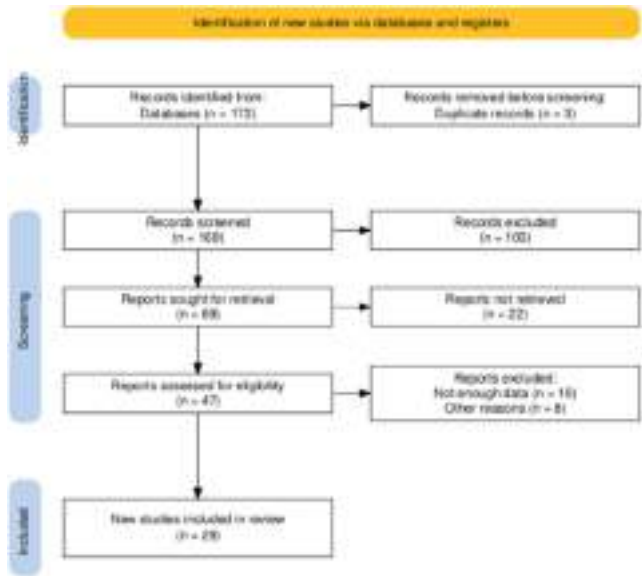


FIGURE 1 Prisma-P flowchart of the process of inclusion of studies in the systematic review.

neurological features and the presence of MSL. Molecular analysis was performed on peripheral blood leukocytes (PBLs) and urinary epithelial cells to determine heteroplasmy levels.

Results: Ten out of 14 family members presented isolated MSL with variable extension. All affected individuals carried the 8344A>G variant. Mean mutation load was higher in affected subjects. In the proband, heteroplasmy exceeded 84% in both tissues. Individuals without MSL showed lower mutation loads. Overall, variant levels were higher in urinary epithelial cells than in PBLs.

Conclusion: This study describes a large family in which MSL was often an isolated manifestation. Differences in heteroplasmy between tissues and between affected and unaffected individuals suggest an apparent threshold effect for the development of MSL. These findings support considering MERRF in families with maternally inherited MSL and warrant further investigation.

Disclosure: Nothing to disclose.

EPO-0482 | Breaking the age barrier: Adult-onset GM2A gangliosidosis with reclassification of two likely pathogenic variants

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Background and aims: GM2 gangliosidosis are rare autosomal recessive lysosomal storage disorders, with a classically fatal infantile onset. GM2 activator protein deficiency (GM2, AB variant), caused by variants in GM2A, is the rarest subtype, with adult-onset cases being exceptionally rare. To date, only one adult-onset case has been reported. We describe two adult siblings with late-onset disease and provide genetic and functional evidence supporting reclassification of two GM2A variants as likely pathogenic.

Methods: Two affected siblings underwent clinical, neurophysiological, imaging, enzymatic, genetic, and functional evaluation, including plasma ganglioside quantification.

Results: A 57-year-old man developed progressive lower limb weakness from age 30. His 53-year-old brother had presented with symmetric areflexic paraparesis, thigh muscle atrophy, gait impairment and Gowers' sign, from age 20. distal sensory involvement was present in one sibling. Electromyography revealed a chronic, long-standing motor neuronopathy associated with a milder sensory neuronopathy. Muscle MRI was significant for quadriceps atrophy, and muscle biopsy showed angular fiber atrophy. β -hexosaminidase activity was normal. Genetic testing identified compound heterozygosity for GM2A variants c.428G>A and c.496G>A, both initially classified as variants of uncertain significance. Plasma ganglioside analysis showed increased GM2 levels compared to controls in both siblings, with a profile comparable to patients with confirmed GM2A pathogenic variants and Tay–Sachs disease, supporting reclassification of both variants as likely pathogenic.

Conclusion: These cases represent an exceptionally rare adult-onset GM2 AB variant, expanding the phenotypic and molecular spectrum of GM2A-related disease and highlighting the diagnostic value of functional studies for variant reclassification in late-onset neuromuscular phenotypes.

Disclosure: Nothing to report.

EPO-0483 | Genotype–phenotype patterns in genetic Parkinson's disease in a tertiary center from Türkiye

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Background and aims: Genetic forms of Parkinson's disease (PD) are clinically heterogeneous and increasingly recognized, particularly in regions with elevated consanguinity such as Türkiye. Despite growing international data, national evidence remains limited. This study evaluates the clinical and genetic characteristics of patients with confirmed pathogenic variants to explore genotype–phenotype relationships.

Methods: In this retrospective cohort, medical records of 1812 patients diagnosed with PD between 2010 and 2024 were reviewed. Thirty-one individuals with confirmed pathogenic or likely pathogenic variants were included and stratified into three groups: PRKN, GBA, and other genetic causes. Demographic, motor, and non-motor features were compared. Logistic regression with correction was used to mitigate small-sample bias, with cognitive involvement as the outcome.

Results: Homozygous or compound heterozygous PRKN variants were the most frequent variations and were associated with earlier onset and a relatively homogeneous, motor-predominant phenotype. Heterozygous GBA variants demonstrated a more heterogeneous profile, with higher frequencies of cognitive impairment and postural instability; exploratory regression indicated increased odds of cognitive impairment (OR $\approx$ 5.7). Rare variants—including ATP13A2, FBXO7, HTRA1, and GCH1—occurred as single cases and were linked to atypical or complex presentations.

Conclusion: This cohort highlights the substantial genotypic and phenotypic diversity of PD in Türkiye. PRKN and GBA predominate, while rare variants contribute to atypical disease profiles.

Disclosure: Nothing to disclose.

EPO-0484 | Genetic contributions in Parkinson's disease and Parkinsonism from the PADUA-CESNE cohort

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Background and aims: In Parkinson's disease (PD), monogenic causes account for 5–10% of cases and are rarer in atypical parkinsonism. In our previous study of 218 selected patients, genetic causes were identified in 20%. The PADUA-CESNE cohort as a biocollection program has now doubled these data, so we aim at further investigating genetic contributions to parkinsonism in our Italian population.

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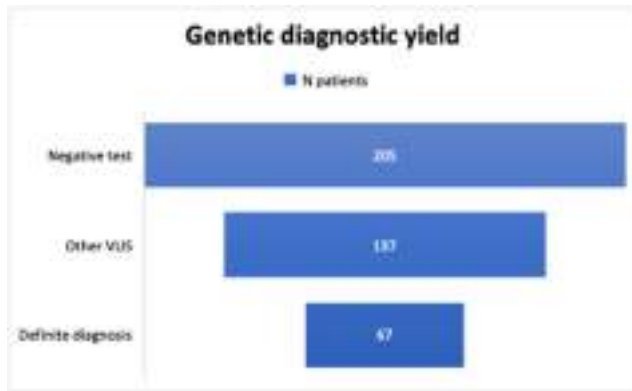


FIGURE 1 Gene panel diagnostic yield in Padua-CESNE cohort.

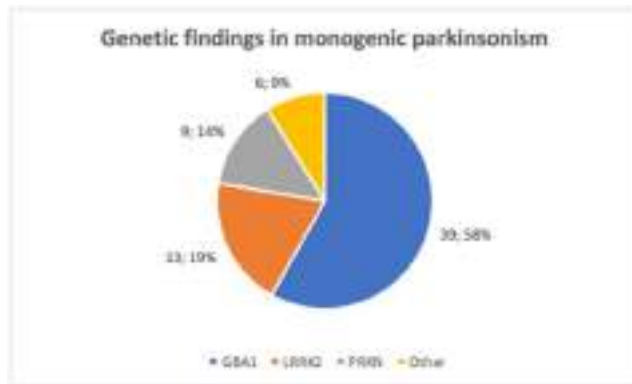


FIGURE 2 Main genetic findings in positive cases.

Methods: NGS multigene panel analysis, MLPA, 409 patients with PD/parkinsonism (377 PD, 32 atypical parkinsonism or DLB).

Results: Overall, 266 genetic variants in genes associated with movement disorders and/or cognitive decline were identified in 204/409 patients; 40 carried multiple variants. According to ACMG-AMP criteria, 98/266 variants were classified as likely pathogenic. Most variants were missense (212/266), followed by frameshift/nonsense variants (23/266); splice-site and copy number variants were less frequent. A diagnosis of monogenic parkinsonism was established in 67 patients (25% of the cohort), all but four with PD. GBA1 mutations were the most frequent finding (39 patients, 58.2% of positive cases), with N370S accounting for 41% of GBA1-PD cases, followed by LRRK2 pathogenic variants (13 patients, 19.4%, half G2019S) and biallelic PRKN variants (9 patients). Rare genetic etiologies (GRN, OPTN) were also identified. Variants of uncertain significance were found in 137 patients, mainly in recessive PD-related genes or other movement disorder genes.

Conclusion: NGS analysis revealed a substantial proportion of monogenic parkinsonism, confirming the central role of GBA1 and LRRK2 and supporting the clinical utility of genetics in movement disorders.

Disclosure: Data partially submitted for 2026 Italian movement disorders congress as abstract/poster Nothing to disclose

Background and aims: Biallelic pathogenic variants in the C19orf12 gene have been shown to cause neurodegeneration with brain iron accumulation 4 (NBIA4), and the same variants have now been identified in Estonia. This study aimed to characterize the phenotype of Estonian NBIA4 patients.

Methods: This retrospective study included eight patients from Tartu University Hospital, born between 1981 and 2010. All patients had radiological and clinical features consistent with NBIA and a homozygous 11-base-pair C19orf12 deletion c.171_181del (p.Gly58Argfs*10). Clinical data were collected from medical records.

Results: In a cohort of eight patients, six were male and two female. At the time of the study, two patients were alive and six were deceased. All patients presented with their first symptoms in early childhood; the mean ($\pm$ SD) age at symptom onset was 4.6 ± 2.1 years. Initial symptoms included vision loss (50%), spasticity of the lower limbs (50%) and gait impairment (37.5%). Optic nerve atrophy was diagnosed in seven of eight patients at a mean age of 9.1 ± 3.1 years. All patients attended school up to the 9th grade and developed movement disorders and neuropsychiatric abnormalities during the disease course. The mean life expectancy of the six deceased patients was 30 ± 4.7 years (range 23–35 years).

Conclusion: The Estonian NBIA4 patients with homozygous C19orf12 deletion c.171_181del exhibited similar clinical features, including pyramidal signs and optic nerve atrophy from early childhood, with survival extending into early adulthood.

Disclosure: An author reports research funding from Estonian Research Council (grant PRG2040).

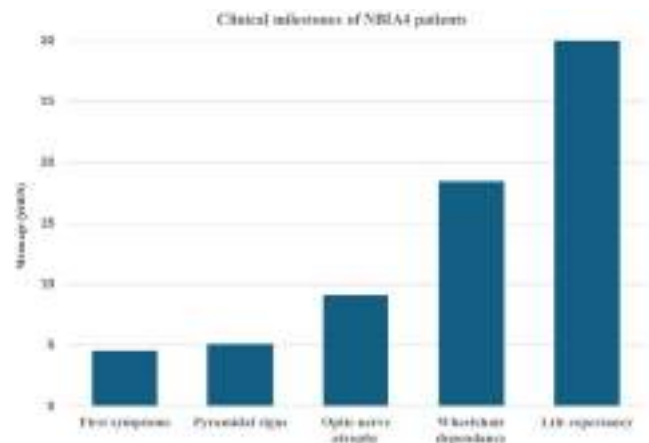


FIGURE 1 Clinical milestones of NBIA4 patients..

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Background and aims: Nuclear factor kappa B1 (NFκB1), a key transcription factor in inflammatory signalling, has been linked to Parkinson's disease (PD) and neurodegeneration. This study intends to clarify the effect of NFκB1 variants in the pathogenesis of PD through a systematic review, and by investigating variants in this gene in the Global Parkinson's Disease (GP2) genotyping data.

Methods: For the systematic review, out of 51 identified records, 11 studies met the inclusion criteria, encompassing a wide methodological spectrum. We also conducted an analysis on genotyping imputed data from GP2 release 10 (33,813 PD cases and 18,714 controls across 11 different ancestries). The NFκB1 gene region was extracted with PLINK2, variants were annotated using ANNOVAR. Fisher's exact test was applied to examine differences between allele frequencies for common variants. Single-nucleotide polymorphism (SNP) associations were also evaluated using logistic regression, with Bonferroni correction applied within each ancestry. To assess the cumulative effect of low-frequency variants we ran burden analyses using RV tests.

Results: Evidence across all studies indicates that NF-κB1 is consistently upregulated in Parkinson's disease and plays a key role in neuroinflammation and dopaminergic neuronal injury. In the large GP2 genotyping data 167 nominal associations with PD risk were observed across multiple ancestries. None remained significant after Bonferroni correction. Burden analyses showed no statistical significance.

Conclusion: While existing literature supports a pathogenic role for NF-κB1 pathway in PD, our large-scale multi-ancestry genetic analysis finds no significant association between NFκB1 variants and PD risk, highlighting the need for future whole-genome sequencing studies to further clarify this relationship.

Disclosure: Nothing to disclose

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Background and aims: The nuclear gene AARS2 encodes the mitochondrial alanyl-tRNA synthetase. Pathogenic biallelic AARS2 variants cause mitochondrial disease, presenting as early-onset cardiomyopathy or leukoencephalopathy, progressive, with ovarian failure (LKENP). LKENP is an autosomal recessive neurodegenerative disorder characterized by gait impairment, ataxia, spasticity, cognitive decline, and premature ovarian failure, usually beginning in young adulthood. Muscle biopsy may show ragged-red fibers and brain MRI typically reveals progressive white-matter abnormalities. We report a patient with LKENP carrying a previously unreported pathogenic AARS2 variant.

Methods: Clinical and neurological data were collected at Sant'Andrea Hospital (Rome). Brain MRI and genetic testing were performed as part of routine diagnostic evaluation.

Results: The patient is a 34-year-old woman with a history of cardiac arrest at birth, early menopause, clumsiness, and

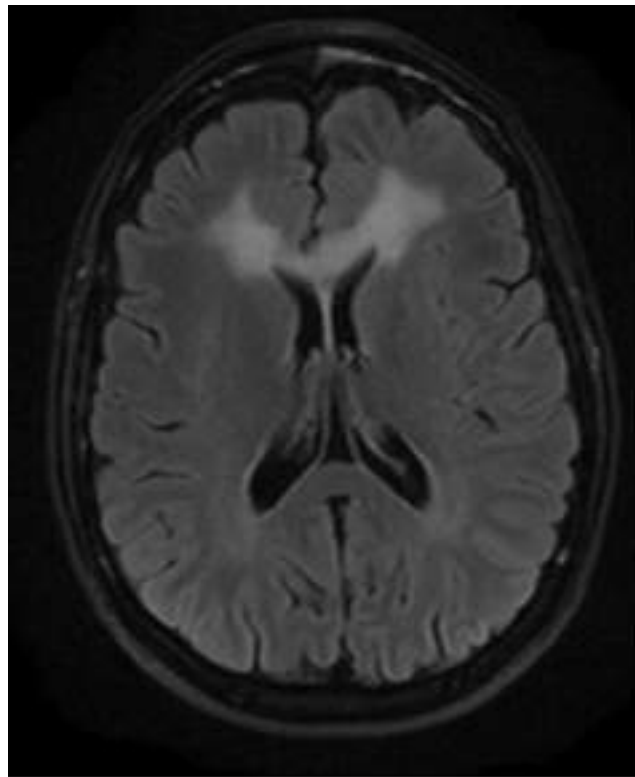


FIGURE 1 Brain MRI (axial FLAIR) obtained at age 34 showing diffuse white matter hyperintensity involving both cerebral hemispheres, mainly in periventricular regions, corpus callosum, and posterior hippocampal areas.

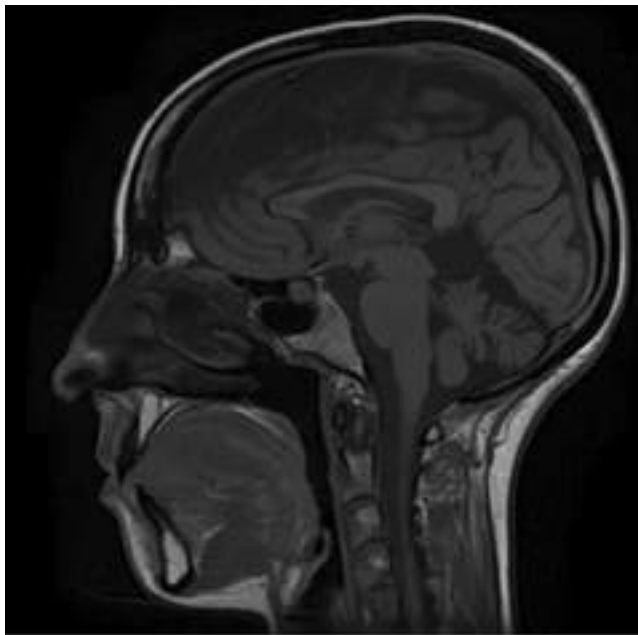


FIGURE 2 Brain MRI (sagittal T1) obtained at age 34 demonstrating thinning of the corpus callosum, bilateral enlargement of frontal pericortical spaces, and mild widening of pericerebellar cisterns.

corrected bilateral strabismus, and a family history of cerebrovascular disease and autism. Karyotype and FMR1 testing at age 24 were normal. At 28, she had a transient loss of consciousness; brain MRI showed small bilateral frontal subcortical lesions, progressing to diffuse white-matter abnormalities five years later. She subsequently developed cognitive decline, dysmetria, and gait impairment. Next-generation sequencing identified two AARS2 variants: a previously unreported frameshift variant c.2821_2832dupTG, p.(Ala945fs*33), classified as pathogenic by ACMG criteria, and the known pathogenic variant c.595C>T, p.(Arg199Cys). Parental testing confirmed compound heterozygosity.

Conclusion: Genetic findings and clinical presentation confirm LKENP diagnosis. No effective therapies are currently available; the patient is receiving symptomatic management, including oral coenzyme Q10 supplementation, which has shown potential benefit in recent reports.

Disclosure: Nothing to disclose

EPO-0488 | Leukoencephalopathy and cerebral venous thrombosis secondary to autosomal recessive MTHFR deficiency: A case report

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Background and aims: Methylenetetrahydrofolate reductase (MTHFR) is an enzyme catalysing the conversion of homocysteine to methionine. Genetic variants that result in MTHFR deficiency may cause hyperhomocysteinemia and its associated complications.

Methods: A middle-aged female presented with progressive unsteady gait and cognitive decline occurring over several years. Clinically, the patient had spastic paraparesis with brisk lower limb reflexes and a scissoring gait. She had a significant background of dietary folate and B12 deficiency which was treated with maintenance B12 and folate supplementation. MRI brain showed confluent periventricular T2-weighted hyperintensities suggestive of an underlying leukodystrophy and identified an incidental venous sinus thrombosis in the junction between the left sigmoid sinus and left internal jugular vein. MRI Whole Spine showed no abnormal cord signal or enhancement, and cerebrospinal fluid analysis was unremarkable. A workup for her venous sinus thrombosis identified a high serum homocysteine (>50.0umol/L) with normal B12, folate levels and low-normal serum methionine levels.

Results: Expanded Exome Proband Analysis confirmed homozygosity for a pathogenic variant c.971A>G in the MTHFR gene. She was diagnosed with autosomal recessive MTHFR deficiency and managed with betaine and B12/folate replacement. Betaine contributes an alternative methyl donor for remethylation of homocysteine to methionine, treating her hyperhomocysteinemia. Apixaban was started to treat her venous sinus thrombosis in the presence of a prothrombotic state.

Conclusion: MTHFR deficiency is a rare but treatable genetic metabolic disorder that may predispose patients to leukoencephalopathy, cognitive decline, spastic paraparesis, spinal cord atrophy, or thrombosis due to hyperhomocysteinemia. It should be suspected in patients with severe hyperhomocysteinemia and hypomethioninemia without folate/B12 deficiency.

Disclosure: Nothing to disclose.

EPO-0489 | Late-onset RANBP2-associated acute necrotizing encephalopathy (ANE1) in two siblings from an Italian family

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Background and aims: Acute Necrotizing Encephalopathy (ANE) is a rare para-infectious encephalopathy of childhood, characterized by symmetric bilateral thalamic brain magnetic resonance imaging (MRI) lesions. Adolescent or adult onset is exceptional. Familial or recurrent forms are linked to heterozygous pathogenic RANBP2 variants (ANE1). We report two siblings with late-onset ANE1, diagnosed after the second presentation.

Methods: Both underwent neurological evaluation, MRI, cerebrospinal fluid analysis, and infectious, autoimmune, and metabolic testing. Genetic investigations included whole-exome sequencing in the second sibling and targeted Sanger sequencing in the first.

Results: The male developed fulminant post-febrile encephalopathy at age 15, rapidly progressing to coma. MRI showed bilateral thalamic, pontine, and external capsule involvement. He recovered with motor and cognitive sequelae. He remained undiagnosed for over 20years. His sister presented at age 20

with milder post-infectious encephalopathy and visual-sensory symptoms. MRI revealed bilateral thalamic lesions involving the external capsules, optic tracts, and mammillary bodies. She received high-dose corticosteroids followed by intravenous immunoglobulin, achieving recovery. Extensive investigations were non-diagnostic in both. In the second sibling, whole-exome sequencing revealed a heterozygous missense RANBP2 variant (c.1754C>T; p.Thr585Met) confirmed by Sanger sequencing in her brother.

Conclusion: A concordant phenotype with variable severity supports a familial ANE1. These cases confirm RANBP2-associated ANE1 as a cause of late-onset encephalopathy and expand the phenotypic spectrum, highlighting intrafamilial variability. Our family is the third reported in Italy and the first with delayed onset and relatively favorable prognosis. Early recognition and immunomodulatory therapy may improve outcomes. Awareness of late-onset ANE1 and RANBP2 testing in adults is warranted.

Disclosure: Nothing to disclose.

EPO-0490 | Genetic insights into somatic symptoms and related disorders versus psychiatric phenotypes

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Background and aims: The genetic mechanisms underlying somatic symptoms and related disorders (SSRD) are poorly known, even though they have a significant impact on health-care. We aimed to identify additional significant loci using statistical genetics methods and first large-scale SSRD GWAS meta-analysis including functional neurological (FND) and somatoform disorders (SomD), and to examine genetic overlap with psychiatric disorders.

Methods: We performed the cross-sectional analysis of SSRD GWAS [Fominykh et al, 2025, 22,203 and 1,831,107 controls] and three psychiatric disorders: anxiety [ANX, N 175,163], major depression [MDD, N 250215] and post-traumatic symptoms disorder [PTSD, N 146,660] from Million Veterans Projects. Genetic correlations were assessed using LDSC. The conditional/conjunctional false discovery rate framework (cond/conjFDR) was used to identify new and shared loci for SSRD. FUMA and Open Targets were used for functional annotations.

Results: LDSC analysis shows significant genetic correlation between SSRD and MDD ($rg=0.8$), ANX ($rg=0.75$) and PTSD ($rg=0.7$) in range with previous publication. We revealed 9 new loci for SSRD using condFDR. ConjFDR analysis identified 2 loci jointly associated with SSRD and MDD, 2 loci – with SSRD and PTSD. The lead SNP common for all three phenotypes was mapped to EXD3 gene. The remaining lead SNPs mapped to BPTF and GPM6A, which is linked to psychiatric and neurological conditions.

Conclusion: This study explores the genetic aspects of the SSRD. We reveal new SNPs associated with SSRD, as well as common SNPs involved in SSRD development and most comorbid psychiatric disorders.

Disclosure: No special disclosures. This work was supported by an RCN grant 324252. MVP dbGaP accession number phs001672.

Ageing and dementia 4

EPO-0491 | Abstract withdrawn

EPO-0492 | Investigating synaptic and neuroinflammation biomarkers in the Alzheimer's disease continuum

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Background and aims: Synaptic dysfunction and neuroinflammation are key mechanisms in Alzheimer's disease (AD), potentially preceding neurodegeneration. Synaptosome-associated protein 25 (SNAP-25) reflects synaptic damage, whereas glial fibrillary acidic protein (GFAP) and soluble TREM2 (sTREM2) index glial activation. Human data on their interplay across the AD continuum remain limited.

Methods: We prospectively enrolled 69 patients with AD (age 70.9 ± 7.0 ; 59% female). Based on Clinical Dementia Rating (CDR), participants were classified as mild cognitive impairment (MCI; CDR=0.5; $n=34$) or dementia (CDR ≥ 1 ; $n=35$). All underwent standardized neuropsychological assessment including MMSE. Cerebrospinal fluid (CSF) levels of GFAP, sTREM2, SNAP-25, and neurofilament light chain (NfL) were measured using Simoa. Associations with AD biomarkers were assessed using Spearman correlations. Longitudinal cognition over two years was analyzed with linear mixed-effects models. Voxel-wise analyses explored relationships with FDG-PET metabolism.

Results: CSF NfL and GFAP were higher in dementia than MCI ($p=0.029$ and $p=0.030$), while sTREM2 and SNAP-25 did not differ. GFAP correlated with p-Tau181 ($\rho=0.41$, $p=0.001$). sTREM2 correlated positively with p-Tau181 ($\rho=0.32$, $p=0.012$) and negatively with the A β 42/40 ratio ($\rho=-0.38$, $p=0.003$). SNAP-25 correlated with t-Tau ($\rho=0.30$, $p=0.019$). Baseline biomarkers were not associated with cognition. Higher NfL predicted faster MMSE decline ($\beta=-0.16$, $p<0.001$). Higher GFAP was associated with reduced FDG uptake in the right medial and orbitofrontal prefrontal cortex ($p<0.001$).

Conclusion: GFAP differentiated clinical stages and was linked to frontal hypometabolism, highlighting the contribution of astrocytic activation to AD-related neurodegeneration. These findings support the relevance of neuroinflammatory biomarkers for staging and monitoring disease progression across the AD continuum.

Disclosure: Nothing to disclose.

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Background and aims: To refine the diagnostic performance of plasma pTau217 for Alzheimer's disease (AD) in a real-world setting, we evaluated double-cutoff strategies and the added discriminative value of GFAP within the pTau217 "gray zone".

Methods: We analyzed 441 consecutive patients from the San Raffaele Memory Center, including 181 with cerebrospinal fluid (CSF) A/T classification. Plasma pTau217, pTau181, GFAP, NFL, and A β 42/40 were measured using automated immunoassays (Lumipulse G600II). The CSF-defined subgroup was used to derive optimal pTau217 double cutoffs across all sensitivity-specificity pairs ≥ 0.90 , quantifying the proportion of indeterminate ("gray") cases. GFAP discriminative ability (AUC) was assessed within each gray zone. Associations between GFAP, age, APOE- ϵ 4, sex, and cognition were explored using generalized additive and linear models.

Results: No pTau217 cutoff combination achieved $\geq 95\%$ sensitivity/specificity while limiting gray cases to $\leq 20\%$. The optimal balanced pair (90% sensitivity, 91% specificity) resulted in 28% indeterminate subjects. Within this gray zone, GFAP discriminated A+ from A- individuals (AUC=0.83), reducing indeterminate cases by 37% when used as a triage marker. GFAP increased with age, particularly in A+ individuals, and remained independently associated with A-positivity after adjustment for APOE and sex ($p < 0.001$). Adding GFAP to pTau217 modestly improved explained MMSE variance ($\Delta R^2 = 0.04$).

Conclusion: Systematic sensitivity-specificity mapping shows that plasma GFAP provides robust complementary value in resolving intermediate pTau217 results. Integrating GFAP into multimarker workflows may enhance diagnostic certainty and support the clinical implementation of blood-based biomarkers for AD.

Disclosure: Giulia Rugarli has nothing to disclose; Giordano Cecchetti received speaker honoraria from Neopharmed Gentili; F Coraglia has nothing to disclose; Edoardo Gioele Spinelli has nothing to disclose; Alma Ghirelli has nothing to disclose; Stefano Pisano has nothing to disclose; Elisa Canu receives research support from the Italian Ministry of Health; Francesca Caso has nothing to disclose; Giuseppe Magnani has nothing to disclose; F Agosta is Associate Editor of NeuroImage: Clinical and the European Journal of Neurology; has received speaker

honoraria from Biogen Idec, Bristol Myers Squibb, Eisai, Eli Lilly, GE Healthcare, Neuraxpharm, and Roche; and receives or has received research supports from the Italian Ministry of Health, the Italian Ministry of University and Research, AriSLA (Fondazione Italiana di Ricerca per la SLA), the European Research Council (ERC), the EU Joint Programme – Neurodegenerative Disease Research (JPND), and Foundation Research on Alzheimer Disease (France). M Filippi is Editor-in-Chief of the Journal of Neurology, Associate Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmed Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla.

EPO-0494 | Integrating retinal OCT and blood biomarkers to characterize cognitive decline across the Alzheimer's disease spectrum

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Background and aims: To investigate the relationship between retinal structural alterations, assessed by optical coherence tomography (OCT), and plasma biomarkers of neurodegeneration across the Alzheimer's disease (AD) continuum, from cognitively unimpaired individuals to patients with dementia.

Methods: A total of 205 participants were recruited at the Neurology Unit, IRCCS San Raffaele Scientific Institute, including subjects with subjective cognitive decline (SCD), mild cognitive impairment (MCI), dementia (DEM), and healthy controls (HC). Plasma levels of A β 42, A β 40, pTau-181, pTau-217, and neurofilament light chain (NfL) were quantified using automated CLEIA technology. OCT was used to measure peripapillary

retinal nerve fiber layer (RNFL), ganglion cell layer (GCL), and inner plexiform layer (IPL) thickness. Correlations between retinal parameters, plasma biomarkers, cognitive performance (MMSE), and carotid ultrasound findings were analyzed, with a one-year longitudinal follow-up in a subsample.

Results: Inner retinal layer thinning—particularly in the GCL and IPL—showed significant associations with plasma NfL and pTau-217 levels, suggesting that retinal structural loss parallels systemic markers of neuroaxonal damage. These correlations were strongest in early disease stages (SCD and SCD_AD) and diminished with advanced cognitive decline. Longitudinal data demonstrated a significant reduction in GCL thickness over one year, reinforcing its sensitivity as an early indicator of neurodegeneration.

Conclusion: Retinal OCT may serve as a non-invasive biomarker for early detection and monitoring of AD-related neurodegeneration. Combining retinal imaging with plasma biomarkers could improve diagnostic accuracy, disease staging, and patient stratification in clinical trials, ultimately advancing personalized therapeutic approaches in Alzheimer's disease.

Disclosure: Giulia Rugarli has nothing to disclose; Giordano Cecchetti received speaker honoraria from Neopharmed Gentili; R Santangelo has nothing to disclose; F Coraglia has nothing to disclose; Edoardo Gioele Spinelli has nothing to disclose; Alma Ghirelli has nothing to disclose; Stefano Pisano has nothing to disclose; Elisa Canu receives research support from the Italian Ministry of Health; Francesca Caso has nothing to disclose; Giuseppe Magnani has nothing to disclose; F Agosta is Associate Editor of *NeuroImage: Clinical* and the *European Journal of Neurology*; has received speaker honoraria from Biogen Idec, Bristol Myers Squibb, Eisai, Eli Lilly, GE Healthcare, Neuraxpharm, and Roche; and receives or has received research supports from the Italian Ministry of Health, the Italian Ministry of University and Research, AriSLA (Fondazione Italiana di Ricerca per la SLA), the European Research Council (ERC), the EU Joint Programme – Neurodegenerative Disease Research (JPND), and Foundation Research on Alzheimer Disease (France). M Filippi is Editor-in-Chief of the *Journal of Neurology*, Associate Editor of *Human Brain Mapping*, *Neurological Sciences*, and *Radiology*; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmed Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla.

EPO-0495 | Early-onset dementia: Etiological and clinical insights from a 10-year cohort

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Background and aims: Early-onset dementia (EOD) represents 5–9% of all dementia cases and differs clinically from late-onset dementia (LOD), often delaying recognition and complicating management. Despite its clinical relevance, large cohort data remain limited, particularly in Türkiye. This study presents a decade-long EOD cohort from a tertiary referral center.

Methods: A total of 1,656 patients were evaluated between 2015–2025. After excluding LODs and secondary dementias, 656 patients with complete CSF assessments were included. Clinical and demographic features were analyzed.

Results: The cohort included 52.1% female. Mean age at onset was similar across groups except for atypical Alzheimer's disease (AD) versus mixed dementia ($p=0.017$). The most common diagnoses were typical AD (24.5%), atypical AD (18.1%), and frontotemporal dementia (FTD) (15.6%). Posterior cortical atrophy (PCA) was the leading atypical AD subtype (30.3%), while behavioral-variant FTD (bvFTD) predominated within FTD (47.1%). When staged at the time of biomarker assessment, dementia was more frequent in typical AD than atypical AD, and in atypical AD than FTD ($p=0.001$), whereas no difference between FTD and typical AD ($p=0.759$).

Conclusion: This cohort resembles previous EOD populations but differs in etiological distribution and clinical stage at diagnosis. Findings underscore the importance of early symptom recognition and timely diagnostic evaluation, given that EOD follows a distinct clinical course compared to LOD. As a decade-long cohort from a high-volume tertiary center, this study may reflect the broader population, addressing gaps in epidemiological and etiological data on EOD globally and within Türkiye.

Disclosure: Nothing to disclose.

EPO-0496 | GFPaint: A novel technique for visualizing alpha-synuclein strains across synucleinopathies

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Background and aims: Misfolded alpha-synuclein aggregates are a defining feature of Parkinson's disease, dementia with Lewy bodies, and multiple system atrophy. However, distinguishing disease-relevant alpha-synuclein strains remains a major challenge. The objective of this study was to develop a single-molecule approach to discriminate alpha-synuclein strain polymorphs derived from different synucleinopathies and defined synthetic conditions based on selective protein interactions.

Methods: We developed GFPaint, a single-molecule fluorescence assay combining cell-free expression of green fluorescent



FIGURE 1 Conceptual schematic of alpha-synuclein fibrillation and strain diversity. Chaperone-mediated proteostasis maintains soluble alpha-synuclein, whereas pathogenic conditions promote formation of distinct fibril strains with differential chaperone engagement.

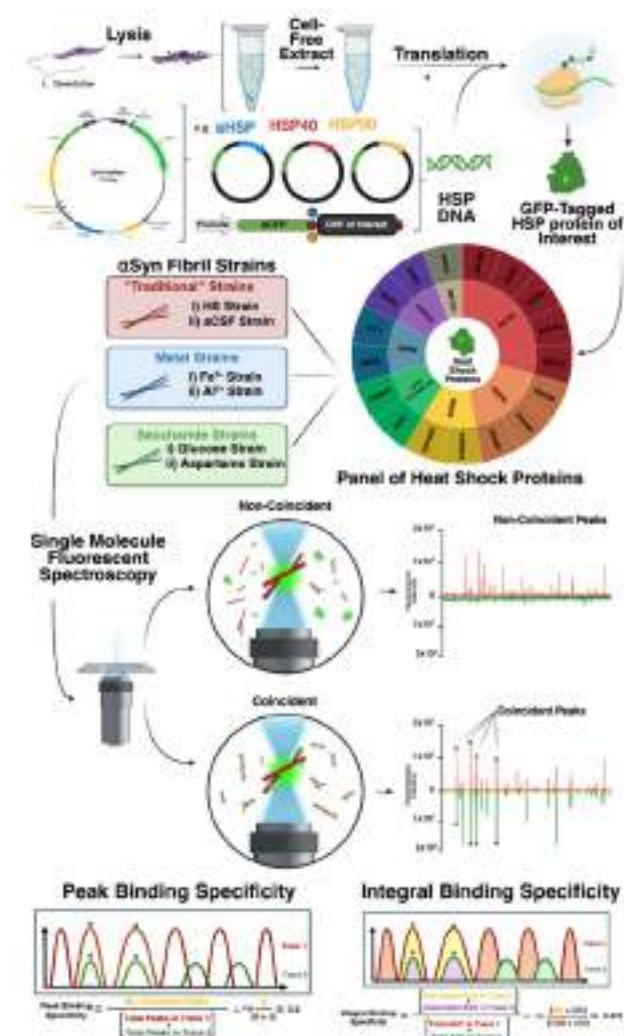


FIGURE 2 Overview of the GFPaint workflow. Green fluorescent protein tagged heat shock proteins are expressed in a cell-free system and incubated with defined alpha-synuclein fibril strains. Dual-channel single-molecule data produced.

protein tagged heat shock proteins with dual-channel coincidence spectroscopy. A panel of chaperones spanning HSP40, HSP70, HSP90, and small heat shock protein families was incubated with alpha-synuclein fibrils generated under traditional, metal ion, and saccharide conditions, as well as with brain-derived samples from Parkinson's disease, dementia with

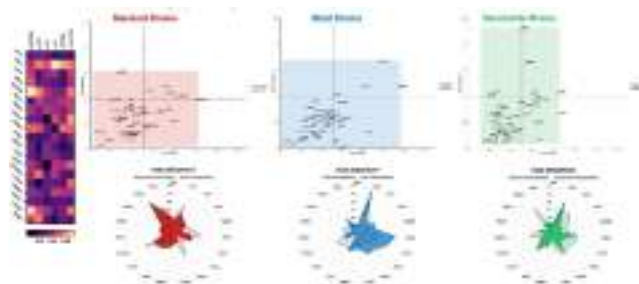


FIGURE 3 Strain-dependent protein interaction profiles across alpha-synuclein fibril classes. Heat maps, quadrant analyses, and radar plots reveal distinct peak and integral binding specificity patterns for standard, metal ion, and saccharide induced aSyn strains.

Lewy bodies, and multiple system atrophy. Binding interactions were quantified using coincidence, binding affinity, and binding specificity metrics, including peak- and integral-based analyses. **Results:** GFPaint revealed pronounced strain-dependent differences in chaperone engagement. HSP40 family members, particularly DNAJB1 and DNAJB6, showed strong and selective binding to specific alpha-synuclein polymorphs, whereas HSP70 and HSP90 family members displayed weak or nonspecific interactions. Distinct binding fingerprints differentiated Parkinson's disease, dementia with Lewy bodies, and multiple system atrophy brain samples and aligned with defined synthetic strain profiles.

Conclusion: GFPaint enables single-molecule strainotyping of alpha-synuclein based on selective protein interactions, providing a scalable platform to distinguish disease-specific alpha-synuclein conformations across synucleinopathies.

Disclosure: Nothing to disclose.

EPO-0497 | Relationship between cortical neurotransmitter target impairment and core features of behavioural variant frontotemporal dementia

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Background and aims: Behavioural variant frontotemporal dementia (bvFTD) is characterized by distinct patterns of neurodegeneration and neurotransmitter deficits, but the relationship between specific neurotransmitter vulnerability and core symptoms of bvFTD is poorly explored. We aimed to investigate the spatial colocalization between hypometabolism and normative distribution of neurotransmitter receptors, assessing their association with the main neuropsychiatric and cognitive symptoms of bvFTD.

Methods: We analysed [18F]FDG-PET scans from bvFTD patients compared to healthy controls to derive relative hypometabolism maps. Using the JuSpace toolbox, we computed spatial correlations between hypometabolism and normative density maps of the neurotransmitter systems target known to be impaired in bvFTD, such as serotonergic (5HT), dopaminergic (D), GABAergic (GABA), and glutamatergic (mGluR) receptors.

Multivariate logistic or linear regression models were applied to evaluate the association between the receptor-specific spatial colocalization coefficients, bvFTD-related subitems of the Neuropsychiatric Inventory (NPI), and executive performance.

Results: In bvFTD patients ($n = 86$; age 73 ± 7 years), the hypometabolism distribution significantly colocalized with the distribution of 5HT1a ($r = -0.24$), 5HT2a ($r = -0.15$), 5HT4 ($r = -0.21$), D1 ($r = -0.29$), D2 ($r = -0.21$), and mGluR5 ($r = -0.21$) receptors (all $p < 0.001$). In multivariate models, apathy presence was associated with 5HT2a vulnerability (OR = 0.029; $p = 0.040$), while disinhibition with D1 (OR = 0.058; $p = 0.038$) and D2 (OR = 0.098; $p = 0.049$) vulnerability. mGluR5 impairment was associated with Trail Making Test A (TMT-A; $B = -0.060$; $p = 0.033$), TMT-B ($B = -0.075$; $p = 0.023$), Corsi test (OR:3.56; $p = 0.03$) and phonemic fluency ($B = 0.76$; $p = 0.042$), while D1 was associated with TMT-B ($B = -0.06$; $p = 0.040$).

Conclusion: Our findings highlighted a distinct receptor vulnerability in bvTFD patients, which correlated with disease-specific symptoms, suggesting possible pharmacological targets. Further studies should evaluate the implications at the individual-patient level, potentially guiding therapeutic strategies.

Disclosure: The authors declare no conflicts of interest.

EPO-0498 | 3D convolutional neural network predicts MCI-to-AD conversion: Systematic review and meta-analysis

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Background and aims: Early identification of patients with Mild Cognitive Impairment (MCI) who are likely to progress to Alzheimer disease (AD) remains a major clinical challenge. In recent years, three-dimensional (3D) deep learning approaches have been increasingly applied to neuroimaging data to improve risk stratification. This meta-analysis examines the performance of 3D deep learning models in predicting MCI-to-AD conversion and compares their results with two-dimensional (2D) models, traditional machine learning techniques, and physician-based assessment.

Methods: A total of 1,987 patients diagnosed with MCI were included. The intervention consisted of 3D deep learning architectures, primarily 3D Convolutional Neural Networks, applied to volumetric neuroimaging modalities, including resting-state functional Magnetic Resonance Imaging and Positron Emission Tomography. Comparator methods included 2D Convolutional Neural Networks, conventional machine learning models, and

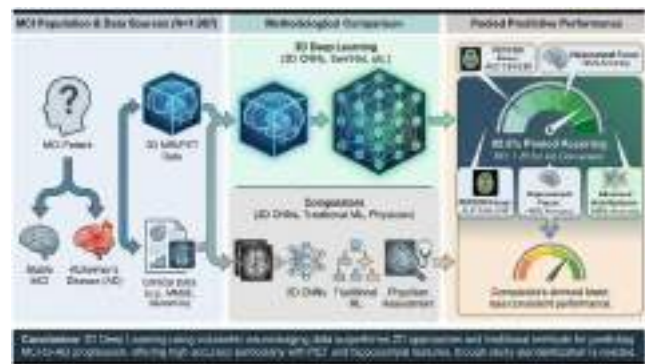


FIGURE 1 Graphical abstract

physician consensus diagnoses. The primary outcomes were the pooled values of classification accuracy and Relative Risk for the conversion to Alzheimer's disease.

Results: Across the five studies, the overall accuracy of the 3D CNN models stood at 92.6% with a relative risk of 1.85 for the conversion to AD, clearly outperforming the consensus of doctors (69.5% accuracy). The AUC values for the discrimination of the FDG-PET 3D CNN were excellent, with values between 0.93 and 0.99. The attention models SwinNet-CATNet had an accuracy of 98.2% for the staging of AD, and the hippocampus-centered PCANet-BLS models surpassed the 95% threshold.

Conclusion: 3D deep learning has a substantial advantage over 2D models, as well as clinical assessment, in predicting MCI-to-AD transition ($p < 0.01$). Models including PET + hippocampal data have high discrimination.

Disclosure: Nothing to disclose.

EPO-0499 | Impact of the 2024 NIA-AA revised criteria on biological AD classification in a clinical Memory cohort from Central Italy

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Background and aims: Updates in Alzheimer's disease (AD) diagnostic criteria have significantly influenced clinical practice over the past decade. The AT (N) research framework introduced by NIA-AA in 2018 stratifies individuals based on biomarkers of amyloid deposition (A), tau pathology (T), and neurodegeneration (N). The 2024 revision introduced two new core biomarkers—p-tau/A β 42 and t-tau/A β 42 ratios—and defined a hierarchy of Core 1 and Core 2 markers, establishing that the presence of just one Core 1 biomarker is sufficient to define biological AD.

Methods: To assess the impact of the updated NIA-AA 2024 criteria, we retrospectively re-evaluated cerebrospinal fluid (CSF) profiles of 81 patients (mean age 68 years; 46 male; mean education 10 years) at our cognitive neurology clinic, including patients with mild cognitive impairment and established dementia.

Results: Of the 81 patients, 27 (33%) didn't meet the 2018 criteria for biological AD. Among these, 10 patients (37%) were reclassified as having biological AD according to the 2024 criteria. All reclassified patients showed pathological values in p-tau/A β 42,

t-tau/A β 42, and A β 42 or A β 42/40 ratios, except for one case, who showed an isolated elevation of the t-tau/A β 42 ratio. This patient was later diagnosed with prion disease, highlighting the need for careful interpretation of neurodegeneration markers in non-AD conditions. P-tau alone was the least informative marker in triggering diagnostic reclassification. In contrast, p-tau/A β 42, A β 42, and A β 42/40 ratio performed consistently and equivalently in identifying amyloid pathology.

Conclusion: The 2024 NIA-AA criteria substantially increase the number of individuals meeting the definition of biological AD.

Disclosure: Nothing to disclose.

EPO-0500 | Fluid Neurofilament Light captures antero/posterior cortex metabolic gradient in behavioral variant of frontotemporal dementia

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Background and aims: Neurofilament light (NfL) is a biofluid marker of axonal injury in frontotemporal lobar degeneration (FTLD), but its regional metabolic correlates on [¹⁸F]FDG PET remain poorly defined. We aimed to characterize cortical [¹⁸F]FDG correlates of NfL in a well-defined bvFTD cohort and to explore associated cognitive effects.

Methods: We studied 48 bvFTD patients who underwent resting-state [¹⁸F]FDG-PET and serum and/or CSF NfL sampling. CSF NfL values were converted to serum-equivalent concentrations using a fixed factor (serum = 0.43 × CSF) and log-transformed. [¹⁸F]FDG uptake was parcellated with the Desikan-Killiany atlas (68 cortical ROIs), normalized to the pons, and related to log-transformed NfL using age-adjusted linear models. ROIs showing significant or trend-level NfL effects were grouped into anterior and posterior composites. From these we derived an A/P gradient and a posterior-residual index (posterior variance unexplained by anterior uptake and age, indexing relative posterior sparing), which were related to logNfL. Associations with neuropsychological scores were tested in separate age-adjusted models.

Results: Higher NfL was significantly associated with lower [¹⁸F]FDG uptake in frontal regions and higher uptake in posterior cortices. Anterior-posterior correlations were weak, and frontal uptake did not predict posterior uptake. The posterior-residual index correlated positively with logNfL ($r=0.43$, $p=0.003$), indicating that increasing NfL values are linked to a steeper A/P metabolic gradient—greater frontal hypometabolism with relative posterior sparing. At the uncorrected level,

the A/P gradient showed small, directionally consistent associations with executive dysfunction.

Conclusion: Integrating NfL with [¹⁸F]FDG PET may help quantify biologically meaningful fronto-posterior imbalance in FTLD and support patient stratification.

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EPO-0501 | Plasma microglia related biomarker predicting faster cognitive decline

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Background and aims: Stratifying subgroups with rapid cognitive decline is a critical challenge in the management and research of Alzheimer's disease (AD) and frontotemporal lobar degeneration (FTLD). Previously identified as a marker of microglial dysfunction in amyotrophic lateral sclerosis, we investigated whether plasma microRNA-214-3p could serve as a predictor for rapid progression across the AD-FTLD spectrum.

Methods: We analyzed a multicenter prospective cohort of 169 participants, including AD, FTLD, and control groups. We evaluated plasma microRNA-214-3p, cerebrospinal fluid (CSF) amyloid and inflammatory markers, APOE4 status, and longitudinal Mini-Mental State Examination (MMSE) scores. Fast progression was defined as an annual MMSE decline of >3 points. Additionally, we assessed phagocytic function and pathology in induced microglial-like cells from participants.

Results: Plasma microRNA-214-3p was the strongest predictor of cognitive decline among all biomarkers. Baseline levels demonstrated excellent accuracy in identifying fast progressors (AUC=0.92), significantly outperforming CSF neurofilament light chain (AUC=0.73). High baseline levels correlated with rapid MMSE decline, exhibiting a synergistic interaction with APOE4. Longitudinal increases in microRNA-214-3p independently predicted decline, particularly in individuals with lower amyloid burden. Mechanistically, fast progressors displayed elevated microRNA-214-3p alongside reduced microglial phagocytosis and increased phosphorylated TDP-43 levels.

Conclusion: Plasma microRNA-214-3p is a sensitive blood-based surrogate for microglial phagocytic dysfunction. It effectively identifies individuals at risk of rapid cognitive decline across the AD-FTLD spectrum, offering significant prognostic value, especially for APOE4 carriers or those with non-amyloid pathologies such as TDP-43.

Disclosure: Funding: This research was supported by a grant of the Korea Dementia Research Project through the Korea Dementia Research Center (KDRC), funded by the Ministry of Health & Welfare and Ministry of Science and ICT, Republic of Korea (grant number: RS-2024-00348451). The authors have no interests to disclosure.

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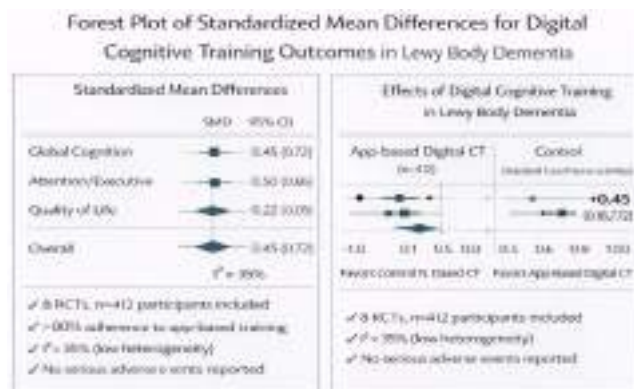
Background and aims: Lewy body dementia (LBD) is associated with rapid cognitive and functional decline, and pharmacological treatment options remain limited. Digital cognitive training (CT) has emerged as a potential non-pharmacological strategy to improve cognitive outcomes in neurodegenerative disorders.

Methods: This systematic review and meta-analysis followed PRISMA guidelines. PubMed, Embase, PsycINFO, Cochrane Library, and ClinicalTrials.gov were searched (January 2010-December 2024) for randomized controlled trials investigating app-based cognitive training in Lewy body dementia. The primary outcome was change in global cognition (MMSE or MoCA). Data were pooled using random-effects models, with standardized mean differences (SMD), 95% confidence intervals (CI), and I^2 for heterogeneity assessment.

Results: From 845 screened records, 8 RCTs ($n=412$) were included. Digital cognitive training significantly improved global cognition compared with control (SMD = 0.45, 95% CI 0.18–0.72; $I^2 = 35\%$). Greater effects were observed in attention and executive function (SMD = 0.58, 95% CI 0.30–0.86). Effects on quality of life were positive but not statistically significant (SMD = 0.22, 95% CI –0.05–0.49). Adherence to app-based training was high (>80%), and no serious adverse events were reported.

Conclusion: Structured digital cognitive training is a safe and effective non-pharmacological intervention for improving cognitive function in Lewy body dementia. These findings support the integration of validated digital applications as adjunct therapy in LBD management.

Disclosure: Nothing to disclose.



Forest plot showing positive effects of app-based digital cognitive training on cognitive outcomes in Lewy body dementia.

EPO-0503 | Making the invisible audible: A real-world connected speech study in myasthenia gravis

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Background and aims: Bulbar involvement in myasthenia gravis (MG) impairs voice and speech, yet subtle changes may be overlooked.

Methods: We conducted a cross-sectional study of consecutively enrolled adults with confirmed MG at a single center. Participants completed standardized voice recordings and contemporaneous clinical assessments (Myasthenia Gravis–Activities of Daily Living [MG-ADL] and revised Myasthenia Gravis Quality of Life [rMG-QOL15]). Tasks included sustained vowels, standardized reading, and spontaneous speech. Acoustic metrics comprised root-mean-square intensity (RMS, dB) and fundamental frequency (F0, Hz).

Results: Seventeen patients were enrolled (median age 58 years; 52.9% female). Median MG-ADL was 6.0 and mean rMG-QOL15 was 16.6, with no significant correlation ($r=0.09$, $p=0.74$). Male participants exhibited significant intensity reductions in connected speech: reading (–3.22 dB, IQR –5.98 to –2.09; $p_{BH}=0.031$; $r=0.84$) and spontaneous speech (–5.46 dB, IQR –6.11 to –4.93; $p_{BH}=0.031$; $r=0.84$). Female participants showed smaller, non-significant changes (–2.2 dB; all $p_{BH} > 0.7$). F0 shifts were modest and non-significant after correction. Across the cohort, MG-ADL correlated with both RMS and F0 drops in unadjusted models but not after adjusting for sex. rMG-QOL15 showed no overall association, although females demonstrated a correlation between F0 drop and QOL burden ($r=0.77$, $p=0.014$).

Conclusion: Connected-speech intensity decrements, particularly in males, represent a robust acoustic marker of bulbar involvement in MG not captured by patient-reported outcomes.

Disclosure: Nothing to disclose related to the submitted manuscript

EPO-0504 | Demyelinating diseases and Sjögren's Syndrome: Clinical intersections and pathogenetic links

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Background and aims: The pathological relationship between demyelinating diseases and Sjögren's syndrome (SS) remains incompletely understood. Autoimmune mechanisms play a central role in the pathogenesis of both conditions, leading to overlapping clinical manifestations and responses to similar immunomodulatory therapies. We aimed to evaluate the frequency of coexisting SS in patients with suspected demyelinating diseases and to assess their clinical, serological, and histological features.

Methods: Over a one-year period, 52 patients admitted to the neurology ward with suspected demyelinating disease and

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Background and aims: Thymoma-associated Myasthenia Gravis (TAMG) represents a clinical entity with poorer prognosis when compared to other MG subtypes. Outcome predictors are lacking. We conducted a retrospective study to identify clinical variables associated with favorable MG outcome as minimal manifestation or better according to MGFA post-intervention status (MGFA-PIS).

Methods: We retrieved information from clinical records of consecutive TAMG patients seen at our Institution between January 2005 and May 2025. Outcome predictors as patient demographics, MG severity (at onset and at maximum severity) and related treatment, pathologic variables, thymoma stage (according to Masaoka classification) and histotypes (according to WHO classification) were assessed by univariate and multivariate analyses performed in R 4.3.1 Software Version. A favorable outcome was defined as achieving MM-or-better.

Results: A total of 118 patients [59 (50%) females, median age (IQR, years)=63 (54–78)] with acetylcholine receptor antibody positive MG (AChR-MG), and histologically confirmed thymoma were included. The outcome was assessed by MGFA-PIS at the last follow-up. Fourteen cases were excluded either due to missing data or to follow-up period < 1 year. The univariate logistic regression revealed that the presence of autoimmune comorbidities (AIC) known or unknown to be linked to thymoma, and occurring at any time of patient history, was significantly associated with a lower chance to achieve ‘MM-or-better’, OR 0.25 (95% CI, 0.08–0.72; $p=0.006$). There were no significant



FIGURE 1: Study population and results.

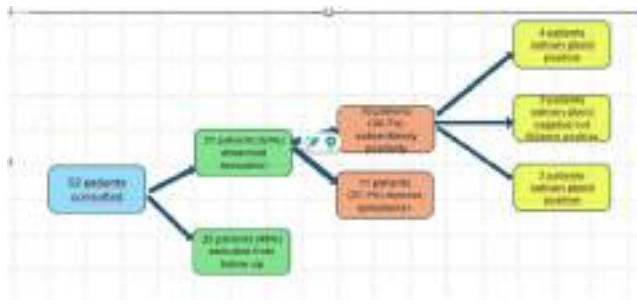


FIGURE 1: Study population and results.



FIGURE 1: Study population and results.

consulted by rheumatology were included. All patients underwent systematic rheumatological evaluation, including assessment of oral and ocular dryness. Antinuclear antibodies (ANA) and ANA profiles were analyzed in all cases. Salivary gland biopsy was performed in patients with sicca symptoms and/or relevant autoantibody positivity. Patients without clinical or serological evidence suggestive of SS were excluded from further evaluation.

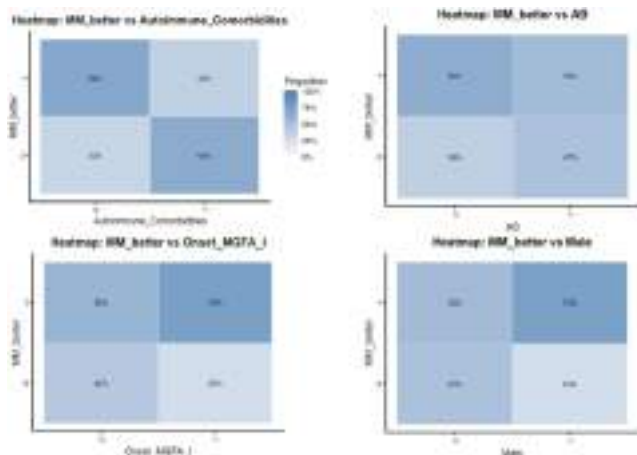
Results: Among the 52 patients, 14 (26.9%) reported oral dryness and 7 (13.4%) ocular dryness. ANA positivity was detected in 11 patients (21.1%), and SS-related autoantibodies were identified in 5 patients (10.5%). Salivary gland biopsy was performed in 27 patients and yielded positive results in 6 cases. Additionally, 3 patients with strong clinical and serological findings but negative biopsy results were diagnosed with SS. Overall, SS was diagnosed in 9 patients (17.3%). Concomitant diagnoses included MS, NMO, and myositis.

Conclusion: Sicca symptoms and autoantibody positivity are more frequent in patients with demyelinating diseases than expected in the general population. Even in the absence of autoantibody positivity, SS should be considered when suggestive clinical features are present.

Disclosure: Nothing to disclose.

Univariate logistic regression (Outcome: MM or better)			
Predictor	OR	95% CI	p
Male sex	2.65	1.23–5.87	0.014
Age at onset (years)	1.00	0.98–1.03	0.868
Late onset (50–65 years)	1.23	0.57–2.63	0.599
Very late onset (> 65 years)	0.70	0.30–2.07	0.611
Autoimmune comorbidities	0.22	0.07–0.63	0.006
Thymoma-associated MG	1.00	0.25–3.52	0.996
MGFA class I at onset	2.18	0.96–5.28	0.071
MGFA II–V at onset	0.56	0.06–4.77	0.564
Thymectomy > 6 months	1.21	0.54–2.72	0.641
1ST start > 6 months	0.61	0.24–1.56	0.302
Cortical histotype	1.24	0.48–3.12	0.652
Antibody positivity	0.63	0.22–1.83	0.389

Univariate Analysis for predictors included across the TAMG cohort followed at our institution



Heatmap by single predictor prevalence across the cohort of the study.

differences in follow-up duration between patients with and without AICs.

Conclusion: AICs can influence clinical outcome of patients with TAMG and should be carefully considered in therapeutic decision-making

Disclosure:

EPO-0506 | Serum NfL and GFAP as differential biomarkers of response to dimethyl fumarate and ocrelizumab in multiple sclerosis

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Background and aims: Multiple Sclerosis (MS) is a chronic inflammatory and neurodegenerative disease. Quantifying neuronal damage is a critical step for patient care and treatment selection. Neurofilament light chain (sNfL) and glial fibrillary

acidic protein (sGFAP) are the two most promising serum biomarkers reflecting neuronal damage and astroglial activation, respectively.

Methods: This study analyzed sNfL and sGFAP in 177 consecutive MS patients and 71 healthy controls (HCs) enrolled at Ospedale San Paolo in Milan using SIMOA technology, classifying patients as Responders (R) or Non-Responders (NR) based on "No Evidence of Disease Activity 3" (NEDA-3) status after two years of treatment. Longitudinal analyses were performed for Dimethyl fumarate (DMF) and Ocrelizumab (OCRE) treatment.

Results: Biomarker-age correlation analysis in HCs confirmed correlation between either NfL and GFAP with age and cut-off values specific for age decades were calculated. Both biomarkers were higher in MS patients compared to HCs. sNfL showed a significant increase in NR patients overall. In contrast, sGFAP was elevated in the low-efficacy treatment agents (LETAs) NR group and also in the DMF NR subgroup, suggesting that it monitors persistent astrogliosis. Longitudinal analysis showed that either biomarkers decreased during DMF treatment after one year. During OCRE treatment sNfL rapidly reduced to HC levels within one year, while sGFAP decreased only after two years.

Conclusion: This study highlights the differential temporal dynamics of sNfL and sGFAP under ocrelizumab and the potential role of sGFAP as treatment-response biomarker in DMF-treated patients.

Disclosure: AM nothing to disclose AC nothing to disclose CM nothing to disclose AZ nothing to disclose CC nothing to disclose TZ received travel support from Merck, Biogen and Novartis FM nothing to disclose MS nothing to disclose GP nothing to disclose PS nothing to disclose MM nothing to disclose AP nothing to disclose FMB received travel support from Roche, Biogen and Novartis and speaking honoraria from Roche

EPO-0507 | Uncovering novel antigenic targets of CNS-reactive autoantibodies in autoimmune neurological syndromes

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Background and aims: The identification of auto-antibodies (auto-Abs) and their antigens has improved the diagnosis and stratification of autoimmune neurological syndromes such as autoimmune encephalitis (AIE) and neuromyelitis optica spectrum disorders (NMOSD). Yet, 7–30% of AIE/NMOSD patients remain seronegative. Auto-Abs are also reported in multiple sclerosis, but their targets and pathogenic relevance remain unclear. Our objective is to identify novel antigenic targets to advance our understanding of disease immunopathology and improve diagnosis/prognosis.

Methods: We established a human induced pluripotent stem cell (hiPSC)-derived astrocyte or neuron cell-based assay (CBA)

to identify patients with astrocyte- and/or neuron-reactive IgG in serum or cerebrospinal fluid. Memory B cells from selected patients were immortalized to generate B lymphoblastoid cell lines (BCLs) secreting monoclonal antibodies (mAbs). These mAbs were screened using the CBA, and B-cell receptor variable regions of autoreactive clones were sequenced for recombinant mAb production, enabling antigen discovery. Enrolled patients presented with inflammatory neurological diseases (IND) including people with MS (pwMS), as well as non-IND control patients.

Results: We identified astrocyte- and neuron-reactive auto-Abs in 10 seronegative AIE/NMOSD patients; 15 astrocyte- and 14 neuron-reactive auto-Abs among 97 pwMS; and astrocyte-reactive auto-Abs in one other-IND (OIND) patient seronegative for known auto-Ags. We successfully produced astrocyte-reactive mAbs from selected patients (AQP4-positive NMO, pwMS and OIND), which are being studied for downstream antigen discovery.

Conclusion: We present a robust pipeline combining hiPSC-derived CBA with patient-derived mAbs for Ag discovery. This approach enables identification of novel autoreactive targets in pwMS and seronegative autoimmune neurological patients, enhancing immunopathological understanding and diagnostic accuracy.

Disclosure: Caroline Pot is supported by the Panacée Foundation, the Swiss MS Society and the Swiss National Science Foundation.

EPO-0508 | Progression independent of relapse and MRI activity in disability accumulation throughout the course of multiple sclerosis

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Background and aims: Progression independent of relapses and MRI activity (PIRMA) is an important driving factor of disability accumulation in relapsing multiple sclerosis (MS) trial-cohorts treated with high-efficacy therapies (HET). It is unclear how PIRMA during HET relates to PIRMA as observed in (untreated) early-stage and primary progressive MS. In this study we assessed PIRMA in 3 independent cohorts to explore applicability of definitions and overlap in clinical characteristics associated with PIRMA.

Methods: Repetitive data on EDSS, PDDS, T25FW, SDMT and walking distance were collected from a retrospective HET-cohort ($N=369$), a prospective first attack of demyelination cohort (PROUD-cohort, $N=458$) and a prospective PPMS cohort

(SPIN-P-cohort, $N=188$). Worsening of these outcomes was defined as potentially attributable to relapses or MRI-activity if any of these events were reported between measurements or <30 days after new measurement. Otherwise, worsening was attributed to PIRMA.

Results: Median age at respective baseline was 34 years (HET-cohort), 33 (PROUD-cohort) and 60 (SPIN-P-cohort). Both in HET-cohort and PROUD-cohort around 70% was female, while in SPIN-P-cohort 52% was female. EDSS and PDDS were the most-used measurements in all cohorts (HET-cohort $N=630$, $N=261$; PROUD-cohort $N=854$, $N=478$; SPIN-P-cohort $N=463$, $N=467$, respectively). Worsening in outcome measures during follow-up was low in all cohorts (20.5% (268/1311), 11.6% (179/1537), 18.5% (320/1729), respectively). The proportion of worsening attributed to PIRMA was 63.1 (HET-cohort), 58.7 (PROUD-cohort) and 92.5% (SPIN-P-cohort) respectively.

Conclusion: PIRMA is proportionally the most contributing factor of disability accumulation throughout the whole disease course of MS. Participant characteristics associated with worsening of EDSS and PDDS will be presented.

Disclosure: Conflicts of interest K. van Tulder: Nothing to disclose. C. Corsten: Nothing to disclose R. Klein Kranenbarg: Nothing to disclose I. Smets: has received lecture fees from Merck, Biogen Idec and Sanofi. J. de Beukelaar: Nothing to disclose. M. M. van Luijn: has received research support from EMD Serono, Merck, Novartis, GSK and Idorsia Pharmaceutical Ltd. B. Wokke: Nothing to disclose J. Smolders: reports grant for scientific research from Roche and Siemens Healthineers and received speaker and/or consultancy fee of Biogen, Merck, Novartis, Roche and Sanofi.

EPO-0509 | Population-normalised smartwatch Z-scores quantify real-world disability in multiple sclerosis

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Background and aims: Wearables quantify real-world function, but missing reference values limit clinical interpretation. Objective: to test whether population-normalised smartwatch Z-scores and a composite digital deviation from norm track disability in multiple sclerosis.

Methods: We pooled Withings ScanWatch data from prospective observational studies. Participants with ≥ 30 valid days

and complete age, sex, body mass index (BMI), and Expanded Disability Status Scale (EDSS) data were included. Steps, moderate-intensity activity, total sleep time, and nightly resting heart rate were converted to matched Z-scores using an aggregated reference dataset of 2,655,433 users (2023–2024). Digital Deviation Composite (DDC2-4) averaged only abnormal deviations across 2–4 domains.

Results: 143/180 participants met criteria. Step and activity Z-scores declined with disability, becoming significantly below norms from EDSS 4.0–4.5 (median step Z-score -0.92) and reaching -1.66 at EDSS ≥ 7.0 (Figure 1). EDSS correlated with steps (Spearman $r = -0.71$) and activity ($r = -0.63$), weakly with resting heart rate ($r = 0.27$), and not with sleep ($r = 0.05$). DDC2, DDC3, and DDC4 correlated with EDSS ($r = 0.72, 0.68, 0.66$) (Figure 2). Serum glial fibrillary acidic protein (GFAP) showed weak positive associations with DDC3 and DDC4, whereas serum neurofilament light chain (NfL) showed none; in migraine controls ($n = 45$), activity Z-scores were near norms.

Conclusion: Population-normalised wearable Z-scores and DDCs provide an interpretable distance-from-norm framework for multiple sclerosis disability and may complement episodic clinic-based scales; a public web app supports Z-score

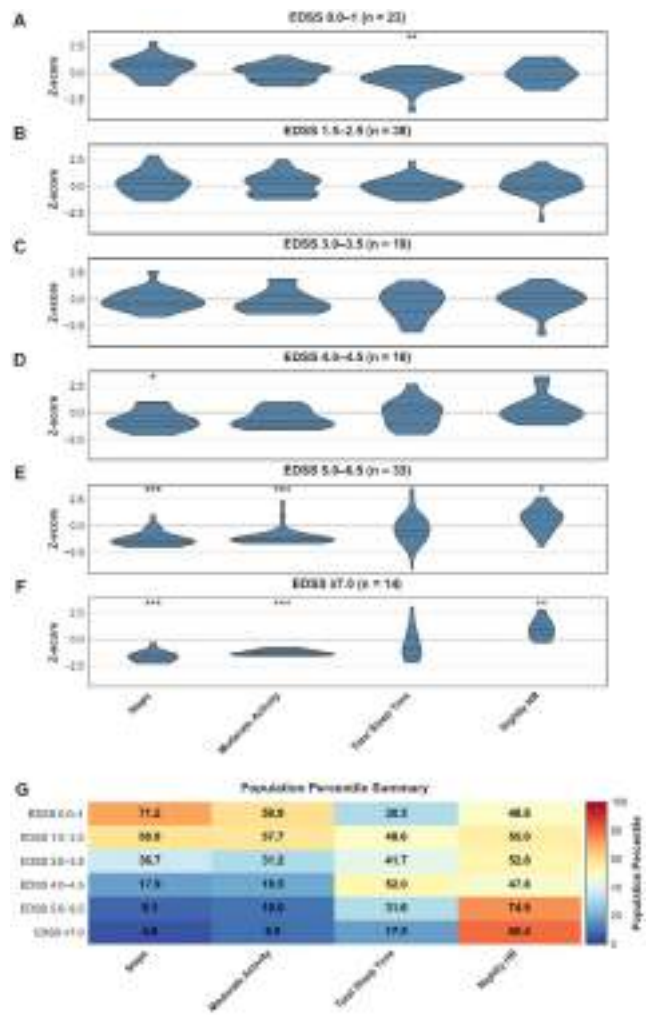


FIGURE 1 Age-, sex-, and BMI-matched smartwatch Z-scores (steps, moderate activity, sleep time, resting heart rate) across Expanded Disability Status Scale (EDSS) groups; heatmap shows median population percentiles. $n = 143$.

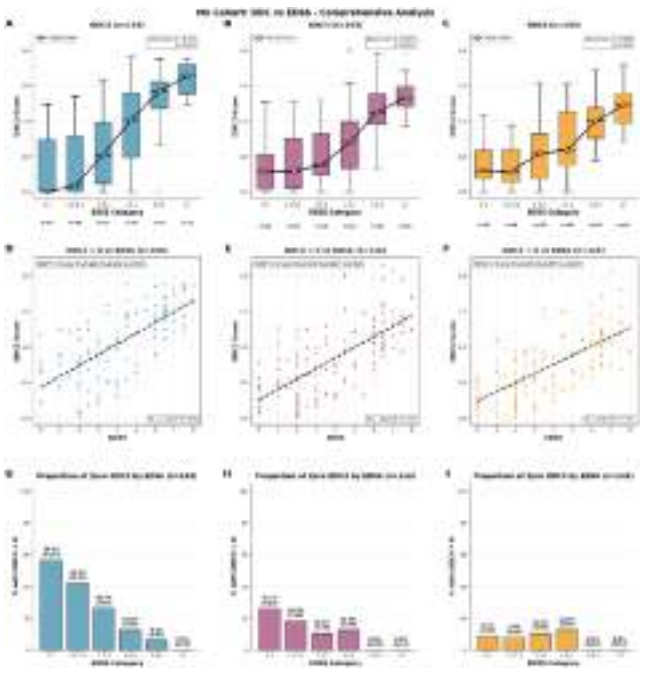


FIGURE 2 Digital Deviation Composite (DDC2-4) versus Expanded Disability Status Scale (EDSS): boxplots by EDSS group, scatter among participants with DDC > 0 (linear fit), and bars showing proportion with DDC = 0. $n = 143$.

calculation. Longitudinal studies should test responsiveness and prognostic value.

Disclosure: Supported by Deutsche Forschungsgemeinschaft (DFG) 493659010; Withings provided aggregated reference data; Benjamin Vittrant is employed by Withings and Bastien Lechat has received Withings research support outside the submitted work; all other authors report no relevant relationships for this abstract.

EPO-0510 | Neurofilament light chain as a biomarker in chronic inflammatory demyelinating polyradiculoneuropathy: Insights from ADHERE

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Background and aims: Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is a debilitating, progressive autoimmune neuropathic disease. Neurofilament light chain (NfL) levels indicate axonal damage and may serve as a biomarker in CIDP. NfL levels were analysed in ADHERE (NCT04281472), the largest CIDP cohort to date.

Methods: Participants with probable/definite CIDP received weekly subcutaneous (SC) efgartigimod PH20 1000 mg (stage A) for ≤ 12 weeks. Responders entered stage B and were randomised (1:1) to weekly efgartigimod PH20 SC 1000 mg or placebo for ≤ 48 weeks. Serum NfL (sNfL) was measured longitudinally in 214 participants, from which NfL z-score (zNfL) was determined.

Results: During stage A, sNfL levels remained stable in responders with baseline levels within healthy reference range (≤ 20 pg/mL, $n = 114$); sNfL reduced by 18% over time in responders with elevated baseline levels (>20 pg/mL, $n = 36$). In participants receiving continuous efgartigimod through stage B, sNfL reduced by 35% by week 24 ($n = 13$). Mean (SD) sNfL levels at stage A baseline were 18.9 (22.6) pg/mL ($n = 214$), corresponding to a mean (SD) zNfL of 0.64 (1.57). Elevated baseline zNfL levels were associated with higher CIDP disease activity status (CDAS 5). zNfL scores at stage A baseline were highest in samples from CIDP-treatment-naïve participants versus those who had previously received CIDP treatment.

Conclusion: At stage A, higher baseline NfL levels correlated with higher (worse) CDAS scores. Among efgartigimod-treated responders with elevated baseline sNfL, we observed a reduction in sNfL in both stages A and B. Further analysis will determine whether NfL can serve as a contextual biomarker and inform on disease monitoring.

Disclosure: Roger Collet Vidiella, Marta Caballero-Avila, Paula Llarch, Lorena Martín-Aguilar: Nothing to disclose. Tineke Casneuf, Erik Hofman, Geoffrey Istas, Arne De Roeck, Bianca Balbino: Employees of argenx. Elba Pascual-Goñi: Speaker honoraria from argenx. Luis Querol: Alnylam, Annexon, argenx, Avilar, Biogen, CIBERER, CSL Behring, Dianthus, Fundació La Marató, GBS-CIDP Foundation International, Grifols, Instituto de Salud Carlos III – Ministry of Economy and Innovation (Spain), Janssen, LFB, Lundbeck, Merck, Novartis, Octapharma, Roche, Sanofi, UCB.

EPO-0511 | **Autoimmune brainstem encephalitis: A cohort study showing wide variety in phenotype and outcomes**

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Background and aims: Autoimmune brainstem encephalitis (ABE) manifests diversely due to the complexity of brainstem nuclei. We aimed to comprehensively characterize the phenotype and paraclinical features to improve diagnostic approaches for ABE, and describe treatment and outcomes.

Methods: Patients suspected of or diagnosed with ABE at Erasmus Medical Center between January 2017 and April 2025 were retrospectively reviewed.

Results: Initial search returned 122 patients. After excluding 25 patients, 97 patients were included. The median age was 61 (range 1–86) years, and 56 (58%) individuals were male. Common brainstem symptoms were: ataxia (47%), eye movement disorders (45%), pyramidal tract signs (44%), and dysarthria (42%), while neuropsychiatric (45%) and movement disorders (25%) were the most common additional symptoms. Cancer was identified in 35 patients (36%): hematologic (12), breast carcinoma (6), colorectal (5), lung cancers (small cell lung cancer 4, adenocarcinoma 1), germ cell (2), neuroendocrine carcinoma (1), urogenital (1), and others (3). Identified antibodies (20%) included:

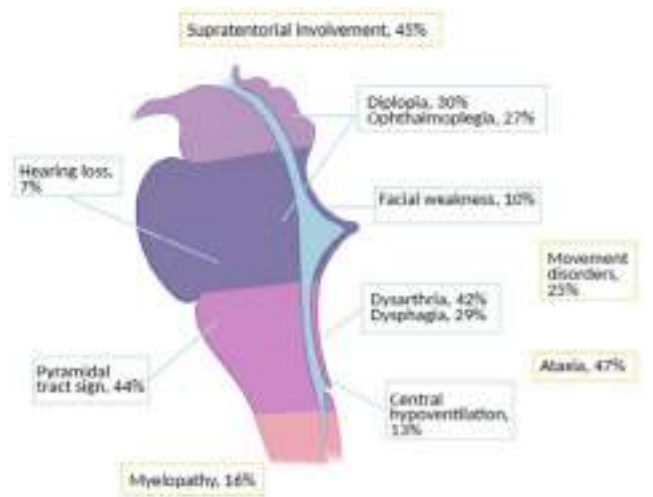


FIGURE 1 Neurological symptom spectrum in autoimmune brainstem encephalitis.

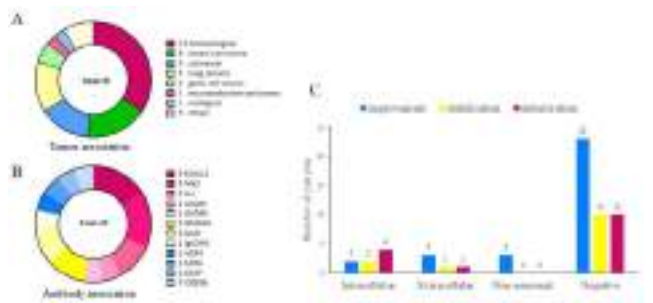


FIGURE 2 A. The tumor associations of autoimmune brainstem encephalitis (ABE). B. The antibody associations of ABE. C. The prognosis of patients with intracellular, extracellular, non-neuronal antibodies or patients without known antibodies.

anti-KLHL11 (3), anti-Ma2 (3), anti-Hu (2), anti-NMDAR (2), anti-glycine receptor (2), anti-IgLON5, anti-amphiphysin, anti-GAD65, anti-AQP4, anti-MOG, anti-GFAP, anti-GQ1b (all $n = 1$). Inflammatory changes in cerebrospinal fluid were observed in 61/88 (69%), and MRI brain abnormalities were observed in 64/95 patients (67%). Outcomes were available in 59 patients: improvement or stabilization was achieved in 44 patients (31 and 13 respectively, 77%). Patients with extracellular or non-neuronal antibodies generally respond better than those with intracellular antibodies, while antibody-negative group tends to be in between.

Conclusion: ABE has complicate aetiologies, broad tumor and antibody associations. Most treated patients respond to immunotherapy.

Disclosure: M.J. is supported by the Chinese scholarship council program (Project ID 202308430038).

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Background and aims: In multiple sclerosis (MS), remyelination is frequently inefficient and highly heterogeneous between individuals. Physical exercise has been shown to enhance oligodendrogenesis and new myelin sheath formation in demyelinating models, and to attenuate disease severity in experimental autoimmune encephalomyelitis (EAE) when performed prior to disease onset. Whether physical activity can promote repair in vivo after disease onset remains unknown.

Methods: We compared two exercise paradigms in EAE induced mice: an earlier-onset, higher-intensity protocol (initiated 36 h after peak disease; 2x15-min treadmill sessions/day, at 5 cm/s, for four consecutive days), with a later-onset, low-intensity protocol (initiated 48 h after peak; 2x10-min sessions/day, at 5 cm/s, for 4 days) and sedentary controls.

Results: Early-onset, higher-intensity exercise failed to improve clinical outcome compared with sedentary mice and was associated with a transient worsening of motor deficits. In contrast, mice subjected to later-onset, low-intensity exercise displayed significantly improved motor performance during remission. At the tissue level, this improvement was associated with enhanced lesion resolution, increased microglial-neuron interactions and higher expression of IGF-1 and P2Y12R in microglia/macrophages, consistent with a pro-repair phenotype and a partial return toward homeostasis.

Conclusion: Together, these findings identify a critical temporal window after demyelination during which, appropriately timed, low-intensity exercise promotes microglial phenotypic changes and improves clinical recovery in mice with EAE. Defining exercise paradigms that are both biologically effective and translatable to patients with MS remains a key challenge.

Disclosure: Nothing to disclose.

EPO-0513 | The Comprehensive Live Cell-Based Cytotoxicity Assay for Predicting Rescue Therapy in Acute Attacks of NMOSD

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Background and aims: Neuromyelitis optica spectrum disorder (NMOSD) lacks serological biomarkers that accurately reflect disease activity and treatment response. We established a novel live cell-based complement-dependent cytotoxicity assay (NMO-CBA-CDC) using endogenous complement to quantify

serum cytotoxicity. This assay was developed to offer a novel tool for monitoring disease activity, assessing therapeutic efficacy, and determining the necessity of rescue therapy during acute attacks.

Methods: 111 samples from 65 aquaporin-4 immunoglobulin G seropositive (AQP4-IgG+) NMOSD patients were collected. We compared serum complement levels (C1q, C5a, soluble membrane attack complex [sC5b-9]), 50% hemolytic complement (CH50), AQP4-IgG titers, and the cytotoxic index (CI) determined by NMO-CBA-CDC between the remission and acute phases, including patients receiving rescue therapies.

Results: Serum C5a and sC5b-9 levels were elevated during acute attacks ($p < 0.05$). Intravenous methylprednisolone

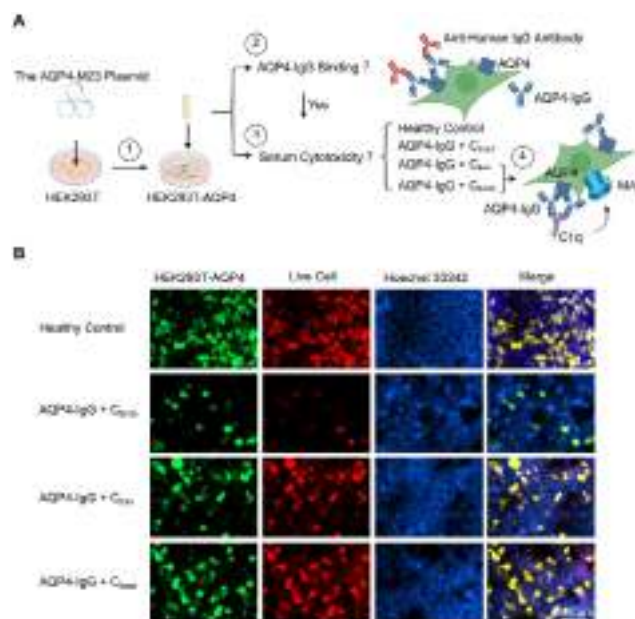


FIGURE 1 Development a comprehensive NMO-CBA-CDC assay to assess serum cytotoxicity in HEK293T-AQP4 cells.

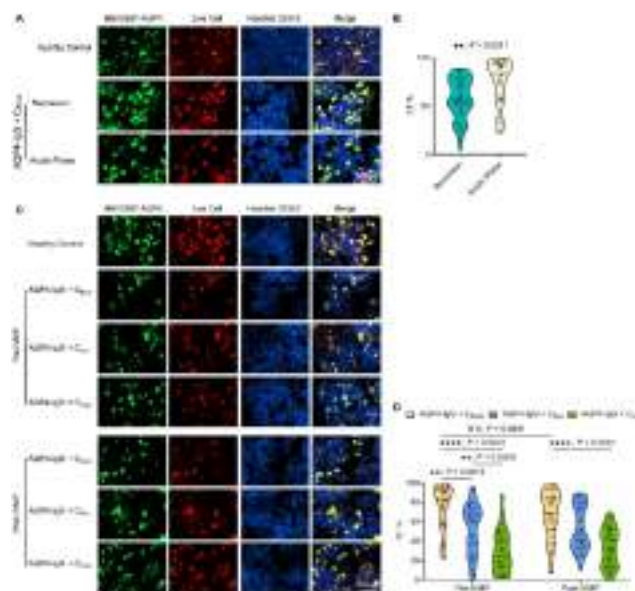


FIGURE 2 The cytotoxic index increased during the acute attack of NMOSD, which failed to be suppressed following intravenous methylprednisolone puls therapy.

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Background and aims: Myasthenia gravis (MG) is a chronic autoimmune neuromuscular disorder needing long-term immunosuppressive therapy. Eculizumab is a promising treatment, but real-world evidence on its efficacy and safety is limited.

Methods: A retrospective cohort study was done across six medical centers, enrolling 60 MG patients on eculizumab.

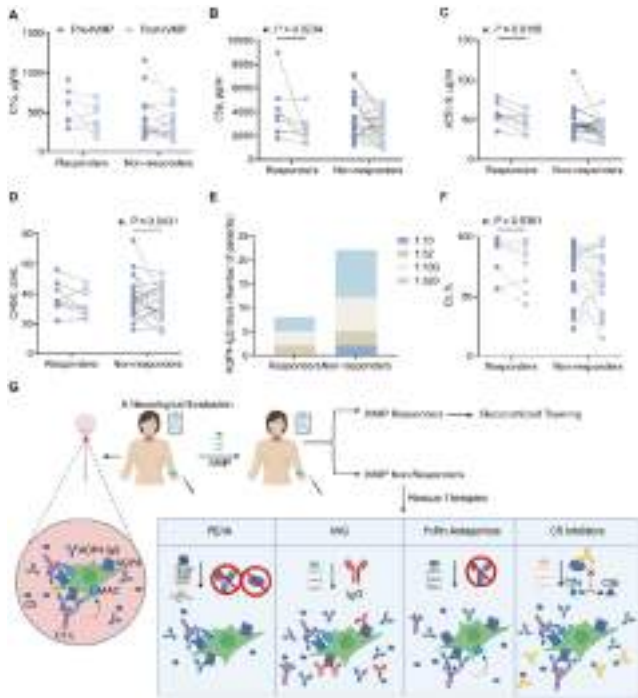


FIGURE 3 The serological indicators for the responsiveness to IVMP during acute attack of NMOSD and the implication for further rescue therapy.

(IVMP) lowered complement components but not AQP4-IgG titers or CI. Notably, the CI was significantly higher in the acute phase than in remission ($p=0.0011$) and effectively distinguished IVMP responders from non-responders. Among rescue therapies, plasma exchange/immunoadsorption (PE/IA) most consistently reduced the CI, while intravenous immunoglobulin (IVIg), neonatal Fc receptor (FcRn) antagonists, and C5 inhibitors showed variable effects.

Conclusion: The NMO-CBA-CDC assay combined endogenous AQP4-IgG and complement activity to comprehensively assess the serum cytotoxicity in AQP4-IgG+ NMOSD patients. The CI served as an efficient tool for monitoring and predicting disease activity, evaluating therapeutic efficacy, and guiding the necessity of rescue therapy. Among rescue therapies, PE/IA demonstrated superior efficacy as reflected by NMO-CBA-CDC assay.

Disclosure: The authors have no conflicts of interest to declare. This study was supported by the National Natural Science Foundation of China (82171277, 82571535, 82171338), and Young Scientific and Technological Talents (Level Three) in Tianjin.

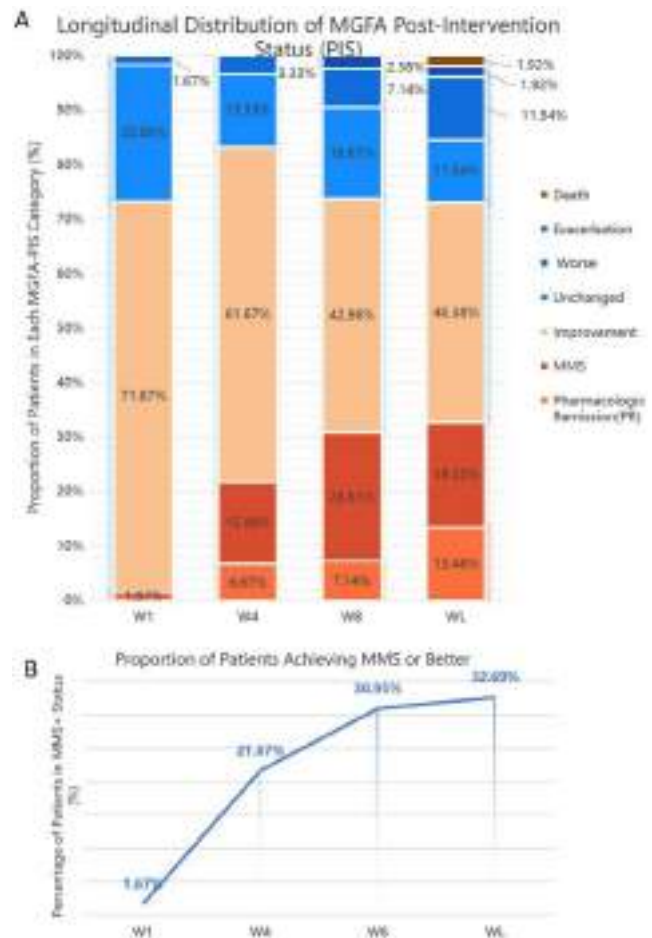


FIGURE 1 Longitudinal Distribution of MGFA Post-Intervention Status (PIS) (A) Percentage of patients classified as MMS+, improved, unchanged, worse, or deceased at baseline, Weeks 1, 4, 8, and the last follow-up. (B) Percentage of patients achieving MMS.

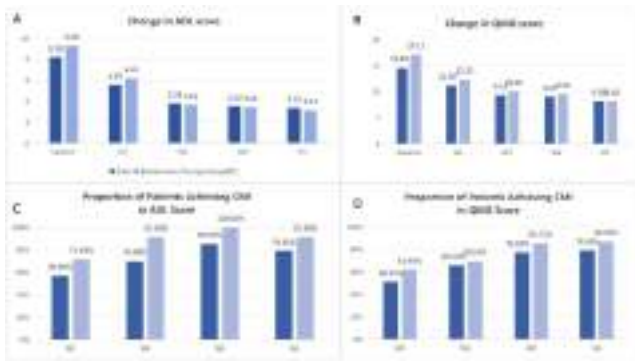


FIGURE 2 ADL and QMG score changes following initiation of eculizumab.

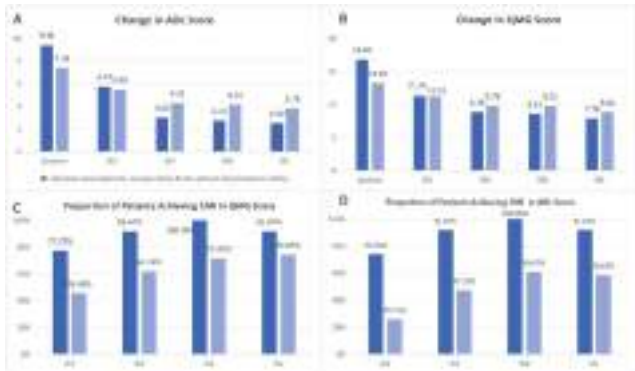


FIGURE 3 ADL and QMG score changes after initiation of eculizumab in the TAMG and non-TAMG groups.

Clinical data, including MG-Activities of Daily Living (ADL) scores, quantitative MG scores (QMG), and oral corticosteroid dosage at different time points (baseline, Weeks 1, 4, 8, and final follow-up before November 2024), were extracted from medical records. Study outcomes included efficacy measures (changes in MG-ADL and QMG scores, Myasthenia Gravis Foundation of America [MGFA] Post-Intervention Status [PIS], glucocorticoid dosage), and safety profile of eculizumab.

Results: In this cohort, 30.95% of patients achieved minimal manifestation status or better by Week 8, increasing to 32.69% at final follow-up. Meanwhile, mean ADL scores significantly dropped from 8.13 ± 3.99 at baseline to 5.59 ± 3.72 by Week 1 and to 3.33 ± 3.56 by the final follow-up ($p < 0.0001$). Clinically meaningful improvement up to 76.27% at final follow-up was also observed. In the thymoma-associated MG (TAMG) subgroup, ADL decreased rapidly compared to baseline ($p < 0.001$), and the non-TAMG cohort also improved significantly at each time point ($p \leq 0.026$). Additionally, no treatment-related adverse events were observed. The quick drop in MG-ADL scores indicates its efficacy, and the steroid-sparing effect is crucial for long-term management. In TAMG, improvement was early, reaching maximum benefit in the first week.

Conclusion: This multicenter analysis shows eculizumab improves clinical outcomes in MG patients, reducing disease activity and corticosteroid use.

Disclosure: Nothing to disclose

EPO-0515 | MRI lesion patterns and embolic sources in patients with embolic stroke of undetermined source

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Background and aims: Embolic stroke of undetermined source (ESUS) represents a significant proportion of ischemic stroke cases. Advanced diagnostic techniques (e.g. transesophageal echocardiography (TEE), implantable loop recorder, and vessel wall MRI (VWMRI)) improve diagnosis but are not widely available. The distribution of lesions on DWI-MRI may provide clues to the underlying embolic source and help guide the diagnostic workup.

Methods: We prospectively included patients with ESUS admitted to five Italian hospitals. At three months, they were classified as “cardio-ESUS” if atrial fibrillation or a probable PFO was detected and “athero-ESUS” if complicated aortic arch plaques on TEE or vulnerable non-stenosing plaques on VWMRI were identified. Patients with no identifiable etiology remained classified as having ESUS. Lesion patterns on DWI-MRI were classified according to a validated classification 1.

Results: A total of 128 patients were included in this study. At three months, 36 patients were diagnosed as athero-ESUS, 19 as cardio-ESUS, and 73 remained ESUS. The posterior cerebral artery pattern was significantly more prevalent in cardio-ESUS group vs ESUS (36,8% vs 8,2%, $p = 0.0014$), and a trend was observed for multiple small scattered lesions (present in ESUS but absent in cardio-ESUS, 16,4% vs 0%, $p = 0.058$). No significant differences were observed between patients with ESUS and athero-ESUS.

Conclusion: The lesion pattern in ESUS patients may suggest the potential embolic source. Patients with no potential embolic sources detected at 3 months resembled those with probable artery-to-artery embolism. The integration of clinical and additional radiological data may further help identify patients who would benefit from targeted diagnostic testing.

Disclosure: Source of funding: Italian Ministry of Health Ricerca Corrente 2022–2024

EPO-0516 | Safety of antiplatelet medication discontinuation more than 12 months after stent-assisted coil embolization

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Background and aims: Antiplatelet maintenance is essential to avoid ischemia following stent-assisted coiling (SAC) procedures. However, indications for antiplatelet medication discontinuation (AMD) remain controversial, and optimal timing of cessation has yet to be determined. Our goal, which we achieved through a multicenter, prospectively enrolled, non-interventional study, was to investigate the safety of AMD conducted more than 12 months after SAC.

Methods: Data were retrieved from the records of 495 consecutive patients prospectively enrolled at 10 institutions during a 3-year period (between January 2021 and December 2023). Each subject had discontinued antiplatelet therapy >12 months after SAC. Maintenance duration and cessation were both at physician discretion, based on patient clinical status. We investigated clinical outcomes for at least 6 months after AMD.

Results: A majority of patients engaged in AMD (292/495, 59.0%) were not at high risk for ischemia. Mean±SD time to AMD was 20.0±12.9 months after SAC. Treated aneurysms were largely confined to the internal carotid artery (332/495, 67.1%), followed by the anterior (95/495, 19.2%) and middle (43/495, 8.7%) cerebral arteries. A laser-cut open-cell stent was most often applied (60.5%); laser-cut closed-cell (22.2%) and braided closed-cell (17.3%) stents were used to a lesser extent. Four patients underwent double stenting. Despite sizeable (41.0%) high-risk group representation, there were no ischemic events in relation to AMD.

Conclusion: Our results suggest that AMD >12 months after SAC procedures is safe in patients who are not at high risk for ischemia. Randomized controlled trials are warranted to confirm these results.

Disclosure: Nothing to disclose

EPO-0517 | Fibrinolytic therapy in late-window stroke: A Bayesian network meta-analysis of randomized trials

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Background and aims: Time from symptom onset to hospital arrival remains the primary determinant for intravenous thrombolysis (IVT) eligibility in acute ischemic stroke (AIS). Although traditional windows were capped at 4.5 hours, recent evidence supports IVT in extended windows. However, the comparative efficacy and safety of different thrombolytic agents remain unclear.

Methods: We searched PubMed, Embase, and Cochrane Central for randomized controlled trials (RCTs) comparing alteplase, tenecteplase, and best medical therapy (BMT) in AIS patients beyond 4.5 hours or with unknown onset. A Bayesian network meta-analysis was performed using JAGS and BUGSnet to estimate odds ratios (OR) with 95% credible intervals (CrI). Ranking was performed via the surface under the cumulative ranking curve (SUCRA).

Results: Ten RCTs ($n = 2,862$) were included. Alteplase significantly improved functional independence (mRS 0–1) compared to BMT (OR 1.53; 95% CrI 1.18–1.96). Tenecteplase showed a numerical trend toward benefit (OR 1.33; 95% CrI 0.98–1.78) but did not reach statistical significance. Alteplase was associated with a higher risk of symptomatic intracranial hemorrhage (sICH) vs BMT (OR 3.82; 95% CrI 1.55–13.39), while tenecteplase demonstrated a more favorable safety ranking (SUCRA 49.1% vs 6.6% for alteplase).

Conclusion: In late-window ischemic stroke, intravenous alteplase confers the highest probability of functional independence but at the cost of increased hemorrhagic risk. Tenecteplase demonstrated a more favorable safety profile with comparable efficacy estimates, suggesting it may represent a balanced alternative for patients at higher bleeding risk. These findings clarify the trade-off between efficacy and safety among fibrinolytic strategies beyond 4.5 hours.

Disclosure: Nothing to disclose.

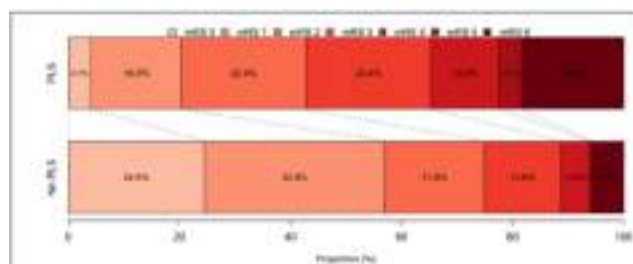
EPO-0518 | Progressive lacunar syndrome and 12-months stroke recurrence in acute ischemic lacunar stroke: Associated factors, long-term outcomes and mortality

G. Kurian; P. Michel; D. Strambo

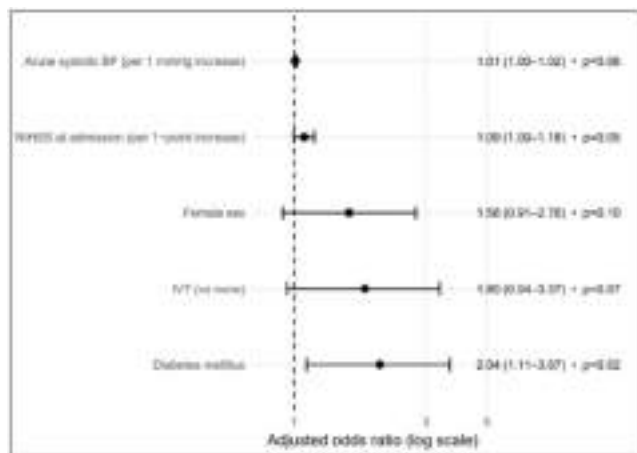
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Background and aims: Acute ischemic strokes (AIS) of microangiopathic etiology usually result in mild-to-moderate neurological deficits and favorable short-term outcomes. However, some patients develop early neurological deterioration, called progressive lacunar syndromes (PLS), or ischemic stroke recurrence (ISR). We aimed to identify predictors of PLS and ISR and assess their long-term prognostic relevance.

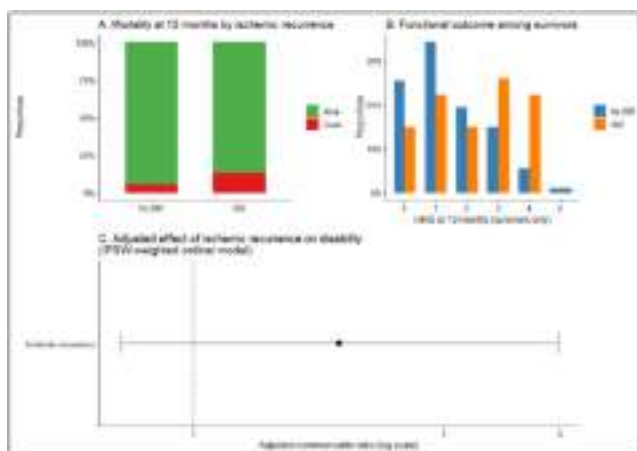
Methods: We included consecutive AIS of microangiopathic etiology from a single-center prospective stroke registry. The first outcome was PLS, defined as early (≤ 24 hours) neurological deterioration (≥ 2 point NIHSS) or fluctuation (≥ 4 point rise and



Distribution of functional outcome distribution at 12 months according to presence of progressive lacunar syndrome (PLS) with Grotta bars.



Multivariable predictors of progressive lacunar syndrome (PLS).



Impact of ISR on mortality and functional outcome. Panel (A) Proportion of patients alive and dead at 1 year according to ISR. Panel (B) Distribution of 1-year functional outcome (mRS) scores among survivors, according to ISR. Panel (C) Adjusted associati.

drop), or progressive worsening (≥ 4 point) within one week. The second outcome was 1-year ISR. Multivariable logistic regression models were developed for each outcome.

Results: Among 843 consecutive lacunar strokes (median age 71, IQR 62–81, 43% female), 74 (8.8%) developed PLS. Among 651 patients with 12-month follow-up, 36 (5.5%) experienced ISR. Only two patients had both PLS and ISR. Higher admission NIHSS (adjusted-OR = 1.09, 95% CI = 0.99–1.8) and diabetes (aOR = 2.04, 95% CI = 1.11–3.67) were independently associated with PLS, while no acute variable predicted ISR. PLS was independently associated with more unfavorable modified-Rankin-scale-shift (mRS) at 3 (acOR = 4.47, 95% CI = 2.56–7.79) and 12 months (acOR = 2.97, 95% CI = 1.55–5.69), as well as higher 12-month mortality (18.4% versus 5.7%, OR = 3.75, $p = 0.003$). ISR occurred in 36 patients (5.5%). After adjustment using the IPSW method among survivors, ISR was not independently associated with worse 12-month functional outcome.

Conclusion: In acute lacunar stroke, PLS is associated with greater baseline stroke severity and diabetes mellitus, as well as worse functional outcome. Distinct mechanisms likely underlie PLS and ISR.

Disclosure: Nothing to disclose

EPO-0519 | Impact of the Absence of Standard Modifiable Cardiovascular Risk Factors on Short- or Long-Term Vascular Outcomes in Patients with Ischemic Stroke

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Background and aims: This study aimed to investigate whether the absence of standard modifiable risk factors (SMuRFs; hypertension, diabetes, dyslipidemia, and current smoking) influences short- and long-term outcomes in patients with ischemic stroke and to explore the prognostic relevance of SMuRF burden.

Methods: This study was an analysis of a prospective, multi-center, stroke registry between January 2011 and December 2023. Patients without any of SMuRFs were classified as no SMuRF. SMuRF burden was defined as the number of risk factors present (0–4). Each risk factor was weighted equally in the analysis. The primary outcome was a composite of stroke, myocardial infarction (MI), and all-cause mortality within 3-month and 1-year.

Results: A total of 67,214 patients (age 68.3 ± 13.4 years, 58.8% male) were finally analyzed. No-SMuRF patients were 9.4%, and those with 1–4 SMuRFs were 27.0%, 34.8%, 22.7%, and 6.2%, respectively. The composite outcome did not differ between patients without and with SMuRFs at 3-months (7.9% vs 7.6%, respectively) or 1-year (12.2% vs 12.3%, respectively). After adjustment, patients with SMuRFs had higher rates of primary composite outcomes at both 3-months and 1-year than those without SMuRFs. SMuRFs were independently associated with increased risk of primary composite outcomes at 3-months (aHR, 1.30[1.19–1.44]) and 1-year (aHR, 1.25[1.06–1.35]), with a graded increase in risk according to the number of SMuRFs.

Conclusion: In patients with ischemic stroke, SMuRF burden—but not the absence of SMuRFs—was independently associated with a higher risk of composite of stroke, MI and all-cause mortality at both 3-months and 1-year, suggesting the prognostic relevance of cumulative vascular risk and the importance of comprehensive risk factor management.

Disclosure: Nothing to disclose

EPO-0520 | Comprehensive prestroke risk factor control and functional outcomes after acute ischemic stroke

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Background and aims: This study evaluated whether comprehensive prestroke risk factor (RF) control—by degree of RF control or comprehensive RF control—is associated with 3-month functional outcomes after ischemic stroke.

Methods: We analyzed ischemic stroke patients from the CRCS-K-NIH registry (2011–2022), a multicenter stroke registry in Korea. Hypertension, diabetes, dyslipidemia, and

atrial fibrillation were considered as major RFs for ischemic stroke. The comprehensive RF control was categorized as well-controlled (all present RFs well-controlled), partially-controlled (some RFs well-controlled), or poorly-controlled (none RFs well-controlled). The primary outcome was 3-month mRS of 0–2, indicating functional independence (good outcome).

Results: Of the 63,570 patients with one or more major RFs, 28.6% had poorly-controlled, 35.6% had partially-controlled, and 35.8% had well-controlled RF control. The rate of 3-month good outcome was 66.0% among those with one RF, 60.8% in two, and 55.0% in three or more. After adjusting for covariates, well-controlled groups had higher odds of good outcomes (aOR[95% CI], 1.16[1.08–1.25] for one RF, 1.34[1.21–1.48] for two, and 1.23[1.03–1.48] for ≥ 3), as did partially-controlled groups (aOR 1.16[1.06–1.26] for two; 1.22[1.05–1.42] for ≥ 3), versus poorly-controlled. Across all RF levels, well-controlled (aOR 1.22[1.15–1.29]) and partially-controlled (aOR 1.14[1.07–1.22]) statuses were significantly linked to higher odds of good 3-month outcomes.

Conclusion: Comprehensive prestroke RF control was independently associated with a higher likelihood of favorable 3-month outcomes, both within and across levels of RF burden. These findings highlight the importance of not only identifying but also optimal comprehensive prestroke RF control to improve post-stroke functional outcomes, regardless of the number of RF present.

Disclosure: Nothing to disclose.

EPO-0521 | Clinical outcomes of endovascular thrombectomy beyond 24 hours in large vessel occlusion stroke: A meta-analysis

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Background and aims: The effectiveness of endovascular thrombectomy (EVT) for acute ischemic stroke due to large vessel occlusion beyond 24 hours from last known well remains unclear, with existing evidence largely derived from observational studies.

Methods: We systematically searched major databases through February 2025 for studies comparing EVT with best medical therapy in patients treated more than 24 hours after symptom onset. Outcomes included 90-day functional independence (mRS 0–2), excellent outcome (mRS 0–1), symptomatic intracranial hemorrhage (sICH), and mortality. Random-effects models were used to pool adjusted and unadjusted odds ratios. PROSPERO (CRD420251274201)

Results: Ten observational studies ($n=1,871$) were included. EVT was associated with higher odds of functional independence (adjusted OR 4.62) and excellent outcome (adjusted OR 5.68). EVT was linked to an increased risk of sICH, while 90-day mortality was not significantly different between groups. Subgroup analyses across imaging strategies, stroke severity, and occlusion territory were consistent with primary findings. Certainty of evidence ranged from low to very low.

Conclusion: EVT beyond 24 hours may improve functional outcomes without increasing mortality, despite a higher

hemorrhagic risk. Randomized controlled trials are needed to validate these findings and refine patient selection in the ultra-late window.

Disclosure: Nothing to disclose.

EPO-0522 | Sex-based inequities in prehospital stroke identification: A systematic review and meta-analysis

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Background and aims: Evidence suggests that sex-related disparities may influence early stroke recognition and management before hospital arrival. This systematic review and meta-analysis evaluated differences between female and male patients across dispatch and emergency medical service (EMS) pathways and identified gaps requiring further investigation.

Methods: A systematic search of PubMed, EMBASE, CINAHL, and EMCARE identified studies reporting sex differences in prehospital stroke care. Random-effects meta-analyses using inverse variance weighting were conducted. The primary outcome was the likelihood of correct prehospital stroke identification. Secondary outcomes included dispatch coding, EMS prenotification, transport destination, and time metrics. PROSPERO (CRD420251274202)

Results: Sixteen studies including 1,193,788 patients (622,764 women; 571,024 men) were analyzed. Women were less likely to receive a correct prehospital stroke diagnosis compared with men (RR 0.92, 95% CI 0.89–0.96), with substantial heterogeneity. No significant sex differences were observed in dispatch stroke coding, EMS prenotification, transport to stroke centers, or call-to-door times. Data were insufficient to draw conclusions regarding on-scene clinical management and interprofessional communication.

Conclusion: Female patients experience lower rates of accurate prehospital stroke recognition. Considerable heterogeneity and limited reporting across care stages highlight the need for standardized, high-quality studies to address sex-based inequities in prehospital stroke systems.

Disclosure: Nothing to disclose.

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Background and aims: Neuromyelitis optica spectrum disorder (NMOSD) is an autoimmune astrocytopathy where disability is largely relapse-driven. Cerebrovascular risk factors (cVRFs) may exacerbate astrocytic and small-vessel injury, therefore we investigated their impact on MRI damage and disability in NMOSD.

Methods: Twenty-eight aquaporin 4-positive NMOSD patients and 56 age- and sex-matched healthy controls (HC) underwent 3T brain and cervical MRI, cVRF and neurological assessment, including Expanded Disability Status Scale (EDSS). Brain T2-hyperintense white matter lesion volume (WMLV), normalized global and regional volumes, normal-appearing WM fractional anisotropy and mean diffusivity, diffusion tensor imaging analysis along the perivascular spaces (DTI-ALPS) index, spinal cord (SC) LV and normalized cross-sectional area (nCSA) were quantified.

Results: cVRFs were present in 57% of NMOSD and 39% of HC, with smoking being the most frequent (FDR- $p=0.734$). Compared to HC, NMOSD showed higher brain WMLV and SCLV, lower normalized brain, deep gray matter volumes (nDGMV), and nCSA (FDR- $p\leq 0.030$). Significant diagnosis×cVRF interactions were found for nDGMV and DTI-ALPS (FDR- $p\leq 0.038$). In NMOSD, higher EDSS correlated with lower DTI-ALPS ($r=-0.46$ /FDR- $p=0.048$) and higher SCLV ($r=0.53$, FDR- $p=0.030$). At moderation analysis, cVRFs amplified the association between DTI-ALPS and disability (FDR- $p=0.045$).

Conclusion: cVRFs amplify CNS damage and the clinical impact of glymphatic dysfunction in NMOSD, suggesting convergent vascular and astrocytic injury.

Disclosure: M. Rubin has nothing to disclose. P. Preziosa received speaker honoraria from Roche, Biogen, Novartis, Merck, Bristol Myers Squibb, Genzyme, Horizon and Sanofi. M. Margoni reports grants and personal fees from Sanofi Genzyme, Merck Serono, Roche, Biogen, Amgen and Novartis. L. Storelli, E. Pagani, P. Valsasina, and M. Albergoni have nothing to disclose. L. Moiola received speaker honoraria from Roche. M. Filippi is Editor-in-Chief of the Journal of Neurology, Associate Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer,

Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmed Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. M.A. Rocca received consulting fees from Biogen, Bristol Myers Squibb, Roche; and speaker honoraria from Alexion, Biogen, Bristol Myers Squibb, Celgene, Horizon Therapeutics Italy, Merck Serono SpA, Mitsubishi-Tanabe Pharma, Neuraxpharm, Novartis, Roche, Sandoz, and Sanofi. She receives research support from the MS Society of Canada, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. She is Associate Editor for Multiple Sclerosis and Related Disorders; and Associate Co-Editor for Europe and Africa for Multiple Sclerosis Journal.

EPO-0524 | Clinical safety and efficacy of stent-assisted coil embolization with ACCERO stent in cerebral aneurysm: Short-term follow-up and precaution for use

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Background and aims: Stent-assisted coil embolization (SAC) is an effective method of treating intracranial aneurysms. The aim of the study was to assess the safety and efficacy of the new ACCERO stent for the treatment of cerebral aneurysms.

Methods: Nine ruptured and 41 unruptured cerebral aneurysms were treated using the ACCERO stent between February 2021 and December 2023. Patient demographics, aneurysm characteristics, procedural parameters, grade of occlusion, complications, and clinical outcomes were analyzed. Follow-up was conducted with magnetic resonance angiography (MRA) or Digital subtraction angiography (DSA) was performed 6 to 12 months after the procedure.

Results: The ACCERO stent deployment was attempted in 51 cases, with replacement by the Neuroform Atlas stent in 1 case. Successful stent deployment was achieved in 50 cases, and appropriate wall apposition to the parent artery. Intimal hyperplasia was observed in 1 case, but no other clinical complications related to the stent occurred. Favorable clinical outcomes were observed in 92% of patients (46/50), including those with subarachnoid hemorrhage. Immediate favorable angiographic outcomes and complete occlusion were achieved in 90% (45/50) and 74% (37/50) of cases, respectively. Among the 45 patients who had imaging follow-up, favorable angiographic outcomes and complete occlusion were observed in 93.3% (43/45) and 82.2% (37/45) of cases, respectively.

Conclusion: The ACCERO stent is a braided-type stent that requires more attention than stents, such as the Neuroform Atlas

or Enterprise stents. However, since the struts of the stent are fully visible, it can be more useful in treating challenging aneurysms once the user becomes familiar with its use.

Disclosure: Nothing to disclose

EPO-0525 | Neuroanatomic correlates of conjugate eye deviation in left hemispheric stroke

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Background and aims: Conjugate eye deviation (ED) is a frequent clinical sign in acute stroke, a marker of large vessel occlusion and a predictor for the occurrence of neglect. While its occurrence has been extensively studied in right hemispheric stroke, data for left hemispheric lesions remain limited.

Methods: We retrospectively analysed patients with first time left hemispheric middle cerebral artery stroke from the Freiburg Large Scale Project registry. ED was quantitatively measured on acute CT or MRI. Neglect was assessed using item 11 of the NIH Stroke Scale (NIH-SS). Lesion-symptom relationships were examined with the novel Streamline-based Lesion-symptom Mapping (SLSM).

Results: Seventy patients met inclusion criteria. Larger ED was associated with the presence of neglect but not with age or lesion volume. SLSM revealed that ED severity was associated with lesions along a number of tracts: superior longitudinal and arcuate fasciculi (dorsal pathway), the extreme capsule complex (ventral pathway), and the middle and inferior longitudinal fasciculi (caudal pathway) (Figure 1). Subgroup analysis (Figure 2) showed that larger ED (in purple) was linked to dorsal and caudal disconnections. Smaller ED (in green) was associated with ventral pathway involvement, because in CT/MRI, no detection of stimuli or reorienting was required.

Conclusion: In left hemispheric stroke, ED at rest without external stimuli reveals disconnection of three distinct networks: dorsal, ventral and caudal, associated with different aspects of neglect: control of eye movements and gaze orientation, attention to novel and behaviourally relevant stimuli, and spatial awareness.

Disclosure: Nothing to disclose.

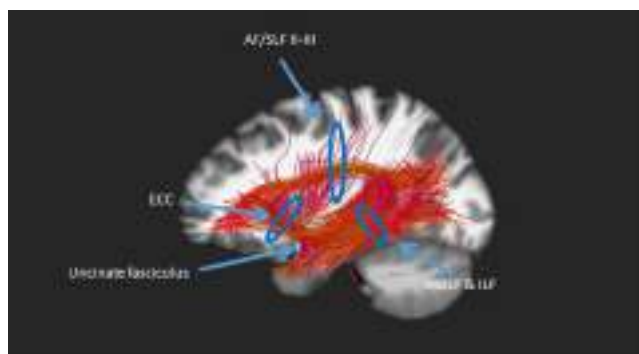


FIGURE 1

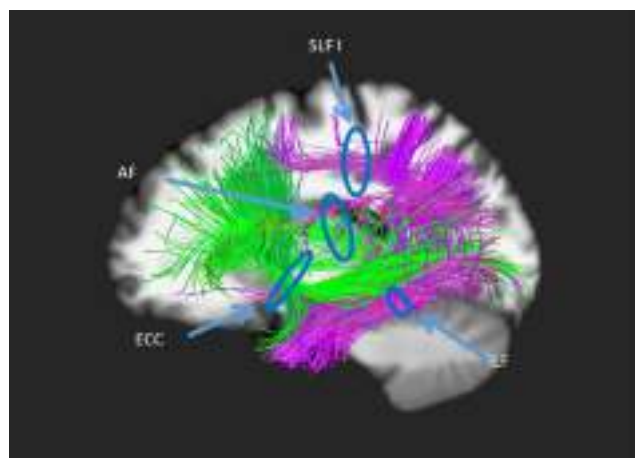


FIGURE 2

MS and related disorders 4

EPO-0526 | Efficacy of Neurofeedback in Cognitive Impairment in Multiple Sclerosis

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Background and aims: Cognitive impairment is a common manifestation of multiple sclerosis (MS), affecting approximately 43–72% of patients and significantly reducing quality of life and social functioning. Pharmacological treatment options for MS-related cognitive deficits remain limited. Therefore, there is a growing interest in non-invasive and non-pharmacological neurorehabilitation approaches, including neurofeedback, aimed at improving cognitive function through modulation of brain activity.

Methods: This study included 40 patients with multiple sclerosis who were divided into two comparable groups. The neurofeedback group ($n=20$) received standard therapy combined with neurofeedback training, while the control group ($n=20$) received standard therapy alone. Cognitive function was assessed using the Montreal Cognitive Assessment (MoCA), Mini-Mental State Examination (MMSE), and Mini-Cog tests. Cortical evoked potentials (P300) were recorded before and after treatment. The neurofeedback protocol consisted of 10–15 training sessions.

Results: At baseline, no significant differences in cognitive performance were observed between the two groups ($p>0.05$). After treatment, the neurofeedback group demonstrated significant improvements in cognitive scores: MoCA increased from 22.6 ± 1.9 to 25.3 ± 1.6 , MMSE from 25.5 ± 1.7 to 28.0 ± 1.3 , and Mini-Cog scores also improved ($p<0.05$). In contrast, cognitive improvements in the control group were less pronounced. A reduction in P300 latency by 18–22 ms was observed only in the neurofeedback group.

Conclusion: Neurofeedback significantly enhances cognitive outcomes in patients with multiple sclerosis when used as an adjunct to standard therapy. This method may be considered an effective and non-invasive approach for cognitive rehabilitation in MS.

Disclosure: Nothing to disclose

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Background and aims: No evidence of disease activity 3 (NEDA-3) is a proposed composite treatment target in multiple sclerosis (MS). Optical coherence tomography (OCT) derived retinal layer thinning is a promising paraclinical marker of future disability progression. We investigated whether integrating OCT into NEDA-3 (NEDA-3+OCT) improves monitoring of response to disease-modifying therapy (DMT).

Methods: We screened two observational cohorts at the Medical Universities of Vienna and Innsbruck for patients with relapsing MS who (i) newly initiated DMT, (ii) underwent MRI and OCT at baseline and 12 months, and (iii) had ≥ 24 months of clinical follow-up. Retinal thinning was defined as $\geq 0.5\%$ reduction in the ganglion cell and inner plexiform layer and/or $\geq 1.0\%$ reduction in peripapillary retinal nerve fibre layer at 12 months. NEDA-3/NEDA-3+OCT status was determined 12 months after DMT initiation. The primary endpoint was confirmed disability progression occurring after month 12.

Results: Overall, 124 individuals were included (72% female; mean age 33.1 ± 7.7 years; median baseline EDSS 2.0 [IQR 0.0–2.5]). Over a median follow-up of 3.4 years, 28 (23%) experienced disability progression. Time to progression did not significantly differ between EDA-3 and NEDA-3 at month 12 ($p=0.067$), and EDA-3 was not significantly associated with progression in multivariable Cox regression (aHR 1.52; 95% LL-CI 0.64; $p=0.173$). In contrast, EDA-3+OCT was associated with increased progression risk (aHR 4.47; 95% LL-CI 1.87; $p=0.002$) and shorter time to progression (RMST 42.5 ± 1.9 vs 51.6 ± 1.1 months; $p < 0.001$).

Conclusion: Adding OCT to NEDA-3 may better identify individuals at risk of future disability progression than NEDA-3 alone, particularly among those receiving high-efficacy DMT.

Disclosure: No disclosures or relationships of interest relevant to the context of this study.

EPO-0528 | Factors associated with progression independent of relapse and magnetic resonance imaging activity in relapsing multiple sclerosis

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Background and aims: The proportion of Progression Independent of Relapse Activity (PIRA) events is significantly reduced when subclinical inflammatory activity detected by magnetic resonance imaging (MRI) is considered. This study aims to identify factors associated with Progression Independent of Relapse and MRI Activity (PIRMA) in patients with relapsing multiple sclerosis (MS).

Methods: This multicenter, retrospective, cohort study used prospectively collected data from the Italian MS Register. Inclusion criteria were clinically isolated syndrome or relapsing–remitting MS, first visit on/after January 1, 2000, ≥ 3 Expanded Disability Status Scale (EDSS) assessments, ≥ 5 years of follow-up. Confirmed Disability Accrual (CDA) was defined as a EDSS progression confirmed at 24 weeks, using roving baseline. PIRA was defined in absence of relapses < 90 days before and < 30 days after CDA onset. In patients undergoing annual brain and/or spinal cord MRI, PIRA events temporally associated with MRI activity were reclassified as PIRMA. Cox proportional hazards models were applied to identify associated factors.

Results: Overall, 30203 patients met inclusion criteria and were followed for a mean of 11.3 years; 18187 (60.2%) experienced at least one CDA, of which 12501 (68.7%) were classified as PIRA. Among 605 patients with annual MRI, 357 (59%) CDA events occurred. The proportion of PIRA decreased from 63.6% to 45.1% after accounting for MRI activity. PIRA was associated with older age (HR = 1.04, 95% CI: 1.02–1.05, $p < 0.001$) and visit density (HR = 1.59, 95% CI: 1.20–2.10, $p = 0.001$). PIRMA was associated with older age (HR = 1.04, 95% CI: 1.02–1.06, $p < 0.001$), higher EDSS (HR = 1.12, 95% CI: 1.01–1.24, $p < 0.047$) and visit density (HR = 1.65, 95% CI: 1.20–2.28, $p = 0.002$).

Conclusion: Approximately one third of PIRA events were associated with MRI inflammatory activity. PIRA and PIRMA showed largely overlapping factors, suggesting similar underlying pathological mechanisms.

Disclosure: E. Portaccio has received compensation for travel, advisory boards and/or speaking activities from Biogen, Merck Serono, Sanofi, Teva, Roche, BMS Celgene, Janssen, and Novartis, and serves on the editorial board of *Frontiers in Neurology and Brain Sciences*. L. Pastò received research support from Novartis and Biogen and speaker honoraria from Teva. A. Lugesesi has served on advisory boards and received honoraria or travel support from multiple companies including Biogen, Merck, Novartis, Roche, Sanofi/Genzyme, and others; her institutions received research grants from Novartis, Roche, and Sanofi/Genzyme. E. Cocco, F. Patti, V.L.A.M. Torri Clerici, D. Ferraro, V. Brescia Morra, G. Salemi, M. Vianello, R. Cerqua, P. Perini, S. Romano, C. Pozzilli, C. Tortorella, A. Di Sapio, P. Annovazzi, P. Iaffaldano, and M. Trojano received grants, honoraria, travel support, and/or served on advisory boards for pharmaceutical companies including Almirall, Alexion, Bayer, Biogen, BMS/Celgene, Janssen, Merck (Serono), Novartis, Roche, Sanofi/Genzyme, Teva, and others. M. Inglese received research grants from NIH, NMSS, and FISM and consultation fees from Roche, Genzyme, Merck, Biogen, and Novartis. M. Filippi is Editor-in-Chief of the *Journal of Neurology* and received consulting fees, speaker honoraria, and research support from multiple pharmaceutical companies and public institutions. M.P. Amato served on advisory boards and received speaker honoraria and research support from Biogen Idec, Merck Serono, Bayer Schering Pharma, and Sanofi Aventis and serves on editorial boards of *Multiple Sclerosis Journal* and *BMC Neurology*. All other authors report no relevant disclosures.

EPO-0529 | Kappa free light chains and oligoclonal bands in the diagnosis of multiple sclerosis: Preliminary results of the PRO-KFLC study

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Background and aims: The 2024-revised McDonald criteria for diagnosing multiple sclerosis (MS) include kappa free light chains (KFLC) alongside oligoclonal bands (OCB) as markers as diagnostic features. However, the role of OCB and KFLC in the diagnosis of MS requires further clarification.

Methods: The PRO-KFLC study (prospective, multicenter, real-world analysis of KFLC in the diagnosis of MS; NCT07183020) is coordinated by the German Society for Cerebrospinal Fluid (CSF) Diagnostics and Clinical Neurochemistry. The study recruits CSF/serum sample pairs from patients with MS, and clinically or radiologically isolated syndromes (CIS, RIS) across international MS-centers. OCB are detected by different externally performed site-specific methods and by the centrally performed silver-staining and immunofixation (Sebia) method. KFLC concentrations are determined using nephelometric and turbidimetric assays on different analytical platforms (Atellica NEPH 630, BN ProSpec II, Optilite).

Results: To date, 764 CSF/serum sample pairs have been analyzed from over 50 centers across Europe. The female-to-male ratio was 1.9, and the median age at diagnosis was 34 years. Among the cohort, 63 patients were diagnosed with CIS or RIS. CSF-specific OCB were detected by center-specific methods in 87% of the patients. An intrathecal KFLC synthesis was identified in 91% using the KFLC index (6.1) and in 96% using Reiber's hyperbolic function (Figure 1).

Conclusion: The preliminary results indicate that KFLC are a valuable biomarker for detecting intrathecal inflammation in patients with MS, CIS and RIS. Ongoing recruitment and analysis are expected to further elucidate the diagnostic performance and clinical utility of KFLC assays and the role of OCB.

Disclosure: This is a prospective, diagnostic biomarker study and none of the authors and centers have to report a conflict of interest. Outside the submitted work: Franz Felix Konen received honoraria from Alexion, Argenx, Merck, Novartis, and Takeda. He received research support from Merck, Siemens and Erwin-Röver-Foundation. Thomas Skripuletz reports research support from Alnylam, CSL Behring, Merck, Novartis, Siemens; honoraria for lectures, travel support for meeting attendance, and/or consultancy fees from Alexion, Alnylam, Amgen, argenx, Bayer, Biogen, Bristol Myers Squibb, Centogene, CSL Behring, Grifols, Hexal AG, Horizon, Janssen, Merck, Novartis, Pfizer, Roche, Sanofi, Siemens, SOBI, Teva, Viatrix. For this study, institutional research support was granted from Erwin-Röver-Foundation. Complete author list: Konen, Franz Felix; Albers, Lina Marie; Bucak, Erda; Streichert, Anna Lena; Schwenkenbecher, Philipp; Jendretzky, Konstantin Fritz; Nay, Sandra; Grote-Levi, Lea Martha; Tauber, Simone; Bohn, Amelie; Bayas, Antonios; Braun, Marie; Ruprecht, Klemens; Höpner, Lena Katharina; Otto, Carolin; Schaebitz, Wolf; Bytyqi, Adonis; Gold, Ralf; Motte, Jeremias; Horstkemper, Lea; Ruck, Tobias; Boeckers, Joshua; Seliger-Behme, Corinna; Lazaridis, Lazaros; Pröbstel, Anne-Katrin; Möhn, Nora; Dressel, Alexander; Bock, Henry; Ziemssen, Tjalf; Goerbing, Tina; Meuth, Sven; Pawlitzki,

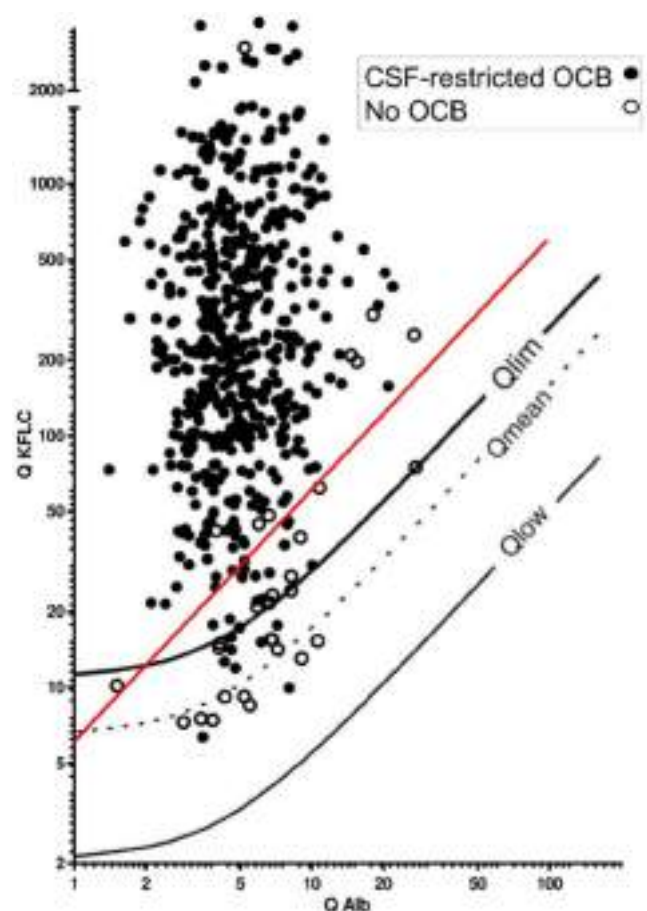


FIGURE 1 The black line “QLim” of the Reiber quotient diagram (kappa free light chain quotient, Q KFLC; albumin quotient, Q Alb) represents the upper reference limit for intrathecal KFLC synthesis. The red line represents the linear KFLC index of 6.1. Black dots i.

Marc; Rothhammer, Veit; Tsaktanis, Thanos; Yalachkov, Yavor; Hauck, Lilith; Koerbel, Kimberley; Wiendl, Heinz; Dersch, Rick; Warnke, Clemens; Svacina, Martin; Pfeuffer, Steffen; Süße, Marie; Budde, Kathrin; Otto, Markus; Bentrup, Jana; Frieese, Manuel A.; Schubert, Charlotte; Wildemann, Brigitte; Groppa, Sergiu; Decker, Yann; Fousse, Mathias; Geis, Christian; Schwab, Matthias; Vay, Sabine; Konitsioti, Agni; Schreiber, Stefanie; Vielhaber, Stefan; Al-Dubai, Marwa; Stephanik, Heike; Kuempfel, Tania; Engels, Daniel; Hemmer, Bernhard; Busch, Theresa; Gross, Catharina; Schulte-Mecklenbeck, Andreas; Witt, Karsten; Linker, Ralf; Angstwurm, Klemens; Winkelmann, Alexander; Zettl, Uwe; Leypoldt, Frank; Ehmke, Lena; Kowarik, Markus; Tieck-Fernandez, Maria; Tumani, Hayrettin; Bachhuber, Franziska; Haarmann, Axel; Schuhmann, Michael; Harrer, Andrea; Kliushnikova, Dariia; Kuhle, Jens; Oechtering, Johanna; Khalil, Michael; Torkildsen, Oivind Fredvik Grytten; Zeman, David; Kusnierova, Pavlina; Rejdak, Konrad; Montalban, Xavier; Arrambide, Georgina; Espejo, Carmen; Alvarez, Gary; Quiroga, Ana; Moccia, Marcello; Rosenstein, Igal; Axelsson, Markus; Novakova, Lenka; Schmierer, Klaus; Gnanapavan, Sharmilee; Galea, Ian; Teunissen, Charlotte; Wessels, Mark; Van Pesch, Vincent; Benyahia, Zohra; Soares, Mafalda; Ladeira, Ana; Laakso, Sini; Tienari, Pentti; Emersic, Andreja; Uros, Rot; Willrich, Maria Alice; Manin, Analisa; Villa, Andres Maria; Tuzun, Erdem; Yilmaz, Vuslat; Skripuletz, Thomas

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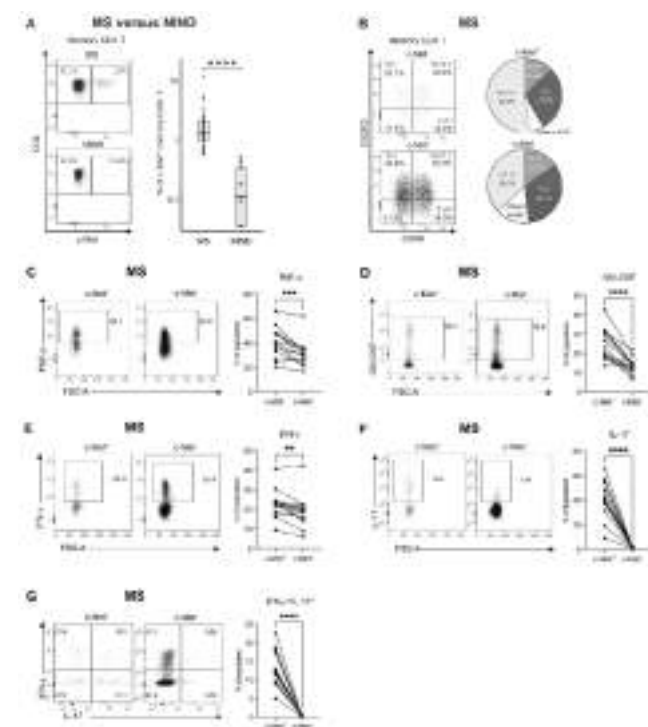
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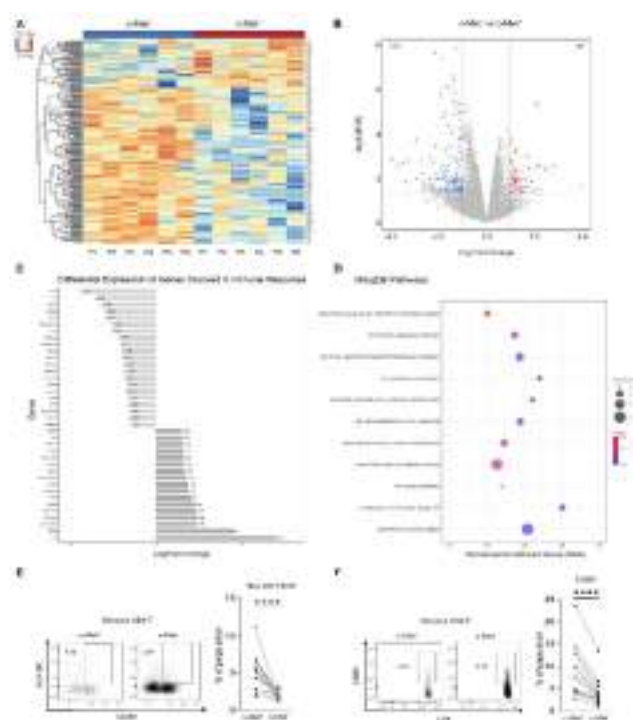
Background and aims: Tyrosine kinase receptor c-Met is the unique receptor for hepatocyte growth factor (HGF). The HGF/c-Met axis is reported to modulate cell migration, cytokine production, and antigen presentation. Here, we analyzed c-Met expression on human CD4⁺ T cells from multiple sclerosis (MS) patients versus patients with non-inflammatory neurological disease (NIND).

Methods: Cells were isolated from blood and CSF samples of MS and NIND patients from December 2020 to December 2022 at Geneva University Hospital. Phenotypic characterization were determined by RNA sequencing (bulk) and flow cytometry. Adhesion and transmigration capacity were studied to further characterize the function of activated c-Met+CD4⁺ T cells.

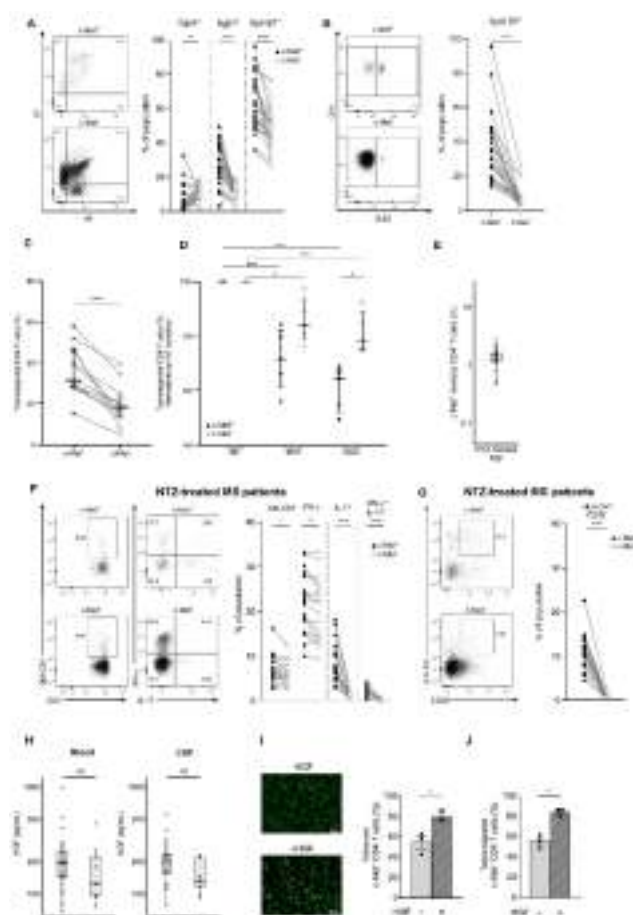
Results: c-Met+CD4⁺ T cells are detected at higher levels in both blood and CSF of MS patients compared to NIND with a blood to CSF gradient. The phenotype of CD4+c-Met+ T cells is oriented toward Th17.1 polarization with a predominance of central memory and effector memory activity profiles. c-Met+CD4⁺ T cells have increased proinflammatory activity, showing higher intracellular levels of IL-17+ and IL-17+IFN γ + compared



Increased frequency of blood c-Met+ CD4⁺ T cells displaying enhanced inflammatory propensity in MS patients.



Bulk RNA-seq analysis of c-Met+ and c-Met- CD4⁺ T cells isolated from the blood of MS patients.



c-Met+ CD4⁺ T cells show increased expression of VLA-4 and LFA-1, and are associated with enhanced migratory capacities in the blood of MS patients.

with c-Met-CD4+ T cells. c-Met+CD4+ T cells showed increased transmigration capacity correlated with increased expression of integrins (Itgα4, Itgβ1, Itgβ7, Itgα4β1, Itgα4β7, ItgαLβ2) in transcriptomic (bulk mRNA sequencing) and surface protein (flow cytometry) analyses compared with c-Met-CD4+ T cells. Anti-Itgα4 (natalizumab) and anti-ItgαLβ2 (odulimomab) inhibit CD4+ T cell transmigration with predominant inhibition of c-Met+CD4+ T cells compared with c-Met-CD4+ T cells.

Conclusion: These results emphasize c-Met as an immune marker of highly pro-inflammatory and pro-migratory CD4+ T lymphocytes in MS.

Disclosure: None

EPO-0531 | Stride-level gait monitoring as a sensitive marker of relapse-associated and relapse-independent disability progression in multiple sclerosis

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Background and aims: In multiple sclerosis (MS), high-efficacy disease-modifying therapies substantially reduce inflammatory activity, yet long-term disability progression persists. Both progression independent of relapse activity (PIRA) and relapse-associated worsening (RAW) contribute to disability accumulation. Sensitive methods to capture these processes are needed.

Methods: We conducted a multicenter, 3-year observational study using an ankle-worn wearable device enabling stride-level gait reconstruction and the computation of the fastest 5% of strides recorded (SV95C). Participants completed one-month periods of daily monitoring at baseline, one year, and every six months over the subsequent two years. The Expanded Disability Status Scale (EDSS) and Timed 25-Foot Walk Test (T25FWT) were performed before each monitoring period. Reliability, clinical validity and sensitivity to change were evaluated.

Results: Seventy-eight participants (median age 48.5 years; EDSS range 0–5.5), including 61 with relapsing-remitting MS (RRMS) and 17 with progressive MS (PMS), were enrolled. SV95C showed excellent reliability (intra-class correlation coefficient of 0.99) and strong correlations with EDSS ($R = -0.65$) and T25FWT ($R = -0.71$; all $p < 0.001$) (Figure 1). SV95C differentiated MS subtypes and EDSS subgroups (Figure 2). Significant SV95C decline was detected in relapse-free PMS (standardized response mean, SRM -0.89 at 1Y; -1.06 at 2Y; both $p < 0.05$) and RRMS (SRM -0.84 at 1.5Y, $p < 0.01$; -0.87 at 2Y, $p < 0.001$)

(Figure 3). EDSS identified change only in PMS (SRM 0.67, $p < 0.05$ at 1Y). Relapses were associated with marked SV95C declines.

Conclusion: Passive, stride-level gait monitoring is a valid and sensitive marker of ambulatory decline in MS, capturing both RAW and PIRA. Full three-year results will be presented at the congress.

Disclosure: This study was funded by F. Hoffmann-La Roche Ltd.

EPO-0532 | Temporal Dynamics of Proteomic Profiling in Multiple Sclerosis: Insights from a retrospective longitudinal study

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Background and aims: Identifying blood-based biomarkers to enhance clinical decision-making in multiple sclerosis (MS) is essential, yet no single protein captures the disease's complex pathophysiology. The MS Disease Activity (MSDA) panel, comprising 18 proteins reflecting neuroinflammation, immunomodulation, myelin biology, and neuroaxonal integrity, has been associated with radiological and clinical outcomes. However, its relationship with evidence of disease activity (EDA) and temporal patterns remains underexplored. This study investigates temporal variations in the MSDA score in relation to MS disease activity and its potential utility in clinical evaluations.

Methods: Retrospective longitudinal data from 102 MS patients (mean±SD age 31.7±7.7 years, 61.8% female, median disease duration 1.3 years [IQR:0.7–6.8]) were analyzed, with a median of 7 samples [IQR:6–9] per patient over a mean±SD follow-up of 10.4±3.4 years. Protein levels were measured using the MSDA panel on the Olink™ platform. Radiological activity was defined as Gadolinium-enhanced or new/enlarging T2 lesions on 3T MRI scans. EDA included clinical relapse, confirmed disability worsening (assessed by Expanded Disability Status Scale scores), or radiological activity within six months of sampling.

Results: Temporal analysis showed a significant MSDA-score increase at relapse ($p < 0.001$), remaining elevated for six months ($p < 0.01$). Inflammation-related pathways peaked at relapse, while myelin and neuroaxonal scores peaked three months later. The MSDA score was higher in patients who developed future EDA within 1.5 years, but only when samples were collected during stable periods ($p < 0.05$).

Conclusion: These findings highlight the value of dynamic proteomic profiling. The MSDA score may help identify patients at risk of future disease activity, potentially improving routine MS care.

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EPO-0533 | Change in excess and relative mortality in multiple sclerosis over time and by sex

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Background and aims: People with multiple sclerosis (PWMS) die at a lower age than matched background populations, but how much the disease shortens lifetime, differ between published studies, older as well as more recent.

Methods: The nationwide Danish Multiple Sclerosis Registry has followed people with multiple sclerosis (MS) for up to 70 years after onset with complete registration of vital status. In this study we describe the trend over time of excess mortality in PWMS and focus on the difference between the sexes.

Results: 28,118 cases had onset of MS between 1950 and 2022. Of them 9875 had died before follow-up at the end of year 2022, whereas only 3579 deaths were expected in matched Danish background population, matched by sex, current age and current calendar year. The relative mortality in terms of standardized

mortality rate (SMR) was 2.84 (95% CI 2.71–2.81). The median survival time from onset was 31.4 years (95% CI 30.6–32.4) as opposed to 49.8 years in the matched population within the same time- and age-frame. The median age of death was 74.2 years in PWMS (95% CI 73.8 – 84.6) and 83.4 years in the matched population. 20-years SMR changed from 5.21 in the 1950–1964 onset cohort to 2.9 in the 1980–1994 onset cohort. The ratio of relative mortalities between women and men was 1.04 ($p=0.085$).

Conclusion: Excess mortality in MS has decreased over the last several decades, but it does not differ between the sexes.

Disclosure: Melinda Magyari has served on the scientific advisory board for Sanofi, Novartis, Merck, and Moderna and has received honoraria for lecturing from Biogen, Merck, Novartis, Roche, Sanofi, Moderna, and Neuroxpharm. Rolf Pringler Holm has received speaker honoraria from Sanofi and Novartis, has served on an advisory board for Novartis, and has received a travel grant from The Danish MS Society. Nils Koch-Henriksen has nothing to disclose.

EPO-0534 | Ponesimod efficacy and safety in relapsing multiple sclerosis patients with mild to moderate disability: Results from OPTIMUM and long-term extension

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Background and aims: OPTIMUM was a phase 3 trial comparing ponesimod with teriflunomide in relapsing multiple sclerosis (RMS). In the long-term extension (LTE), all participants received ponesimod 20 mg for up to 5 years. This post hoc analysis assessed efficacy and safety in patients with baseline Expanded Disability Status Scale (EDSS) ≤ 3.5 at core study entry.

Methods: Annualized relapse rate (ARR) was estimated using a negative binomial model. Disability outcomes included 24-week confirmed disability accumulation (24wCDA). Magnetic resonance imaging activity was assessed using cumulative combined unique active lesions (CUAL), defined as T1 gadolinium-enhancing lesions and new or enlarging T2 lesions. Safety assessments included serious and fatal adverse events (AE).

Results: Overall, 946 patients were included (core study: 472 ponesimod; 474 teriflunomide). During the core period, ARR was lower with ponesimod versus teriflunomide (0.17 vs 0.29), rate ratio 0.59 (95% confidence interval [CI] [0.47–0.74]). Over the combined period, ARR remained lower in patients receiving continuous ponesimod versus those switching from teriflunomide (0.13 vs 0.21), rate ratio 0.63 (95% CI [0.51–0.79]). 24wCDA estimates at 360 weeks were 20.5% (95% CI [16.7–25.0]) with continuous ponesimod versus 24.6% (95% CI [20.5–29.3]) in switchers. No CUALs were observed in 69.7% and 75.9% of patients receiving continuous ponesimod at end of core and extension studies visits respectively. 12.3% of patients experienced severe AE in the ponesimod group.

Conclusion: In patients with baseline EDSS ≤ 3.5 , ponesimod showed sustained clinical and magnetic resonance imaging efficacy across OPTIMUM and the LTE with a favorable safety profile.

Disclosure: O.H. reports consulting and lecturing fees from Bayer Schering, Merck, Teva, Genzyme, Novartis, Almirall and Biogen Idec, travel grants from Novartis, Teva, Genzyme, Merck Serono and Biogen Idec and research support from Roche, Merck and Novartis, all of the above unrelated to this work. M.H, S.G, A.A and A.S are employees of Juvise Pharmaceuticals.

EPO-0535 | Defining Progression Independent of Relapse Activity in Early Multiple Sclerosis

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Background and aims: Progression independent of relapse activity (PIRA) and relapse-associated worsening (RAW) are primary mechanisms of disability accumulation in multiple sclerosis (MS). Various PIRA definitions in literature limit cross-study comparability, and most studies rely on clinical trials rather than real-world data. We aimed to determine a clinically applicable PIRA definition in early MS by comparing 54 detection models.

Methods: We included 392 relapsing-remitting MS patients meeting 2024 McDonald criteria, evaluated within one year of symptom onset, with ≥ 2 years follow-up. Missing EDSS data were imputed by employing a 90-day interval linear interpolation-based imputation method, in which missing values were estimated along the trajectory defined by the preceding and subsequent EDSS assessments. Fifty-four PIRA detection models were compared across four parameters: (1) baseline relapse-free criteria (3 months, 6 months, 12 months), (2) confirmation timeframe (6 months, 12 months, end-of-follow-up), (3) confirmation type (threshold-based vs. absolute), and (4) worsening relapse-free criteria (90 days before worsening, no relapses baseline-to-worsening, no relapses baseline-to-confirmation).

Results: Among 392 patients (257 female), median follow-up was 6.4 years (IQR: 3.2–10.2) and median age at disease onset was 26.4 years (IQR: 21.9–33.3). During up to 5 years of follow-up (1,592.5 patient-years), PIRA prevalence ranged from 1.5% to 7.4% across definitions. We identified the 3-month relapse-free roving baseline, 12-month absolute confirmation with baseline-to-worsening relapse-free criteria as optimal for clinical use. This model yielded 3.3% PIRA and 7.9% RAW prevalence.

Conclusion: We propose 3-month relapse-free roving baseline, 12-month absolute confirmation with baseline-to-worsening relapse-free criteria as the most clinically applicable PIRA definition in early MS.

Disclosure: This study was funded by the Turkish Neuroimmunology Association.

EPO-0536 | Can Immune Cell Dynamics After Short Course Cladribine Tablets Predict MRI Lesion Activity in MS? Insights From The 4 year MAGNIFY MS Study

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Background and aims: It is unknown whether immune reconstitution patterns during years (Y)3–4 (treatment-free period) after cladribine tablets (CladT) treatment relate to MRI-lesion activity. We investigated lymphocyte reconstitution dynamics and MRI-lesion activity during Y3–4 in people with MS (PwMS) after treatment with CladT.

Methods: Absolute and relative changes from baseline (Month [M] 0) in 14 predefined T-cell and 9 B-cell subsets were compared in PwMS with/without Y3–4 MRI-lesion activity. 215 B- and T-cells ratios were also compared. Data were evaluated using Wilcoxon rank tests for univariate comparative analyses, and adjusted, multivariate Cox regression analyses using M15–48 data (post-lymphocyte-count-nadir). $p < 0.05$, and $p < 0.1$ for sensitivity analysis with adjustment for multiple testing, considered nominally significant.

Results: 159/182 PwMS without lesion-activity after treatment completion (M18–24) were evaluated in the comparative analysis; 59 experienced Y3–4 MRI-lesion activity and 100 remained lesion free. Patients with new/active lesions had increased B-memory, B-activated and B-transitional cells, proportionally decreased B-naïve cells, and increased central memory (CM) and Th1 T-cells (Figure 1). Multivariate Cox regression confirmed increased risk for MRI-lesion activity in patients with higher levels of these cells in addition to plasma B-cells, and decreased risk with higher levels of CD8-naïve cells (Figure 2). Adjustment for multiple testing supported the relevance of B-activated and B-plasma cell ratios to CD4 naïve and/or Th2 T-cells (Figure 3).

Conclusion: CM and Th1 T-cells, along with plasma (CD38+ and short-lived), B-memory, and B-activated cells were associated with MRI-lesion activity. Assessing reconstitution dynamics, especially B- vs T-cell ratios, during the treatment-free period may help predict disease activity and guide long-term disease management.

Disclosure: TD: Scientific ad boards: Actelion (Janssen/J&J), Alexion, Amgen, Biogen, Celgene (Bristol Myers Squibb), GeNeuro, Merck, Novartis, Roche, Sanofi. Funding for travel and/or speaker honoraria: Biogen, Merck, Novartis, Roche, Sanofi. Research support: Alexion, the EU, Novartis, Roche,

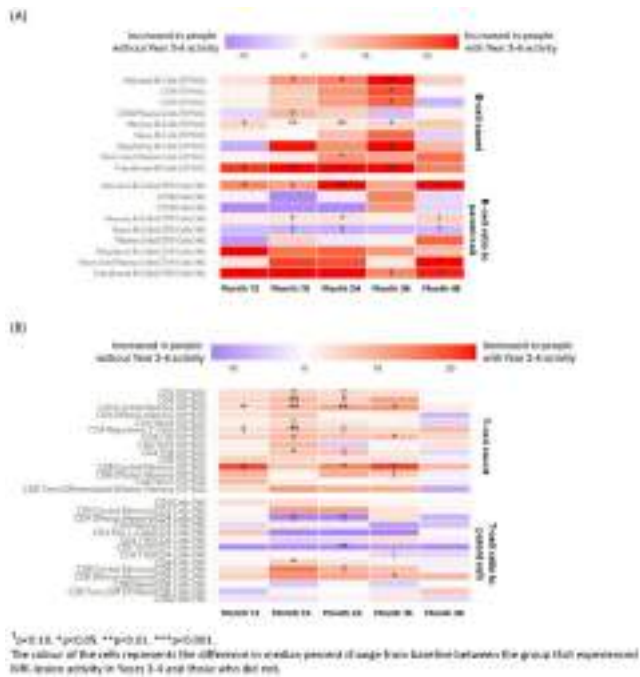


FIGURE 1 Difference in median percent change from baseline in (A) B-cells and (B) T-cells comparing patients resuming MRI lesion activity during Years 3–4 with those remaining lesion-free.

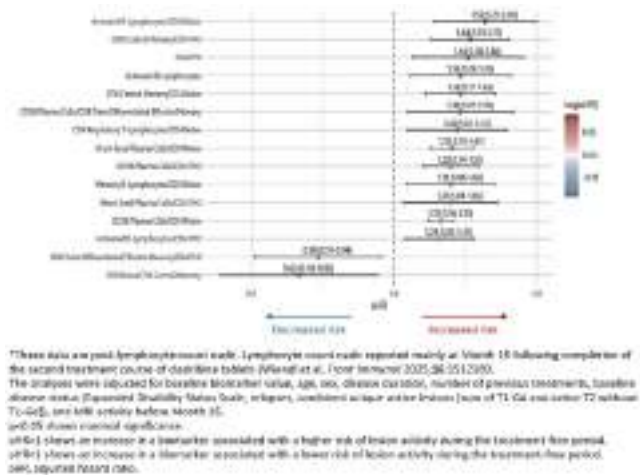


FIGURE 2 Multivariate Cox-regression analyses of MRI activity according to immune cell markers during Months 15–48* (absolute values and ratios; $n = 197$): top 15 associations ($p < 0.05$).

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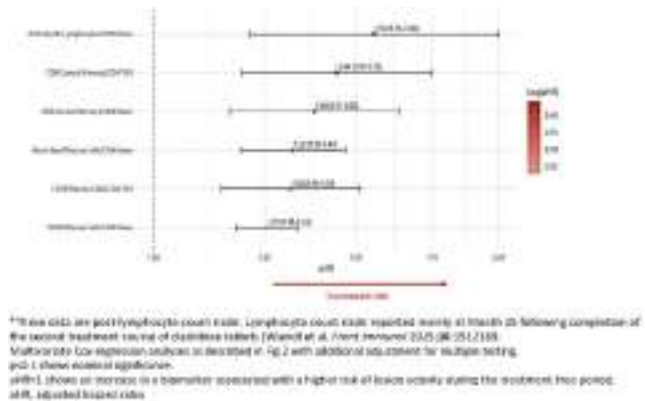


FIGURE 3 Multivariate Cox-regression analyses of MRI activity according to immune cell markers during Months 15–48 (absolute values and ratios; $n = 197$): adjusted for multiple testing ($p < 0.1$).

PSL Group, Red Nucleus, Roche, Samsung, Sangamo, Sanofi, Springer, StreamedUp, Swiss MS Society, Teladohealth, Toleranzia, Teva, UCB, Uvet, Viartis, WebMD global. Scientific Ad Boards: Alexion, Argencx, Biocryst, Bristol Myers Squibb, Cellerys, Galapagos, Janssen, Merck, Novartis, Sandoz-Hexal, uniQure biopharma B.V FS: Scientific ad boards, clinical trial steering committees, consultancy, support for congress participation, speaker honoraria, or research support for his laboratory: Biogen, Celgene (Bristol Myers Squibb), EMD Serono, Merck, Novartis, Roche, Sanofi, Teva NdS: Honoraria: Biogen, Celgene (Bristol Myers Squibb), Genzyme, Immunic, Novartis, Roche, Teva. Advisory boards: Biogen, Immunic, Merck, Novartis, Roche, Sanofi. Research grant support: Italian Ministry of University and Research, Fondazione Italiana Sclerosi Multipla. Associate Editor of Neurological Sciences and co-founder of Siena-Imaging s.r.l DPM: Employee of Ares Trading SA, Eysins, Switzerland, an affiliate of Merck KGaA DG: Employee of Merck AB, Solna, Sweden, an affiliate of Merck KGaA KT: Employee of MeDaStats, LLC, Tampa, USA, funded by Merck KGaA to perform statistical analyses LG: Employee of EMD Serono Research & Development Institute, Inc, Billerica, USA, an affiliate of Merck KGaA KS: Chief investigator of ChariotMS (NCT04695080) supported by NIH, MS Societies, Barts Charity, Merck, and AttackMS (NCT05418010) supported by Biogen & Sandoz. Co-chief investigator of AssistMS supported by UK Department of Health and Social Care and icometrix. Advisory committee: Merck. Research and NHS service support: Merck, Neuraxpharm, Novartis, Roche, Sanofi. Speaking honoraria and/or advisory role: Biogen, icometrix, Merck, Neuraxpharm, Novartis, Roche, Sanofi; remuneration for teaching activities from AcadeMe & Medscape

EPO-0537 | Brain parenchyma displacement and deformation in patients with Spontaneous Intracranial Hypotension (SIH)

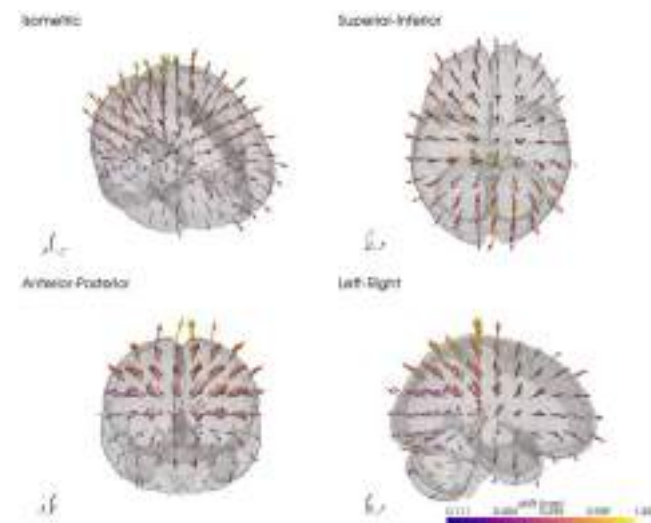
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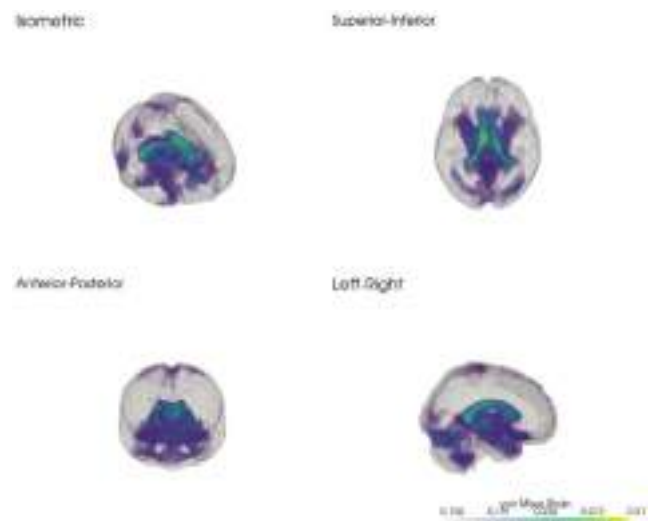
Background and aims: Spontaneous intracranial hypotension (SIH) is mainly associated with orthostatic headache, but can also cause neurological symptoms, with symptoms mimicking behavioral-type frontotemporal dementia in severe cases. SIH is known to lead to brain sagging. The aim of this study was to investigate whether brain MRI imaging in SIH patients shows quantifiable displacement, strain, and compression of brain structures.

Methods: We designed an AI-driven image analysis pipeline for automated segmentation, volumetry and diffeomorphic registration of pre- and postoperative MRI imaging (AssemblyNet, Synthmorph CNNs). We calculated voxel-wise perioperative spatial displacements for all brain areas in 96 patients with SIH. We calculated the Jacobian determinant as a measure of local brain expansion and compression, and the von Mises equivalent strain as a measure of local distortion.

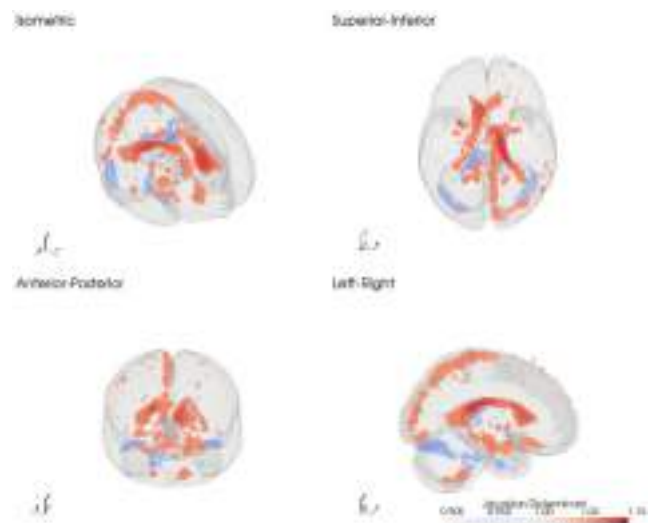
Results: Brain structures demonstrated consistent predominantly upward-outward postoperative shift. Most major brain regions exhibited statistically significant movement, with the highest magnitude seen in the parietal lobes and the left lateral ventricle ($>0.7\text{ mm}$, $p < 0.0001$). Ventricles and transverse sinuses showed relevant perioperative expansion and contraction, respectively. Apart from the ventricles, high strain was observed in temporomesial structures and the ventromedial prefrontal cortex, indicating structural strain.



Three-dimensional illustration of perioperative brain parenchyma shifts.



Three-dimensional illustration of perioperative deviatoric strain in brain parenchyma.



Three-dimensional illustration of Jacobian determinant as a measure of local brain expansion.

Conclusion: Surgical CSF leak closure in SIH is associated with consistent, quantifiable postoperative brain shifts on MRI. We demonstrate strain in brain regions of SIH patients usually affected by frontotemporal dementia, which could explain a similar clinical presentation in severe cases of SIH. AI-assisted image analysis in SIH may provide valuable prognostic information in clinical practice, potentially aiding the assessment of surgical outcome.

Disclosure: Nothing to disclose

A. Gardoni¹; E. Sarasso²; M. Gandolfi³; S. Basaia¹; E. Canu⁴; E. Sibilla¹; C. Tripodi¹; A. Sandri⁵; I. Di Vico⁵; M. Fiorio³; G. Pedrotti³; A. Paolicelli⁵; P. Barone⁶; M. Pellecchia⁶; R. Erro⁶; S. Cuoco⁷; I. Carotenuto⁷; C. Vinciguerra⁷; A. Botto⁸; M. Amboni⁷; M. Russo⁹; G. Mansueto³; F. Pizzini³; M. Barillari³; M. Lauriola⁵; M. Tozzi⁵; F. Rusciano⁵; C. Geroi³; M. Fasoli³; A. Marotta³; E. Pizzolla³; F. Salaorni³; I. Lozzi³; F. Bombieri³; G. Squintani⁵; S. Mariotto⁵; S. Tamburin³; F. Paio³; G. De Biasi⁷; G. Piscosquito⁷; M. Tinazzi³; M. Filippi¹⁰; F. Agosta⁴

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Background and aims: Preliminary evidence in functional movement disorders (FMD) suggested that symptoms might result from faulty neural processing. This study aimed at investigating alterations in resting-state brain functional connectivity (FC) and deep gray matter volume in patients with FMD.

Methods: Sixty patients with FMD and 65 age- and sex-matched healthy controls underwent brain MRI. We assessed the FC within the main resting-state networks using independent component analysis (MELODIC), cerebellar volumes using SUIT, and deep gray matter (GM) volumes using FMRIB's Integrated Registration and Segmentation Tool and FreeSurfer.

Results: FMD patients relative to healthy subjects exhibited an increased FC of bilateral caudate, and right putamen/pallidum, insula, inferior/superior frontal gyrus and olfactory gyrus within the basal ganglia network; an increased FC of bilateral precuneus and left cuneus within the ventral default-mode network (DMN); an increased FC of right middle cingulum within the precuneus network; and an increased FC of left cerebellum crus I-II within the primary visual network. Moreover, FMD patients showed increased volume of vermis crus II, VIIIA/b, and left lobules VIIIA/b. The altered FC within the basal ganglia and

ventral DMN correlated with worse alexithymia, depression and fatigue in FMD patients.

Conclusion: The observed alterations of cerebellar volumes and resting-state FC may represent a mismatch between sensorimotor, emotional, and cognitive systems, leading to impaired self-awareness and motor intention, heightened attention to bodily signals, and compromised voluntary movement control mechanisms. These results support the neural basis of FMD, confirming that distinct mechanisms underlie the symptoms of this complex disorder.

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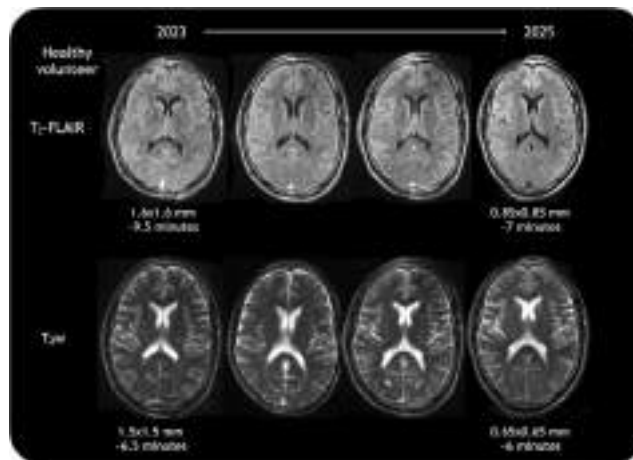
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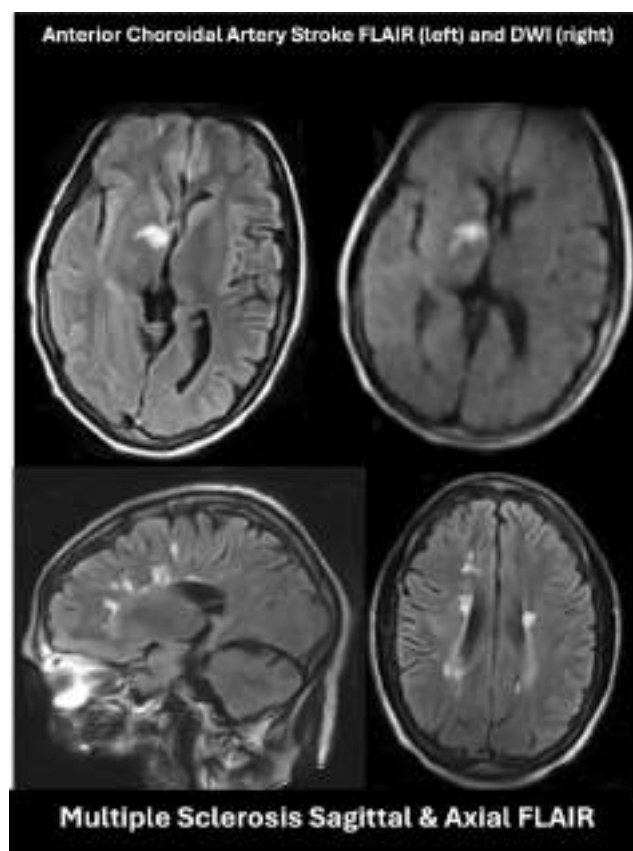
Background and aims: Ultra-low field MRI (0.064T) is a point of care, portable imaging tool that has a small physical foot print, runs off of a standard electric outlet, does not require specialized shielding, and a low technician operating burden. It could improve access to brain imaging in resource limited or remote locations and is ideal for patients who can't tolerate a conventional MRI study. Technical advances in image reconstruction have improved image quality and user satisfaction. AIM: demonstrate improvements in scan-rescan reproducibility and illustrative case examples of lesion detection in MS and Stroke.

Methods: Healthy volunteers were scanned multiple times on Hyperfine Inc's portable Swoop 64mT MRI scanner before and after software upgrade. Image acquisition included Fluid Attenuated Inversion Recovery (FLAIR, thickness=5mm; before: 7.5min, in-plane resolution=1.7mm; after: 7min, in-plane resolution=0.85mm) and T2-weighted (thickness=5mm; before: 5.5min, in-plane resolution=1.5mm; after: 6min, in-plane resolution=0.65mm) sequences. The Structural Similarity Index Measure (SSIM), measuring image contrast, luminance and structure, was used to assess reproducibility of scans before and after this software upgrade.

Results: The average SSIM scores before and after upgrade increased (T2w: 0.958 to 0.962, FLAIR: 0.931 to 0.943), showing improved reproducibility. Qualitatively, both periventricular and juxtacortical demyelinating white matter lesions could be



Sequential improvements in image quality with subsequent software versions.



Illustrative example of stroke and MS lesions visible at 0.064T Ultra Low Field MRI.



Hyperfine Swoop Ultra Low Field 0.064T MRI.

identified, with further improvements seen in identifying infratentorial lesions.

Conclusion: Image quality acquired on an ultra-low field portable MRI scanner has improved with subsequent iterations of scanner software. Potential applications include monitoring of multiple sclerosis patients on treatment, rapid relapse assessment, assessing MS patients with poor mobility, rapid assessment of stroke, and neurocritical care.

Disclosure: E Knopp is an employee of Hyperfine Inc.

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Background and aims: Olfactory dysfunction is among the earliest and most prevalent clinical features of several neurodegenerative disorders, including Alzheimer's disease and Parkinson's disease. Despite its diagnostic and prognostic relevance, the neuroanatomical substrates linking hyposmia to cognitive syndromes across aging and neurodegeneration remain undefined.

Methods: Seventy-four participants divided in cognitively unimpaired (CU), mild cognitive impairment (MCI), and Parkinson's disease-cognitive impairment (PD-CogImp), completed the Brief Smell Identification Test (B-SIT) and underwent high-resolution structural MRI. Cortical thickness (CT) measure was obtained using FreeSurfer. Partial correlation analyses assessed associations between olfactory identification and CT across 34 bilateral cortical regions, controlling for age, sex, education, and APOE ϵ 4 carrier status. Analyses were conducted within diagnostic groups to characterize syndrome-specific structure–olfaction relationships.

Results: Olfactory identification differed across groups, with lowest B-SIT scores in PD-CogImp, followed by MCI and CU. In CU individuals, higher performance was associated with greater CT, predominantly right-hemispheric, including the lateral orbitofrontal cortex ($p=0.04$), cuneus ($p=0.04$), pericalcarine cortex ($p=0.01$), and insula ($p=0.04$). Trend-level associations were observed in left cuneus ($p=0.54$), pericalcarine cortex ($p=0.53$), and rostral middle frontal cortex ($p=0.53$). MCI showed no significant associations (p range = -0.29 to 0.27 ; all $p \geq 0.35$). PD-CogImp showed absent or inverse structure–olfaction relationships in posterior and associative cortices, consistent with syndrome-specific neuroanatomical disruption.

Conclusion: Olfactory identification shows syndrome-specific associations with cortical thickness across CU aging, MCI, and PD-CogImp, supporting its utility for differential diagnosis and neurobiological characterization of neurodegenerative disease.

Disclosure: Richard Doty is president and major shareholder of Sensonics International, the manufacturer of the olfactory test used in this study. All the other authors have no competing interests and nothing to disclose.

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Background and aims: Brain connectivity is an important determinant of disability in multiple sclerosis (MS). It varies across the disease course and can lead to “network collapse”, causing clinical deficits. We investigated how changes in short- and long-range functional and structural connectivities within the sensorimotor network (SMN) contribute to disability and physical performance worsening over one year in MS patients.

Methods: We studied 100 MS patients and 89 healthy controls (HC), who underwent 3.0-Tesla MRI and clinical evaluation, including Nine-Hole Peg Test (9-HPT), Timed 25-Foot Walk Test (T25FWT), and Expanded Disability Status Scale (EDSS). MS patients were reassessed clinically after one year. Clinical worsening was defined using the EDSS-Plus (EDSS progression or $\geq 20\%$ deterioration in 9-HPT or T25FWT). Group comparisons between HC and MS, and between stable/worsened MS patients, evaluated functional connectivity (FC) and structural connectivity (SC) of the SMN at intra- and inter-hemispheric levels.

Results: Compared with HC, MS patients had worse baseline motor performance ($p \leq 0.001$) and presented low disability levels (median EDSS = 1.5). They showed reduced inter-hemispheric SMN SC ($p \leq 0.001$), while other connectivity measures did not differ. After one year, 36 patients were classified as worsened by EDSS-Plus. No baseline demographic or clinical differences emerged between stable and worsened patients. Worsened patients showed higher intra-hemispheric SMN SC at baseline ($p=0.013$), which significantly decreased at follow-up ($p=0.006$). No differences were observed in other SMN connectivity measures.

Conclusion: SMN connectivity evolves nonlinearly in MS. Inter-hemispheric SC disruption appears early, while increased intra-hemispheric connectivity may reflect compensatory reorganization and is associated with subsequent clinical worsening.

Disclosure: F Romanò, S Zucchelli, E Pagani, P Valsasina have nothing to disclose. P. Preziosa received speaker honoraria from Roche, Biogen, Novartis, Merck, Bristol Myers Squibb, Genzyme, Horizon and Sanofi. M.A. Rocca received consulting fees from Biogen, Bristol Myers Squibb, Roche; and speaker honoraria from Alexion, Biogen, Bristol Myers Squibb, Celgene, Horizon Therapeutics Italy, Merck Serono SpA, Mitsubishi-Tanabe Pharma, Neuraxpharm, Novartis, Roche, Sandoz, and Sanofi. She receives research support from the MS Society of Canada, the Italian Ministry of Health, the Italian Ministry of University and Research, and FISM. She is Associate Editor for Multiple Sclerosis and Related Disorders; and Associate Co-Editor for Europe and Africa for Multiple Sclerosis Journal. M. Filippi is Editor-in-Chief of the Journal of Neurology, Associate

Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharmed Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and FISM.

EPO-0542 | Clinical relevance of baseline brain MRI-derived risk groups for accelerated disability progression in Multiple Sclerosis

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Background and aims: MRI metrics offer potential for assessing disease progression in patients with Multiple Sclerosis (pwMS). We aim to validate whether baseline MRI metrics can estimate the risk of faster progression.

Methods: The study utilized a subset of the RECLAIM dataset. A parametric Weibull survival model was trained to predict time-to-event for reaching Expanded Disability Status Scale (EDSS) ≥ 3 on 1002 pwMS with baseline EDSS < 3 . Features included 29 MRI-based metrics computed by icobrain, age, and sex. Model's risk predictions were used to stratify an independent test set (329 patients) into low-risk group (LRG; $n = 294$) and high-risk group (HRG; $n = 35$). We assessed the risk group characteristics in terms of EDSS, brain and lesion volumes.

Results: The MRI-based risk clustering showed statistically significant faster disease progression of HRG compared with LRG ($p = 0.0006$) over a 15 year follow-up (Figures 1 and 2). HRG showed significantly lower brain tissue volumes (1467.06 ± 65.98 ml vs. 1555.71 ± 51.45 ml in LRG) and higher lesion volumes (19.55 ± 10.94 ml vs. 3.41 ± 3.36 ml in LRG) at baseline, suggesting a link between brain atrophy, lesion burden, and a higher risk for conversion to EDSS ≥ 3 (Figure 3).

Conclusion: These findings support the hypothesis that brain tissue loss and lesion accumulation contribute to an increased risk of disease progression. With further validation, this approach could help clinicians better interpret brain MRI volumetric measurements for making clinical decisions.

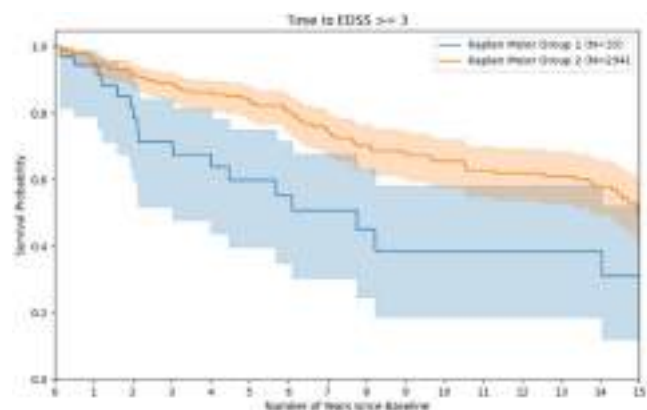


FIGURE 1 Faster progression represented by an accelerated decrease of the survival probability (Kaplan-Meier curve) over a 15-year follow-up in the high-risk group (Group 1 in blue) in comparison with the low-risk group (Group 2 in orange).

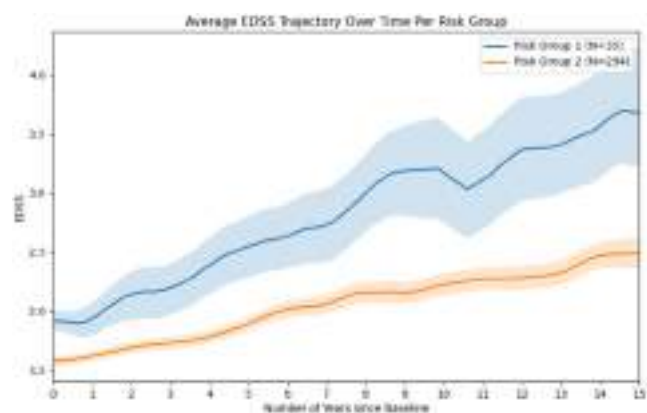


FIGURE 2 Faster progression represented by accelerated increase of disability (EDSS trajectory) over a 15-year follow-up in the high-risk group (Group 1 in blue) in comparison with the low-risk group (Group 2 in orange).

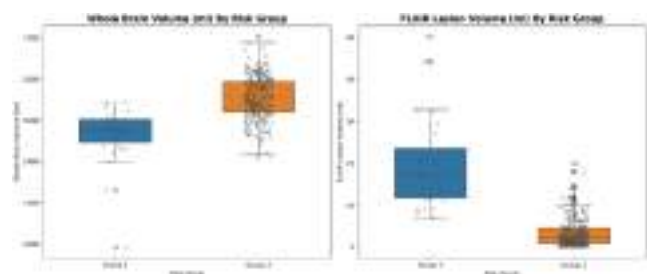


FIGURE 3 Boxplot showing the distribution of the whole brain volumes normalized for head size (left) and FLAIR lesion volumes (right) of the high risk group (Group 1 in blue) and the low-risk group (Group 2 in orange).

Disclosure: 1 icometrix, Leuven, Belgium 2 Ruhr University Bochum, St. Josef Hospital, Institute of Neuroradiology, Bochum, Germany 3 Ruhr University Bochum, St. Josef Hospital, Department of Neurology, Bochum, Germany 4 Department of Neurology, Charité - Universitätsmedizin Berlin, Berlin, Germany 5 Department of Neurology and Centre of Clinical

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EPO-0543 | Superior anatomical fidelity of multi-shell (MS) vs single-shell (SS) tractography for deep brain stimulation (DBS) targeting in tremor disorders

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Background and aims: Tremor improvement following DBS is increasingly linked to modulation of the dentatorubrothalamic tract (DRTT). Targeting the DRTT, surgical planning often relies on SS diffusion tractography, which may limit anatomical fidelity in areas with complex fibre architecture. This study compared the reliability and anatomical precision of MS vs SS tractography for DRTT reconstruction in patients with Parkinson's disease, essential tremor and dystonic tremor.

Methods: We conducted a unicentral, retrospective analysis of 41 movement disorder patients undergoing DBS using 3-Tesla SS and MS diffusion MRI. DRTT reconstruction was performed on individual patient data without template registration. Evaluation focused on reliability of DRTT visualisation within the posterior subthalamic area (PSA, **Figure 1.i**) and detection of decussating fibres in the brainstem (**Figure 1.ii**) across streamline thresholds from >150 to >500 streamlines per voxel (spv).

Results: MS tractography consistently demonstrated higher reliability than SS. Bilateral DRTT reconstruction was achieved in all patients using MS data, whereas SS imaging failed to detect the DRTT in 27–29% of cases at >150 and >200 spv ($p < 0.001$). Decussating fibres were identified in 98% of cases using MS compared to 70–73% using SS ($p < 0.006$). These findings were consistent across higher streamline thresholds.

Conclusion: Patient-specific MS diffusion tractography provides superior reliability and anatomical fidelity for DRTT reconstruction compared to SS imaging. Our results support the use of MS reconstructions at lower streamline thresholds to

refine DBS targeting which may translate into improved tremor outcomes.

Disclosure: Nothing to disclose.

EPO-0544 | Lesion Histogram Features for Distinguishing Multiple Sclerosis And Systemic Lupus Erythematosus

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Background and aims: Evolution of white matter lesions in the course of autoimmune inflammatory disorders differs depending on severity of demyelination and recovery dynamic. Multiple Sclerosis (MS) and Systemic Lupus Erythematosus (SLE) with neuropsychiatric presentation may be difficult to distinguish in clinical practice. We aimed identify the features of the signal distribution of T1-weighted and FLAIR sequences in the lesions and the cortex in the MS and SLE populations.

Methods: We investigated distributions of regions of interest signal using skewness and kurtosis and creating 2D histograms to differentiate data from 42 MS patients and 35 SLE patients acquired at 3T MR 750 GE scanner. The FLAIR and T1-weighted images were coregistered, bias field corrected and segmented using SAMSEG pipeline (Puonti 2021). Then extracted skewness and kurtosis using python script and computed diagnostic performance of the receiver operator characteristic curves.

Results: The most effective features discriminating systemic lupus erythematosus patients from multiple sclerosis were lesion 2D PCA receiver operator characteristic area under the curve Skewness 0.77 and Kurtosis 0.69. For cortex 2D PCA receiver operator characteristic area under the curve skewness 0.59 and kurtosis 0.51. For each mode separately FLAIR lesion kurtosis AUC is 0.76 and for skewness 0.64; for cortex kurtosis 0.48 and skewness 0.52. For T1-weighted lesion signal both kurtosis and skewness were 0.62; for the cortex skewness was 0.43 and kurtosis was 0.60.

Conclusion: The multimodal 3D MRI acquisitions enables generation of reliable 2D signal histograms characterized by signal features that help distinguish populations of patients with multiple sclerosis and systemic lupus erythematosus.

Disclosure: Mikolaj A. Pawlak: Speakers fee from Merck range \$500–4900 two years ago. J. Nikadon: nothing to disclose. A.Wypych: nothing to disclose. A. Kalinowska received honoraria and/or consultation fees and/or support for

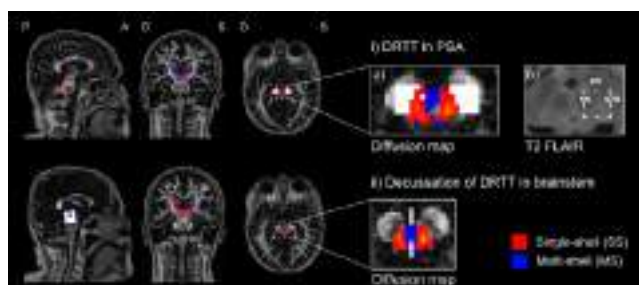


FIGURE 1i, SS and MS DRTT projections within the PSA (white box), shown on diffusion image (a) and T2-FLAIR (b), highlighting the subthalamic nucleus (STN), substantia nigra (SN) and red nucleus (RN). **ii**, Decussating fibres crossing the brainstem midline.

attending meetings and/or travel from: Bayer, Biogen, Bristol Myers Squibb, GSK, Janssen-Cilag, Merck, Novartis, Roche, Sandoz, Sanofi-Genzyme.

EPO-0545 | Amide Proton Transfer Imaging Detects Hippocampal Proteostasis Disturbance Induced by Simulated Microgravity at 7.0 T MRI

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Background and aims: Many studies have confirmed that spacelight and simulated microgravity can affect learning and memory, resulting in cognitive decline; however, the effects of simulated microgravity on cognitive function remain unclear. The relationship between cognitive decline and proteostasis disorders in the brain under microgravity has not been elucidated. While amide proton transfer (APT) imaging is a chemical exchange saturation transfer technique that utilizes amide protons in proteins and peptides, it can serve as a non-invasive imaging biomarker for identifying brain proteostasis disorders.

Methods: We employ a 14-day hindlimb unloading (HU) rat model to simulate microgravity conditions and utilize APT imaging on HU Rats and control rats. To explore the mechanisms of microgravity-induced cognitive impairment in spaceflight through mass spectrometry-based proteomics analysis combined with hematoxylin and eosin (HE) staining and Nissl staining.

Results: Our results show that 14 days of tail suspension can cause disturbed proteostasis, and the brain APT signal increases with increasing tail suspension time, which is significantly higher in HU rats than in normal rats, and this signal showed a negative correlation with the number of viable neurons counted in Nissl staining. Through mass spectrometry-based proteomics analysis, differentially expressed proteins within the protein homeostasis network pathway were identified in the hippocampi of both normal and HU rats, providing biological evidence for the altered APT signal.

Conclusion: This study shows that APT imaging can monitor the disturbance of protein homeostasis in the brain of simulated microgravity rats in vivo and provides more references for the study of cognitive decline in simulated microgravity rats.

Disclosure: Nothing to disclose

EPO-0546 | Double Dat: Clinical impact of repeated brain dopaminergic neurotransmission nuclear imaging - A retrospective cohort

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¹Neurology Service, Local Health Unit of Coimbra, Coimbra, Portugal; ²Nuclear Medicine Service, Local Health Unit of Coimbra, Portugal

Background and aims: The clinical role of repeating dopamine transporter studies remains unclear, particularly in the diagnostic workflow for parkinsonian syndromes. Objectives: To characterize patients undergoing repeated dopaminergic imaging and evaluate its repetition on clinical management.

Methods: Retrospective descriptive analysis of patients who underwent multiple dopamine transporter imaging studies between 2007 and 2025 at a Portuguese center.

Results: 2930 studies were performed (90.0% DATScan, 10.0% PET-BCIT) in 2828 patients. Ninety-seven patients (50.5% male; 49.5% female) underwent repeated scanning (94.8% twice; 5.2% thrice). Median age at first examination was 66.0 years (57.0–72.0). Tremor was the most frequent motor symptom (44.2%), followed by akinesia (27.9%) and gait disturbances (16.3%). Median symptom duration was 4.0 years (1.0–7.5) and median MDS-UPDRS-III score was 12.0 (8.0–25.0). DATScan was the first requested exam in 92.8% of cases; 62.0% were initially normal. Median inter-scan interval was 43.0 months (22.0–72.0). While 65.6% of patients initially evaluated with DATScan repeated it, all PET-BCIT patients later had DATScan. Imaging results changed in 28.3% of cases (16.3% from “abnormal” to “normal”; 12.0% from “normal” to “abnormal”). Normalization occurred in 78.6% of patients who underwent PET-BCIT after an initially abnormal DATScan. The second scan led to diagnostic changes in 20.5% and therapeutic modifications in 39.2% of patients.

Conclusion: Most patients did not benefit from repeat imaging. High rates of normalization were found in the transition from DATScan to PET-BCIT.

Disclosure: Nothing to disclose.

EPO-0547 | Noninvasive Prediction of Ki-67 expression in gliomas using MRI-based artificial intelligence: A systematic review and meta-analysis

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Background and aims: Gliomas are aggressive central nervous system tumors, and the Ki-67 proliferation index is a key prognostic marker. However, assessment via immunohistochemistry (IHC) is invasive and subject to sampling bias. Magnetic resonance imaging (MRI)-based artificial intelligence (AI) has emerged as a promising noninvasive alternative, but its diagnostic performance remains uncertain. This meta-analysis evaluates the diagnostic accuracy of MRI-based AI in predicting Ki-67 status in gliomas.

Methods: A systematic search was performed for studies through August 2025. Eligible studies compared MRI-based AI models with IHC as the reference standard. Pooled sensitivity, specificity, and area under the summary receiver operating characteristic (SROC) curve were estimated using a univariate random-effects model. Heterogeneity was assessed using the I² statistic. Statistical analyses were performed using RStudio (R version 4.2.3)

Results: From 1,704 identified records, eleven studies comprising 893 patients were included. MRI-based AI models achieved a pooled sensitivity of 0.849 (95% confidence interval (CI): 0.730–0.921) and a pooled specificity of 0.744 (95% CI: 0.636–0.829) for Ki-67 prediction. The SROC curve yielded an



FIGURE 1 Forest plots of sensitivity (left) and specificity (right) across included studies.

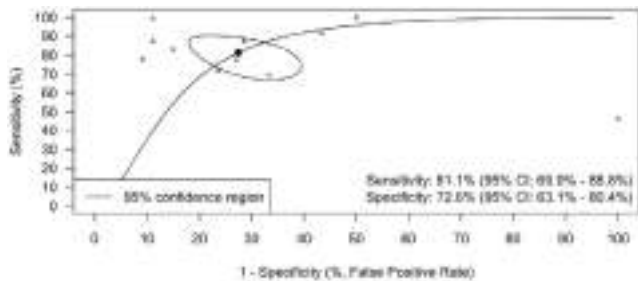


FIGURE 2 SROC curve derived from bivariate analysis for MRI-based AI models predicting Ki-67 expression in gliomas.

area under the curve (AUC) of 0.82 (95% CI: 0.676–0.872), reflecting good diagnostic accuracy. Heterogeneity was substantial for sensitivity ($I^2=77\%$) and moderate for specificity ($I^2=40\%$). Variability likely stemmed from differences in MRI protocols, AI architectures, and Ki-67 thresholds across studies.

Conclusion: MRI-based AI demonstrates good diagnostic accuracy (AUC=0.82) for noninvasive prediction of Ki-67 status in gliomas. These findings highlight its potential to support preoperative decision-making. However, standardized imaging protocols, consistent thresholds, and robust prospective multicenter studies are crucial for clinical implementation

Disclosure: Nothing to disclose

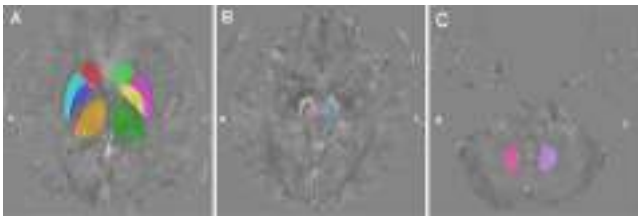
EPO-0548 | The value of quantitative susceptibility mapping in differential diagnosis of Parkinson's disease and atypical parkinsonian syndrome

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The Union Hospital Affiliated to Fujian Medical University, Fuzhou, China

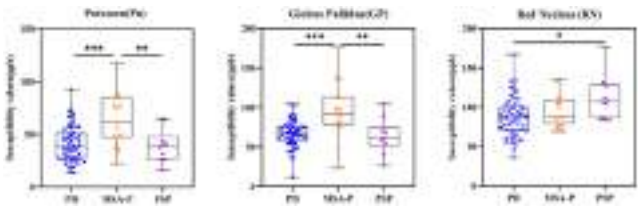
Background and aims: To assess brain iron deposition features in Parkinson's disease (PD) and atypical parkinsonian syndromes (APS) using Quantitative Susceptibility Mapping (QSM) and evaluate its differential diagnostic value.

Methods: This study included 80 patients (with 2-year follow-up for diagnosis confirmation) and 33 matched controls. Regional susceptibility values were obtained via manual ROI sketching. Statistical analyses (including logistic regression and ROC curve analysis) identified differential features and evaluated diagnostic performance.

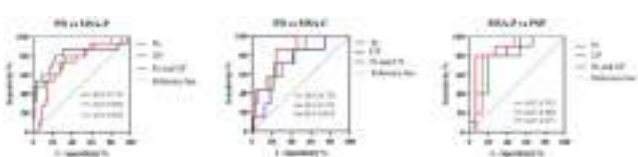
Results: 1) Specific brain regions showed higher susceptibility in each group versus controls. 2) MSA-P and PSP groups had higher UPDRS-III scores and H-Y stages than PD. 3) Significant inter-group differences were found in susceptibility of the putamen, globus pallidus, and red nucleus. 4) In PD only, disease severity scores correlated with susceptibility in the putamen and red nucleus. 5) Combined putamen/globus pallidus susceptibility effectively differentiated MSA-P from PD (AUC=0.838) and



QSM Region of Interest (ROI) Mapping Diagram



Intergroup Comparison of QSM Susceptibility Values for Different Nuclei.



ROC Curve of QSM in the Differential Diagnosis of PD.

from PSP (AUC=0.873). Red nucleus susceptibility differentiated PD from PSP (AUC=0.760).

Conclusion: QSM reveals distinct iron deposition patterns (PD: substantia nigra; MSA-P: putamen/globus pallidus; PSP: red nucleus). These patterns, along with clinical correlations in PD, demonstrate the utility of QSM for differential diagnosis among Parkinsonian disorders.

Disclosure: Nothing to disclose.

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EPO-0549 | Neurological Manifestations of Preeclampsia

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Tashkent State Medical University, Tashkent, Uzbekistan

Background and aims: Preeclampsia is one of the leading causes of maternal morbidity and is frequently associated with central nervous system involvement, increasing the risk of neurological and cognitive complications.

Methods: A total of 120 pregnant women in the third trimester were examined: 80 patients with preeclampsia and 40 with normal pregnancy. All participants underwent clinical and neurological examination, blood pressure assessment, evaluation of proteinuria, and cognitive testing using the Montreal Cognitive Assessment (MoCA). Statistical analysis included Student's t-test, chi-square test, and Pearson correlation analysis. Differences were considered statistically significant at $p<0.05$.

Results: Neurological symptoms were observed in 67.5% of patients with preeclampsia compared to 12.5% in the control group

($p < 0.001$). The most common manifestations were headache (58.7%), visual disturbances (32.5%), and dizziness (28.7%). The mean MoCA score was significantly lower in the preeclampsia group (24.1 ± 2.3) than in controls (27.6 ± 1.8 , $p < 0.01$). A positive correlation was found between systolic blood pressure and severity of neurological symptoms ($r = 0.62$; $p < 0.01$), while proteinuria showed a negative correlation with cognitive performance ($r = -0.54$; $p < 0.05$).

Conclusion: Neurological and cognitive impairments are common in preeclampsia and are significantly associated with disease severity, highlighting the importance of early neurological monitoring in affected patients.

Disclosure: Nothing to disclose.

EPO-0550 | Long-term cognitive, emotional and quality-of-life outcomes after recovery from prolonged disorders of consciousness

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Background and aims: Epidemiological studies on prolonged disorders of consciousness (PDOC) usually focus on recovery of consciousness and functional independence. However, whether recovery is meaningful to patients depends on cognitive, emotional and behavioural functioning and health-related quality of life (HRQoL) as well. We studied these factors in a nationwide PDOC cohort.

Methods: Prospective cohort study including PDOC patients ≥ 16 years admitted to the nationwide Dutch PDOC chain of care (2019–2023) with follow-up until 2 years post-admission. Outcomes included cognition (Montreal Cognitive Assessment, MoCA), emotional functioning (Hospital Anxiety and Depression Scale, HADS), neuropsychiatric symptoms (Neuropsychiatric Inventory, NPI), and HRQoL (Quality of Life after Brain Injury questionnaire, QOLIBRI). Longitudinal changes were analysed using generalized estimating equations.

Results: For 61 patients, data were available at two-year follow-up (mean age 37 years; 68% traumatic aetiology). Cognitive functioning improved significantly over time, but remained impaired in most patients: only 11% achieved a normal MoCA score at two years. Anxiety and depression decreased over time, yet persisted in a substantial proportion of patients who could complete questionnaires (15% and 30%, respectively). Neuropsychiatric symptoms were absent in 67% of patients at two years. Condition-specific HRQoL was within the normal range in 32% of patients able to complete the QOLIBRI.

Conclusion: Patients recovering from PDOC constitute a highly heterogeneous group with wide variability in cognitive, emotional and quality-of-life outcomes. While a meaningful recovery is achievable for a subset of patients, persistent cognitive and emotional sequelae are common and should be central in long-term outcome evaluation and shared decision-making.

Disclosure: Nothing to disclose

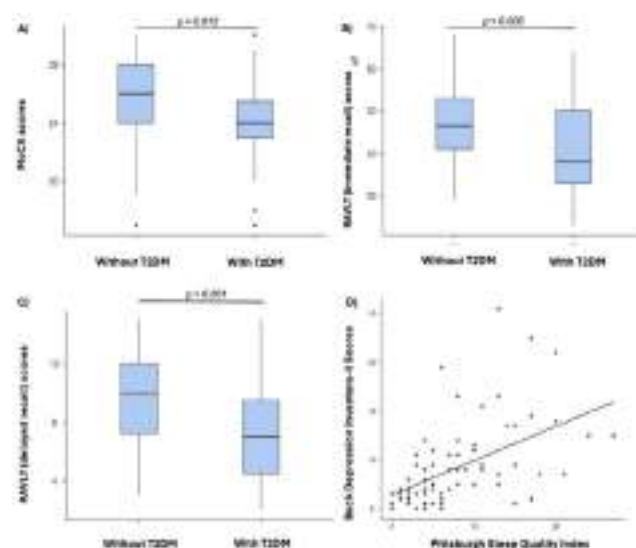
EPO-0551 | Neuropsychological assessment in obesity: Cognitive vulnerability in patients with Type 2 diabetes mellitus

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Department of Medical, Surgical and Neurological Sciences, University of Siena, Siena, Italy

Background and aims: This study aims to assess cognitive functions, mood, and sleep quality in obese patients and evaluate their association with comorbidities, given the elevated risk of neurodegenerative diseases linked to obesity and the limited understanding of these associations

Methods: One hundred and one obese patients (80 females) were recruited and underwent a neuropsychological assessment including Montreal Cognitive Assessment (MoCA), a Rey Auditory Verbal Learning Test (RAVLT), the Beck Depression Inventory (BDI-II), and the Pittsburgh Sleep Quality Index (PSQI).

Results: Comorbidities were carefully recorded. A non-parametric statistical analysis was performed with age and BMI as covariates. The median age of our cohort was 48 years [25th–75th percentile 34–55]; the median body weight was 116 kg (25th–75th percentile 105–129); the median BMI was 43.1 (25th–75th percentile 40–45.7), and 27 patients had type 2 diabetes. The median education level was 12 years, with no difference between diabetic and non-diabetic obese individuals. Comparing the subcohort of diabetic obese patients with those without diabetes, we found significantly lower scores for MoCA ($p = 0.012$; median value 24 vs 26; 25th–75th percentile 23–25.5 vs 25th–75th percentile 24–28), immediate RAVLT ($p = 0.005$; median value 38 vs 46.5; 25th–75th percentile 33–50 vs 25th–75th percentile 41–53) and delayed recall ($p < 0.001$; median value 7 vs 10; 25th–75th percentile 4.5–9.5 vs 25th–75th percentile 7.25–12). BDI-II scores correlated with PSQI scores ($r = 0.54$; $p < 0.001$).



Cognitive and psychological measures in participants with and without type 2 diabetes mellitus (T2DM). (A–C) Boxplots comparing performance on the MoCA (A), RAVLT immediate recall (B), and RAVLT delayed recall (C). Participants with T2DM scored significantly lower.

Conclusion: Our ongoing study indicates a potential link between type 2 diabetes mellitus, obesity, and reduced cognitive performance, as evidenced by borderline/impaired MoCA scores.

Disclosure: No disclosure to declare.

EPO-0552 | Core components of apraxia differentially predict disability after stroke: Evidence from principal component analysis

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Background and aims: Apraxia is a common but heterogeneous higher-order motor disorder after stroke, affecting 30–50% of survivors and strongly associated with disability. However, the relationship between specific apraxic deficits and functional independence remains unclear. Traditional subtypes (ideational, ideomotor, constructional) may not capture functionally relevant dimensions, and prior studies were limited by small samples and multicollinearity. Here, we identify core apraxia components that independently predict disability in the largest UK post-stroke cohort comprehensively assessed for apraxia.

Methods: Eighty-five stroke patients (mean age 60.5 ± 11 years; 42% female; median 3.6 years post-stroke) completed a comprehensive apraxia battery assessing gesture production, object use, imitation, visuospatial processing, biological motion, and executive sequencing. Eleven composite z-scores underwent principal component analysis with varimax rotation following KNN imputation. Factorability was assessed using KMO and Bartlett's test. Multiple regression examined independent predictors of instrumental activities of daily living (Frenchay Activities Index), comparing rotated and unrotated solutions.

Results: Data showed excellent factorability (KMO=0.807, Bartlett's $p < 0.001$). Varimax rotation revealed three components explaining 62% of variance: (1) Gesture/Praxis (23.2%); (2) Visuospatial/Imitation (21.4%); and (3) Executive/Sequential (17.4%). All three independently predicted iADL performance (Adj- $R^2 = 0.338$, $p < 0.001$), with Gesture/Praxis showing the strongest effect ($\beta = 0.82$, $p < 0.0001$), followed by Visuospatial/Imitation ($\beta = 0.70$, $p < 0.0001$) and Executive/Sequential abilities ($\beta = 0.36$, $p = 0.038$).

Conclusion: Apraxia fractionates into three dissociable components with differential functional impact, challenging unitary deficit models. Gesture production is the strongest determinant of independence, explaining divergent functional outcomes among patients with similar overall severity. These components likely reflect distinct neural mechanisms and argue for targeted, component-specific rehabilitation strategies.

Disclosure: Nothing to disclose

EPO-0553 | Metabolic and bioimpedance changes associated with dietary ketosis in early cognitive decline: A pilot study

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Background and aims: Systemic metabolic alterations and chronic low-grade inflammation are increasingly recognized as contributors to early neurodegenerative processes. Bioelectrical impedance vector analysis (BIVA), particularly phase angle, has emerged as a potential marker of cellular integrity and inflammatory status in neurological disorders.

Methods: In this single-center pilot study, patients aged 60–85 years with Mild Cognitive Impairment (MCI) or Subjective Cognitive Decline (SCD) followed one of three dietary interventions for three months: a modified ketogenic diet, a Mediterranean diet supplemented with medium-chain triglycerides (MCT), or a standard Mediterranean diet. Anthropometric measures, ketonemia, and BIVA-derived parameters (fat mass, fat-free mass, phase angle) were assessed at baseline and follow-up, alongside clinical neurological evaluation.

Results: The ketogenic diet was associated with a significant reduction in body weight and body mass index compared to the Mediterranean diet. Although changes in fat mass, lean mass, and phase angle did not reach statistical significance, patients following the ketogenic intervention showed a consistent favorable trend toward increased phase angle, suggestive of improved cellular integrity. These changes occurred in parallel with modest increases in circulating ketone levels. No adverse effects on lean mass were observed.

Conclusion: Diet-induced ketosis was associated with favorable metabolic and bioimpedance trends in patients with

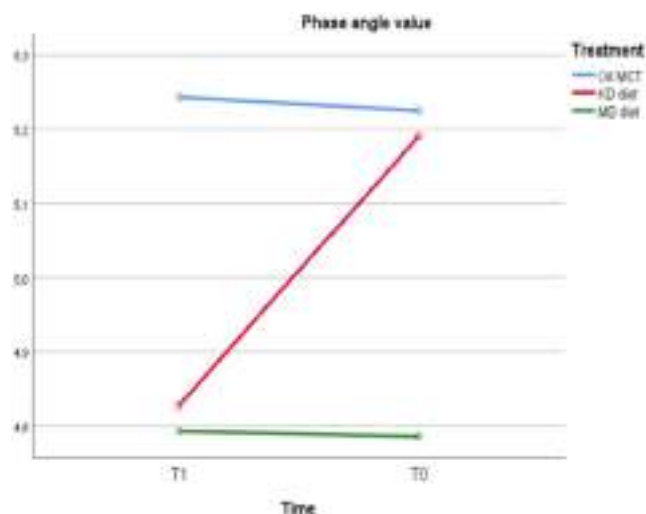


FIGURE 1: The graph highlights a metabolic improvement in patients on the ketogenic diet, evidenced by an increase in phase angle, which is indicative of a reduction in systemic inflammatory status.

Disclosure: Nothing to disclose.

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Methods: An electronic search was conducted in PubMed and APA PsycINFO for articles published between 1972 and July 2025, following PRISMA guidelines. A total of 503 studies were screened by title and abstract, and 62 full texts were assessed for eligibility. Each study's characteristics related to tools, populations, sociocultural effects, normative and validation data were



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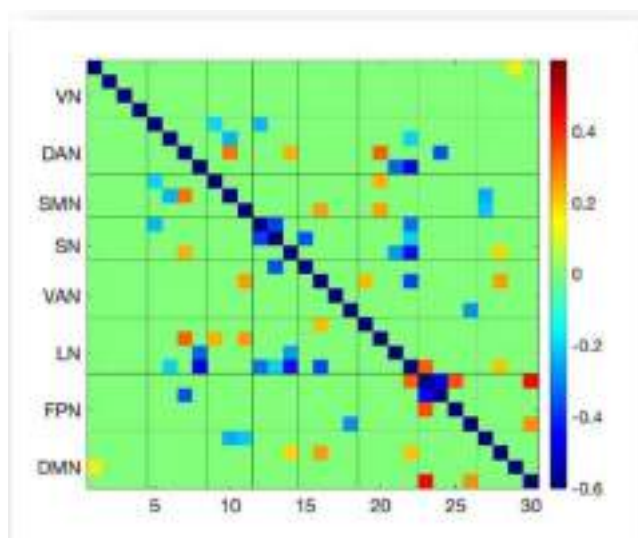
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Background and aims: The insular cortex is considered a cerebral hub involved in multiple cognitive domains. Ischemic strokes involving the insular cortex are often clinically under-rated, as routine assessments across acute/subacute and chronic phases often fail to capture the multifaceted complexity of this subtype of stroke. This study aims to characterize the neuropsychological and resting-state Functional Connectivity (rs-FC) profiles of patients with ischemic stroke involving the insular cortex, evaluated during the subacute and chronic stages.

Methods: Fifteen patients with subacute ischemic stroke were enrolled: seven with insular involvement and eight with strokes in other cerebral regions. All participants underwent a comprehensive neuropsychological assessment and resting-state functional MRI (rs-fMRI). Six patients with insular stroke completed a follow-up evaluation at 3–3.5 months.

Results: Compared with patients with non-insular strokes, patients with insular involvement showed significant impairments on the Trail Making Test and phonemic fluency, along with trend-level reductions on the Reading the Mind in the Eyes Test, the Ekman 36-item Faces Test, and the Rey–Osterrieth Complex Figure recall. At follow-up, emotion recognition and verbal fluency improved, whereas attentional/executive functions and visuospatial memory did not show significant recovery. Rs-FC analysis revealed reduced intra-network connectivity within the Salience Network and decreased anticorrelation between the Default Mode Network and the Frontoparietal and Dorsal Attention Networks. At follow-up, inter-network connectivity



Functional connectivity matrix showing between-group differences (two-sample t-test) between patients with insular stroke and those with non-insular stroke during the acute phase.

involving the Salience and Ventral Attention Networks showed a partial tendency toward normalization.

Conclusion: Insular stroke is associated with crucial Salience Network dysfunction and related executive–attentional deficits, with only partial recovery during the first months after the acute event.

Disclosure: Nothing to disclose.

EPO-0556 | **Perfusion Abnormality is Associated with Cognitive Impairment in Patients with Chronic Carotid or MCA Artery Occlusion**

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Background and aims: Cognitive impairment is well known to be associated with Chronic occlusion of large vessel occlusion like carotid or middle cerebral artery. It is still unclear what factors influence the occurrence of cognitive impairment in these patients. Here, we investigate the radiological and clinical parameters associated with cognitive dysfunction in the patients with chronic occlusion of carotid artery.

Methods: From unicentric, prospectively collected stroke registry, we enrolled the patients who had chronic carotid or MCA occlusion and who took MMSE at admission and 2 years later simultaneously. We excluded patients with severe neurologic deficit or aphasia or the patients with stroke recurrence. We analyzed the results of MMSE, the clinical history, and radiological findings in these patients.

Results: Among the patients with chronic carotid or MCA artery occlusion, a total of 105 patients were enrolled. The mean age was 76 ± 15 years and 62 patients were men. During the median follow-up of 26.8 months, 75 patients showed aggravation of MMSE score. The multivariate analysis demonstrated the old age (75 or older), diabetes mellitus, atrial fibrillation and perfusion abnormality in lesion area. The site of occlusion, HbA1c level, or type of antithrombotic agent was not associated with cognitive deterioration.

Conclusion: Cognitive aggravation is common in the patients with chronic occlusion of carotid or MCA artery occlusion. Perfusion status at baseline appears to be a good predictor of cognitive impairment, suggesting that interventions aimed at improving perfusion deficits may have beneficial effects.

Disclosure: Nothing to disclose.

EPO-0557 | **Selective Executive and Attentional Dysfunction in Chronic Obstructive Pulmonary Disease**

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Background and aims: Chronic obstructive pulmonary disease (COPD) is associated with chronic hypoxia, systemic

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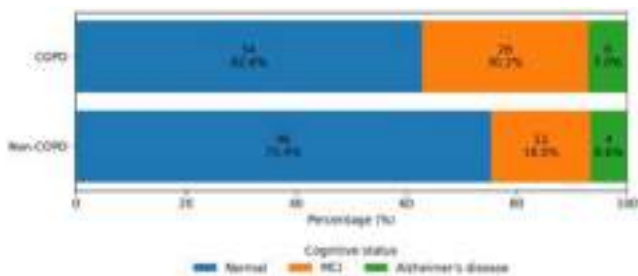


FIGURE 1 Distribution of cognitive status in individuals with and without COPD.

TABLE 1 comparison of cognitive test results in individuals with and without COPD.

Variables	Non-COPD (n=61)		COPD (n=86)		p
	n	%	n	%	
Cognitive status					
Normal	49	78.7	34	39.5	0.000
MCI	11	18.0	38	44.1	
AD	1	1.6	14	16.3	
MMSE score	Mean ± SD Median (Min-Max)		Mean ± SD Median (Min-Max)		p
MMSE score	26.25 ± 5.32 21 (17-30)		21.83 ± 5.43 22 (11-30)		0.119
MoCA score	25.90 ± 2.88 27 (16-30)		21.27 ± 3.14 28 (13-30)		0.236
Semantic fluency	18.72 ± 6.17 17 (9-25)		18.33 ± 6.36 21.5 (7-25)		0.381
Phonemic fluency	17.75 ± 6.00 18 (7-27)		17.41 ± 6.81 17 (7-28)		0.737
Clock drawing test	8.64 ± 3.77 9 (3-16)		8.33 ± 3.89 9 (6-20)		0.119
Stroop word reading time (s)	44.40 ± 12.33 40 (32-60)		46.59 ± 16.73 46 (32-82)		<0.000*
Stroop color naming time (s)	43.25 ± 12.80 37 (25-70)		47.44 ± 16.32 48 (34-74)		0.019

inflammation, and oxidative stress, mechanisms also implicated in neurodegenerative disorders. While an increased prevalence of dementia has been reported in COPD, data regarding mild cognitive impairment (MCI) and Alzheimer's disease (AD) remain limited.

Methods: This cross-sectional case-control study included 147 participants: 86 patients with COPD and 61 age- and sex-matched controls. Cognitive assessment comprised the Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), semantic and phonemic fluency tests, clock drawing test, and Stroop test. Clinical characteristics, pulmonary function parameters, oxygen saturation (SpO₂), inflammatory markers, and COPD severity indices were recorded. Group comparisons, correlation analyses, and multivariable path analysis were performed.

Results: The prevalence of MCI was higher in patients with COPD compared to controls (30.2% vs. 18.0%), whereas AD prevalence was similar between groups (Figure 1). MMSE and MoCA did not differ significantly. COPD patients demonstrated significantly prolonged Stroop word reading times, indicating impaired executive function and attention (Table-1). Lower SpO₂ levels and higher dyspnea severity were significantly associated with worse cognitive performance. In multivariable path analysis adjusting for age, sex, smoking status, body mass index, and C-reactive protein, COPD presence remained independently associated with prolonged Stroop word reading time ($\beta=0.22$, $p=0.004$), explaining 14.7% of the variance.

Conclusion: COPD is associated with selective cognitive impairment, predominantly affecting executive functions and attention, independent of systemic inflammation. Hypoxemia and dyspnea may play an important role in this association. Patients with COPD may benefit from cognitive screening targeting executive functions.

Disclosure: Nothing to disclose.

Background and aims: Alzheimer's disease can begin as mild cognitive impairment (MCI), a prodromal stage that requires sensitive detection. Traditional screening may show insufficient sensitivity and access to neuropsychological testing is limited. As speech problems could precede other clinical symptoms, this study reviews digital/automated speech-based methods and evaluates their diagnostic performance.

Methods: A systematic search was conducted in PubMed, EMBASE, WoS, SCOPUS, IEEE-Xplore, ACM Digital Library, and Cochrane Central in studies published between 2015 and 2025. Observational studies involving adults with MCI and cognitively unimpaired (CU) controls using automated speech biomarkers and reporting diagnostic accuracy were included. A diagnostic accuracy meta-analysis was performed using hierarchical models to pool sensitivity, specificity, and summary ROC estimates. Quality and risk of bias were assessed using QUADAS-2.

Results: After duplicate removal, 3,735 records were screened, of which 34 studies met the predefined inclusion criteria; 68% were observational, cross-sectional studies. Overall, 9,957 participants were analyzed, including 1,852 individuals with MCI and 3,375 CU control. Most of the studies employed automated analysis (67.7%) with supervised machine learning and hybrid acoustic-linguistic biomarkers (41.2%). Diagnostic accuracy demonstrated considerable heterogeneity, with pooled analysis

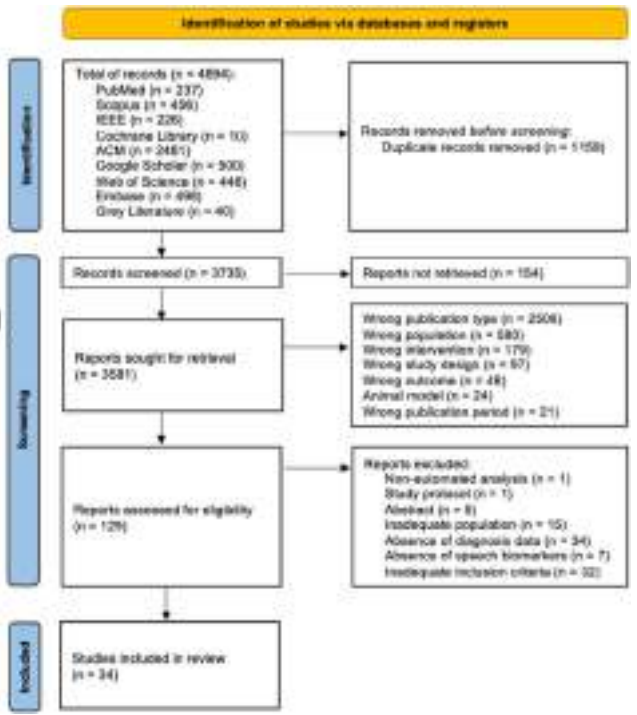


FIGURE 1 PRISMA 2020 flow diagram.

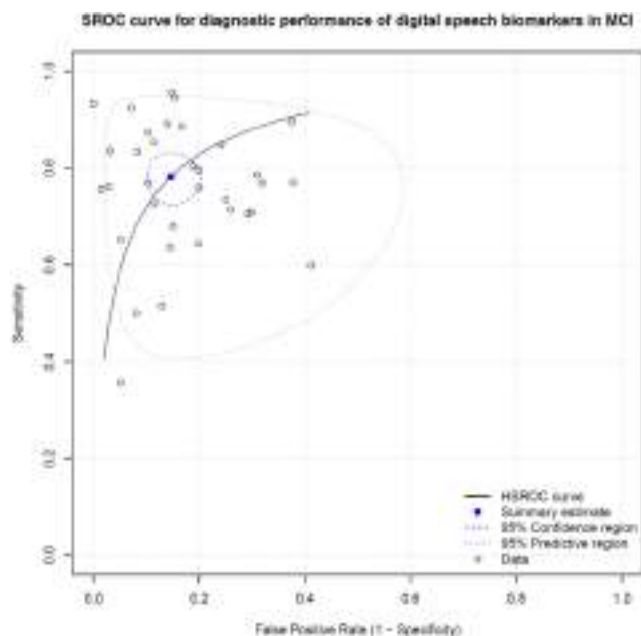


FIGURE 2 SROC curve for diagnostic performance of digital speech biomarkers in MCI. The positive likelihood ratio was 5.34 (95% CI 4.06–7.02) and the negative likelihood ratio was 0.26 (95% CI 0.21–0.31).

sensitivity and specificity of 0.78 (95% CI=0.74–0.82) and 0.85 (95% CI=0.81–0.89), respectively. There was moderate between-study heterogeneity for sensitivity ($\sigma^2=0.23$) and substantial heterogeneity for specificity ($\sigma^2=1.13$).

Conclusion: Automated speech-based biomarkers show promising diagnostic performance for MCI, supporting potential integration into dementia screening pathways.

Disclosure: Nothing to disclose.

EPO-0559 | Cognitive, neuropsychiatric and electrophysiological changes in post COVID-19 patients - A 6 months trajectory

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Background and aims: Post-Coronavirus disease 2019 (COVID-19) condition is frequently associated with persistent cognitive decline, neuropsychiatric symptoms, and fatigue. This study aimed to evaluate the trajectory of cognitive function, neuropsychiatric manifestations, and electrophysiological changes in post-COVID-19 patients over a 6-month follow-up period.

Methods: This prospective, hospital-based observational study included patients with a confirmed history of COVID-19 infection at least 6 months prior to enrolment. Cognitive function, anxiety, depression, and fatigue were assessed using standardized clinical scales, and P300 event-related potentials were recorded at baseline and after 6 months.

Results: Among 102 patients, cognitive impairment was observed in 38.2% at baseline, predominantly mild in severity. At

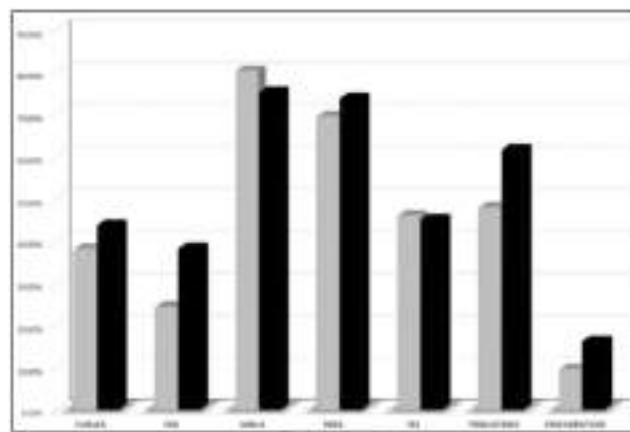


FIGURE 1: Bar graph illustrating the proportion of patients with abnormal scores on neuropsychiatric scales at baseline and at 6-month follow-up. Anxiety, depression, fatigue and P300 abnormalities remained highly prevalent with an observed increase over.

TABLE 1 Comparison of neuropsychiatric and electrophysiological parameters at baseline and 6-month follow-up.

Test	Timeline 1 n=102 Median (Q1-Q3)	Timeline 2 n=73 Median (Q1-Q3)	P value
II-MoCA	26 (24–27)	26 (24–28)	0.088
FAB	16 (15–18)	16 (14–17)	0.043*
HAM-A	17 (9–25.25)	16 (7.5–25)	0.305
HDRS	13 (6–20)	16 (7–22.5)	<0.001*
FSS	31 (14.75–50)	32 (15.5–51)	<0.001*
P300 LATENCY (ms)	343 (322–361)	354 (340.5–393.5)	0.005*
P300 AMPLITUDE (μV)	12.1 (9.27–16)	8.8 (7.25–13.15)	<0.001*

* Statistically significant ($p < 0.05$)

Abbreviations:

II-MoCA, Hindi version of Montreal Cognitive Assessment; FAB, Frontal Assessment Battery; HAM-A, Hamilton Anxiety Rating Scale; HDRS, Hamilton Depression Rating Scale; FSS, Fatigue Severity Scale; P300, event-related potential.

6-month follow-up, cognitive dysfunction was present in 43.8% of patients. Anxiety (80.4%), depression (69.6%), and fatigue (46.1%) were highly prevalent **Figure 1** Electrophysiologically, prolonged P300 latency was noted in 48% of patients at baseline, increasing to 61.6% at follow-up (Table 1). Patients with baseline cognitive impairment demonstrated significantly worse frontal lobe function, anxiety, depression, and fatigue scores compared to cognitively preserved individuals (p 0.0001, 0.002, 0.003 and 0.043 respectively) (Table 2). Notably, P300 abnormalities were also observed in patients without clinically evident cognitive decline.

Conclusion: Persistent and progressive cognitive, neuropsychiatric, and electrophysiological abnormalities were observed in post-COVID-19 patients over a 6-month period. Cognitive dysfunction, depression, and fatigue may worsen over time, even long after acute infection. P300 abnormalities may precede clinically apparent cognitive decline, highlighting their potential

TABLE 2 Baseline comparison of neuropsychiatric and electrophysiological parameters between cognitively preserved and cognitively impaired groups.

Parameter	Cognitively preserved		Cognitively impaired	P value†
	n = 63	n = 39		
	Median (Q1–Q3)	Median (Q1–Q3)		
FAB	17 (16–18)	15 (12–16)		<0.001*
HAM-A	13 (7–23)	22 (13–29)		0.002*
HDRS	11 (5–17)	17 (10–25)		0.003*
FSS	39 (13–48)	34 (22–52)		0.043*
P300 latency (ms)	344 (330–361)	344 (325–371)		0.579
P300 amplitude (µV)	11.4 (9.1–17.1)	12.7 (9.9–14.7)		0.087

* Statistically significant ($p < 0.05$)

† Mann–Whitney U test

Abbreviations:
H-MoCA, Hindi version of Montreal Cognitive Assessment; FAB, Frontal Assessment Battery; HAM-A, Hamilton Anxiety Rating Scale; HDRS, Hamilton Depression Rating Scale; FSS, Fatigue Severity Scale; P300, event-related potential.

role as an objective marker for subclinical cognitive dysfunction in post-COVID-19 condition.

Disclosure: Nothing to disclose.

EPO-0560 | Multidimensional fatigue relates to disability severity and personal well-being in multiple sclerosis

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Background and aims: Fatigue is one of the most common symptoms of multiple sclerosis (MS), negatively affecting daily functioning and well-being. Understanding the relationships between different fatigue domains, disability status, and personal well-being is necessary for practical interventions. The aim of this study was to examine the relationships between multidimensional fatigue, disability status, and personal well-being in people with MS.

Methods: A total of 141 MS subjects participated in the study, with an average age of 47 years, of whom 104 (74%) were female. The median EDSS was 3, and the average disease duration was 9 years. All subjects completed the Modified Fatigue Impact Scale (MFIS) and the Personal Wellbeing Index – Adult (PWI-A), while an expert neurologist completed the Expanded Disability Status Scale (EDSS).

Results: Pearson correlation analyses showed moderate to strong positive associations between all fatigue domains (physical, cognitive, psychosocial, and total) and disability ($r=0.43$ – $.56$, $p<0.001$), and strong negative associations with personal well-being ($r=-.71$ to $-.74$, $p<0.001$).

Conclusion: Higher multidimensional fatigue is strongly associated with higher disability status and lower personal well-being.
Disclosure: This study was conducted as part of the Croatian Science Foundation project [HRZZ-IP-2022-10-6203] at the Department of Neurology, University Hospital Split, and the Department of Neuroscience, Laboratory for Human and Experimental Neurophysiology (LAHEN), University of Split School of Medicine.

Epilepsy 4

EPO-0561 | IP3R2 deletion alters glutamate dynamics and increases mortality in a mouse model of temporal lobe epilepsy

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Background and aims: Inositol 1,4,5-trisphosphate receptor 2 (IP3R2)-mediated Ca²⁺ signaling has been implicated in epilepsy pathogenesis, potentially through Ca²⁺-dependent astrocytic release of glutamate.

Methods: In this study, we examined the contribution of IP3R2 to activity-evoked astrocytic Ca²⁺ signals and extracellular glutamate dynamics during epileptogenesis in a mouse model of temporal lobe epilepsy with hippocampal sclerosis. To do this, we used adeno-associated viral vectors to genetically express fluorescent reporters for intracellular Ca²⁺ and extracellular glutamate in hippocampal slices from mice

Results: We found that IP3R2 knockout mice showed markedly increased mortality in the days following kainate-induced status epilepticus. Among survivors, activity-evoked glutamate responses were reduced 10 days after status epilepticus, which may reflect enhanced neuronal loss.

Conclusion: Overall, our findings indicate that IP3R2-mediated Ca²⁺ signaling, despite its proposed proconvulsant role in acute seizures, may be neuroprotective during epileptogenesis by supporting survival and preserving activity-evoked glutamate signaling.

Disclosure: Nothing to disclose

EPO-0562 | Continuous versus routine EEG impact on the management of critically ill adults: Analysis of the CERTA study

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Background and aims: Continuous electroencephalogram (cEEG) is increasingly utilized in the intensive care unit for consciousness-impaired patients with various conditions. It is however costly in terms of material and human resources, and its impact on prognosis compared with routine EEG (rEEG) remains debated. We aimed to characterize the effect of cEEG vs rEEG on clinical management modifications, and to determine if these modifications were linked to a better outcome.

Methods: This is an ancillary analysis using data of the patients enrolled in the CERTA trial (NCT03129438), a randomized control trial comparing cEEG versus repeated rEEG in critically ill adults with altered consciousness. Clinical management modifications due to the EEG intervention, defined as performed within 60 hours of EEG start, were prospectively recorded (anti-seizure medication -ASM, sedation, new radiological examination, surgical intervention, intensive care withdrawal). Correlation with the EEG type were analyzed by 2x2 tables; Mantel-Haenszel tests explored the impact on mortality and functional outcome at 6 months.

Results: We included 182 patients each in the cEEG and rEEG groups. ASM were modified in 22.0% (cEEG) and 12.1% (rEEG) ($p=0.012$), while 58.2% of cEEG and 48.4% of rEEG patients had any change of management ($p=0.018$) (ASM, sedation, new radiological examination, new surgical intervention and care withdrawal). Adjusting for the impact of ASM modifications, mortality ($p=0.845$) and functional outcome ($p=0.729$) remained similar across EEG intervention groups.

Conclusion: In critically ill adults, cEEG is statistically associated with modifications of clinical management, particularly regarding ASM, compared to repeated rEEG, but does not translate into improved patients' outcomes.

Disclosure: Nothing to disclose.

EPO-0563 | The many faces of Lafora progressive myoclonus epilepsy – the Swiss cohort

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Background and aims: Lafora progressive myoclonus epilepsy (PME) is an ultra-rare autosomal-recessive neurodegenerative disorder with onset in adolescence. The prevalence is estimated around four cases per million individuals with higher numbers in Mediterranean countries. It is caused by loss-of-function mutations in EPM2A or EPM2B/NHLRC1 genes. With currently no directed therapy available and increasing pharmacoresistance in the disease course, most patients die within a decade after symptom onset. Comprehensive data on the electroclinical disease course are rare due to scarcity of the disease, delayed diagnosis, and rapid disease progression.

Methods: We retrospectively analysed clinical and electroencephalographic data from the medical records of three patients with a genetic diagnosis of Lafora PME at the Swiss epilepsy center between 2019 and 2025. To our knowledge, these are currently the only Lafora patients in Switzerland.

Results: Patient 1 presented with discrete bilateral myoclonia, followed by focal visual seizures, clustering of atypical absences, and progressing to super-refractory non-convulsive

status epilepticus within 8 months, being rescued only by the implantation of deep brain stimulation into the anterior nucleus of the thalamus (ANT-DBS). Patient 2 was diagnosed after an initial bilateral tonic-clonic seizure and later developed prominent myoclonic-tonic-clonic seizures. Patient 3 showed frequent eyelid myoclonia and eye closure sensitivity on EEG, leading to the initial diagnosis of epilepsy with eyelid myoclonia (formerly Jeavons syndrome). All patients showed progressive drug-resistant myoclonus, progressive cognitive deterioration and electrographic status epilepticus with background slowing over time.

Conclusion: Lafora disease can present with many different initial symptoms, clinically prominent features, and dynamics of disease course.

Disclosure: Nothing to disclose.

EPO-0564 | Duration matters: Prognostic cut-offs of status epilepticus beyond established predictors

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Background and aims: Status epilepticus (SE) is a time-dependent neurological emergency. While baseline clinical predictors are well established, the prognostic role of SE duration remains incompletely defined. We aimed to identify clinically relevant duration cut-offs for major outcomes and to assess the impact of diagnostic delay on SE duration in nonconvulsive SE (NCSE).

Methods: We retrospectively analyzed 73 consecutive adult patients admitted for SE between January 2024 and August 2025. Outcomes were SE resolution, 30-day mortality, and functional worsening at discharge. Multivariable models included established prognostic predictors and SE duration. Receiver operating characteristic analysis identified prognostic duration cut-offs. In NCSE, the association between onset-to-electroencephalography time and total SE duration was evaluated.

Results: SE resolved in 82.2% of patients; 30-day mortality was 39.7%, and functional worsening occurred in 57.5%. When compared with established predictors, SE duration showed an independent prognostic value for non-resolution and functional worsening. A duration ≥ 108 hours was associated with an approximately eleven-fold increased risk of non-resolution, while a duration ≥ 52 hours increased the risk of functional worsening nearly twelve-fold. SE duration was not independently associated with 30-day mortality. In NCSE, longer onset-to-electroencephalography time was significantly associated with longer SE duration.

Conclusion: SE duration provides prognostic information beyond established predictors, particularly for refractoriness and functional outcome. In NCSE, diagnostic delay contributes to prolonged SE, highlighting electroencephalography timing as a modifiable determinant of outcome.

Disclosure: Nothing to disclose.

EPO-0565 | Real-world effectiveness and safety of fenfluramine in adults with Dravet syndrome and Lennox–Gastaut syndrome: A retrospective single-centre study

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Republic

Background and aims: Evidence for fenfluramine in Dravet syndrome (DS) and Lennox–Gastaut syndrome (LGS) is largely derived from paediatric cohorts, while real-world data in adults are scarce. We evaluated the effectiveness and safety of fenfluramine in adults with long-standing, drug-resistant epilepsy.

Methods: We retrospectively identified adults with DS or LGS who initiated fenfluramine between January 2024 and August 2025. Fifteen patients were included (DS $n=9$; LGS $n=6$). All DS cases had confirmed SCN1A variants; LGS etiologies were structural ($n=2$), genetic ($n=1$), or unknown ($n=3$). Patients were highly pharmacoresistant (9–17 lifetime antiseizure medications; 2–5 at initiation). Median disease duration was 31 years. Effectiveness was assessed as percentage seizure reduction at 6 months ($n=15$) and 12 months ($n=9$). Safety and discontinuations were recorded retrospectively.

Results: At 6 months, 86.7% achieved a clinical response and 13.3% were non-responders, most commonly with $\geq 50\%$ seizure reduction. At 12 months, all evaluable patients (100%) showed sustained benefit. Treatment was discontinued in 13.3% due to inefficacy with or without adverse events. Adverse events occurred in 53.3%, mainly gastrointestinal symptoms, fatigue/somnolence, reduced appetite with weight loss, and behavioural changes; 20% reported improved attention.

Conclusion: In this real-world adult cohort with long-standing, highly refractory DS and LGS, fenfluramine was associated with substantial and sustained seizure reduction and an acceptable safety profile. These findings support the use of fenfluramine as an effective add-on therapy beyond childhood and highlight its potential benefit even in chronically treated adult patients.

Disclosure: Nothing to disclose.

EPO-0566 | Fast-ripples are an emergent property of neural networks

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Background and aims: Fast-ripples have been proposed as a promising biomarker in epilepsy, but their specificity remains unclear. In particular, it is unclear whether they reflect chance coincident neural activity or distinctly generated pathological entities.

Methods: Here, we used a multi-modal approach combining in silico simulations, neuronal cultures, a rodent model of temporal lobe epilepsy and microwire recordings in humans with epilepsy to test, using a within-subjects design, whether fast-ripples occur more frequently than predicted by the random firing of neurons. For chance level, we used signal shuffling to disrupt any distinct pathological entities and to generate only fast-ripples arising from stochastic aggregation of action potentials. Furthermore, since evidence suggests that fast-ripples incidence varies across the sleep–wake cycle, we assessed whether the relative proportion of “chance” fast-ripples is modulated across vigilance states.

Results: We found that, both in vitro and in sleeping rodents, the incidence of fast-ripples did not exceed chance levels. Our simulations predicted an increased rate of fast-ripples during wakefulness, when firing rates are higher and less synchronized across neurons. This was confirmed in a rodent model of epilepsy, where fast-ripples were also more frequent than expected by chance but still represented a minority of all detected events. This was also the case in awake human recordings.

Conclusion: Our findings suggest that most fast-ripples reflect general network activity rather than distinctly generated pathological entities, challenging prevailing assumptions about their pathogenesis and biomarker specificity.

Disclosure: M.W. has acted as a consultant for Seer and EpilepsyGtx. He is a founder shareholder in EpilepsyGtx. He has received honoraria from Eisai, Angelini, and UCB pharma.

EPO-0567 | Can statins influence the risk of remote seizure after a first new onset status epilepticus? Data from the adult population of Modena, Northern Italy

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Background and aims: Preclinical evidence supports the role of statins as antiepileptogenic agents. In this study, we investigated the risk of remote unprovoked seizures (RS) according to the use of statin therapy in a cohort of first ever status epilepticus (SE) survivors.

Methods: Observational, single-center, retrospective study in adult patients with a first SE who were consecutively admitted to Modena Academic Hospital, Italy, from September 2015 to December 2023. Kaplan–Meier survival analyses were used to calculate the probability of RS following the index event, while Cox proportional hazard regression models were used to assess predictors of RS occurrence. Competing-risk regression model was used as sensitivity analysis to assess the impact of mortality as a competing event with the occurrence of RS.

Results: 314 patients were included (mean age 70 y/o; 63% females), 86 (27.4%) of whom were taking statins. Overall, 67

patients (21.3%) developed RS (mean follow-up: 31.5 months). Cumulative probability of RS occurrence was 16% and 32% at 12 months and 5 years after SE, respectively. Cumulative probability of RS was significantly lower in statin users compared to non-users (log rank $p=0.038$). After adjusting for potential confounders, the risk of RS was lower in patients who assumed statins after the index event (HR 0.48 95% CI 0.26 – 0.90; $p=0.023$).

Conclusion: In our cohort, statin exposure was associated to a lower incidence of RS. Our findings expand the knowledge regarding a possible neuroprotective and antiepileptogenic role of statins after SE and support further studies on this field.

Disclosure: Nothing to disclose

EPO-0568 | Real-world evidence on the use of Perampanel in the management of brain tumor-related epilepsy

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Background and aims: Seizures are a frequent complication in patients with brain tumors, with more than 50% developing epilepsy that is often pharmacoresistant [1]. This study aimed to

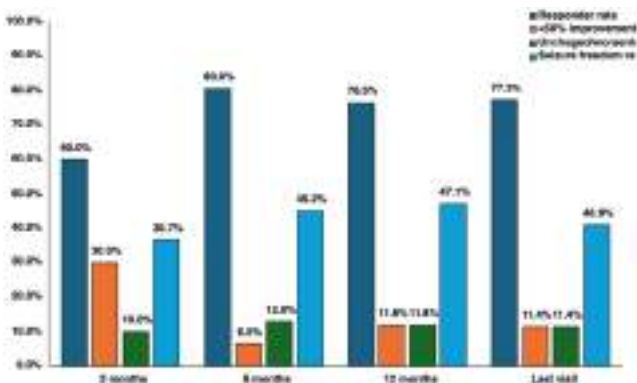


FIGURE 1 Clinical response to add-on Perampanel treatment in the overall population. The figure shows the proportion of responder patients, patients achieving seizure freedom, patients 3with

Table 1. Adverse events in early and late add-on patient

	Early add-on	Late add-on
Tolerability population	21	25
Subjects with any adverse events, n. (%)	-	7 (15.2)
Withdrawal due to adverse events, n. (%)	-	3 (6.5)
Type of adverse event, n. (%)		
Behavioral disorders	-	4 (8.7)
Irritability/Nervousness	-	1 (2.2)
Dizziness/Vertigo	-	1 (2.2)
Other	-	2 (4.3)

Adverse events in early and late add-on patient.

assess the efficacy and tolerability of perampanel (PER) as the sole additional antiseizure medication (ASM) in brain tumor-related epilepsy (BRTE).

Methods: we performed a subgroup analysis of patients with BRTE included in the PEROC study cohort [2], a retrospective, longitudinal, multicentre observational study enrolling patients with focal or generalized epilepsy receiving PER added to a single concomitant ASM. Retention, responder rate, seizure freedom and adverse event (AE) frequency at 3, 6 and 12 months after PER introduction were evaluated, also comparing early (≤ 1 prior ASM) vs late PER add-on.

Results: forty-six patients were included (23 female, median age: 51 years). The median PER dose at 12 months was 4 mg. Retention rates were 90%, 82.9%, and 70.8% at 3, 6, and 12 months. Responder rates were 60% at 3, 80.6% at 6, and 76.5% at the same time points. Seizure freedom was achieved in 36.7%, 45.2% and 47.1% of patients after 3, 6 and 12 months. AEs were reported in 15.2% of patients, leading to treatment discontinuation in 6.5%. No significant differences were observed between early and late add-on groups for any outcome.

Conclusion: PER proved to be an effective and well-tolerated treatment option for BTRE, providing satisfactory seizure control and good treatment retention. Although confirmation from larger prospective studies is needed, PER may represent a valuable strategy to improve seizure management and quality of life in this population

Disclosure: Nothing to disclose.

EPO-0569 | Clinical characteristics, comorbidities, and healthcare utilization in patients with psychogenic nonepileptic seizures: A retrospective study

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Background and aims: Psychogenic nonepileptic seizures (PNES) are paroxysmal events that resemble epileptic seizures but occur without ictal epileptiform activity. Diagnosis is frequently delayed, and many patients are initially misdiagnosed with epilepsy, leading to unnecessary anti-seizure medication (ASM) use and increased healthcare utilization. Regional data describing the clinical characteristics and burden of PNES remain limited.

Methods: We retrospectively reviewed patients evaluated for suspected psychogenic nonepileptic seizures (PNES) using outpatient video-EEG (1–6 hours) at King Abdullah University Hospital between 2018 and 2022. PNES was defined as a typical clinical event captured without ictal EEG changes. Patients with exclusively subjective symptoms were excluded. Demographic, clinical, diagnostic, treatment, and healthcare utilization data were analyzed descriptively.

Results: Among 59 patients with PNES, 57.6% were female and psychiatric comorbidities were common. Events were captured on video-EEG in 83.1%, with 74.6% being typical habitual events, and epileptiform discharges were uncommon (13.6%). Semiology was heterogeneous using Pooya and Wadwekar classifications. MRI was mostly normal or non-specific, ≈68% had received ASMs, often polytherapy, and healthcare utilization prior to diagnosis was high.

Conclusion: Psychogenic nonepileptic seizures are associated with heterogeneous clinical presentations, high psychiatric comorbidity, substantial healthcare utilization, and frequent exposure to anti-seizure medications prior to diagnosis. This study adds regional, real-world data on the clinical characteristics, diagnostic evaluation, and healthcare utilization associated with PNES, reinforcing existing evidence while highlighting the burden of delayed diagnosis in routine practice.

Disclosure: Nothing to disclose

EPO-0570 | An optimized clinical algorithm for selecting DEE-SWAS patients for whole exome sequencing

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Background and aims: Developmental and epileptic encephalopathy with spike-wave activation in sleep (DEE-SWAS) is a clinically heterogeneous disorder with a substantial genetic contribution. Whole exome sequencing (WES) is not routinely accessible for all patients, highlighting the need for rational selection strategies.

Methods: A retrospective cohort of 125 pediatric patients with DEE-SWAS was analyzed. Clinical characteristics, cognitive outcomes, treatment response, family history, and brain magnetic resonance imaging (MRI) findings were collected.

Electroencephalography (EEG) recordings during non-rapid eye movement sleep were reviewed, and spike-wave index (SWI) was calculated. A scoring algorithm was developed assigning one point to each of seven criteria: SWI >85% or pronounced SWAS pattern, full-scale intelligence quotient (FSIQ) <70, pharmacoresistant epilepsy or need for intensified therapy, absence of a convincing structural MRI cause, epilepsy onset ≤4 years, positive family history of epilepsy or febrile seizures, and parental consanguinity. Patients were stratified into low (0–2), moderate (3–4), and high (≥5) genetic risk groups. Multivariable logistic regression was performed to identify independent predictors of monogenic etiology.

Results: High SWI, severe cognitive impairment, pharmacoresistance, absence of structural MRI abnormalities, positive family history, and parental consanguinity were independently associated with monogenic disease (odds ratios 1.8–4.9; all $p < 0.05$).

Conclusion: A weighted clinical, EEG, and MRI-based algorithm effectively stratifies DEE-SWAS patients according to genetic risk and supports targeted referral for WES. This approach improves diagnostic efficiency and may facilitate earlier etiological clarification in routine clinical practice.

Disclosure: Nothing to disclose.

EPO-0571 | Vagus Nerve Stimulation in Failed Epilepsy Surgery: 36 Month Outcomes From the CORE-VNS Study

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Background and aims: The safety and effectiveness of VNS Therapy are evaluated in individuals who have (+Hx) and have not (-Hx) previously undergone brain surgical procedures for epilepsy.

Methods: The impact of +Hx on the safety profile and change in seizure frequency of severe seizure types (Focal Impaired Awareness Motor [FIA-M], Focal Impaired Awareness Non-Motor [FIA-NM], Focal-to-Bilateral Tonic-Clonic [FBTC], and Generalized Tonic-Clonic [GTC]) is evaluated.

Results: Of the 464 participants with -Hx 446 were included in the modified safety population (mSAF). Lesionectomy, callosotomy, and lobar resection were the 3 most frequently reported therapeutic brain surgeries in the 84 participants with +Hx in the mSAF. At 36 months, participants with +Hx experienced a median percentage change in frequency of FIA-M by -36.1% ($n=28$), FIA-NM by -83.3% ($n=18$), FBTC by -91.7% ($n=19$), and GTC by -84% ($n=5$). In the -Hx participants this was -86.1% ($n=131$), -100% ($n=72$), -96.7% ($n=73$), and -83.5%

($n = 78$) respectively. In those with a +Hx and -Hx, complete seizure freedom (zero seizures over the previous 3 months) was reported at the first 3 month visit in 6.9% (5/72) and 8.8% (29/329) respectively. At the 36-month visit, 8.8% (6/68) and 18% (58/323) reported complete seizure freedom, respectively. The overall incidence of adverse events (AEs) was similar between groups, 36.9% +Hx and 41.0% -Hx. Serious AEs were observed in 3.6% and 7.2% respectively.

Conclusion: Adjunctive VNS Therapy was found to have consistent safety and effectiveness in participants with +Hx and -Hx epilepsy brain surgery. In addition, SF was reported in 8.8% of participants.

Disclosure: EN, KN, GG, CG, MD, and AM are employees of LivaNova PLC (or a subsidiary) and hold stock or stock options with the company, who is the manufacturer of the VNS Therapy System. All other authors are investigators associated with the CORE-VNS Study, and in that capacity they or their institutions receive compensation from LivaNova for study-related activities. No author received direct compensation from LivaNova related to this abstract.

EPO-0572 | VNS Therapy is Associated with Reduced Disabling Seizure Burden in Children Early in Treatment (3 M) of DRE with Continued Improvement Over 36 Months

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Background and aims: The CORE-VNS represents the first large cohort of prospectively gathered pediatric data on the effectiveness of VNS Therapy for DRE.

Methods: The CORE-VNS (NCT03529045) study collected data on seizure and non-seizure outcomes following treatment with VNS Therapy. Participants 4- <18 (children) who received their first VNS Therapy were selected for this analysis. Seizure frequency reduction of defined most severe seizures and patient-described outcome measures (quality of life and quality of sleep) were collected at baseline and at 3, 6, 12, 24, and 36 months.

Results: 213 met the criteria for this analysis. Certain seizure types were more commonly described in this analysis as most disabling (focal impaired aware motor (FIA-M), focal impaired aware non-motor (FIA-NM), focal to bilateral tonic-clonic (FBTC), and generalized tonic-clonic seizures (GTC)). Seizure frequency reduction was reported in all seizure types across age groups early (3-month visit) in the study. At the 36-month follow-up, children had a median seizure reduction of 86.7% FIA-M, 100% FIA-NM, 100% FBTC, and 75.2% GTC. At 36 months, 80.3% reported their quality of life as very good/pretty good/good and bad. The most reported (~5%) adverse events were cough and dysphonia.

Conclusion: Adjunctive VNS Therapy reduced the most severe disabling seizures in children early after implantation with sustained effectiveness and improved quality of life at 36 months.

Disclosure: GG, RV, KN, FB, CG, MD, and DU are employees of LivaNova PLC (or a subsidiary) and may hold stock or stock options with the company, who is the manufacturer of the VNS Therapy System. All other authors are investigators associated with the CORE-VNS Study, and in that capacity they or their institutions receive compensation from LivaNova for study-related activities. No author received direct compensation from LivaNova.

Headache 4

EPO-0573 | Venous sinus stenting in idiopathic intracranial hypertension: A prospective interventional study of safety and efficacy

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Background and aims: Idiopathic Intracranial Hypertension (IIH) with bilateral transverse-sigmoid venous sinus stenosis is a debilitating condition with limited effective treatments. This study aimed to evaluate the safety and effectiveness of venous sinus stenting (VSS) in medically refractory IIH patients, focusing on clinical outcomes and objective physiological parameters.

Methods: A prospective, single-arm interventional study was conducted across two tertiary centers in Cairo, Egypt, over nine months. Twenty-four patients (median age 35.5 years; 95.8% female) with IIH and bilateral venous sinus stenosis confirmed by magnetic resonance venography underwent VSS using self-expanding stents across stenotic segments. Inclusion criteria included failure of medical therapy, papilledema grade $\geq$ II, and trans-stenotic pressure gradient $>8-10$ mmHg. Pre- and post-intervention assessments comprised intracranial pressure (CSF opening pressure), venous pressure gradients, optical coherence tomography, visual evoked potentials, and visual field perimetry. Follow-up was performed at 1 and 6 months.

Results: All patients demonstrated significant improvement in symptoms and objective measures. Headache severity reduced markedly ($p < 0.001$); papilledema resolved in 100% of affected



Pre venous sinus stenting pressure gradient.



Post venous sinus stenting pressure gradient.

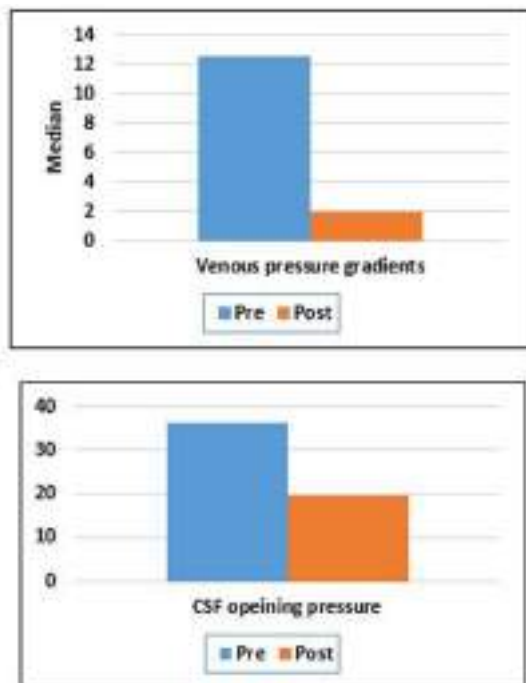


Figure (4): VPG & CSF of pre and post Intervention.

VPG & CSF of pre and post Intervention.

cases. Median venous pressure gradient decreased from 12.5 to 2 mmHg, and cerebrospinal fluid opening pressure reduced from 36.45 ± 5.83 to 19.54 ± 2.6 Cm H₂O ($p < 0.001$). Visual

evoked potential parameters improved significantly, reflecting enhanced optic nerve function. No complications, stent migration, or symptom recurrence occurred during follow-up.

Conclusion: Venous sinus stenting is a safe and effective therapeutic option for medically refractory IIH with venous sinus stenosis. It significantly lowers intracranial pressure, resolves papilledema, and improves visual function, supporting its role as a minimally invasive alternative in selected patients.

Disclosure: The authors declare no conflicts of interest or financial support related to this study.

EPO-0574 | Increased neuroimmune and neurotrophic responses to a migraine trigger after prenatal immune activation

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Background and aims: Prenatal immune activation (PIA) induced by maternal lipopolysaccharide (LPS) exposure is considered as a factor capable of altering neuroimmune reactivity in adulthood. In this study, we analyzed how prenatal LPS exposure modifies migraine responses by assessing biochemical markers in a model of migraine induced by a single nitroglycerin (NTG) injection.

Methods: This study included 16 adult male Wistar rats. Levels of CGRP, TNF-alpha, IL-1beta, IL-6 and BDNF were measured in homogenates of isolated brain regions (hippocampus, cerebral cortex and brainstem) using ELISA.

Results: At baseline, the animals exposed to PIA demonstrated significantly decreased IL-1beta levels in all examined brain regions ($p < 0.05$) and reduced BDNF levels in the cortex ($p = 0.04$) compared to the control group. Levels of TNF-alpha, CGRP, IL-6 were nearly equal or slightly decreased, indicating the absence of active neuroinflammation and the development

TABLE 1 Levels of CGRP, IL-6, IL-1beta, TNF-alpha, BDNF in hippocampus, cortex, brainstem in experimental groups (mean $\pm$ SD); Mann-Whitney U test; significant $p < 0.05$ in bold.

Marker, pg/ml	Brain region	Groups		p-value	Groups		p-value
		Control	PIA		Migraine	PIA + Migraine	
CGRP	Hippocampus	18.3 $\pm$ 0.9	21.3 $\pm$ 1.3	0.08	22.3 $\pm$ 0.5	56.2 $\pm$ 0.2	0.04
	Cortex	17.5 $\pm$ 0.7	20.9 $\pm$ 1.1	0.6	21.0 $\pm$ 0.5	16.2 $\pm$ 1.1	0.05
	Brainstem	19.0 $\pm$ 0.9	15.3 $\pm$ 1.2	0.3	14.2 $\pm$ 0.6	52.2 $\pm$ 0.8	0.04
IL-6	Hippocampus	0.51 $\pm$ 0.04	0.5 $\pm$ 0.04	1	0.4 $\pm$ 0.1	0.4 $\pm$ 0.01	0.1
	Cortex	0.4 $\pm$ 0.09	0.4 $\pm$ 0.08	0.14	1.1 $\pm$ 0.02	1.06 $\pm$ 0.01	1
	Brainstem	1.7 $\pm$ 0.2	1.2 $\pm$ 0.1	0.58	1.5 $\pm$ 0.2	0.4 $\pm$ 0.01	0.02
IL-1 β	Hippocampus	0.95 $\pm$ 0.08	0.5 $\pm$ 0.04	0.05	2.4 $\pm$ 0.3	0.3 $\pm$ 0.02	0.08
	Cortex	1.55 $\pm$ 0.7	0.74 $\pm$ 0.05	0.05	1.9 $\pm$ 0.3	0.3 $\pm$ 0.1	0.3
	Brainstem	1.85 $\pm$ 0.5	0.85 $\pm$ 0.01	0.05	2.3 $\pm$ 0.4	1.1 $\pm$ 0.2	0.1
TNF- α	Hippocampus	26.3 $\pm$ 1.2	18.8 $\pm$ 3.0	0.7	18.1 $\pm$ 0.5	31.1 $\pm$ 0.2	0.04
	Cortex	23.2 $\pm$ 1.8	22.5 $\pm$ 0.7	0.1	14.1 $\pm$ 0.9	23.1 $\pm$ 0.01	0.04
	Brainstem	38.2 $\pm$ 4.0	28.0 $\pm$ 0.5	0.08	22.5 $\pm$ 0.5	60.4 $\pm$ 0.8	0.05
BDNF	Hippocampus	3794.7 $\pm$ 533.0	1418.7 $\pm$ 148.0	0.2	2513.3 $\pm$ 510.4	2861.5 $\pm$ 55.9	0.6
	Cortex	2547.8 $\pm$ 427.8	1174.1 $\pm$ 0.5	0.04	2686.2 $\pm$ 279.9	1975.8 $\pm$ 191.7	0.3
	Brainstem	3090.8 $\pm$ 597.0	2553.4 $\pm$ 154.3	0.3	2133.9 $\pm$ 153.5	1857.9 $\pm$ 54.3	0.2

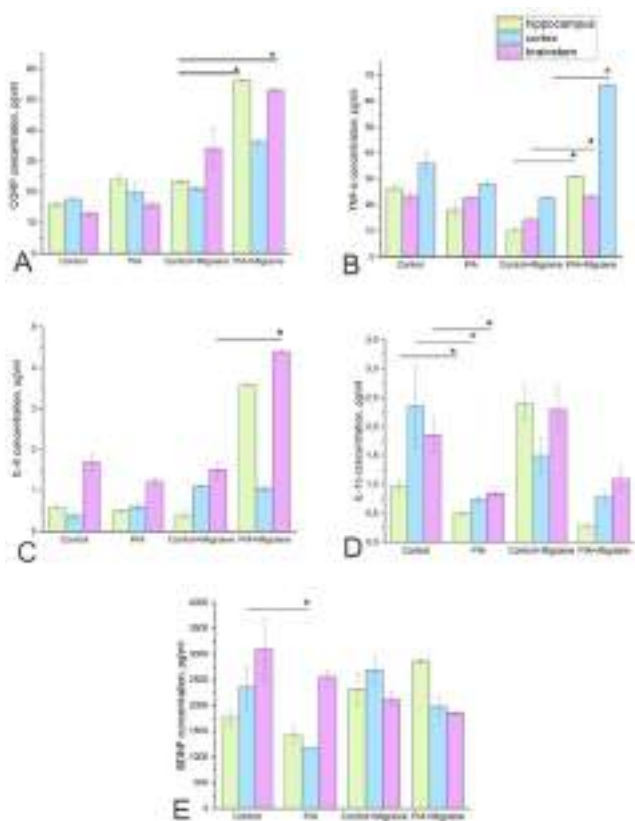


FIGURE 1 Comparison of CGRP, TNF- α , IL-6, IL-1 β , and BDNF levels in the hippocampus, cerebral cortex and brainstem between control and prenatal immune activation (PIA) groups under baseline conditions and after migraine induction. * $p < 0.05$.

of a compensated state. After administration of NTG, a region-dependent increase in the central inflammatory response was observed in the PIA group. CGRP concentrations increased significantly in the hippocampus and brainstem compared to control animals with migraine ($p < 0.05$), with a similar trend in the cortex. TNF- α levels were significantly elevated in all examined brain regions ($p < 0.05$), IL-6 level increased only in the brainstem, while in the cortex remained equal. IL-1 β levels tended to be lower in the PIA group and BDNF levels did not change significantly.

Conclusion: Thus, PIA does not lead to a sustained increase of inflammatory markers, but induces heightened sensitivity to the migraine trigger, manifested by an enhanced and heterogeneous biochemical response from central brain structures.

Disclosure: This study was supported by the Kazan Federal University Strategic Academic Leadership Program (PRIORITY-2030).

EPO-0575 | Genetic associations of TRPM8 with migraine chronification

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Background and aims: TRPM8 receptors are highly expressed in migraine pathophysiology. Genome-wide association studies have previously identified the strong association of single nucleotide polymorphism rs10166942 with migraine. However, its specific role in disease progression remains unstudied. Here we evaluated the genotype frequency distribution of rs10166942 in the TRPM8 gene in healthy individuals and patients with episodic (EM) and chronic migraine (CM) to test its influence on susceptibility to these forms of migraine.

Methods: The study included 238 participants - patients with migraine (47 EM and 42 CM) and 149 healthy controls. DNA from peripheral blood was used to test rs10166942 using allele-specific PCR.

Results: The genotype and allele frequency distributions in EM were comparable with control ($p = 0.77$ and $p = 0.7$). In the CM group distribution of genotypes differed significantly from both control and EM groups (both $p = 0.03$), (Table 1). Notably, in the CM group, in contrast to control and EM groups, the TT genotype frequency was nearly halved, while the TC and CC genotypes frequencies increased nearly twofold (Figure 1). Allele analysis confirmed this trend and showed a significant accumulation of the C allele in CM patients ($p = 0.03$).

Conclusion: These are the first indications of distinctive involvement of rs10166942 TRPM8 genotypes in EM and CM. Our data reveal a different predisposition to chronic pain in migraine and propose that this SNP play a potential role in migraine chronification.

Disclosure: This study was supported by the Kazan Federal University Strategic Academic Leadership Program (PRIORITY-2030).

TABLE 1 Genotypes and alleles distributions of rs10166942 in TRPM8 gene in the control group, episodic and chronic migraine patients.

Groups	Genotypes, n (%)			Alleles, n (%)	
	TT	TC	CC	T	C
Control (n=149)	82 (55)	57 (38)	10 (7)	221 (74)	77 (26)
Episodic migraine (n=47)	28 (60)	17 (36)	2 (4)	73 (77)	21 (23)
Chronic migraine (n=42)	13 (31)	24 (57)	5 (12)	50 (60)	34 (40)

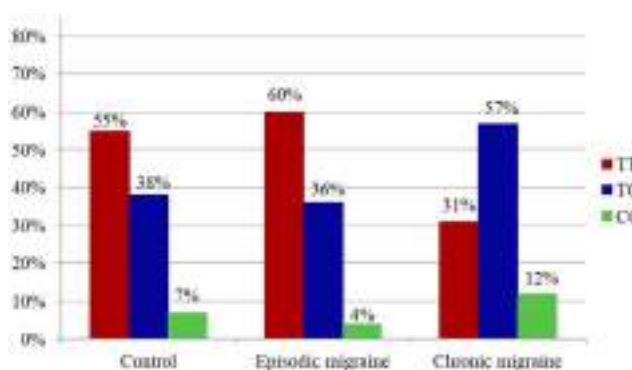


FIGURE 1 TRPM8 rs10166942 genotypes distribution in the control group, episodic and chronic migraine patients.

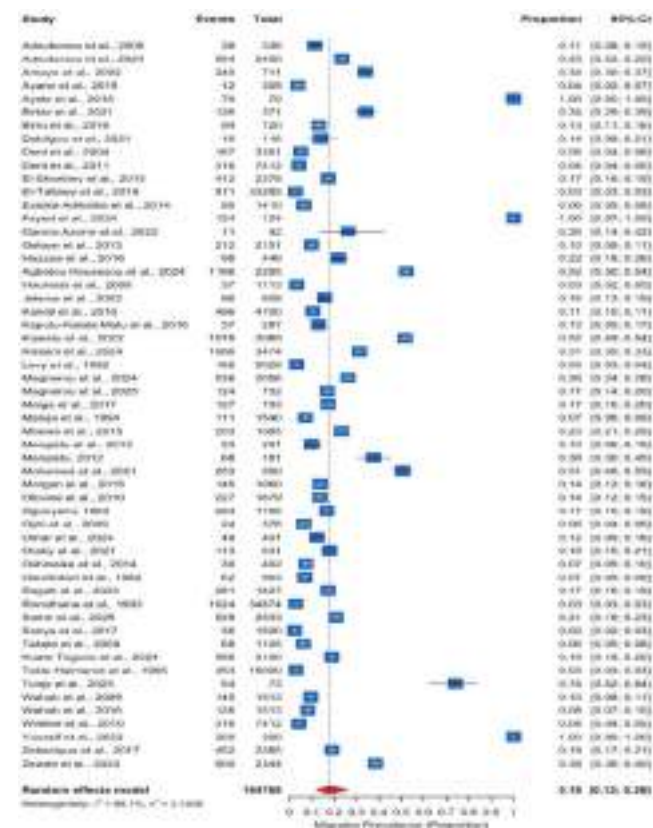
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Background and aims: Migraine is a leading cause of years lived with disability worldwide, yet data from Africa are limited. We aimed to estimate migraine prevalence, patterns, and associated burden across African populations.

Methods: We systematically searched PubMed, Scopus, Web of Science, African Journals Online, and Google Scholar (inception–August 2025) for community-based studies reporting migraine prevalence or burden. Two reviewers independently screened studies, extracted data, and assessed quality using the Joanna Briggs Institute checklist. Random-effects meta-analyses estimated pooled prevalence, with subgroup analyses by sex, migraine subtype, and setting. Heterogeneity and publication bias were assessed.

Results: Sixty-one studies ($n = 201,437$) from 18 African countries were included. Overall migraine prevalence was 18% (95% CI: 12–26%), with high heterogeneity ($I^2 = 99.7\%$). Standardized prevalence was 140 per 1,000 population (14%; 95% CI: 11–17%). Females were more affected than males (63% vs. 37%; female-to-male ratio $\approx 1.7:1$). Migraine without aura predominated, with 84% lower odds of aura. Urban–rural differences were inconsistent. Migraines accounted for 31% of all headaches. Burden



was substantial, with moderate-to-severe disability, reduced quality of life, productivity loss, and low healthcare utilization. Evidence of small-study effects and potential publication bias was detected.

Conclusion: Migraine affects a considerable proportion of Africans, disproportionately females, and are associated with significant disability. Heterogeneity across regions and populations highlights the need for improved recognition, standardized diagnostics, and better resource allocation for headache disorders in Africa.

Disclosure: Nothing to disclose.

EPO-0577 | **Temporal burden synchronization and machine learning-identified dietary nuances in migraine-anxiety comorbidity**

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Background and aims: Migraine and anxiety disorders are highly prevalent and frequently comorbid, leading to substantial functional impairment. However, the long-term temporal evolution of this comorbidity and the role of modifiable dietary factors remain poorly understood. We aimed to characterize the evolving disease burden in the USA and identify specific dietary micronutrients and whole-food patterns associated with anxiety risk in individuals with migraine.

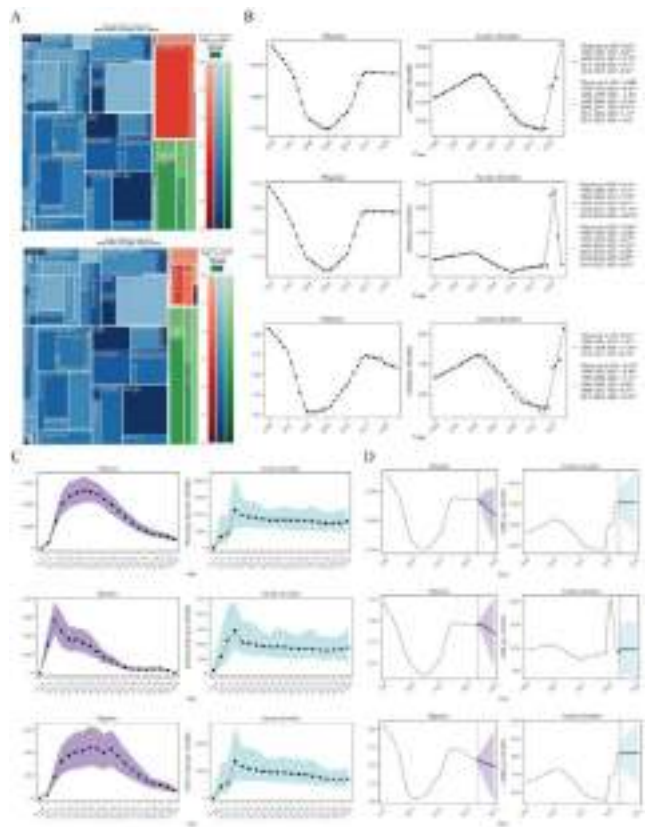
Methods: We utilized data from the Global Burden of Disease (GBD) Study 2023 to analyze US trends (1990–2023) and generate projections to 2030 using ARIMA models. We further integrated NHANES (2001–2004) data and the MyPyramid Equivalents Database to assess dietary correlates in migraineurs ($n=984$). Six machine-learning algorithms were benchmarked, with Random Forest selected for interpretation via SHAP and LIME frameworks to identify key dietary predictors.

Results: GBD analysis revealed a stable migraine burden but a sharp increase in anxiety disorders after 2018, peaking during the COVID-19 pandemic. A "burden-synchronization window" was identified between ages 20 and 49, where peak disability-adjusted life years (DALYs) for both conditions overlapped. Random Forest demonstrated superior predictive performance. SHAP analysis identified caffeine, theobromine, and protein as protective factors, while higher intakes of vitamin E, dietary fiber, and polyunsaturated fatty acids were unexpectedly associated with increased risk. Red/orange vegetables and whole grains were inversely associated with anxiety risk.

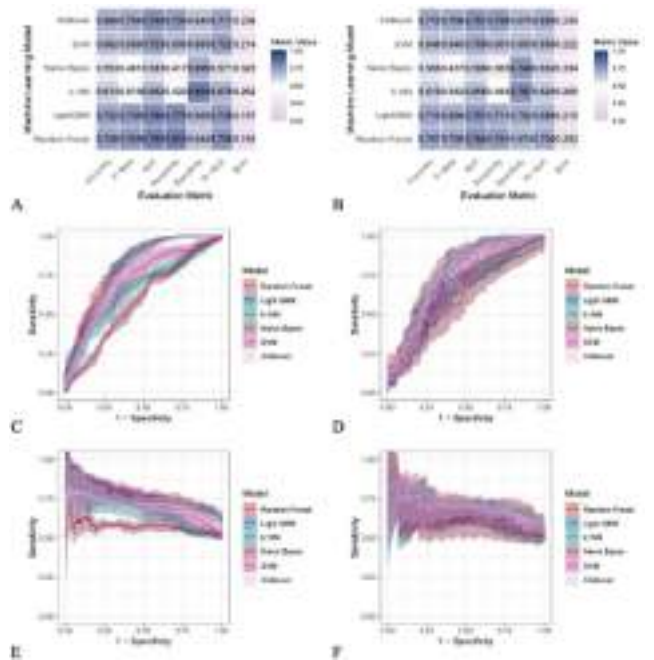
Conclusion: The rising burden of anxiety among migraineurs during mid-adulthood necessitates integrated clinical



Flowchart of participant selection.



Trends in the burden of migraine and anxiety disorders in the United States (1990–2023) and projections based on GBD 2023. (A) Heatmap comparison of age standardized DALY rates for leading diseases in the United States between GBD 2021 and GBD 20



Time-series analysis validating the machine-learning results based on the MyPyramid Equivalents Database.

management. Specific dietary interventions may modulate anxiety risk, challenging simplified antioxidant theories and emphasizing personalized nutritional strategies.

Disclosure: Nothing to disclose

EPO-0578 | Development and Assessment of a Headache Medicine Fellowship for Advanced Practice Providers (APPs)

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Background and aims: There exists a mismatch between the volumes of individuals with headache disorders and providers possessing the skills and inclination to manage those individuals effectively. To help redress this imbalance we developed and assessed a 1 year headache medicine fellowship program intended for APPs.

Methods: One of us (JR) developed a 1 year fellowship curriculum blending didactic instruction, self-learning, in-clinic observation and subsequent clinical work involving a progressive increase in independence. In the last quarter of the fellowship we conducted a prospective study to assess clinical concordance and patient satisfaction for the APP fellow vs an MD neurologist with subspecialty training in headache (JR). All patients initially were evaluated by the fellow and then subsequently by the neurologist blinded to the results of the fellow's evaluation.

Results: Two nurse practitioners (NPs:AK and KH) successfully completed the 1 year fellowship, including the last quarter's prospective study. In 98 and 96 of the 100 consecutive new headache patients evaluated by each NP, diagnoses matched those of the neurologist. There were no major differences in the management recommended by the NPs versus the neurologist. Patient satisfaction with the NPs was slightly higher than with the neurologist.

Conclusion: Assessing the clinical effectiveness of a fellowship-trained APP vs that of a subspecialist physician, we found patient satisfaction to be essentially equal and both diagnostic and management concordance to be high. Utilization of appropriately trained APPs may represent one means of coping more effectively with the existing imbalance between headache patients seeking expert care and the volume of providers available.

Disclosure: This research was funded by AbbVie, Inc and the Inova Health Care system of Northern Virginia. All 3 authors are full-time employees of Inova Health and the University of Virginia School of Medicine, and Dr. Rothrock has served as a paid consultant and speaker for AbbVie. His parent institutions have received funding for research he has conducted on behalf of AbbVie.

EPO-0579 | Early wearing-off of onabotulinumtoxin A in chronic migraine: A retrospective study

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Background and aims: OnabotulinumtoxinA is a well-established prophylactic therapy for chronic migraine, administered every 12 weeks. However, many patients report a wearing-off of efficacy prior to the end of the dosing cycle. This

Table 1. Patients in Onabotulinumtoxin A toxic treatment for migraine (total 89 patients 100%)

	Gender female	Wearing off after 12 weeks	Baseline 10 days prior	Injection 10 weeks	Dose 195 U	GON block performed	Rimegepant	Ubrogepant	Atogepant	MMB
Mean	11	11	0	11	21	11	0	11	4	10
%	12%	12%	0%	12%	24%	12%	0%	12%	4%	11%

Table 2. Patients in Onabotulinumtoxin A toxic treatment for migraine presenting wearing-off (24) or not (65)

	Gender female	Wearing off after 12 weeks	Baseline 10 days prior	Injection 10 weeks	Dose 195 U	GON block performed	Rimegepant	Ubrogepant	Atogepant	MMB
Wearing off < 12	11	11	0	11	0	0	0	0	0	0
%	100%	100%	0%	100%	0%	0%	0%	0%	0%	0%
Not wearing off > 12	11	11	1	11	0	0	0	0	0	0
%	11%	11%	100%	100%	0%	0%	0%	0%	0%	0%
p value	-	0.007	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000

Patients using Onabotulinumtoxin A.

retrospective review investigates the frequency of early wearing-off in patients receiving onabotulinumtoxin A and explores associated clinical factors.

Methods: Medical records of chronic migraine patients treated with onabotulinumtoxinA were reviewed. Patient demographics, headache frequency, use of rescue therapies, dosing regimens, and adjunctive procedures were recorded. Patients were divided into two groups based on whether they experienced wearing-off before 12 weeks.

Results: Among the 89 patients (91% female), 24 (26.9%) reported wearing-off prior to 12 weeks. It was significantly associated with higher baseline headache frequency (79% vs. 47.7%, $p=0.007$), earlier injection requirements (87.5% vs. 1.5%, $p=0.000$), and Greater Occipital Nerve blocks (GON blocks) for recurrent pain (41.6% vs. 9.2%, $p=0.000$). Patients with wearing-off prior to 12 weeks received the full 195 U dose of onabotulinumtoxin A more frequently (58.3% vs. 20%, $p=0.000$). Ubrogepant was also more commonly used in the wearing-off group (33.4% vs. 6.2%, $p=0.000$). No significant differences were noted for Rimegepant (16.7% vs 6.2%, $p=0.123$), Atogepant (8.3% vs 3.0%, $p=0.288$) or Monoclonal antibodies against CGRP (12.5% vs 10.8%, $p=0.818$) use.

Conclusion: Wearing-off was associated with higher baseline migraine frequency, earlier reinjection intervals, and increased use of acute therapies, suggesting that this subgroup may require tailored management strategies, including reassessing injection technique, dose optimization, shorter injection intervals (10 weeks), GON blocks inter procedures, optimizing other preventive treatment or using Gepants to improve outcomes.

Disclosure: Nothing to disclose.

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Background and aims: Atogepant is a novel migraine preventive drug acting as calcitonin gene-related peptide (CGRP) receptor antagonist. We conducted a prospective, open label study aimed to evaluate whether clinical response to atogepant is associated with biomolecular changes in subjects with high-frequency episodic migraine (HFEM - at least 8 migraine days/month - MMDs). As a secondary aim, we assessed differences between Responders, those achieving at least a 50% reduction in MMDs after atogepant treatment, and Non-Responders.

Methods: We enrolled 40 subjects (75% women, age 35.5 ± 21.9 , MMDs 9.9 ± 2.3). All participants underwent a biomolecular profiling at baseline (T0) and after a 3-month daily atogepant 60 mg treatment (T1). We analyzed the expression of endocannabinoid enzymes, monoacylglycerol lipase (MAGL) and fatty-acid amide hydrolase (FAAH), and microRNAs (miR-155, miR-34a, miR-382-5p) expression in peripheral blood mononuclear cells, and neuropeptides (CGRP, PACAP) plasma levels.

Results: At T1 70% of participants classified as Responders (28/40). At T1, MAGL gene expression decreased in Responders (T0: 9.8 ± 5.4 relative quantification - RQ vs. T1: 13.2 ± 7.9 RQ, $p=0.021$), without changes in Non-Responders (T0: 7.4 ± 4.3 RQ vs. T1: 8.3 ± 4.4 RQ, $p=0.552$). At T0, MAGL was higher in future Responders compared to Non-Responders ($p=0.022$). No baseline differences or after-treatment modifications were found for FAAH expression, microRNAs expression, or neuropeptides levels.

Conclusion: We confirmed atogepant effectiveness in HFEM. We found a modulation of MAGL expression in Responders participants. Our data suggests an indirect modulation of the endocannabinoid system homeostasis during atogepant treatment.

Disclosure: Nothing to disclose

EPO-0581 | Migraine-free days following acute rimegepant treatment in the real-world: Findings from CONFIDENCE

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¹King Abdullah University of Science and Technology, Thuwal, Saudi Arabia and King's College London, London, UK; ²Montefiore Medical Center and Albert Einstein College of Medicine, Bronx, USA; ³Pfizer R&D UK Ltd, Tadworth, Surrey, UK; ⁴Aptar Digital Health, Paris, France; ⁵The Headache and Facial Pain Service, Guy's and St. Thomas' NHS Foundation Trust, London, UK;

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⁷Department of Neurology, Mayo Clinic, Scottsdale, USA; ⁸MIST Research, Wilmington, USA; ⁹Pfizer AB, Stockholm, Sweden

Background and aims: CONFIDENCE (NCT06467370) was a prospective observational study evaluating the effectiveness of acute rimegepant treatment over multiple migraine attacks. As rimegepant is also a preventive migraine treatment, this analysis evaluated duration of migraine freedom after acute use.

Methods: Participants with 3-14 headache days in the past 30 completed a 28-day migraine diary via the Migraine Buddy® app. Preventive therapy was permitted. This analysis included all acutely treated (prescription/over-the-counter) migraine attacks recorded >3 days before the end of study and without missing diary data before the next attack. Generalized linear mixed models with a logit link were used to calculate the odds ratio for ≥ 1 , ≥ 2 , ≥ 3 , or ≥ 4 consecutive migraine free days following the day of acute treatment with rimegepant vs other medications.

Results: 429 participants recorded 2563 eligible migraine attacks. Independent of which acute treatment was used, 64.9% of attacks were followed by ≥ 1 , 47.6% by ≥ 2 , 35.4% by ≥ 3 , and 25.0% by ≥ 4 migraine-free days. In models evaluating treatment alone, rimegepant was associated with a significantly higher chance of migraine-freedom in the following ≥ 1 (77%), ≥ 2 (71%), ≥ 3 (58%), and ≥ 4 days (44%) vs other acute medications (Table 1). In models controlling for migraine days during the study, pain level, and functional ability at the time of treatment, rimegepant was associated with a significantly higher chance of migraine-freedom in the following ≥ 1 (54%), ≥ 2 (48%), ≥ 3 days (30%) vs other acute medications.

Conclusion: Higher odds of migraine-free days following treatment was observed with rimegepant vs other acute migraine medications.

TABLE 1 Odds ratios for consecutive migraine-free days following rimegepant vs other acute medication treatment of a migraine attack.

	Odds ratio (95% CI) of consecutive migraine-free days following acute treatment			
	>1 days after migraine-free (n=1683 attacks)	>2 days after migraine-free (n=1218 attacks)	>3 days after migraine-free (n=937 attacks)	>4 days after migraine-free (n=642 attacks)
Model 1 - Only including acute treatment as the independent variable				
Rimegepant (ref other*)	1.77 (1.43, 2.19) $P=0.001$	1.71 (1.38, 2.11) $P=0.001$	1.58 (1.26, 1.99) $P=0.001$	1.46 (1.11, 1.93) $P=0.005$
Model 2 - With covariates				
Rimegepant (ref other*)	1.54 (1.25, 1.89) $P=0.001$	1.46 (1.16, 1.83) $P=0.001$	1.33 (1.03, 1.63) $P=0.001$	1.13 (0.86, 1.46) $P=0.335$
Activity impairment at treatment	0.99 (0.87, 1.13) $P=0.888$	0.99 (0.87, 1.12) $P=0.890$	1.03 (0.89, 1.17) $P=0.650$	1.05 (0.89, 1.20) $P=0.541$
Pain level at treatment	1.02 (0.92, 1.13) $P=0.632$	1.06 (0.97, 1.16) $P=0.188$	1.02 (0.92, 1.13) $P=0.184$	0.97 (0.87, 1.07) $P=0.594$
Number of migraine days during the study	0.92 (0.90, 0.94) $P=0.001$	0.79 (0.77, 0.81) $P=0.001$	0.76 (0.74, 0.78) $P=0.001$	0.74 (0.71, 0.76) $P=0.001$
*Other medications were reported by category: over the counter drug, triptan, other prescription drug, unknown (ref). Some attacks were treated with >1 type of acute medication. Attacks were included in the rimegepant group if rimegepant was used, with or without other acute medications.				
The generalized linear mixed models had a binomial distribution, logit link, and random intercepts for participants that were fit to account for repeated attacks nested within individuals. Activity impairment at treatment was as reported by the participant in the diary using a four-step scale: 1=no disability to 4=severely impaired. Pain level at treatment was as reported by the participant in the diary using a seven-step scale: 0=not present to 6=extremely severe. Number of migraine days during the 28-day observational period was calculated from the complete diary (actual range 1 to 28). All were entered as continuous covariates.				

Disclosure: This study was sponsored by Pfizer. PJG reports consulting fees from Aeon, AbbVie, CoolTech, Dr Reddy's Laboratories, Eli Lilly, Epalex, Ipsen, Kallyope, Linpharma, Lundbeck, Orion Pharma, Pfizer, PureTech Health, Satsuma, Seaport Therapeutics, Shiratronics, and Teva; personal fees for advice from Gerson Lehrman Group, Guidepoint, SAI Med Partners, and Vector Psychometric; fees for educational materials from CME Outfitters and WebMD; publishing royalties or fees from Massachusetts Medical Society, Oxford University Press, UpToDate, and Wolters Kluwer. RBL reports research support from NIH, FDA, and the National Headache Foundation; research grants, consultant, advisory board member or honoraria from Aeon, Allergan/AbbVie, Amgen, Axsome, Dr Reddy's Laboratories, Eli Lilly, GSK, Ipsen, Lundbeck, Pfizer, Merck, Teva, and Vedanta; royalties from Informa and Wolff's Headache (8th edition, Oxford University Press, 2009); and stock options in Biohaven Pharmaceuticals, CoolTech, and NuVieBio. AU is an employee of Aptar Digital Health, which was a paid consultant to Pfizer for the conduct of this study. GL reports consulting fees from AbbVie, Dr Reddy's Laboratories, Eli Lilly, Lundbeck, Novartis, Pfizer, and Teva. PP-R reports in the last 36 months consultant or speaker fees/support from AbbVie, Almirall, Dr. Reddy's, Eli Lilly, Lundbeck, Medscape, Novartis, Organon, Pfizer, and Teva; research grants from AbbVie, AGAUR, EraNet Neuron, FEDER RIS3CAT, Instituto Investigación Carlos III, MICINN, Teva; funding for clinical trials to her research group from AbbVie, Amgen, Eli Lilly, Ipsen, Lundbeck, Novartis, Pfizer and Teva. TJS reports in the last 24 months consulting fees from AbbVie, Lundbeck, and Salvia; royalties from UpToDate; stock options/ownership interest in Allevalux and Nocira; research grants from AbbVie, American Heart Association, Flinn Foundation, Henry Jackson Foundation, National Headache Foundation, United States National Institutes of Health, Patient Centered Outcomes Research Institute, Pfizer, and United States Department of Defense. KMF is the Managing Director of MIST Research, which has received research funding from AbbVie, AESARA, Aptar, CoolTech, Migraine Canada, NYC Langone Health, Pfizer, UC Irvine, Ipsen. LA and KHB are employees of and own stock/options in Pfizer.

EPO-0582 | Rimegepant for the Acute Treatment of Migraine in People With ≥ 1 Lapsed Triptans, With and Without Current Triptan Use, in the CONFIDENCE study

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¹Montefiore Medical Center and Albert Einstein College of Medicine, Bronx, USA; ²Pfizer R&D UK Ltd, Tadworth, Surrey, UK; ³Aptar Digital Health, Paris, France; ⁴The Headache and Facial Pain Service, Guy's and St. Thomas' NHS Foundation Trust, London, UK; ⁵King Abdullah University of Science and Technology, Thuwal, Saudi Arabia and King's College London, London, UK; ⁶Headache Unit, Neurology Department, Vall d'Hebron Hospital, and Headache Research Group, Vall d'Hebron Research Institute (VHIR), Universitat Autònoma de Barcelona, Barcelona, Spain; ⁷Department of Neurology, Mayo Clinic, Scottsdale, USA; ⁸MIST Research, Wilmington, USA; ⁹Pfizer AB, Stockholm, Sweden

Background and aims: The prospective, observational CONFIDENCE study (NCT06467370) evaluated the effectiveness of rimegepant for the acute treatment of migraine over multiple attacks. This analysis evaluated rimegepant treatment outcomes in participants with ≥ 1 previously discontinued (lapsed) triptans, both with/without current triptan use.

Methods: CONFIDENCE recruited participants using rimegepant for the acute treatment of migraine from the Migraine Buddy® app. Participants completed a baseline survey and 28-day migraine diary. On days with a migraine attack, treatment characteristics, time to meaningful pain relief (MPR), and meaningful functional improvement (MFI) were collected. Generalized linear mixed models with a logit link were used to control for within person correlation and evaluate predictors of MPR and MFI within 2 and 4 h of rimegepant intake.

Results: Participants ($n=370$) with ≥ 1 lapsed triptan reported 1948 rimegepant-treated migraine attacks. Of these, 662 (34%) attacks were in participants with 1 lapsed triptan, 719 (37%) with 2, and 567 (29%) with ≥ 3 ; 538 (28%) were in participants who currently used a triptan to treat some attacks. Models identified significantly lower odds of MPR within 2 (58%) and 4 h (50%), and MFI within 2 (54%) and 4 h (57%) of rimegepant treatment in participants with ≥ 3 lapsed triptans (vs 1; Table 1). Having 2 lapsed triptans (vs 1) and current triptan use were associated with numerically lower odds of MPR and MFI but were not consistently statistically significant.

Conclusion: Rimegepant efficacy was higher in participants with lower numbers of lapsed triptans and without current triptan use, possibly reflecting clinical differences between subgroups.

Disclosure: This study was sponsored by Pfizer. RBL reports research support from NIH, FDA, and the National Headache Foundation; research grants, consultant, advisory board member or honoraria from Aeon, Allergan/AbbVie, Amgen, Axsome, Dr Reddy's Laboratories, Eli Lilly, GSK, Ipsen, Lundbeck, Pfizer, Merck, Teva, and Vedanta; royalties from Informa and Wolff's Headache (8th edition, Oxford University Press, 2009); and stock options in Biohaven Pharmaceuticals, CoolTech, and NuVieBio.

TABLE 1 Predictors of meaningful pain relief and meaningful functional improvement following acute rimegepant treatment.

	Odds ratio (95% CI) of achieving MPR	
	Within 2 h of rimegepant intake	Within 4 h of rimegepant intake
Number of migraine attacks during the 28-day observational period		
6 to 10 (vs 1 to 5)	1.01 (0.89, 1.14) P=0.98	0.99 (0.87, 1.13) P=0.97
11 to 24 (vs 1 to 5)	0.61 (0.39, 1.25) P=0.16	0.38 (0.16, 0.86) P=0.02
Age (continuous)	1.00 (0.98, 1.02) P=0.97	1.00 (0.97, 1.02) P=0.71
Female sex at birth (vs other)	0.67 (0.39, 1.15) P=0.16	0.91 (0.46, 1.82) P=0.79
White race (vs other)	1.62 (0.88, 3.26) P=0.13	1.39 (0.48, 2.57) P=0.85
Number of lapsed triptans		
2 (vs 1)	0.65 (0.42, 1.00) P=0.07	0.55 (0.33, 1.04) P=0.07
≥ 3 (vs 1)	0.42 (0.26, 0.67) P<0.001	0.50 (0.27, 0.91) P=0.02
Current triptan use (vs none)	0.62 (0.40, 0.95) P=0.03	0.60 (0.35, 1.03) P=0.06
	Odds ratio (95% CI) of achieving MFI	
	Within 2 h of rimegepant intake	Within 4 h of rimegepant intake
Number of migraine attacks during the 28-day observational period		
6 to 10 (vs 1 to 5)	0.99 (0.79, 1.20) P=0.94	0.94 (0.60, 1.47) P=0.79
11 to 24 (vs 1 to 5)	0.61 (0.32, 1.15) P=0.12	0.48 (0.22, 1.05) P=0.06
Age (continuous)	1.00 (0.98, 1.01) P=0.87	0.99 (0.97, 1.01) P=0.53
Female sex at birth (vs other)	0.60 (0.43, 1.31) P=0.27	1.30 (0.65, 2.21) P=0.55
White race (vs other)	1.59 (0.86, 2.94) P=0.14	1.17 (0.54, 2.53) P=0.69
Number of lapsed triptans		
2 (vs 1)	0.76 (0.50, 1.15) P=0.19	0.57 (0.34, 0.97) P=0.04
≥ 3 (vs 1)	0.46 (0.30, 0.72) P<0.001	0.43 (0.25, 0.75) P<0.003
Current triptan use (vs none)	0.61 (0.54, 1.20) P=0.29	0.74 (0.45, 1.22) P=0.24

MFI = meaningful functional improvement; MPR = meaningful pain relief. Shown outcomes from generalized linear mixed models including listed covariates. Shading indicates P<0.05.

AU is an employee of Aptar Digital Health, which was a paid consultant to Pfizer for the conduct of this study. GL reports consulting fees from AbbVie, Dr Reddy's Laboratories, Eli Lilly, Lundbeck, Novartis, Pfizer, and Teva. PJG reports consulting fees from Aeon, AbbVie, CoolTech, Dr Reddy's Laboratories, Eli Lilly, Epalex, Ipsen, Kallyope, Linpharma, Lundbeck, Orion Pharma, Pfizer, PureTech Health, Satsuma, Seaport Therapeutics, Shiratronics, and Teva; personal fees for advice from Gerson Lehrman Group, Guidepoint, SAI Med Partners, and Vector Psychometric; fees for educational materials from CME Outfitters and WebMD; publishing royalties or fees from Massachusetts Medical Society, Oxford University Press, UpToDate, and Wolters Kluwer. PP-R reports in the last 36 months consultant or speaker fees/support from AbbVie, Almirall, Dr. Reddy's, Eli Lilly, Lundbeck, Medscape, Novartis, Organon, Pfizer, and Teva; research grants from AbbVie, AGAUR, EraNet Neuron, FEDER RIS3CAT, Instituto Investigación Carlos III, MICINN, Teva; funding for clinical trials to her research group from AbbVie, Amgen, Eli Lilly, Ipsen, Lundbeck, Novartis, Pfizer and Teva. TJS reports in the last 24 months consulting fees from AbbVie, Lundbeck, and Salvia; royalties from UpToDate; stock options/ownership interest in Allevalux and Nocira; research grants from AbbVie, American Heart Association, Flinn Foundation, Henry Jackson Foundation, National Headache Foundation, United States National Institutes of Health, Patient Centered Outcomes Research Institute, Pfizer, and United States Department of Defense. KMF is the Managing Director of MIST Research, which has received research funding from AbbVie, AESARA, Aptar, CoolTech, Migraine Canada, NYC Langone Health, Pfizer, UC Irvine, Ipsen. LA and KHB are employees of and own stock/options in Pfizer.

EPO-0583 | Efficacy of eptinezumab in adults with CM and MOH who also received patient education: Country-level results from the RESOLUTION trial

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Background and aims: These country-level analyses evaluated the migraine-preventive efficacy of eptinezumab versus placebo in adults with chronic migraine (CM) and medication-overuse

headache (MOH), together with a brief educational intervention (BEI).

Methods: The multinational phase 4 RESOLUTION (NCT05452239) trial included a 12-week double-blind, placebo-controlled period. Adults (18–75yrs) with CM and MOH were randomized 1:1 to IV eptinezumab 100mg or placebo at baseline; all participants received a BEI on MOH before infusion. Post hoc subgroup analyses were conducted by country (those with ≥ 20 participants per arm). Endpoints included change from baseline in monthly migraine days (MMDs) and monthly days with acute medication use (Weeks 1–12; full-analysis set).

Results: The full-analysis set included 602 of 608 randomized participants. Countries with ≥ 20 participants per arm included Denmark, France, Georgia, Italy, the Netherlands, and Spain (Table 1). In comparison with placebo with BEI, eptinezumab with BEI improved treatment outcomes, with directionally consistent results seen across countries. Participants from the Netherlands had large reductions in acute medication use in both treatment groups, but concurrently the largest difference between both treatment groups in change from baseline in MMDs.

Conclusion: In this post hoc analysis of the RESOLUTION trial, the treatment effect of eptinezumab among participants with CM and MOH who received patient education appeared to be homogenous across countries, supporting the robustness of the intervention and its relative independence of country effects. Results from the Netherlands suggest that, while patient education can help reduce acute medication use, concurrent initiation of preventive treatment with eptinezumab has additional benefit for reversing migraine chronification.

TABLE 1 Change from baseline over Weeks 1–12 in MMDs and MAMDs.

MMDs				
Country	Treatment arm	N	Baseline, mean	CFB, Weeks 1-12, LS mean (SE)
Denmark	Eptinezumab	22	22.1	-7.1 (1.14)
	Placebo	25	22.3	-4.3 (1.07)
France	Eptinezumab	90	19.8	-6.5 (0.38)
	Placebo	91	21.2	-3.4 (0.57)
Georgia	Eptinezumab	36	19.9	-12.5 (1.01)
	Placebo	35	20.6	-9.9 (1.05)
Italy	Eptinezumab	58	22.0	-6.8 (0.77)
	Placebo	56	20.6	-3.8 (0.78)
The Netherlands	Eptinezumab	21	20.9	-9.7 (1.20)
	Placebo	21	18.5	-4.9 (1.20)
Spain	Eptinezumab	48	21.6	-9.4 (0.95)
	Placebo	47	21.4	-6.8 (0.96)
MAMDs				
Country	Treatment arm	N	Baseline, mean	CFB, Weeks 1-12, LS mean (SE)
Denmark	Eptinezumab	22	20.8	-12.7 (1.30)
	Placebo	25	20.2	-9.5 (1.22)
France	Eptinezumab	90	19.4	-8.1 (0.52)
	Placebo	91	20.2	-4.7 (0.52)
Georgia	Eptinezumab	36	19.6	-12.5 (0.95)
	Placebo	35	19.9	-10.3 (0.98)
Italy	Eptinezumab	58	20.8	-8.3 (0.75)
	Placebo	56	21.4	-5.0 (0.76)
The Netherlands	Eptinezumab	21	18.8	-14.8 (0.84)
	Placebo	21	16.7	-14.3 (0.84)
Spain	Eptinezumab	48	20.8	-11.6 (0.92)
	Placebo	47	21.1	-8.5 (0.93)

CFB, change from baseline; LS, least-squares; MAMDs, monthly days of acute migraine medication use; MMDs, monthly migraine days; SE, standard error.

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EPO-0584 | Migraine Prevention with Eptinezumab in Real Life – A Retrospective Swiss Cohort Study

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Background and aims: To evaluate real-world data to establish safety and efficacy of eptinezumab for the prevention of episodic and chronic migraine in clinical practice in Switzerland.

Methods: This multicenter retrospective cohort study included patients treated with eptinezumab at eleven Swiss headache centers. Over 12-months, patients received up to four infusions of eptinezumab 100 mg or 300 mg administered every three months. Data on efficacy and tolerability were extracted from patients' charts, including prospective headache diaries. The primary efficacy endpoint was the proportion of patients achieving a $\geq 50\%$ reduction in monthly migraine days (MMDs) three months after the first infusion.

Results: A total of 201 patients were included. Mean baseline MMDs were 17.5. After eptinezumab administration, 56% (95% CI [48.7, 63.1]) of patients reached the primary endpoint of $\geq 50\%$ response. Mean MMDs decreased significantly from baseline by 7.3 days (95% CI [6.19, 8.33], $p < 0.001$). This was observed for both, the 100 mg and 300 mg doses (100 mg, -7.2 , $p < 0.001$; 300 mg, -7.4 , $p < 0.001$). Prior treatment failure with CGRP ligand-targeting antibodies was associated with a significantly lower $\geq 50\%$ response rate (OR = 0.42, 95% CI 0.2–0.89, $p = 0.024$). Efficacy was not related to comorbid anxiety or depression. Adverse events within three months after infusion were reported by 15.1% of patients, most commonly rhinosinusitis (8%), constipation (6%), and fatigue (4%).

Conclusion: In a real-world clinical setting, eptinezumab for the prevention of episodic and chronic migraine was effective and well tolerated. Prior treatment failure with CGRP ligand-targeting antibodies was associated with reduced treatment response.

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institution (University Hospital of Basel) received speaker fees/fees for advisory boards/for consulting from Sanofi-Genzyme, Eli Lilly, AbbVie, Lundbeck, Pfizer and Teva. She received travel support from Bayer AG, Abbvie, Teva and Hoffmann-La Roche. Her research was/is being supported by the University and University Hospital of Basel, the Swiss Multiple Sclerosis Society, the "Stiftung zur Förderung der gastroenterologischen und allgemeinen klinischen Forschung sowie der medizinischen Bildauswertung", the "Freie Akademische Gesellschaft Basel" and the Swiss National Science Foundation (Project numbers: P300PB_174480 and currently: PZ00P3_216468). Philipp Balcerak: Advisory Board/Speaker for Novartis, AbbVie and Alexion.

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EPO-0585 | Limb-specific cervical spinal cord microstructure in Parkinson's disease: A clinically focused re-analysis of an open quantitative MRI dataset

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Background and aims: Gait disturbance and postural instability are major contributors to disability in Parkinson's disease (PD), yet conventional brain MRI and global clinical scores only partially account for these deficits. The cervical spinal cord—particularly the dorsal columns—may contribute to limb-specific motor impairment but remains understudied.

Methods: We performed a cross-sectional re-analysis of an open NeuroLibre dataset including quantitative cervical spinal cord MRI (C2–C5) from 68 individuals with PD and 38 healthy controls. Metrics included cord cross-sectional area, diffusion tensor imaging, and NODDI measures in the dorsal columns, magnetisation transfer ratio (MTR) in white and grey matter, and grey-matter T2. Motor impairment was assessed using UPDRS III total and limb-specific subscores.

Results: Cord cross-sectional area did not differ between groups, but PD showed altered dorsal column microstructure, with higher orientation dispersion and lower axial diffusivity. Within PD, dorsal column fractional anisotropy, orientation dispersion, and intracellular volume fraction were independently associated with worse lower-limb/gait impairment, explaining ~23–25% of variance. Grey-matter MTR preferentially correlated with upper-limb impairment. Lower-limb-dominant patients exhibited greater dorsal column microstructural disruption, with lateralisation to the clinically more affected side and progression across disease stages. An exploratory model combining dorsal column metrics with age achieved modest discrimination between PD and controls (AUC = 0.64).

Conclusion: Cervical dorsal column microstructure is selectively altered in PD and closely linked to lower-limb and gait impairment, motor phenotype, and disease stage, even in the absence of cord atrophy. Grey-matter metrics show a complementary relationship with upper-limb dysfunction.

Disclosure: Nothing to disclose.

EPO-0586 | Knowledge, perceptions, and attitudes toward influenza vaccination in people with Parkinson's disease

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Background and aims: Influenza infection represents a significant health risk for people with Parkinson's disease (PD), being associated with increased morbidity, functional decline, and hospitalization. Although annual influenza vaccination is recommended for older adults and individuals with chronic neurological disorders, reported vaccination rates in PD remain low. The aim of this study was to evaluate knowledge, perceptions, and attitudes toward influenza vaccination in patients with Parkinson's disease and to determine influenza vaccine acceptance rates in this population.

Methods: We conducted a cross-sectional observational study including 123 people with PD. Participants completed a structured questionnaire assessing socio-demographic variables, disease duration, comorbidities, influenza vaccination history, knowledge of influenza and vaccination, perceived risks and benefits, sources of information, and attitudes toward vaccination.

Results: The mean age of participants was 68.4 ± 8.9 years, and 56% were male. Only 11.4% ($n = 14$) reported receiving the influenza vaccine during the most recent season. Although 74% of patients considered influenza a potentially serious disease, only 23% correctly identified vaccination as the most effective preventive measure. Fear of adverse effects was reported by 61% of unvaccinated patients, concerns regarding worsening of Parkinson's symptoms by 56%, and doubts about vaccine efficacy by 42%. A lack of explicit recommendation from a health-care professional was reported by 53% of participants.

Conclusion: Influenza vaccination acceptance among patients with Parkinson's disease was extremely low. Despite moderate awareness of influenza-related risks, misconceptions, safety concerns, and insufficient physician recommendations remain major barriers.

Disclosure: Nothing to disclose.

EPO-0587 | Local and global microstructural and functional thalamomotor connectivity alterations in Parkinson's disease following motor learning

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Background and aims: Diffusion tensor imaging (DTI) provides a sensitive approach to detect microstructural decline in Parkinson's disease (PD) and is emerging as a method to identify

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Background and aims: Sublingual apomorphine (SL-APO) is an on-demand therapy for the management of OFF episodes in Parkinson's disease. This analysis describes the post-marketing safety profile of SL-APO in Europe during the first 18 months after launch.

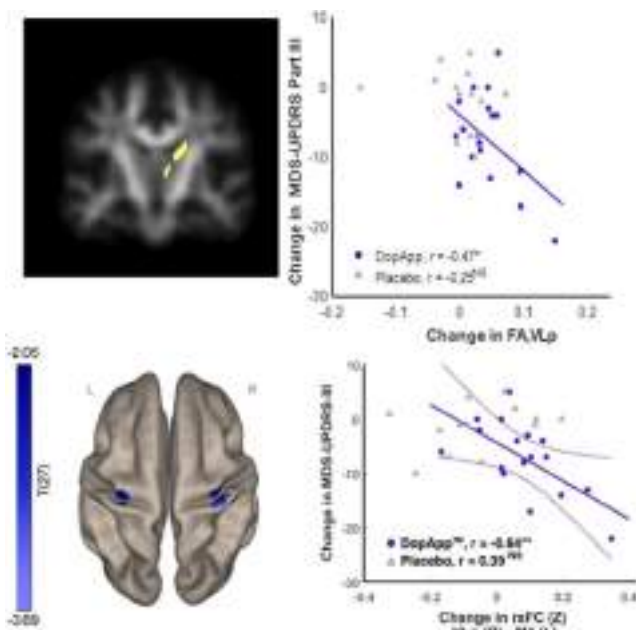
Methods: The drug global safety database was searched for European valid post-marketing reports (spontaneous reports, health authorities, literature reports, non-interventional studies, excluding marketing studies) from launch (April 2025) to October end 2025. Expectedness was assessed based on the current European Summary of Product Characteristics (SmPC) for SL-APO.

Results: A total of 346 events were collected from 159 valid cases: 94 male (59%), mean age 65years, from Germany (90, 57%), Portugal (44, 28%) and Spain (25, 16%). The 3 most common reported events were “nausea” (48, 14%), “dizziness” (22, 6%) and “vomiting” (15, 4%), consistent with known safety profile of SL-APO. Oropharyngeal events accounted for 47 (14%) of all events, most commonly “aphthous ulcer”(7) and “oral pain”(4) (Table1). Overall, 326 (94%) events were considered non-serious, and “syncope”(4) was the only serious event reported more than once. SL-APO was withdrawn, temporarily or permanently, in 72 (45%) cases, including 18 (11%) due to oropharyngeal events.

Conclusion: Reported events were predominantly non-serious and consistent with SL-APO known safety profile, supporting its favorable tolerability in real-world practice. The apparent discontinuation rate should be interpreted cautiously, given the reporting bias inherent to spontaneous post-marketing data and

TABLE 1 Summary of 346 reported events from 159 cases with sublingual apomorphine (SL-APO) in European post-marketing surveillance.

Event type	# Events	% of total
Non-serious	326	94,2%
Serious	20	5,8%
Most common events (≥ 3% of total)		
Nausea	48	13,9%
Dizziness	22	6,4%
Vomiting	15	4,3%
Drug ineffective	14	4,0%
Somnolence	13	3,8%
Hypotension	12	3,5%
Oropharyngeal events	47	13,6%
Most common (≥ 1% of total)		
Aphthous ulcer	7	1,9%
Oral pain	4	1,1%
Swollen tongue	4	1,1%



Neuroimaging analysis results and correlation with clinical measures

rapid learning induced white-matter changes, yet such evidence in PD remains limited.

Methods: In this prospective, double-blind, randomized, placebo-controlled study, 32 levodopa-treated patients were randomized to a 3-week digital intervention (DopApp) or placebo app. The DopApp involved a digital spatial navigation protocol combining egocentric and allocentric strategies with multisensory-deprivation principles, alongside psychological and rehabilitation modules. Patients were assessed pre- and post-treatment using clinical and psychological scales and MRI scans, including resting-state fMRI (rsFC) and DTI, to investigate motor learning neuroplasticity.

Results: DopApp treatment significantly improved the total MDS-UPDRS score compared to placebo-app, driven by gains in motor function (Part III) and activities of daily living (Part II). Neuroimaging revealed increased fractional anisotropy in the right ventrolateral posterior (VLP) thalamus and strengthened functional connectivity between VLP and precentral gyrus (PreCG), both correlated with clinical motor improvements. Graph-theory analysis indicated increased local efficiency in PreCG and adjacent motor network, suggesting enhanced structural integration.

Conclusion: These findings demonstrate regionally specific, short-term plasticity in PD, and support DTI's sensitivity to capture clinically relevant motor training-related white-matter adaptations beyond dopaminergic medication effects.

Disclosure: Amir Amedi is a co-founder and employee of Remepy Health Ltd. and holds shares in Remepy Inc, the company that developed the DopApp.

increased vigilance during the early post-launch period. These observations underscore the importance of ongoing pharmacovigilance, proactive management of tolerability issues, and patient education to optimize adherence.

Disclosure: Supported by Bial-Portela & C^a, S.A.

EPO-0589 | Advancing Deep Brain Stimulation management in Parkinson's Disease through wearable technologies: A systematic review

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Background and aims: Deep Brain Stimulation (DBS) is a well-established advanced treatment in Parkinson's disease (PD) [1]. The effects of DBS have been investigated using laboratory-based systems [2], which are restricted to specialized clinical settings and therefore limit broader clinical integration. In contrast, wearable sensors have emerged as a complementary approach to objectively assess PD symptoms in both clinical and real-world settings [3], thereby extending traditional assessments. This study aims to conduct a systematic review to examine the application of wearable technology in supporting DBS management.

Methods: The literature review was conducted analyzing Pubmed and Scopus databases. Studies were eligible for inclusion if they involved individuals with PD treated with DBS and used wearable devices. Studies were excluded if they involved non-commercial prototype devices, did not specify the device used, and were not peer-reviewed.

Results: After full-text screening, 71 articles were included and categorized into two main research topics: 49 studies (69%) focused on digital monitoring, aiming to evaluate DBS efficacy; 22 studies (31%) investigated wearable-based approaches to support DBS programming, exploring brain-activity patterns during disease-related symptoms. The majority of studies were conducted in clinical settings (53, 74%) and used a single device (42, 58%). Considerable heterogeneity was observed across study protocols in terms of tasks, treatment conditions, and time measurements.

Conclusion: Current literature, while heterogeneous and largely exploratory, highlights the promising potential of wearables to assess treatment efficacy, characterize motor impairments alongside associated brain activity patterns, and support personalized approaches to DBS management. Large-scale studies employing standardized protocols are required to improve their generalizability.

Disclosure: Nothing to disclose.

EPO-0590 | Alpha-fetoprotein level in dystonia patients: Follow - up

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Background and aims: In our previous study, we observed elevated alpha-fetoprotein (AFP) levels in patients with dystonia. The aim of this project was to monitor AFP levels over one-year period in dystonic patients with elevated AFP, to exclude other causes of increased AFP (tumor, hepatopathy, or gene-related persistence), and to compare clinical characteristics between patients with normal and elevated AFP levels.

Methods: Patients with primary idiopathic dystonia (including negative WES) and healthy controls were enrolled. AFP levels were measured using an immunoassay.

Results: Of 171 patients with primary idiopathic dystonia, 156 (47 men) had normal AFP levels (3 ± 1.7 ug/L), whereas 15 (2 men) had elevated AFP levels (17.7 ± 22 ug/L). At one-year follow-up, AFP levels normalized in only two patients; in the remaining 13 patients, AFP levels either remained stable or increased. Oncological screening was negative in all patients and mild hepatopathy was observed in two cases, HNF-1 mutations, (causing AFP persistence) were excluded. All 57 age- and sex-matched healthy controls had normal AFP levels (3.3 ± 1.8 µg/L) and no significant differences between groups were detected.

Conclusion: These findings suggest that AFP elevation in dystonia was predominantly long-term and occurred without an identifiable triggering cause. This unexpected persistence raises the possibility of an association between AFP elevation and neurodevelopmental pathology. Increased AFP levels have been observed in neural tube defects, spina bifida, and in autosomal recessive cerebellar ataxias. We conclude that long-term AFP elevation may be present in patients with dystonia; however, its clinical relevance and relationship to disease pathophysiology remain unclear.

Disclosure: Nothing to disclose.

EPO-0591 | Concomitant recording of streamed local field potentials from the STN and GPI to investigate Parkinson's disease neurophysiology: A feasibility study

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Background and aims: Globus pallidus internus (GPi) and subthalamic nucleus (STN) deep brain stimulation (DBS) are

both effective treatments for Parkinson's disease (PD). The development of stimulators able to stream local field potentials (LFP) such as Medtronic Percept® allows to study the effect of DBS in chronic implants, and to elucidate the role of STN and GPi neurophysiology in the parkinsonian system. We recorded simultaneously from two patients with dual STN and GPi implant, to assess the feasibility of concomitant recording, and to investigate the effect of DBS on the interaction between nuclei.

Methods: 2 patients with dual STN-GPi Medtronic Percept® implants underwent resting-state simultaneous LFP recording with increasing levels of stimulation (0 to 4 mAmp, 0.5 mAmp steps). After synchronization, we computed time-frequency resolved power, coherence, and granger causality at each step.

Results: We successfully synchronized the STN and GPi signal in both patients. Stimulation of either nucleus resulted in suppression of high-beta peak power in both the stimulated and non-stimulated nucleus, with accompanying coherence loss at the same frequency. Granger analysis showed an STN to GPi directionality of high-beta connectivity, which was abolished with increasing stimulation to either nucleus.

Conclusion: In this preliminary study we demonstrate the feasibility of simultaneous LFP recording from the STN and GPi. We identify an indirect modulatory effect of stimulation on the high beta peak of the non-stimulated nucleus and show that high beta-band connectivity within the subthalamo-pallidal system is directed from the STN to the GPi, in line with previously proposed models.

Disclosure: Nothing to disclose.

EPO-0592 | Non-motor symptom burden does not reliably discriminate between PSP subtypes

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Background and aims: Progressive supranuclear palsy (PSP) can be sub-classified into Richardson's syndrome (RS), cortical, and subcortical subtypes. We aimed to characterise symptom severity across PSP variants using validated scales, adjusting for disease duration, to determine whether phenotypes exhibit differential patterns of non-motor burden.

Methods: 92 patients with PSP were classified into cortical ($n=17$), subcortical ($n=28$) and RS subtypes ($n=47$). Each was assessed with PSPRS, ACE-III, SCOPA-AUT, BDI, CBI, NPI, and SF-36. Descriptive statistics summarised total-cohort and subtype distributions. Group differences were examined using multivariate general linear modelling.

Results: PSP subtypes demonstrated a significant overall multivariate effect on PSPRS domain scores ($p<0.001$).

Univariate tests showed differences in Mentation ($p=0.029$), Bulbar ($p<0.001$), Ocular ($p=0.008$), Limb ($p<0.001$) and Gait ($p=0.014$) with PSP-RS exhibiting greatest impairment and PSP-Subcortical least. No significant subtype differences were detected across autonomic symptom domains by SCOPA-AUT. While there was no overall multivariate subtype effect on cognitive profile, higher fluency scores characterised PSP-Subcortical versus PSP-RS ($p=0.047$). In the neuropsychiatric domain, neither BDI scores nor CBI caregiver-informed behavioural scores differed significantly between subtypes. Multivariate analysis did not display an overall subtype effect on total NPI scores. Quality-of-life did not differ significantly between subtypes across any SF-36 domain.

Conclusion: Our results suggest that motor manifestations remain the principal differentiator between PSP phenotypes, with PSP-RS demonstrating most severe bulbar, ocular, limb and gait impairment. In contrast, non-motor domains—including cognition, autonomic function, mood and behaviour—did not reliably discriminate between subtypes, suggesting a shared non-motor burden across the PSP spectrum.

Disclosure: No conflicts of interest to disclose. Acknowledgment given to the PSP Association of Ireland for providing funding to support this research.

EPO-0593 | Motor, nonmotor and cognitive correlates of iron deposition within subcortical nuclei in early drug-naïve Parkinson's disease patients

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Background and aims: Iron deposition using Quantitative Susceptibility Mapping (QSM) has been reported in several cortical/subcortical areas within the dopaminergic pathways in patients with Parkinson's disease (PD). This study was aimed at exploring 3T MRI-derived iron deposition content within bilateral subcortical nuclei in early drug-naïve PD patients compared to healthy controls (HC), and as well as potential associations with motor, non-motor and cognitive/behavioral symptoms.

Methods: 3T MRI images of 59 drug-naïve PD patients were analyzed and compared with 18 age-, sex-matched HC. QSM values were extracted from several subcortical deep gray matter nuclei and 16 thalamic subregions using the HybraPD atlas. Partial Spearman correlation analysis was performed between QSM metrics significantly different in PD compared to HC, and motor, non-motor and cognitive/behavioral variables. Finally, a ROC curve analysis was performed to test the ability to discriminate PD patients from HC using QSM values.

Results: Compared to HC, PD patients showed significantly higher iron deposition in the bilateral substantia nigra pars compacta, left substantia nigra pars reticulata, left subthalamic nucleus, and in the right red and intralaminar thalamic nuclei. Correlation analyses revealed greater motor and non-motor severity associated with increased iron within the right intralaminar thalamic and red nuclei, respectively. ROC analysis demonstrated that QSM values may accurately discriminate PD patients from HC.

Conclusion: Our findings demonstrated that using a disease-specific HybraPD atlas, a specific iron accumulation within sub-cortical nuclei can be found in PD patients even at early stages, and may be proposed as a potential biomarker of dopaminergic neurodegeneration.

Disclosure: Disclosures: no disclosures to declare

EPO-0594 | MRgFUS thalamotomy may reduce long-term motor complications in tremor-dominant Parkinson's disease: A five-year prospective case-control study

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Background and aims: Magnetic resonance-guided focused ultrasound (MRgFUS) thalamotomy targeting the ventral intermediate nucleus (VIM) is an established treatment for medication-refractory tremor in tremor-dominant Parkinson's disease (TDPD). Long-term data on tremor improvement, dopaminergic therapy management and development of motor complications after MRgFUS are limited. This study evaluated the long-term impact of VIM MRgFUS thalamotomy compared with optimized medical therapy in TDPD.

Methods: We conducted a prospective case-control study including patients with TDPD without baseline motor fluctuations or dyskinesias. Patients treated with unilateral VIM MRgFUS thalamotomy (PD-FUS) were compared with patients receiving optimized drug therapy alone (PD-ODT). Groups were matched 1:2 for sex, age, disease duration and baseline levodopa equivalent daily dose (LEDD). Assessments were performed in On-medication state at baseline, one and five years. The primary outcome was motor complications severity assessed by the Movement Disorder Society–Unified Parkinson's Disease Rating Scale (MDS-UPDRS) Part IV.

Results: 21 PD-FUS and 38 PD-ODT patients were included. At five years, median MDS-UPDRS-IV scores were lower in PD-FUS than PD-ODT (3 [0;4] vs 5 [4;7] respectively; $p=0.016$) (Figure 1). Tremor improved in PD-FUS, with a greater median reduction in total tremor score than PD-ODT ($-60\%[-75; -27]$ vs $-13\%[-43; 25]$; $p=0.049$). Over five years, total LEDD increased in both groups but remained lower in PD-FUS (median increase 185 mg [83–463] vs 419 mg [303–690]; $p=0.014$) (Figure 2). Adverse events were mild and transient.

Conclusion: VIM MRgFUS thalamotomy provides durable tremor control in TDPD and is associated with reduced

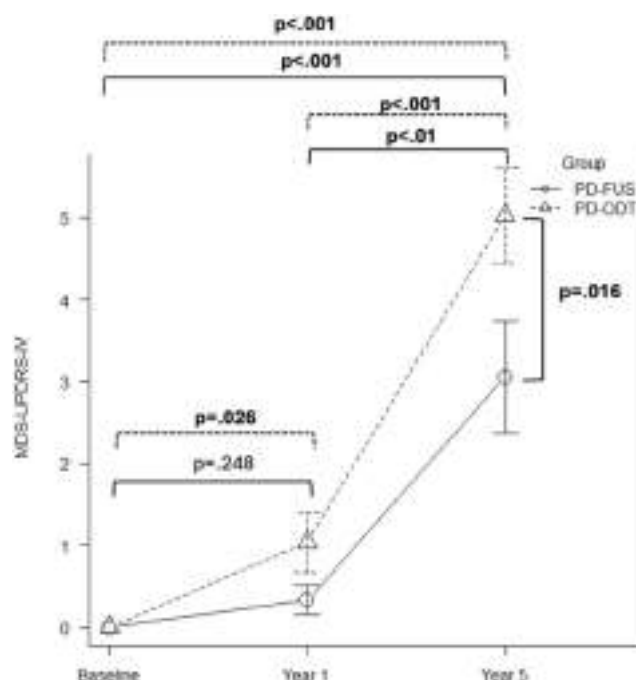


FIGURE 1 Changes in MDS-UPDRS Part IV scores over follow-up in patients treated with magnetic resonance-guided focused ultrasound (PD-FUS) and optimized drug therapy (PD-ODT). p values indicate between-group comparisons.

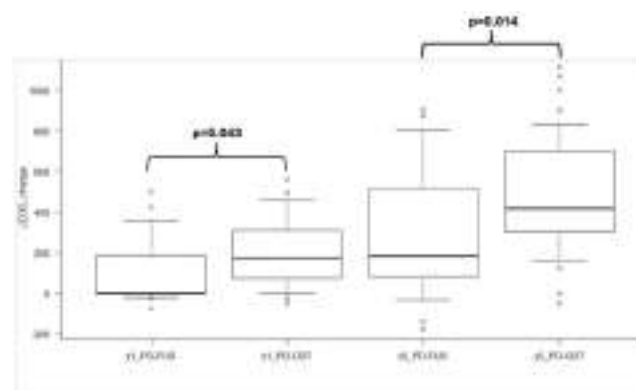


FIGURE 2 Changes in total levodopa equivalent daily dose (LEDD) from baseline to 1 and 5 years in PD-FUS and PD-ODT patients. p values indicate between-group comparisons.

long-term severity of motor complications and lower increase in LEDD.

Disclosure: Nothing to disclose.

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Background and aims: Olfactory impairment is a recognised early marker of Parkinson's disease (PD). However, the extent to which graded hyposmia severity relates to longitudinal cognitive performance remains unclear. This study examined longitudinal cognitive outcomes and baseline resting-state functional connectivity (rs-fMRI) across five olfactory categories: normosmia, mild, moderate and severe hyposmia, and anosmia.

Methods: Data were obtained from the Parkinson's Progression Markers Initiative (PPMI) database. Participants were classified into five olfactory groups based on age-adjusted percentile scores from the University of Pennsylvania Smell Identification Test (UPSIT). Longitudinal Montreal Cognitive Assessment (MoCA) scores were analysed using linear mixed-effects models in SPSS. Baseline rs-fMRI data were processed using CONN. ROI-to-ROI and seed-to-voxel analyses focused on regions involved in olfactory and cognitive networks, including the insula, orbitofrontal cortex, anterior cingulate cortex, amygdala, hippocampus and thalamus.

Results: A total of 254 PD patients and 133 healthy controls showed comparable demographic characteristics. Greater olfactory impairment severity significantly predicted lower MoCA scores ($p=0.004$), independent of time. Each incremental increase in olfactory severity category was associated with a 0.14-point reduction in MoCA score. Baseline rs-fMRI analyses (PD=91; controls=17) revealed no significant functional connectivity differences across olfactory categories within pre-defined regions of interest.

Conclusion: Greater olfactory impairment severity is associated with reduced cognitive performance in PD, even without detectable baseline functional connectivity differences. These findings suggest olfactory testing may be a sensitive marker of early cognitive vulnerability, while connectivity alterations may emerge later or require alternative imaging approaches to detect subtle network changes.

Disclosure: Nothing to disclose.

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Background and aims: Deep Brain Stimulation (DBS) is an effective treatment for the motor symptom of Parkinson's disease (PD), but it seems to have a limit effect on caregiver burden. Caregivers continually confront the dual challenges of navigating symptom fluctuations and handling the DBS device. Moreover, there are limit intervention were used to decrease caregiver burden of PD patients post DBS. Therefore, we have constructed an caregiver intervention program based on Omaha system and to verify it's effectiveness on caregiver burden.

Methods: A randomized controlled trial was designed. The stratified block randomization was used. 120 caregivers were randomly separated in either intervention (Nursing care based on Omaha system) or control (General nursing care) group. The demographic information, score of ZBI, GSES and KPPDQ were collected at baseline (T0), 3- and 9-month follow.

Results: Using repeated-measures analysis of variance to compare difference between two groups. And Caregiving knowledge only revealed a significant main effect of time ($F(1.83, 215.83)=17.66, p<0.001, \eta^2=0.13$). A significant main effect of group ($F(1, 118)=9.19, p=0.003, \eta^2=0.072$) and a significant time-by-group interaction ($F(2, 236)=5.23, p=0.006, \eta^2=0.042$) for caregiver self-efficacy. Caregiver burden revealed a significant main effects of time ($F(2, 118)=4.13, p=0.017, \eta^2=0.034$) and group ($F(1, 118)=8.08, p=0.005, \eta^2=0.064$), as well as the time-by-group interaction ($F(2, 118)=5.20, p=0.006, \eta^2=0.042$).

Conclusion: Neuromodulation Specialist Nurse-Led support system for caregivers of PD patients post DBS based on Omaha system provide a long-term, multi-dimensional and multi-form to improve the knowledge, self-efficiency and burden of caregivers.

Disclosure: The work described in this paper was supported in full by grants from the Fu Xing Nursing Scientific Research Fund of Fudan University, Shanghai, China [grant number



TABLE 1 Baseline characteristics.

Caregiver Characteristic Variable	Intervention Group n=90	Control Group n=60	P value
Age, n(%)			0.418
<30	0	2	
30-39	29	28	
40-49	29	26	
50-59	2	4	
60+	0	0	
Sex, n(%)			0.815
Male	31	32	
Female	29	28	
Education, n(%)			0.1
Low	7	16	
Medium	25	23	
High	28	21	
Employment status, n(%)			0.842
Employed	34	34	
Non-employed	6	8	
Retired	18	18	
Marital status, n(%)			0.500
Partnered	56	54	
Unpartnered	4	6	
Relation, n(%)			0.72
Spouse	25	21	
Descendant	33	37	
Extended kin	2	2	
Residence, n(%)			0.866
Urban	41	42	
Town	10	8	
Rural	9	10	
Ethnicity, n(%)			0.1
Han	40	39	
Other	0	1	
Religious belief, n(%)			0.717
No	55	37	
Yes	5	3	
Health status, n(%)			0.893
Bad	4	3	
Normal	35	37	
Health	21	20	
Household income, n(%)			0.398
Low	2	5	
Medium	30	32	
High	28	23	
Caregiving duration, n(%)			0.198
Short-term (<3 years)	13	14	
Medium-term (3-10 years)	33	24	
Long-term (>10 years)	14	22	
Mean caregiving hours a day(SD), h	4.26(3.34)	4.39(7.10)	0.017
Mean accuracy of knowledge(SD), %	69.23(16.19)	69.64(16.63)	0.748
Mean score of GSES(SD)	22.40(5.54)	22.72(5.62)	0.445
Mean score of ZHI(SD)	28.50(16.77)	29.43(17.40)	0.906
Personal burden	16.47(8.78)	17.40(9.19)	0.648
Responsibility burden	6.90(5.52)	7.20(6.17)	0.56

TABLE 2 Within- and between-group comparison for self efficacy.

Comparison	Control	MD	SE	p ^a	95% CI
Within-group					
Intervention	T0 vs T1	-1.05	0.308	0.588	[-3.012, 0.912]
	T0 vs T2	-2.233 [*]	0.831	0.025	[-4.256, -0.211]
	T1 vs T2	-1.183	0.807	0.436	[-3.143, 0.777]
Control	T0 vs T1	1.6	0.804	0.15	[-0.362, 3.562]
	T0 vs T2	1.367	0.831	0.31	[-0.656, 3.389]
	T1 vs T2	-0.233	0.807	1	[-2.193, 1.727]
Between-group					
T0 Intervention vs Control		-0.317	0.961	0.743	[-2.227, 1.594]
T1 Intervention vs Control		2.333 [*]	0.828	0.006	[0.694, 3.972]
T2 Intervention vs Control		3.283 [*]	0.857	0	[1.566, 4.981]

^ap < .05

^bBonferroni correction was applied for multiple comparisons.

TABLE 3 Within- and between-group comparison for caregiver burden.

Comparison	Control	MD	SE	p ^a	95% CI
Within-group					
Intervention	T0 vs T1	1.267	2.167	1	[-3.997, 6.53]
	T0 vs T2	8.035 [*]	2.2	0.008	[2.689, 13.377]
	T1 vs T2	6.767 [*]	1.91	0.002	[2.128, 11.406]
Control	T0 vs T1	-2.717	2.167	0.637	[-7.98, 2.547]
	T0 vs T2	-1.481	2.2	1	[-6.827, 3.861]
	T1 vs T2	1.233	1.91	1	[-3.465, 5.871]
Between-group					
T0 Intervention vs Control		-0.95	3.128	0.762	[-7.144, 5.244]
T1 Intervention vs Control		-4.833	2.603	0.084	[-10.088, 0.221]
T2 Intervention vs Control		-10.467 [*]	1.907	0	[-14.546, -6.388]

^ap < .05

^bBonferroni correction was applied for multiple comparisons.

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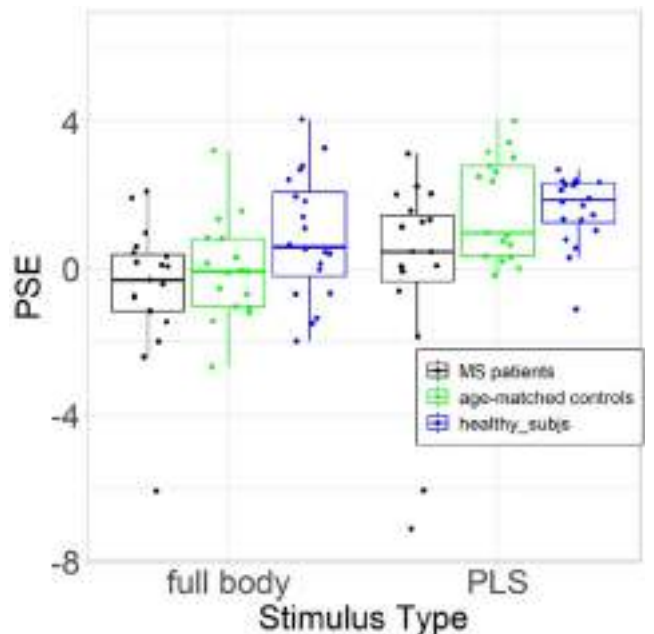
Neurorehabilitation 2

EPO-0597 | Biological motion recognition in multiple sclerosis: Toward behavioral biomarkers for personalized neurorehabilitation

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Background and aims: Multiple sclerosis (MS) is a chronic disease associated with complex neurorehabilitation needs. The Action Observation Network (AON) supports motor learning and the prediction of biological motion (BM). However, evidence on AON functioning and BM recognition in MS remains limited. We aimed to (i) characterize BM recognition performance in people with MS (pwMS), (ii) compare their performance with that of young and age-matched healthy controls (HCs), and (iii) investigate the role of clinical, neuroimaging, and neuropsychological covariates.

Methods: In this cross-sectional study, 37 pwMS, 30 age-matched HCs, and 22 young HCs were enrolled. Participants



Performance on the BM at the two levels of detail (i.e., full-body and point-light stimuli) in pwMS, age-matched HCs, and young HCs.

completed a computerized BM task in which they observed arm movements displayed as either photorealistic avatars or point-light stimuli to estimate perceptual temporal extrapolation at the point of subjective equality (PSE). Clinical, neuropsychological, and subjective fatigue measures were collected; structural neuroimaging was available for pwMS.

Results: Two response patterns emerged: a “good” psychometric curve that allows PSE estimation and an inverted (“bad”) pattern. Among “good” participants, pwMS showed significantly altered temporal extrapolation compared with both control groups, independent of stimulus type. Age-matched HCs exhibited a bimodal distribution for impoverished stimuli, and performance was associated with fatigue: increased fatigue was associated with greater temporal extrapolation (similar to young HCs), whereas reduced fatigue was associated with diminished extrapolation.

Conclusion: Preliminary findings indicate altered BM recognition in pwMS, possibly reflecting AON dysfunction. Fatigue appears to modulate BM perception not only in pwMS but also in physiological aging. These results support the potential of BM recognition as a biomarker for personalizing action-observation-based rehabilitation.

Disclosure: Nothing to disclose.

EPO-0598 | **Multimodal integrative interventions in Korean medicine for persistent postural-perceptual dizziness: A systematic review and network meta-analysis**

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Background and aims: Persistent Postural-Perceptual Dizziness (PPPD) is a chronic functional vestibular disorder with complex pathophysiology. Evidence regarding multimodal integrative strategies remains limited. This study compared the relative effectiveness of Korean medicine-based single and multimodal interventions for PPPD using Bayesian network meta-analysis.

Methods: We analyzed RCTs published between 2000 and September 2024 involving patients with PPPD or chronic functional dizziness. Interventions included herbal medicine, acupuncture, physical/psychological therapy, and combinations. Primary outcomes were Dizziness Handicap Inventory (DHI) and emotional well-being, with treatment hierarchies estimated via SUCRA values.

Results: Ten RCTs (956 participants) were analyzed across three subnetworks (Figure 1, Figure 2). In the control-monotherapy subnetwork, psychological therapy ranked highest (SUCRA=99%). Within the VRT-based subnetwork, CBT-informed VRT was most effective (SUCRA=86.1%). In the pharmacologic-integrative subnetwork, multimodal interventions consistently outperformed monotherapy, with combined psychological, physical, and pharmacological therapy ranking highest (SUCRA=99.71%) (Figure 3).

Conclusion: This first network meta-analysis on PPPD highlights the clinical relevance of multimodal strategies addressing both physical and psychological dimensions. Although findings are promising, evidence certainty is low due to small sample

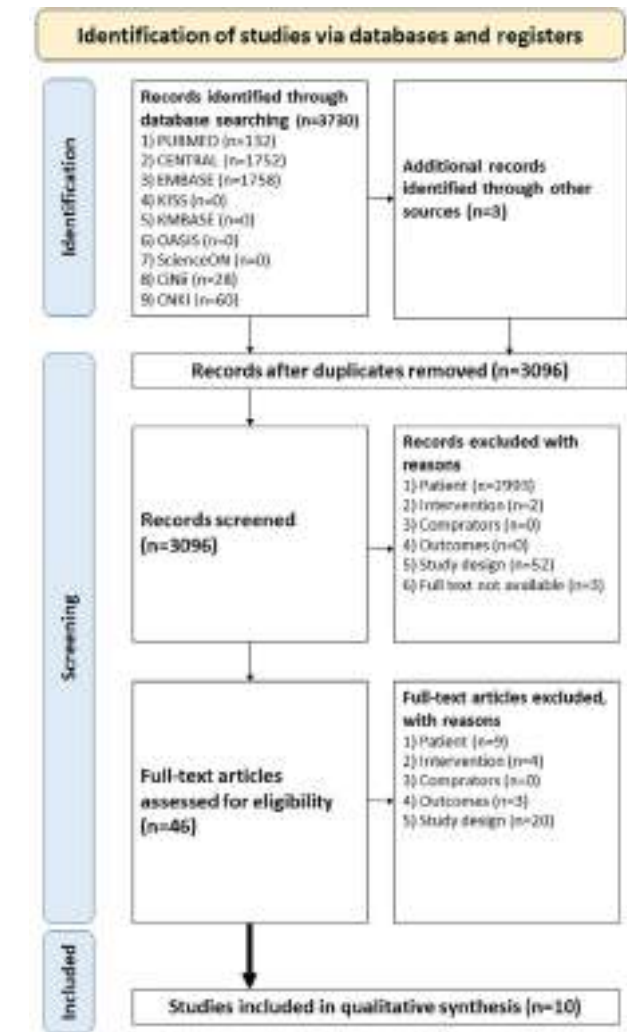


FIGURE 1 PRISMA Flow Diagram of Study Selection. From an initial 3,733 records identified across nine databases, 10 RCTs involving 956 participants were finally included for the network meta-analysis after a rigorous screening based on PICOS criteria.



FIGURE 2 RoB of the included studies. Most included studies (8/10) were classified as having “some concerns,” while two studies were judged as “high risk” primarily due to issues with missing outcome data.

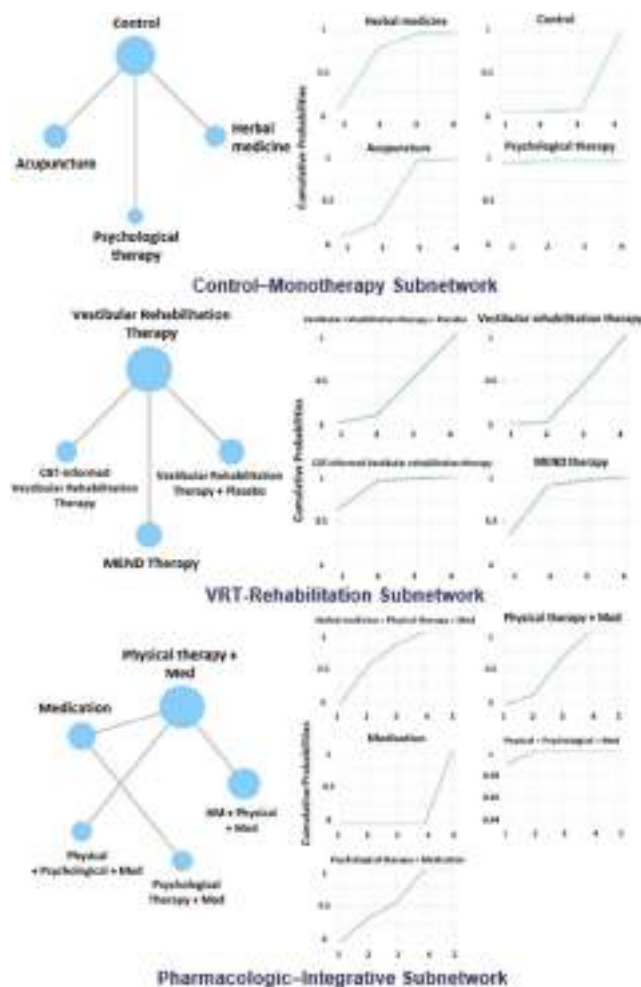


FIGURE 3 Multimodal strategies (SUCRA 99.71%) outperformed monotherapy. Psychological therapy (99%) and CBT-informed VRT (86.1%) ranked highest in their subnetworks, demonstrating that integrated physical and psychological care is most effective.

sizes and fragmented network structure. Future large-scale randomized trials are needed to strengthen the evidence base.
Disclosure: Nothing to disclose.

EPO-0599 | Abstract withdrawn

EPO-0600 | Polypharmacy Deprescribing Does Not Affect Muscle Mass, Strength, and Physical Function in Post-Stroke Geriatric Patients with Sarcopenia

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FIGURE 1 Forest plot for SMI (Skeletal Muscle mass Index).



FIGURE 2 Forest plot for HG (Hand grip Strength).



FIGURE 3 Forest plot for FIM (Functional Independence Measure) Motor.

Background and aims: One-third of geriatric patients with multimorbidity take five or more prescribed medications (polypharmacy). We evaluated whether polypharmacy deprescribing affect muscle mass, strength, and physical function in post-stroke geriatric patients with sarcopenia.

Methods: Relevant studies were identified from PubMed, Scopus, and Web of Science until December 2025. Observational studies involving post-stroke geriatric patients with sarcopenia and polypharmacy were included, comparing deprescribing and non-deprescribing groups. Two reviewers independently screened studies and assessed risk of bias. Forest plots were generated, with SMI (Skeletal Muscle mass Index), HG (Hand Grip strength), FIM (Functional Independence Measure), BRS (Brunnstrom Recovery Stage) as the outcome.

Results: Four studies comprising 432 participants in the deprescribing group and 534 in the non-deprescribing group were included. There were no significant differences between groups in SMI (MD -0.04 kg/m²; 95% CI -0.20 to 0.11; I²=0%), hand-grip strength (MD -0.17 kg; 95% CI -1.93 to 1.59; I²=37%), FIM motor scores (MD 0.25; 95% CI -7.21 to 7.71; I²=72%), BRS of the upper limb (MD -0.07; 95% CI -0.46 to 0.31; I²=0%), and lower limb (MD -0.25; 95% CI -0.59 to 0.08; I²=0%).

Conclusion: This study showed that polypharmacy deprescribing was not associated with difference in muscle mass, muscle strength, or physical function. Limited study numbers and heterogeneity highlight the need for future prospective studies focusing on the effects of specific drug classes on sarcopenia.

Disclosure: Nothing to disclose.

EPO-0601 | Combined high-frequency rTMS over the non-lesional hemisphere and rPMS for severe upper limb paralysis: Functional compensation via ipsilateral fibers

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Background and aims: Standard repetitive transcranial magnetic stimulation (rTMS) for stroke-induced upper limb paralysis

often shows limited efficacy in severe cases. This study investigates a novel therapeutic approach combining high-frequency (10Hz) rTMS over the non-lesional hemisphere and repetitive peripheral magnetic stimulation (rPMS) of paretic proximal muscles. The objective was to activate ipsilateral descending pathways, such as the anterior corticospinal and reticulospinal tracts, to promote functional compensation in severe paralysis.

Methods: We included 16 patients (mean age 61.4 +/- 10.0 years) with severe paralysis, defined as a Fugl-Meyer Assessment for Upper Extremity (FMA-UE) score of 22 or less. Participants received 10Hz rTMS over the non-lesional motor cortex for two weeks and rPMS on paralyzed shoulder muscles (e.g, deltoid) for two to four weeks. Clinical outcomes were evaluated using FMA-UE scores, comparing pre- and post-treatment results via the Wilcoxon signed-rank test.

Results: Total FMA-UE scores significantly improved from 12.5 ± 4.4 to 14.8 ± 4.6 ($p=0.0051$). Sub-score analysis revealed a significant increase in Part A (proximal function) from 10.8 ± 3.8 to 12.6 ± 3.8 ($p=0.0074$). Conversely, no significant differences were observed in Parts B, C, or D. No serious adverse events were reported during the intervention.

Conclusion: Simultaneous central and peripheral stimulation significantly improved proximal upper limb function in severe stroke cases. This combined protocol likely promotes functional

activation of ipsilateral descending pathways, offering a valuable new strategy for functional recovery in patients with severe paralysis where conventional treatment options are extremely limited.

Disclosure: This work was supported by JSPS KAKENHI Grant Number 24K14390. The authors declare no other conflicts of interest associated with this manuscript.

EPO-0602 | Exploring the relationship between balance and quality of life in individuals with Parkinson's disease: Preliminary findings

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Background and aims: The aim of this study was to investigate the relationship between health-related quality of life (QoL) and commonly used clinical balance and functional performance tests in individuals with PD.

Methods: Nineteen individuals with PD at Modified Hoehn and Yahr stages ≤ 2.5 were included in the study. Health-related quality of life was assessed using the Parkinson's Disease Questionnaire-39 (PDQ-39). Balance and lower extremity function were evaluated using the 30-Second Sit-to-Stand Test (30STS), Six-Spot Step Test (SSST), Four Square Step Test (FSST), 3-Meter Backward Walk Test (3MBWT), Timed Up and Go Test (TUG), and the Berg Balance Scale (BBS). The Modified Falls Efficacy Scale (MFES) was used to assess the perceived fear of falling.

Results: No significant relationships were found between the PDQ-39 total score and the performance-based balance tests (30STS, SSST, FSST, 3MBWT, and TUG) ($p > 0.05$). In contrast, statistically significant associations were observed between the total PDQ-39 scores and both the BBS and MFES scores ($p < 0.05$). The simple linear regression analyses indicated that BBS and MFES explained 20% and 32% of the variance in QoL, respectively.

Conclusion: The findings of this study suggest that the reduced QoL in individuals with PD is more strongly associated with balance-related perceptual factors, such as fear of falling, and balance confidence, than with objective, performance-based balance and mobility measures alone. These results highlight the importance of incorporating interventions that not only aimed physical performance but also address fear of falling, and enhance balance confidence within physiotherapy and rehabilitation programs.

Disclosure: Nothing to disclose.

EPO-0603 | Longitudinal Effects of Two Courses of HF-rTMS and Intensive Speech Therapy for Aphasia

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Background and aims: The longitudinal effects of combining intensive speech therapy (ST) with two separate courses of high-frequency repetitive transcranial magnetic stimulation (HF-rTMS) for aphasia remain unclear. This study retrospectively

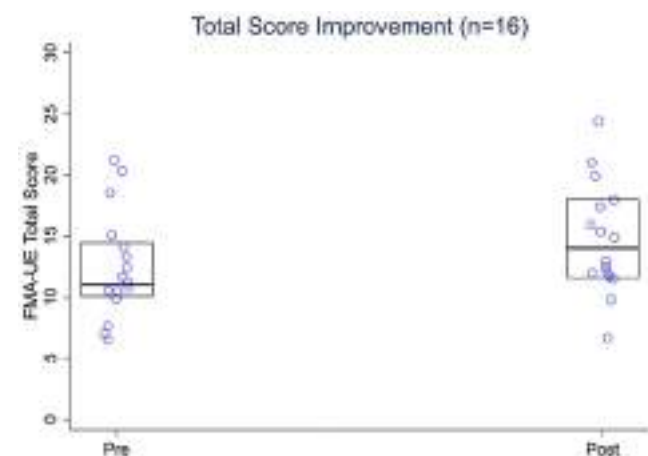


FIGURE 1 Changes in total FMA-UE scores before and after the combined stimulation protocol.

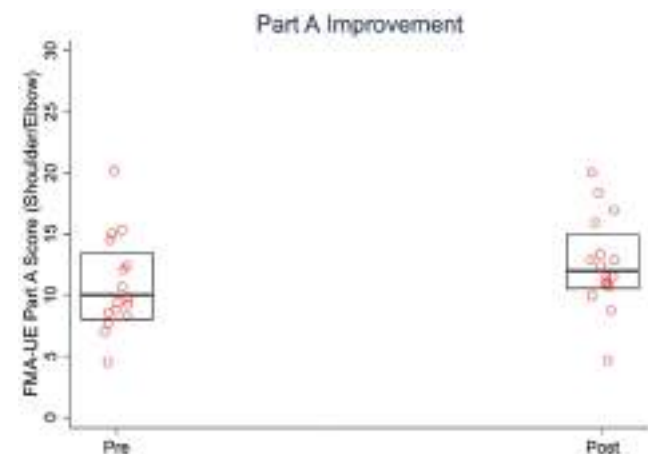


FIGURE 2 Changes in FMA-UE Part A scores before and after the combined stimulation protocol.

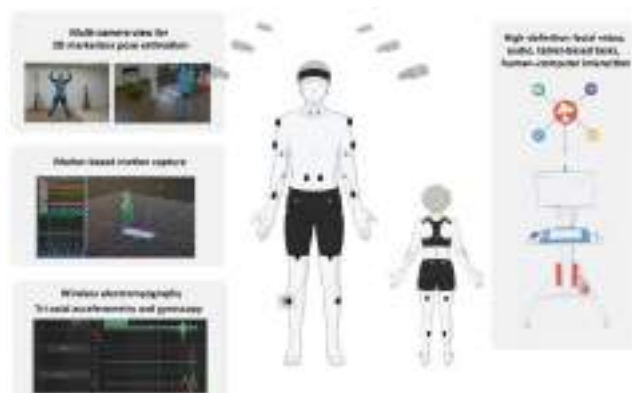


FIGURE 3 Multimodal data acquisition from anterior and posterior views, integrating synchronised multi-view video, motion capture, surface EMG with IMU's, and additional streams including facial video, audio, tablet tasks, and human–computer interaction.

longitudinal tracking, and data-driven inference. This strengthens behavioural examination as a quantitative foundation for biomarker discovery and treatment optimisation.

Disclosure: Nothing to disclose.

EPO-0605 | Treatment effects on spasticity and motor outcomes in pediatric cerebral palsy: A systematic review and meta-analysis

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Background and aims: Botulinum toxin type A (BoNT-A) is a cornerstone intervention for managing focal spasticity in children with Cerebral Palsy (CP). Despite its widespread use, variability in trial designs has complicated the assessment of its magnitude, durability, and impact on functional gains. This study aimed to quantify the short-term therapeutic effects of BoNT-A and synthesize evidence regarding dosing and rehabilitation contexts.

Methods: We conducted a systematic review of Randomized Controlled Trials (RCTs) published between January 2015 and November 2025 across four major medical databases (PubMed, Cochrane, Web of Science, Google Scholar). Inclusion was restricted to pediatric CP populations receiving pharmacological spasticity management. Primary outcomes included spasticity severity (Modified Ashworth Scale, MAS) and gross motor function (GMFM). Data were synthesized to calculate pooled mean differences and evaluate heterogeneity across limb targets and dosing strategies.

Results: Eighteen RCTs involving 892 participants met the inclusion criteria. Meta-analysis of placebo-controlled lower-limb studies demonstrated a statistically significant reduction in spasticity at 4–6 weeks (pooled mean difference on MAS: −0.31). While tone reduction was consistent across licensed doses, functional improvements (GMFM) were modest and primarily observed when injections were paired with structured rehabilitation (1–3 months post-intervention). The therapeutic

effect typically attenuated by week 12. Adverse events were predominantly mild and transient.

Conclusion: BoNT-A is effective for short-term focal spasticity reduction in pediatric CP. However, translation into functional motor gains is inconsistent and strongly dependent on concurrent rehabilitation. Clinicians should view BoNT-A as an adjunct to therapy rather than a standalone solution, with re-administration required to sustain benefits.

Disclosure: Nothing to disclose.

EPO-0606 | Comparison of Respiratory Functions and Physical Fitness Levels in Individuals Diagnosed with Multiple Sclerosis and Healthy Individuals

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Background and aims: Multiple sclerosis (MS) is defined as a chronic, inflammatory and autoimmune disease affecting the white matter of the central nervous system. Depending on the location of the lesions, symptoms may vary and can significantly affect the daily life of patients. The study aimed to investigate the comparison of pulmonary function and physical fitness levels in individuals with Multiple Sclerosis and healthy individuals.

Methods: The study included 34 individuals with MS with EDSS scores of ≤4.5 - 6 and 34 healthy individuals of similar age and gender. Respiratory function was measured by spirometer, inspiratory muscle strength by respiratory muscle strength meter, inspiratory muscle endurance by ascending threshold loading test, biceps and quadriceps muscle strength by digital hand dynamometer, cardiorespiratory endurance by Six Minutes Walking Test, flexibility with sit and reach and back scratch test and balance by interactive balance system.

Results: There were significant differences between the MS group's respiratory function test and inspiratory muscle strength and biceps, quadriceps muscle strength, 6MWT walking distance, expected percentage, stability test parameters and the values of the healthy group ($p < 0.05$).

Conclusion: As a result of our study we determined that respiratory functions and physical fitness parameters were lower in MS individuals than in healthy individuals. We think that appropriate evaluation and treatment should be made by considering the effects of these factors in physiotherapy and rehabilitation programs specific to MS individuals and developing them.

Disclosure: Nothing to disclose

TABLE 1 Demographic characteristics of participants.

	MS n (M)	Healthy Subjects n (M)	p
	Mean(SD) (Min-Max)	Mean(SD) (Min-Max)	
Age	46.53(11.17)(21-65)	46.78(10.82)(25-60)	0.999
Height (cm)	1.67(0.11)(1.3-1.89)	1.68(0.08)(1.34-1.93)	0.794
Weight (kg)	78.78(19.67)(45-131)	72.65(11.89)(60-97)	0.100
BMI (kg/m ²)	28.01(3.19)(17.03-36.87)	26.14(3.16)(19.93-37.48)	0.213
Gender (F/M) (%)	28(18.5)/8(5.5)	35(26.3)/3(23.3)	

1: Independent one sample t test, 2: Mann-Whitney U test, MS: Multiple sclerosis, SD: Standard deviation, BMI: Body mass index

TABLE 2 Comparison of pulmonary function test results of MS and healthy group.

	MS (n=34) Mean±SD (Min-Max) Median (Q1-Q3)	Healthy Subjects (n=34) Mean±SD (Min-Max) Median (Q1-Q3)	p
FVC(L)	2.28±0.94 (1.5-3.21) 2.07 (2.54-4.11)	2.47±0.6 (2.33-3.34) 2.77 (3.26-4.3)	0.001 [†]
FEV ₁ (L)	2.12±0.99 (1.09-4.65) 2.00 (2.48-3.78)	2.92±0.77 (1.98-4.65) 2.79 (3.07-3.13)	0.001 [†]
FEV ₁ /FVC(%)	86.31±15.29 (52.96-96.78.75) (95.7) 77.4)	94.87±14.37 (59.58-117.83) (92.5-93)	0.004 [†]
PEF(L/s)	2.78±1.18 (1.04-5.47) 2.40 (3.77-4.48)	4.34±1.32 (0.96-8.78) 4.23 (5.09-5.74)	<0.0001 [†]
FEF ₂₅₋₇₅ (L/s)	2±1.05 (0.15-4.48) 1.83 (1.54-2.27)	3±1.28 (0.75-7.07) 2.7 (2.08-5.6)	<0.0001 [†]
Mean±SD (Min-Max) 47 (35-58)	56.15±16.19 (31-108) 67.3 (32-80)	0.001 [†]	
HR (bats)	7.06±3.34 (1.5-6.7) 2.7 (2.2-3.3)	6.92±4.94 (1.1-7.7) 4.1 (3.2-5)	<0.0001 [†]
Volume (L)	1.96±0.7 (0.8-3.1) 1.7 (1.4-3.3)	2.34±0.64 (1.3-4.1) 2.2 (1.9-3.6)	0.004 [†]
Respiratory muscle endurance (min:HR:HRmax)	102.58±36.76 (4.11-133.13) (90) 86.41 (74.93-115.29)	90.87±34.78 (39.11-120) 98.73 103.00 (70.6-168)	0.001 [†]
Maximal Borg Scale	8.5 (3.1-12.7) (8) 8 (4-7)	8.1 (4.1-14.2) (8) 8 (4-8)	0.001 [†]

[†]Independent two sample t test, * Mann Whitney U test, HR: heart rate, SPO2/Oxygen saturation, FVC: Forced vital capacity, FEV₁: Forced expiratory volume in 1 second, FEV₁/FVC: The ratio of Forced Expiratory Volume in 1 second (FEV₁) to Forced Vital Capacity (FVC), PEF: Peak expiratory flow, FEF₂₅₋₇₅: Forced Expiratory Flow at 25-75% of Forced Vital Capacity, MV: Minimum voluntary ventilation

[†]Independent two sample t test. * Mann Whitney U test. HR: Heart rate. SPO2: Oxygen saturation. FVC: Forced vital capacity. FEV₁: Forced expiratory volume in 1 second. FEV₁/FVC: The ratio of Forced Expiratory Volume in 1 second (FEV₁) to Forced Vital Capacity (FVC). PEF: Peak expiratory flow. FEF₂₅₋₇₅: Forced Expiratory Flow at 25–75% of Forced Vital Capacity. MVV: Maximum voluntary ventilation.

TABLE 3 Comparison of physical fitness test results of MS and healthy group.

	MS (n=34) Mean±SD (Min-Max) Median (Q1-Q3)	Healthy Subjects (n=34) Mean±SD (Min-Max) Median (Q1-Q3)	p
Biceps muscle strength (kg)	117.1 (34.4-193.5-140) 118 (86-125)	145.7 (15.111-174) 145 (104-133)	<0.0001 [†]
Quadriceps muscle strength (kg)	118±29.7 (53.8-158) 118 (65-129)	144.9±25.0 (5.208) 143 (28-146)	<0.0001 [†]
60 and reach Test (cm)	116 (11-53.4) 89 (5-125)	176 (92-34) 109 (79)	0.001 [†]
Back Scratch Test (cm)	-1.2 (3.5-4) 8 (4-1)	-1.3 (2.6-4.2) 11 (3-1)	0.667 [†]
2MWT (%)	55.05±18.78 (16.65-84.78) 59.3 (40.35-88.84)	75.66±9.24 (58.54-87) 75.66 (68.53-81.45)	<0.0001 [†]
2MWT walking distance (m)	773±143.4 (178-819) 778 (518-819)	917.6±61.44 (716-1,031) 917.5 (853-952)	<0.0001 [†]
ST(NH)	22.86±15.14 (7.11-70.89) 26.77 (11.3-60.07)	15.54±5.41 (7.87-33.71) 22.18 (9.6-33.6)	0.001 [†]
ST(NC)	47.12±39.15 (1.01-217.73) 33.39 (1.84-61.39)	16.25±5.69 (7.65-26.99) 16.43 (5.56-30.68)	<0.0001 [†]
ST(PC)	26.39±18.13 (5.14-62.21) 30.8 (9.41-59.01)	31.25±12.57 (5-52.88) 25.69 (1.51-50.47)	0.001 [†]
ST(PC)	33.11±19.82 (11.24-218.07) 44.72 (1.34-87.82)	30.06±12.70 (6.72-136.09) 25.79 (23.43-31.49)	<0.0001 [†]
ST(NH)	51.21±62.47 (1.93-198.67) 56.86 (2.38-98.87)	30.46±10.01 (9.59-62.96) 19.69 (5.58-31.84)	<0.0001 [†]
ST(NC)	49.71±46.15 (1.37-240.6) 33.22 (18.67-70.12)	30.65±5.91 (9.36-57.93) 19.69 (17.42-22.82)	0.001 [†]
ST(PC)	51.37±48.38 (1.11-126.2) 39.81 (13.09-70.88)	31.76±7.81 (13.91-60.49) 29.38 (17.5-34.64)	<0.0001 [†]
ST(NH)	49.81±42.16 (1.05-211.54) 31.11 (23.68-64.21)	19.66±5.62 (1.84-35.78) 16.97 (1.12-32.33)	<0.0001 [†]

1. Independent two sample t test. 2. Mann Whitney U test. ST: Stability index. ST: Static position eyes open. NC: Static position eyes closed. PC: On pillow eyes open. PC: On pillow eyes closed. NH: Eyes closed head turned right. HL: Eyes closed head turned left. HB: Eyes closed head turned backwards. HF: Eyes closed head turned forward onto chest.

EPO-0607 | Stride width can be a descriptor of reduced ability of gait modulation in people with Multiple Sclerosis

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Background and aims: Gait modulation—the ability to adapt gait to maintain stability while minimizing energetic cost across different contexts—can be impaired in people with Multiple Sclerosis (pwMS) due to proprioceptive deficits and motor rigidity, increasing fall risk. Traditional gait measures such as stride velocity describe overall performance but limited insight into dynamic balance. Stride width (SW), the mediolateral distance between the feet, is a sensitive marker of lateral stability but is typically restricted to lab-based assessment. We monitored SW and stride velocity in healthy subjects (HS) and pwMS during in-lab and ecological walking, to examine how different contexts shape gait modulation.

Methods: Ten healthy subjects (HS) and ten pwMS (EDSS 1–6) performed a 2-min walking test (2MWT), an in-lab simulation of daily activities (SDA), and a 2.5-h free-living session while wearing a validated wearable system estimating stride velocity and SW (Figure 1). Linear mixed-effects models evaluated the impact of walking condition and group on these parameters.

Results: Stride velocity was consistently lower in pwMS than HS, while SW revealed context-specific patterns (Figure 2).

Conclusion: Wearable-based SW captures context-dependent gait adaptation in pwMS that stride velocity alone cannot. SW variability was reduced in pwMS during 2MWT and SDA, indicating limited modulation of the base of support, whereas free-living walking showed wider SW, suggesting a compensatory

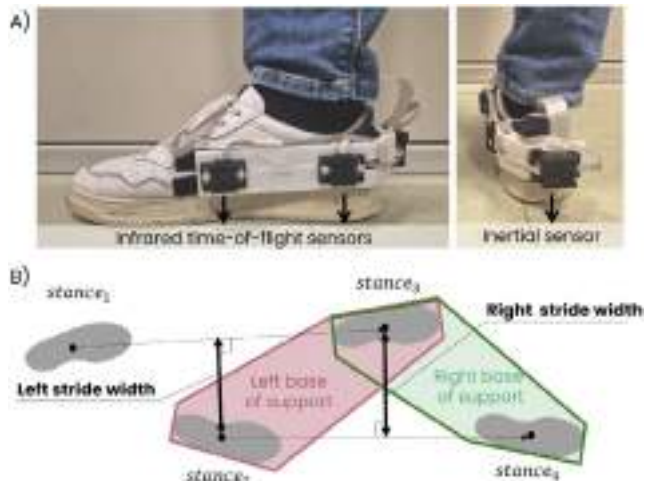


FIGURE 1 Participant's right foot instrumented with a novel miniaturized wearable system (A) which enables to estimate bilateral stride width (B).

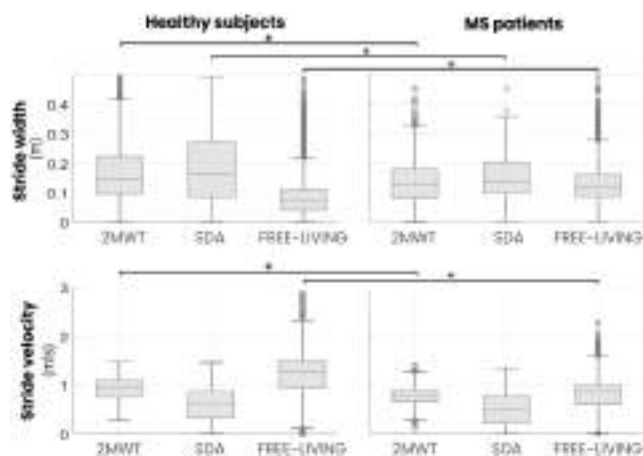


FIGURE 2 Distributions of stride width (above) and stride velocity (bottom) of healthy subjects (left) and people with Multiple Sclerosis (right) across different walking conditions. Over 45000 steps were analyzed. * $p < 0.05$.

strategy prioritizing postural safety over gait economy. Impaired SW modulation may represent a novel biomarker of lateral instability, proprioceptive deficits, and motor rigidity. Future work will investigate its longitudinal relationship with clinical disability and fall incidence.

Disclosure: Part of the project NODES, MUR – M4C2 1.5 of PNRR funded by the European Union - NextGenerationEU (ECS00000036 – CUP E13B22000020001).

EPO-0608 | Validation of a Vision Screening Tool for Detecting Visual Abnormalities in Acute Stroke Care

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Background and aims: Visual abnormalities are common after stroke but are under-recognised in acute care. The Vision Impairment Screening Assessment (VISA) was developed to enable structured bedside screening by non-specialist stroke clinicians. We aimed to validate the Norwegian translated version of VISA against comprehensive neuro-ophthalmic examination and describe the prevalence of post-stroke visual abnormalities in a hyperacute stroke population.

Methods: StrokeVIS was a prospective diagnostic accuracy study including consecutive patients admitted for acute stroke management at a tertiary thrombectomy centre. Participants underwent VISA screening and neuro-ophthalmic examination within 72 hours of admission. VISA was administered by trained stroke staff blinded to the reference standard. Diagnostic performance was assessed using sensitivity, specificity, predictive values, and overall accuracy. Secondary analyses examined prevalence across visual domains and evaluated the performance of VISA and the visual items of the National Institutes of Health Stroke Scale (NIHSS).

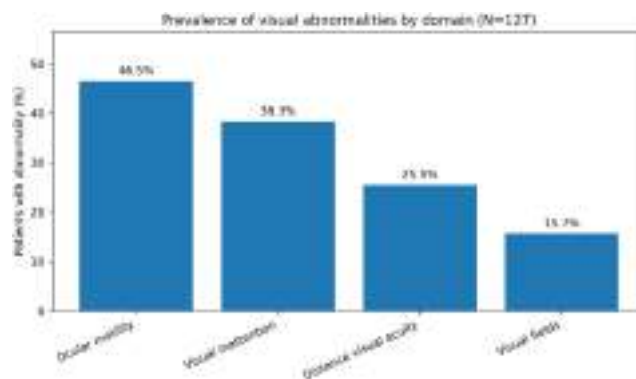


FIGURE 1 Prevalence of visual abnormalities detected on neuro-ophthalmic examination in the full study cohort (N=127). Percentages represent patients with at least one abnormality per domain. Multiple abnormalities per patient were allowed.

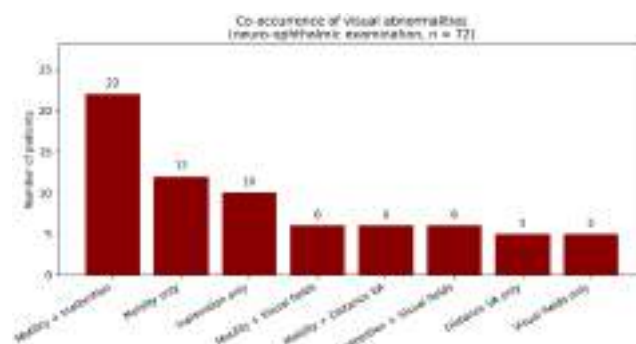


FIGURE 2 Co-occurrence of visual abnormalities identified on neuro-ophthalmic examination among patients with complete data across all domains (n = 72). Bars show single-domain abnormalities and the most frequent multi-domain combinations.

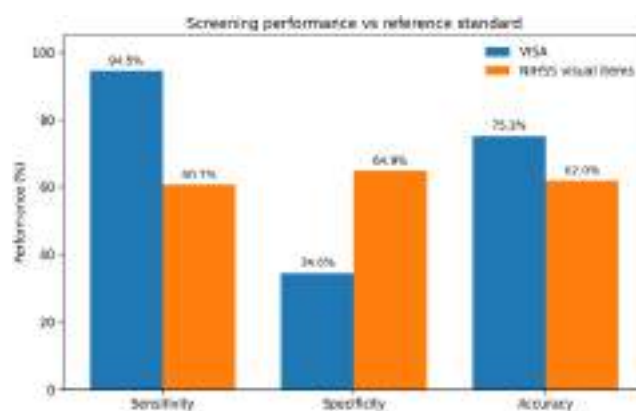


FIGURE 3 Screening performance of the Vision Impairment Screening Assessment (VISA) and visual items of the National Institutes of Health Stroke Scale (NIHSS) compared with neuro-ophthalmic examination as the reference standard.

Results: A total of 127 patients were included, of whom 81 completed both assessments. Neuro-ophthalmic examination identified visual abnormalities in 70.1% of patients, most frequently ocular motility disturbances (46.5%), visual inattention (38.3%), reduced distance visual acuity (25.5%), and visual field loss (15.7%). VISA demonstrated high sensitivity (94.5%) but

low specificity (34.6%), with an overall accuracy of 75.3%. The NIHSS visual items showed lower sensitivity (60.7%) and accuracy (62.0%).

Conclusion: The Norwegian version of VISA demonstrates high sensitivity for detecting early post-stroke visual abnormalities in acute care. Given the high prevalence of visual abnormalities and the limited sensitivity of routine neurological assessment, structured visual screening may improve early identification of vision-related deficits in stroke pathways.

Disclosure: Nothing to disclose.

Movement disorders 6

EPO-0609 | Disease course in primary brain calcification: A monocentric longitudinal study from an Italian Cohort

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Background and aims: Primary Brain Calcification (PBC) is a rare neurodegenerative disorder characterized by brain calcium deposition, featuring movement disorders and psychiatric/cognitive disturbances[1–3]. Quantitative longitudinal data describing disease progression are limited[2–4]. We here investigate motor-symptoms evolution and clinical predictors of functional outcome.

Methods: 83 PBC patients from Padova University, diagnosed by CT-scan and metabolic screening; NGS genetic test; neurological assessment (MDS-UPDRS-III, H&Y, SARA, UHDRS, BFMDS, TETRAS, MoCA); mean prospective follow-up of 3.5±2years, combined with retrospective data collection covering 9±4years; linear-mixed-models/hinge-LMM, Kaplan-Meier-survival-curves, multivariate analysis.

Results: Neurological symptoms were documented in 64 subjects; movement disorders were the most frequent manifestation ($n=52$, predominantly parkinsonism); over time, combined motor and cognitive/psychiatric involvement occurred in 65% of patients. Mean age-at-onset was 53.3 years but neuropsychiatric symptoms presented earlier (mean 42 years, $p=0.002$). Genetic diagnosis was achieved in 46%, mostly involving MYORG and SLC20A2 mutations. Parkinsonism, cerebellar signs and cognitive decline predicted worse functional outcome ($p=0.01$), older age was a risk-factor of symptom development ($p=0.004$), genetics influenced specific symptoms penetrance. Clinical progression occurred in 63% of the cohort, with significant worsening in UPDRS-III, SARA and MoCA ($p<0.001$) scales. Hinge-LMM, aligning to symptom onset, demonstrated progression rates of +1.7/year at UPDRS-III, +0.7/year at SARA ($p<0.001$). Isolated

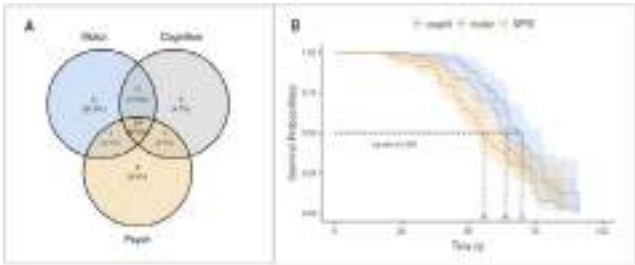


FIGURE 1 A) Combination of clinical symptoms at last evaluation (Venn diagram). B) Kaplan-Meier survival curves for core symptoms over time.

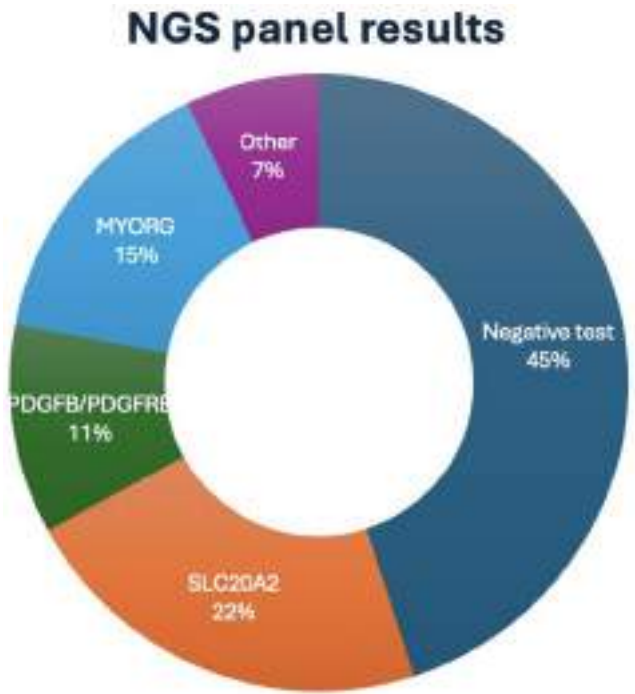


FIGURE 2 Genetic findings in the cohort.

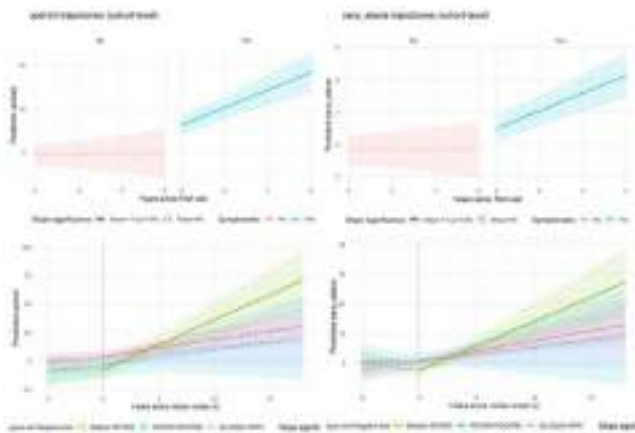


FIGURE 3 Disease trajectories for significant motor scales (UPDRS-III and SARA).

dystonia/tremor showed minimal progression. MYORG mutation carriers showed faster progression compared to other genetic subgroups (UPDRS-III:+3.6/y, $p=0.01$; SARA:+1.7/y, $p=0.01$).

Conclusion: PBC is a progressive disorder with distinct disease trajectories influenced by symptom profile and genetic background. Comprehensive assessment and longitudinal monitoring are essential to define individual disease prognosis and support future therapeutic trials.

Disclosure: Data partially submitted to 2026 Italian Movement Disorders Congress as abstract Nothing to disclose

EPO-0610 | Baseline variables associated with sublingual apomorphine film maintenance dose

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Background and aims: This analysis aimed to identify baseline demographic and clinical characteristics associated with the final titrated dose of sublingual apomorphine (SL-APO) in patients with Parkinson's disease experiencing OFF episodes, to support more individualized titration strategies and optimized treatment.

TABLE 1 Baseline demographic and clinical characteristics associated with the final titrated dose of sublingual apomorphine.

Baseline Characteristics	10 mg (51)	15 mg (11)	20 mg (17)	25 mg (20)	30 mg (24)	Univariate Ordinal Logistic Regression OR (95%CI)	p
Sex							
Female	21	34	21	17	5	1.26 (0.81;1.94)	0.299
Male	42	47	46	33	19		
Age (years)	63.0 ± 9.51	64.0 ± 8.45	63.8 ± 9.47	65.1 ± 7.68	64.4 ± 9.21	1.01 (0.99;1.00)	0.308
Height (cm)	177.9 ± 9.42	171.2 ± 9.67	172.7 ± 10.36	171.8 ± 10.06	173.7 ± 8.4	1.00 (0.99;1.00)	0.813
Weight (kg)	85.9 ± 17.55	81.18 ± 14.48	80.3 ± 17.58	81.6 ± 20.24	81.2 ± 18.99	1.00 (0.99;1.01)	0.568
BMI	28.3 ± 5.08	27.7 ± 4.89	26.9 ± 4.78	27.5 ± 6.30	28.3 ± 5.90	1.00 (0.98;1.00)	0.432
Hoehn & Yahr	2.1 ± 0.58	2.1 ± 0.55	2.2 ± 0.73	2.1 ± 0.64	2.5 ± 0.81	1.07 (0.93;1.23)	0.344
Mini Mental State	28.7 ± 1.38	29.3 ± 1.01	28.8 ± 1.34	28.9 ± 1.28	29.3 ± 1.07	1.01 (0.98;1.03)	0.942
MDS-UPDRS Part I	9.0 ± 5.11	10.2 ± 5.38	11.7 ± 6.14	13.5 ± 9.78	13.7 ± 8.32	1.07 (1.00;1.14)	<0.001
MDS-UPDRS Part II	12.7 ± 6.82	12.6 ± 6.42	15.0 ± 8.52	16.6 ± 7.58	15.4 ± 7.49	1.00 (1.00;1.00)	<0.001
MDS-UPDRS Part III	16.6 ± 13.85	18.9 ± 15.12	41.5 ± 14.00	44.7 ± 14.87	47.7 ± 14.56	1.00 (1.00;1.00)	<0.001
MDS-UPDRS Total	61.6 ± 21.86	62.5 ± 19.32	67.4 ± 18.13	72.8 ± 22.77	66.7 ± 19.99	1.02 (1.01;1.03)	<0.001
Levodopa Dose (mg)	944.4 ± 362.60	921.8 ± 552.98	1094.1 ± 768.12	1391.5 ± 981.62	1542.6 ± 1223.83	1.07 (1.04;1.10)	<0.001
PD Duration (years)	9.1 ± 4.09	7.1 ± 3.71	8.6 ± 4.33	8.6 ± 3.78	10.5 ± 4.04	1.02 (0.99;1.00)	0.136
Motor Fluctuations							
Duration (years)	6.5 ± 5.11	2.9 ± 3.87	3.8 ± 2.56	6.1 ± 2.55	5.8 ± 3.11	1.01 (0.95;1.00)	0.694
Morning Akinesia							
No	31	32	23	22	8	1.28 (0.85;1.92)	0.238
Yes	12	49	44	28	16		
Wearing-off							
No	2	2	1	3	0	1.46 (0.27;7.52)	0.562
Yes	81	79	66	47	24		
Delayed ON							
No	23	32	34	16	8	1.33 (1.05;1.68)	0.018*
Yes	40	49	43	36	16		
Sudden OFF							
No	33	48	40	27	13	1.04 (0.68;1.58)	0.858
Yes	28	33	27	23	11		
Number of OFF Episodes	1.8 ± 1.08	3.7 ± 1.10	6.6 ± 1.07	6.3 ± 2.00	4.3 ± 0.56	1.25 (1.07;1.46)	0.000*
Duration of a single OFF episode	76.7 ± 80.39	81.0 ± 64.27	94.4 ± 57.89	58.7 ± 53.26	57.0 ± 25.16	1.00 (0.99;1.00)	0.424
Tremor							
No	22	19	30	15	11	0.80 (0.52;1.23)	0.333
Yes	41	61	37	35	12		

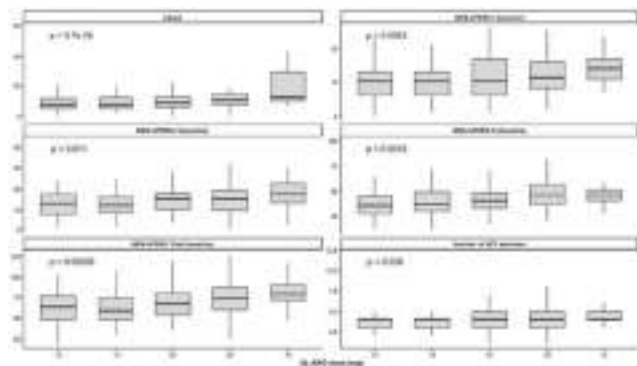


FIGURE 1 Baseline demographic and clinical characteristics associated with the final titrated dose of sublingual apomorphine.

Methods: Baseline characteristics from the CTH-301 dose-optimisation phase were examined across five SL-APO doses (10–30 mg) from de novo patients. Associations between variables and SL-APO dose were evaluated using univariate ordinal logistic regression. Odds ratios with 95% confidence intervals were calculated for continuous and categorical variables, and statistical significance was set at $p < 0.05$.

Results: Several baseline factors showed significant associations with higher SL-APO doses. Patients with greater motor severity, reflected in higher MDS-UPDRS Part I, II, III, and Total scores (all $p < 0.001$), were more likely to titrate to higher doses. A higher total daily levodopa dose was also significantly associated with higher SL-APO doses ($p < 0.001$). In addition, the presence of delayed ON ($p = 0.018$) and a greater number of daily OFF episodes ($p = 0.005$) were associated with requiring higher doses. No significant associations were observed for age, sex, BMI, Hoehn&Yahr stage, cognitive status, PD duration, motor fluctuations duration, tremor, wearing-off, morning akinesia, sudden OFF, or duration of OFF episodes.

Conclusion: Patients who required higher SL-APO doses presented with more severe baseline motor symptoms, higher levodopa requirements, delayed ON, and more frequent OFF episodes. These findings indicate that individuals with a greater motor burden and more complex fluctuation profiles may require higher SL-APO doses, providing useful guidance for clinicians during dose titration to achieve an optimal therapeutic effect.

Disclosure: Supported by Bial-Portela & Ca, S.A.

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Background and aims: Opicapone is used to manage motor fluctuations in Parkinson's patients, and its earlier initiation at wearing-off onset demonstrated benefits in OFF- and Good-ON-time without increasing dyskinesia over 1 year. This post-hoc analysis evaluated opicapone's long-term safety and tolerability in Parkinson's patients with early fluctuations.

Methods: Data were pooled from double-blind, placebo-controlled BIPARK I and II studies (3.5 months) and their 1-year open-label extensions. Analyses included participants randomized to placebo or opicapone 50mg who continued opicapone during the open-label phase and had developed wearing-off within ≤2years prior to screening. Treatment-emergent adverse

TABLE 1 Safety and tolerability during the double-blind phase (14–15 weeks).

	Opicapone 50mg n=118	Placebo n=110
All TEAEs, n (%)	66 (55.9)	57 (51.8)
Mild	58 (48.2)	46 (41.8)
Moderate	30 (25.4)	16 (14.5)
Severe	4 (3.4)	4 (3.6)
Serious	3 (2.5)	2 (1.8)
All related TEAEs, n (%)	39 (33.0)	22 (20.0)
Serious	0 (0)	2 (1.8)
Related TEAEs occurring in ≥2% in at least one arm, n (%)		
Dyskinesia	11 (9.3)	4 (3.6)
Insomnia	4 (3.4)	0 (0)
Dry mouth	3 (2.5)	2 (1.8)
Blood CPK increased	3 (2.5)	0 (0)
Nausea	2 (1.7)	3 (2.7)
Headache	2 (1.7)	2 (1.8)
Somnolence	2 (1.7)	2 (1.8)
Constipation	2 (1.7)	0 (0)
Dysgeusia	2 (1.7)	0 (0)
Hypertension	2 (1.7)	0 (0)
Weight decreased	2 (1.7)	0 (0)
Dizziness	1 (0.8)	2 (1.8)
Hyperhidrosis	0 (0)	2 (1.8)

CPK, creatine phosphokinase; TEAE, Treatment-emergent adverse events

TABLE 2 Safety and tolerability during the open-label extension phase (1 year).

	Opicapone 50mg- Opicapone n=118	Placebo- Opicapone n=110
All TEAEs, n (%)	86 (72.9)	77 (70.0)
Mild	69 (58.5)	69 (62.7)
Moderate	51 (43.2)	42 (38.2)
Severe	13 (11.0)	12 (10.9)
Serious	11 (9.3)	10 (9.1)
Leading to discontinuation	4 (3.4)	0 (0.0)
All related TEAEs, n (%)	47 (39.8)	39 (35.5)
Serious	3 (2.5)	0 (0)
Leading to discontinuation	3 (2.5)	0 (0)
Related TEAEs occurring in ≥2% in at least one arm, n (%)		
Dyskinesia	10 (8.5)	15 (13.6)
Parkinson's Disease	3 (2.5)	5 (4.5)
Blood CPK increased	5 (4.2)	4 (3.6)
Insomnia	2 (1.7)	4 (3.6)
Nausea	2 (1.7)	4 (3.6)
Dry mouth	1 (0.8)	3 (2.7)
Nightmare	1 (0.8)	3 (2.7)
Weight decreased	3 (2.5)	3 (2.7)
Drug effect decreased	5 (4.2)	2 (1.8)
Hallucination, visual	3 (2.5)	2 (1.8)
Tremor	3 (2.5)	2 (1.8)
Hypertension	3 (2.5)	1 (0.9)
Deaths, n (%)	0 (0)	0 (0)

CPK, creatine phosphokinase; TEAE, Treatment-emergent adverse events

events (TEAEs), TEAEs considered related to treatment, and TEAEs leading to discontinuation were evaluated.

Results: Safety Set included 218 patients with recent-onset wearing-off (placebo, $n=110$; opicapone, $n=118$). During the double-blind phase, TEAEs occurred in 55.9% of opicapone-treated and 51.8% of placebo-treated patients, with treatment-related TEAEs being reported in 30.5% and 20.0%, respectively (Table 1). Most frequent treatment-related TEAEs with opicapone were dyskinesia (9.3%) and insomnia (3.4%). In the 1-year open-label extension, treatment-related TEAEs occurred in 39.8% of patients continuing opicapone and 35.5% of those switching from placebo, most commonly dyskinesia (8.5% and 13.6%, respectively) (Table 2). Treatment-related TEAEs leading to discontinuation were infrequent (2.5% and 0.0%, respectively); no deaths were reported.

Conclusion: Long-term opicapone exposure was generally safe and well-tolerated in patients with recent-onset fluctuations. Most TEAEs were mild/moderate and consistent with dopaminergic therapy. Dyskinesia occurred less frequently in early versus later starters during the open-label phase, supporting opicapone's favorable safety profile when initiated at wearing-off onset.

Disclosure: Supported by Bial.

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Background and aims: Parkinsonism, particularly Parkinson's disease (PD), is a progressive neurodegenerative disorder with motor and non-motor symptoms that negatively affect quality of life and increase caregiver burden. Neuropalliative care has recently emerged for chronic neurological disorders, including PD. This study evaluated awareness and perceptions of palliative care among patients with parkinsonism, their caregivers, and physicians in Korea, and identified factors associated with caregiver burden.

Methods: A multicenter, cross-sectional study was conducted with 112 patients, 110 caregivers, and 61 physicians. Structured questionnaires assessed awareness and attitudes toward neuropalliative care. Caregiver burden was measured using validated Korean version of the Parkinson's Disease Questionnaire-Carer (PDQ-Carer).

Results: Awareness of palliative care differed markedly: 31.3% of patients and 27.5% of caregivers versus 90.2% of physicians. Caregivers were stratified by PDQ-Carer score into low- (≤ 34 , $n = 63$) and high-burden (> 34 , $n = 47$) groups. High-burden caregivers supported patients with longer disease duration, poorer cognition, higher Hoehn and Yahr stage, and greater levodopa equivalent daily doses (LEDD). Regression analysis showed that caregiver demographics were not associated with burden, whereas caregiving duration, patient cognitive decline, Hoehn and Yahr stage, LEDD, and disease duration were correlated. In the multivariable model, only patient disease duration remained significant ($B = 2.048$, 95% CI 0.083–4.012, $p = 0.041$).

Conclusion: There is a significant awareness gap in palliative care between physicians and patients/caregivers. The caregiver burden was mainly linked to disease duration as the strongest predictor. Education and structured neuropalliative strategies are needed to support patients with PD and their families

Disclosure: The authors declare no conflicts of interest.

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Background and aims: Subthalamic deep brain stimulation elicits short- and long-latency cortical responses reflecting activation of hyperdirect and polysynaptic basal ganglia–thalamo–cortical pathways (Miocinovic et al, 2018). While dopaminergic medication alters network synchrony and basal ganglia coupling (Kühn et al, 2008; Tinkhauser et al, 2017), its influence on DBS-evoked cortical responses remains unclear. Human EEG and primate studies suggest a medication-related dampening of late cortical components (Eusebio et al, 2009; Campbell et al, 2022). However, systematic MEG studies examining the influence of dopaminergic state on DBS-evoked cortical activity in Parkinson's disease are lacking. The present study aimed to determine whether dopaminergic state modulates the amplitude, latency, or spatial distribution of STN-DBS-evoked cortical responses.

Methods: Nineteen patients with Parkinson's disease and STN-DBS underwent MEG recordings during low-frequency (6 Hz) stimulation of the left STN. Stimulation was applied omnidirectionally at the clinically effective contact using the individual therapeutic amplitude and a pulse width of 60 μ s. Each patient was recorded in medication ON and OFF states. Source activity was reconstructed using linearly constrained minimum variance beamforming, focusing on the primary motor cortex and supplementary motor area. Peak amplitudes and latencies were compared between medication states using paired two-tailed t-tests.

Results: DBS evoked consistent early (~6 ms), mid (~13 ms) and long-latency (~21 ms) cortical responses in both conditions. No significant differences in response amplitude, latency, or cortical topography were observed between medication states.

Conclusion: DBS-evoked cortical responses were robust and reproducible in both medication states, suggesting that DBS programming based on evoked cortical responses is feasible without medication withdrawal.

Disclosure: Nothing to disclose.

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Background and aims: Continuous subcutaneous infusion of foslevodopa/foscarbidopa (CSFI) is an effective therapy for advanced Parkinson's disease (PD). However, the role of concomitant opicapone (OPC) during CSFI has not been systematically assessed. This study evaluated the impact of OPC co-administration on clinical outcomes, dopaminergic requirements, and safety.

Methods: We performed a single-center, retrospective study including 59 patients with advanced PD treated with CSFI.

Motor outcomes (MDS-UPDRS-III and IV), infusion parameters, levodopa equivalent daily dose (LEDD), and adverse events were compared between patients treated with and without concomitant OPC.

Results: The cohort comprised 36 men and 23 women (mean age 66.7 years; mean disease duration 14.5 years). Twenty-nine patients received OPC (24 continued from baseline, 5 initiated de novo). Treatment retention was high; only two patients discontinued OPC for non-safety reasons, with one requiring prompt reintroduction due to severe OFF worsening. Pre-infusion LEDD was significantly higher in the OPC group. At 8 months, both groups showed comparable motor improvement with identical infusion flow rates (0.28 mL/h). Mean improvement in MDS-UPDRS-III was similar between groups, while a trend toward greater improvement in UPDRS-IV was observed in OPC-treated patients. Total LEDD remained higher in the OPC group at follow-up. Neuropsychiatric adverse events were more frequent in OPC-treated patients, though differences were not statistically significant.

Conclusion: OPC appears to be a feasible adjunct during CSF1, supporting higher dopaminergic requirements without increasing infusion flow rates. While generally well tolerated, careful monitoring for neuropsychiatric adverse events is recommended.

Disclosure: G Lazzeri has received speaker honoraria by Zambon, abbvie, Everpharma IU Isaías is a Newronika S.p.A. consultant and shareholder and received funding for research activities from Newronika S.p.A. IUI received lecture honoraria and research funding from Medtronic Inc. IUI is Adjunct Professor at the Department of Neurology, NYU Grossman School of Medicine. The other authors declare no conflict of interest.

EPO-0615 | Wearable sensors may detect subtle gait alterations associated with autonomic dysfunction in drug-naïve Parkinson's disease patients

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Background and aims: Gait and balance disorders significantly affect functional activities of daily life in patients with Parkinson's disease (PD). Autonomic dysfunction has been associated with increased risk of falls in patients with advanced PD. Recent studies suggest that earlier development of dysautonomia is associated with more rapid disease progression and shorter survival time. We aimed at investigating potential correlations between gait and autonomic dysfunction in early drug-naïve PD patients using wearable sensors.

Methods: Fifty drug-naïve PD patients (disease duration ≤ 3 years, Hoehn & Yahr ≤ 2.5) and 20 age and sex-matched healthy controls were consecutively enrolled. Gait and balance parameters were acquired using six Opal V2R wearable sensors, when performing the Timed up and go, 7 and 10 Meters walking tests in single and dual task (ST/DT) conditions. Motor and autonomic symptoms were evaluated using the Unified Parkinson's

disease Rating scale part III (UPDRS-III) and the Scales for Outcomes in Parkinson's Disease - Autonomic Dysfunction (SCOPA-AUT). Bivariate correlation analysis between clinical and wearable sensors metrics were performed.

Results: Higher SCOPA-AUT scores significantly correlated with worse gait and postural performances as detected by wearable sensors in both ST and DT conditions. Interestingly, no correlations have been found between SCOPA-AUT and UPDRS III, including axial and gait subscores.

Conclusion: Our findings revealed that application of wearable sensors could provide useful information for the characterization of drug-naïve PD patients, even in the absence of clinically detectable gait and postural disturbances. These subtle changes are already associated with autonomic dysfunctions, suggesting a potential role to predict worse clinical outcome.

Disclosure: Nothing to disclose.

EPO-0616 | Short- and long-term effects of continuous dopaminergic infusion on cortical excitability and plasticity in Parkinson's disease.

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Background and aims: Continuous subcutaneous infusion of foslevodopa/foscarbidopa has been recently developed for patients with Parkinson's disease (PD) experiencing motor complications inadequately controlled by oral dopaminergic therapy. The pathophysiology of motor complications involves abnormally reduced plasticity of the primary motor cortex (M1) in PD. Once motor complications emerge, intermittent dopaminergic stimulation fails to adequately influence motor cortical plasticity. The aim of the present study was to evaluate whether continuous dopaminergic stimulation can modulate cortical plasticity in patients with PD.

Methods: Fifteen patients (7 female, age 67 ± 6.41) with complicated PD receiving oral L-dopa were consecutively enrolled. Clinical evaluation included standardized scales assessing motor and non-motor manifestations. Neurophysiological evaluation of M1 plasticity was performed using intermittent theta-burst stimulation (iTBS), transcranial magnetic stimulation paradigm. The after-effects of iTBS were evaluated by measuring motor evoked potential amplitudes recorded before and at 5, 15, and 30 minutes after stimulation. Patients were assessed while still receiving oral therapy (T0), immediately after initiation of foslevodopa/foscarbidopa infusion (T1), and after 24 weeks of continuous treatment (T2).

Results: Foslevodopa/foscarbidopa treatment was associated with a significant improvement in motor and non-motor manifestations, including a reduction in the severity of motor complications. At T2, foslevodopa/foscarbidopa treatment induced an improvement in abnormally reduced M1 plasticity in patients with PD, as evidenced by an increased response to the iTBS protocol compared with baseline (T0).

Conclusion: In contrast to intermittent dopaminergic stimulation, continuous dopaminergic stimulation induced by

foslevodopa/foscarbidopa can modulate M1 plasticity in patients with PD. This mechanism may contribute to the clinical effects of this therapeutic approach.

Disclosure: Nothing to disclose.

EPO-0617 | Plasma NfL, GFAP and P-Tau181 as stratifiers of alpha-synucleinopathies, from prodromal to early stages

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Background and aims: plasma markers have recently been implemented in neurodegenerative disorders as they might contribute to stratify subjects at different risk for Parkinson Disease (PD) or Lewy Body Disease (DLB). Rem Sleep Behaviour disorders (RBD) patients seem to be biologically closer to DLB rather than PD. Aim of the study was to evaluate the distribution of NfL, GFAP and pTau 181 in PD, DLB and subjects with different prodromal risk factors for alpha-synucleinopathies

Methods: early PD, DLB, as well as subjects with idiopathic Sleep behavioral disorders or isolated hyposmia and matched controls RBD and 136 Healthy Controls (HC), underwent an extensive PD prodromal marker evaluation and plasma markers assessment using Single molecule array (Simoa) technology. Cut-off values were calculated based on 1 standard deviation of healthy controls.

Results: The study included 559 individuals, 108 DLB, 206 PD, 91 idiopathic RBD and 136 Healthy Controls (HC) of which 18 hyposmic, DLB globally showed higher levels of GFAP and a significant increase of pTau181 when compared to PD. iRBD exhibited higher NfL and GFAP levels compared to HC and PD, and a lower score at sniffing test suggesting that hyposmia can be detected together with RBD. GFAP levels seemed to be higher in subjects at risk for alpha synucleinopathies.

Conclusion: Plasma neurodegenerative markers stratify people at risk for alpha-synucleinopathies and are higher in DLB patients. In prodromal cases, the increase of GFAP correlates with clinical risk of conversion. Future goal is identifying which prodromic symptoms correlate the more with a biomarker increase.

Disclosure: no disclosures

EPO-0618 | Results from the Global Phase 3 Trial (IB1001-303) Evaluating Levacetylleucine in Ataxia-Telangiectasia

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Background and aims: Ataxia-Telangiectasia (A-T) is a rare, inherited cerebellar ataxia. Levacetylleucine (also known as N-acetyl-L-leucine) is a modified amino acid that enters enzyme-controlled pathways to correct metabolic dysfunction and restore cellular communication. In this Phase 3 randomised, double-blind, placebo-controlled trial (IB1001-303), we evaluated the safety and efficacy of levacetylleucine for pediatric and adult individuals with A-T.

Methods: IB1001-303 enrolled A-T participants aged 4 years or older across 10 multinational trial sites. Primary efficacy endpoint was the Scale for the Assessment and Rating of Ataxia (SARA). Secondary endpoints included Spinocerebellar Ataxia Function Index (SCAFI), International Cooperative Ataxia Rating Scale (ICARS), Neurology Quality of Life-Upper Extremity Function (NeuroQOL-UEF), Quality of Life (EQ-5D), and investigator, caregiver, and patient Clinical Global Impression of Improvement (CGI-I). Safety assessment included adverse event incidence and severity.

Results: Seventy-three participants aged 5 to 50 were enrolled. The primary endpoint was met; mean change in the SARA total score with levacetylleucine was -1.92 (SD=2.81) and -0.14 (SD=2.38) with placebo; Linear Mixed Model (LMM) treatment effect -1.88 (SD=0.41) [95% CI -2.70, -1.06; $p < 0.0001$]. The trial met secondary endpoints, including ICARS and CGI-I. NALL was observed to be safe and well-tolerated, consistent with its established safety profile.

Conclusion: Levacetylleucine demonstrated a significant benefit versus placebo, and clinically meaningful improvements in neurological manifestations of A-T, functioning, and quality of life, and a favorable benefit-risk profile for the treatment of A-T.

Disclosure: MCP, BZ, JR, JK, AH, TF and IB are employees of and may have stock option ownership in IntraBio, Inc. MS received speaker's honoraria from Abbott, Actelion, Auris Medical, Biogen, Eisai, Grünenthal, GSK, Henning Pharma, Interacoustics, MSD, Otometrics, Pierre-Fabre, TEVA, UCB, Viatrix. Consultant for Abbott, Actelion, Auris Medical, Decibel, Heel, and Sensorion. Scientific founder and shareholder of IntraBio. TB received speaker's honoraria and consultancy fees from Actelion, Sanofi-Genzyme and Zevra as well as blinded video-rater fees from IntraBio.

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Background and aims: Progressive supranuclear palsy (PSP) is a rare neurodegenerative disorder characterized by tau pathology and rapid clinical progression. Reliable cerebrospinal fluid (CSF) biomarkers reflecting disease-specific molecular mechanisms are still lacking. Proteomic analysis of CSF may provide insight into pathophysiological processes and identify potential biomarkers for diagnosis and disease monitoring.

Methods: In this single-center study, CSF samples from 24 PSP patients and 12 neurologically healthy age-matched controls were analyzed. Samples were collected using standardized procedures. High-resolution mass spectrometry-based proteomics was performed, enabling the identification and quantification of approximately 2,500 CSF proteins. Differential expression analyses were conducted to compare PSP subgroup with controls, followed by functional and pathway enrichment analyses.

Results: Proteomic profiling revealed distinct CSF protein signatures in PSP patients compared with controls. Differentially expressed proteins were mainly associated with neurodegeneration-related pathways, including synaptic dysfunction, neuroinflammation, cytoskeletal organization, and protein aggregation. Several proteins involved in tau-related processes and glial activation showed altered abundance in PSP. Exploratory analyses suggested potential protein panels that may discriminate PSP from controls.

Conclusion: CSF proteomics identified disease-related molecular alterations in PSP and highlighted biological pathways relevant to its pathogenesis. These findings support the utility of CSF proteomic profiling as a tool for biomarker discovery in PSP. Further studies in larger, multicenter cohorts are warranted to validate candidate biomarkers and assess their clinical applicability.

Disclosure: Nothing to disclose.

EPO-0620 | Individualisation of LECIG therapy in advanced Parkinson's disease: Adapting treatment to suit patients' lives

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Background and aims: Advanced Parkinson's disease (PD) is a heterogeneous condition requiring an individualised treatment approach. Levodopa–entacapone–carbidopa intestinal gel (LECIG) is a device-aided therapy for advanced PD designed to

TABLE 1 Characteristics and outcomes of patients treated with LECIG at our centre.

Parameter	Patient 1	Patient 2	Patient 3
Gender	Female	Female	Male
Age, years	51	76	75
Duration of T2E, years	20	30	18
Exponent value to (T2E)	0.26, 16 hours/day Morning sleep: 1.0 min Continuous sleep: 3.0 min/day Catheterless: 2.5 d	1.04, 24 hours/day Morning sleep: 1.0 min Sleep: 2.1 min while driving the truck Continuous sleep: 2.0 min/day Sleep device: 2.0 min Pump 2.1 min while asleep Continuous sleep: 2.3 min/day Sleep device: 2.3 min	1.00, 26 hours/day Morning sleep: 1.0 min Sleep: 5.5 min while driving the truck Continuous sleep: 0.9 min/day Sleep device: 0.3 min Pump 2.1 min while asleep Continuous sleep: 5.0 min/day Sleep device: 0.3 min
Urethral leak, authorized	The patient used a catheterically active and a Prolapsed Tissue Tissue Pump	She is intubated and leads a spinal device	He is intubated and leads a spinal device
Urethral leaking device (Urethral catheterization)	4.3 ml Flow rate: 1.77 min/day (at night) Flow rate: 2.1 min/day Flow rate: 2.7 min/day	1.8 ml Flow rate: 1.1 min/day Flow rate: 2.2 min/day Flow rate: 2.1 min/day (at night) Flow rate: 2.2 min/day (at night)	2.8 ml Flow rate: 1.2 min/day Flow rate: 2.2 min/day Flow rate: 2.4 min/day (at night) Flow rate: 2.4 min/day (at night)
Duration of catheterization	13 hours	14 hours	14 hours
Catheter device	1.0 ml	2.1 ml	3.0 ml
Urethral, bladder, pelvic catheter	Flow rate can be adapted to daily needs	Urethral, bladder, and pelvic catheter (catheterization)	Urethral, bladder, and pelvic catheter (catheterization)

LOS, levodopa-carbidopa-methyl dopa; UETC, levodopa-entacapone-carbidopa methyl dopa.

allow flexible dosing, enabling adaptation to patients' specific and fluctuating motor needs throughout the day.

Methods: We describe clinical cases from our centre illustrating how LECIG therapy, administered via the Crono® LECIG pump, can be adjusted to achieve optimal motor control over a 24-hour period and across different levels of daily activity (Table 1).

Results: (1) A 51-year-old woman with PD from age 35, treated with levodopa-carbidopa intestinal gel (LCIG) infusion since 2015; switched to LECIG in 2023 due to disabling dyskinesias and evening motor worsening. She now receives 24-hour LECIG infusion with three adjustable flow rates tailored to daily demands. (2) A 76-year-old woman with PD since age 44, treated with LCIG since 2011; switched to LECIG in 2025 due to evening motor deterioration and technical difficulties managing two pumps required for 24-hour LCIG infusion. She currently uses LECIG at three flow rates, with marked motor improvement and better caregiver quality of life. (3) A 75-year-old man with PD since 2011, treated with high-dose 24-hour LCIG infusion since 2020; switched to LECIG due to unpredictable and uncontrollable OFF periods and now shows marked clinical improvement using three flow rates.

Conclusion: The use of multiple flow rates with the Crono® LECIG pump allows individualised medication delivery aligned with daily activities and symptom fluctuations, supporting functional independence and an active lifestyle.

Disclosure: NS is an employees of Britannia Pharmaceuticals Ltd. Other authors have nothing to disclose.

MS and related disorders 5

EPO-0621 | Shifting paradigms in multiple sclerosis: Long-term outcomes across diagnostic eras

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Background and aims: Advances in diagnostic criteria, neuroimaging, and disease-modifying therapies have substantially changed the clinical course of multiple sclerosis (MS). This study compared long-term outcomes of patients diagnosed with MS in two different diagnostic eras to assess the real-world impact of evolving clinical practice.

Methods: In this retrospective cohort study, patients diagnosed with MS in two eras were analyzed: Group 1 (1990–1996, $n=133$) and Group 2 (2008–2014, $n=579$). Demographic characteristics, time from symptom onset to diagnosis, Expanded Disability Status Scale (EDSS) scores at diagnosis and at 10-year follow-up, annualized relapse rates (ARR), and conversion to secondary progressive MS (SPMS) were recorded.

Results: A total of 712 patients were included (mean age: 50.7 ± 12.2 years; mean disease duration: 20.7 ± 8.8 years; 70.4% female). Time from symptom onset to diagnosis was significantly shorter in Group 2 compared with Group 1 (3.2 ± 4.6 vs 4.2 ± 4.7 years; $p < 0.005$). After 10 years, disability outcomes remained significantly better in Group 2 (mean EDSS: 1.85 ± 2.02) compared with Group 1 (3.97 ± 2.23 ; $p < 0.001$). ARR was also lower in Group 2 (0.18 ± 0.20 vs 0.23 ± 0.20 ; $p < 0.05$). Conversion to SPMS occurred in 52.6% of Group 1 patients versus only 5.4% of Group 2.

Conclusion: Patients diagnosed in the more recent era demonstrated markedly improved long-term disability outcomes, lower relapse activity, and substantially reduced progression to SPMS, underscoring the positive impact of advances in MS diagnosis and treatment.

Disclosure: Nothing to disclose.

EPO-0622 | Serum IgM Index as a Biomarker of Motor and Cognitive Performance in Multiple Sclerosis

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Background and aims: The immunoglobulin M (IgM) index, reflecting intrathecal IgM synthesis, has been proposed as a biomarker of disease activity in multiple sclerosis (MS). This study examined functional differences between MS patients with persistently elevated IgM index values and those with normal IgM index.

Methods: This retrospective observational study included MS patients with elevated IgM index values and long-term clinical follow-up. A control group with normal IgM index was selected from a larger cohort of 4,191 MS patients and matched for disease duration and baseline Expanded Disability Status Scale (EDSS). Motor performance was assessed using the Timed 25-Foot Walk (T25FW) and Nine-Hole Peg Test (9HPT). Cognitive function was evaluated with the Symbol Digit Modalities Test (SDMT), Brief Visuospatial Memory Test-Revised (BVM-T-R), and California Verbal Learning Test-II (CVLT-II).

Results: A total of 268 patients with high IgM index and 150 patients with normal IgM index were analyzed. The high IgM index group demonstrated significantly worse motor performance on the T25FW ($p=0.040$) and 9HPT ($p=0.001$). In cognitive assessments, patients with normal IgM index achieved higher scores on the SDMT ($p=0.022$) and BVM-T-R ($p=0.011$), indicating better processing speed and visuospatial memory. No significant difference was observed in CVLT-II scores ($p=0.173$).

Conclusion: MS patients with elevated IgM index showed poorer motor performance and selective cognitive impairment compared with those with normal IgM index. These findings support the IgM index as a potential prognostic biomarker and

emphasize the value of integrating laboratory markers into long-term functional evaluation in MS.

Disclosure: Nothing to disclose.

EPO-0623 | Spinal Cord Lesions and Atrophy Predict Disability Progression and PIRA in Multiple Sclerosis

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Background and aims: Spinal cord pathology is a major contributor to long-term disability in multiple sclerosis (MS). This study aimed to evaluate spinal cord lesion patterns and spinal cord atrophy and to determine their association with long-term disability progression, progression independent of relapse activity (PIRA), and conversion to SPMS.

Methods: We retrospectively analyzed 223 patients with MS followed for at least three years. Spinal cord MRI was assessed for lesion presence, level, axial distribution, number, length, gadolinium enhancement, and spinal cord atrophy. Outcomes included confirmed disability accumulation, attainment of EDSS ≥ 3 , PIRA, and SPMS conversion. Kaplan–Meier analyses and multivariate Cox regression models were applied.

Results: Spinal cord lesions were present in 172 patients (77.1%) and were associated with higher disability, increased risk of EDSS ≥ 3 , PIRA, and conversion to SPMS. Lesion-specific characteristics did not independently predict long-term outcomes. In contrast, spinal cord atrophy independently predicted EDSS ≥ 3 (HR 1.98) and PIRA (HR 2.29). Despite similar baseline disability, patients with spinal cord lesions showed progressive divergence in disability over time.

Conclusion: Spinal cord atrophy is a strong, lesion-independent prognostic marker of disability progression and PIRA in multiple

TABLE 1 Kaplan-Meier survival and log-rank analyses for time to EDSS ≥ 3 and PIRA according to spinal cord lesion status.

A. Survival summary						
Outcome / Group	n	Events (n, %)	Censored (n, %)	Median time (years)	5-year survival	10-year survival
EDSS ≥ 3 – Lesion present	172	99 (57.5%)	113 (65.7%)	13.7 $\pm$ 1.1 (95% CI 13.9–22.2)	0.804 $\pm$ 0.027	0.735 $\pm$ 0.043
EDSS ≥ 3 – Lesion absent	51	5 (9.8%)	46 (90.2%)	Not reached	—	—
PIRA – Lesion present	172	39 (22.7%)	133 (77.3%)	23 $\pm$ 1.4 (95% CI 20.3–25.8)	0.837 $\pm$ 0.023	0.847 $\pm$ 0.018
PIRA – Lesion absent	51	3 (5.9%)	48 (94.1%)	Not reached	—	—
B. Log-rank comparison by baseline variables – EDSS ≥ 3						
Variable	Comparison	p (Log-rank)	Interpretation			
Sex	Male vs Female	0.36	No significant difference			
Smoking	Yes vs No	0.48	No significant difference			
Onset type	PPMS / ACMS / LOMS	0.048	LOMS patients progressed earlier			
Baseline EDSS	<3 vs ≥ 3	<0.001	Strongly predict faster disability			
Cord atrophy	Present vs Absent	0.009	Atrophy linked to earlier EDSS ≥ 3			
Spinal Cd enhancing lesion	Present vs Absent	0.32	Trend toward higher risk			
Brainstem lesion	Present vs Absent	0.81	ns			
OCB positivity	Positive 2/3 vs Negative	0.64	ns			
IgG index	<0.7 vs ≥ 0.7	0.38	ns			
Lesion length	Short vs Long/Extensive	0.19	ns			
Axial distribution	Peripheral vs Central	0.58	ns			
Thalamic lesions	Present vs Absent	0.13	ns			

Abbreviations: CI, confidence interval; EDSS, Expanded Disability Status Scale; Cd, gadolinium; IgG, immunoglobulin G; ns, not significant; OCB, oligoclonal bands; PIRA, progression independent of relapse activity.

TABLE 2 Cox proportional hazards models for predictors of EDSS ≥ 3 and PIRA among patients with spinal cord lesions ($n = 172$).

Outcome / Predictor	β (SE)	p	HR (Exp β)	95% CI for HR	Interpretation
EDSS ≥ 3					
Baseline EDSS ≥ 2	1.12 (0.28)	<0.001	3.07	1.63–5.23	3+ higher risk of disability
Cord atrophy (present)	0.88 (0.27)	0.002	1.96	1.16–3.37	2+ higher risk
GL-enhancing lesion (present)	0.63 (0.37)	0.072	1.88	0.95–3.71	Trend toward higher risk
Model fit: $\chi^2 = 23.26$, $p < 0.001$					
PIRA					
Baseline EDSS ≥ 2	0.906 (0.351)	0.007	2.46	1.29–4.71	2.5+ higher risk of PIRA
Cord atrophy (present)	0.626 (0.336)	0.062	2.29	1.20–4.37	2+ higher risk of PIRA
GL-enhancing lesion (present)	-0.347 (0.356)	0.33	0.71	0.35–1.42	no (net) effect
Model fit: $\chi^2 = 13.17$, $p = 0.004$					

Abbreviations: CI, confidence interval; EDSS, Expanded Disability Status Scale; GL, gadolinium; HR, hazard ratio; PIRA, progression independent of relapse activity; SE, standard error.



FIGURE 1 Graphical abstract.

sclerosis. While lesion presence identifies patients at risk, lesion morphology has limited prognostic value, highlighting the importance of quantitative spinal cord assessment in longitudinal follow-up.

Disclosure: Nothing to disclose.

EPO-0624 | Prevalence and clinical correlates of sexual dysfunction in multiple sclerosis: A single-center cross-sectional study

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Background and aims: Sexual dysfunction (SD) is a common but under-recognized complication of multiple sclerosis (MS) that substantially affects patients' quality of life. Although the etiology of SD in MS is multifactorial, reported prevalence and associated clinical factors vary widely across studies.
Methods: We conducted a single-center cross-sectional observational study including MS patients followed at our Neuroimmunology-MS Unit between June and October 2025. Sexual function and satisfaction were assessed using self-administered questionnaires: the Multiple Sclerosis Intimacy and Sexuality Questionnaire (MSISQ-15), the New Sexual Satisfaction Scale (NSSS), the Female Sexual Function Index (FSFI), and the International Index of Erectile Function (IIEF). Demographic and clinical variables were collected. Bivariate analyses and multivariable logistic regression were performed.

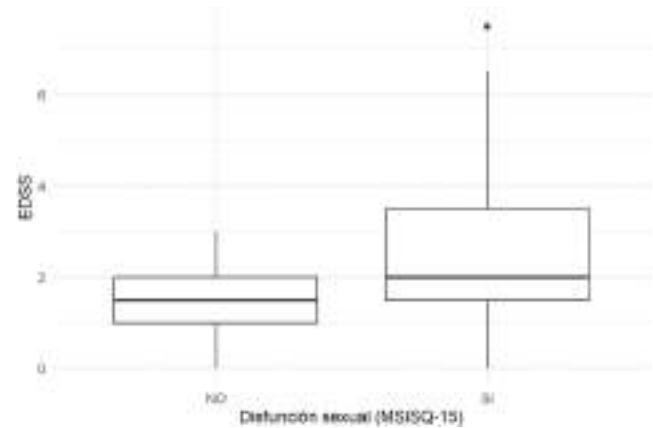


FIGURE 1 Neurological disability according to sexual dysfunction status.

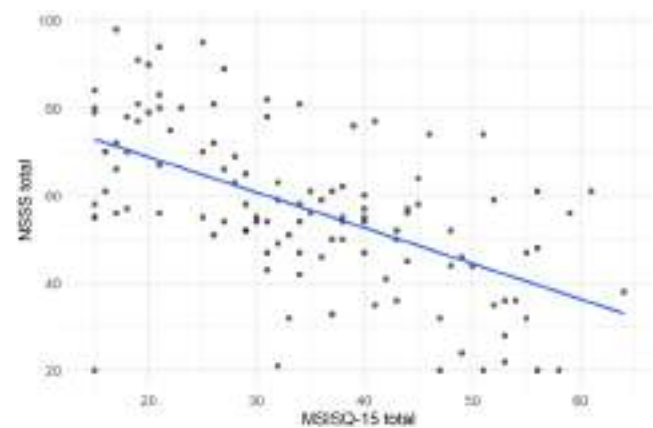


FIGURE 2 Correlation between sexual dysfunction severity and sexual satisfaction.

Correlations were assessed using Spearman's rank correlation coefficient.

Results: 115 patients were included of whom 69 (60.5%) were female, with a mean age of 46, and with 90 (78.9%) being relapsing-remitting forms. A total of 100 (87.0%) patients showed SD based on MSISQ-15 score. Patients with SD had greater neurological disability than those without SD (median EDSS 2.0 vs 1.5, $p = 0.018$). In multivariable analysis, EDSS remained independently associated with SD (OR 2.29, 95% CI 1.23–5.24). SD severity was strongly and inversely correlated with sexual satisfaction measured by the NSSS ($\rho = -0.57$, $p < 0.001$). In men, MSISQ-15 scores were strongly and negatively correlated with IIEF scores, whereas in women the correlation with FSFI scores did not reach statistical significance.

Conclusion: SD is highly prevalent in patients with MS and is strongly associated with neurological disability and reduced sexual satisfaction.

Disclosure: Nothing to disclose.

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Background and aims: Neuromyelitis optica spectrum disorder (NMOSD) and myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) predominantly affect women of reproductive age. This study aimed to assess pregnancy impact on disease activity

Methods: This retrospective study included informative pregnancies in women with NMOSD or MOGAD followed in the Oxford national service. Annualized relapse rates (ARR) were calculated in patients who had a pregnancy after disease onset.

Results: In MOGAD, 72 of 105 informative pregnancies occurred after disease onset. Mean ARR in the two years before pregnancy was 0.23 ± 0.30 and decreased significantly during pregnancy. Post-pregnancy ARR showed a numerical increase in the early months (0.39 ± 1.19 at 0–3 months), followed by a decline by 12 months, without significant differences compared with the pre-pregnancy period. Spontaneous miscarriage occurred in 5/72 pregnancies (6.9%). In NMOSD, 43 of 51 informative pregnancies occurred after disease onset. Mean ARR before pregnancy was 0.36 ± 0.47 . During pregnancy, ARR was significantly lower in the first and third trimesters, and similar to pre-pregnancy in the second trimester (0.37 ± 1.18). Post-pregnancy ARR showed a numerical increase at 0–3 months (0.74 ± 1.80) and declined thereafter, without significant differences compared with pre-pregnancy. Spontaneous miscarriage occurred in 10/43 pregnancies (23.2%). Post-pregnancy disease onset occurred in 31 MOGAD (4.62 ± 3.10 months) and 7 NMOSD (3.97 ± 3.43 months) patients.

Conclusion: Disease activity was significantly reduced during pregnancy in MOGAD. In both NMOSD and MOGAD, ARR showed a numerical increase in the early post-pregnancy period (0–3 months), followed by a decline over the first year.

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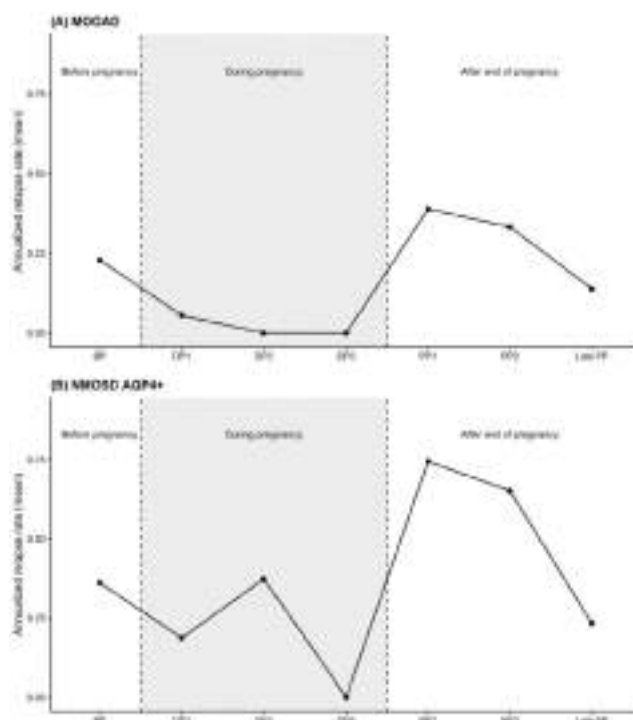


FIGURE 1 Annualized relapse rate across time periods in relation to pregnancy in (A) MOGAD and (B) NMOSD: before pregnancy (BP), during pregnancy by trimester (DP1, DP2, DP3), post-pregnancy 0–3 months (PP1), 3–6 months (PP2), and 6–12 months (late PP).

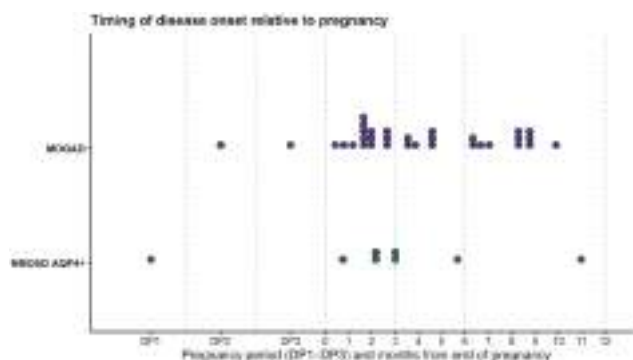


FIGURE 2 Timing of disease onset relative to pregnancy in NMOSD and MOGAD. Each dot represents one patient. Disease onset is shown during pregnancy by trimester (DP1–DP3) and after the end of pregnancy (in months).

Guthy-Jackson Foundation, a board member of the European Charcot Foundation, a member of MAGNIMS, the UK NHSE IVIG Committee, and has been an ECTRIMS Council member since June 2023. She currently serves on ABN advisory groups for MS and neuroinflammation and is a medical advisor to The Lancet Neurology editorial board. Maria Isabel Leite is funded by the NHS (Myasthenia & Related Disorders Service; National Specialised Commissioning Group for Neuromyelitis Optica, UK) and the University of Oxford. She has received research grants from Myaware, MDUK, and the University of Oxford. She has served on advisory boards for UCB Pharma, argenx, and Amgen, and has received speaker honoraria and travel grants from UCB Pharma, argenx, and Amgen. She has provided consultancy services for UCB Pharma. Patricia Faustino has

TABLE 1 Clinical and pregnancy characteristics of the study cohort: interim analysis. Values are presented as mean $\pm$ SD or *n* (%). Continuous variables were compared using the Wilcoxon rank-sum test, and categorical variables using Fisher's exact test.

	MOGAD (n = 67)	NMOGD (n = 29)	p-value
Age at disease onset (years), mean $\pm$ SD	26 $\pm$ 9	27 $\pm$ 8	0.382
Autoimmune comorbidities, n (%)	6 (9%)	12 (41%)	<0.001
Informative pregnancies, n	305	91	
Maternal age at pregnancy (years), mean $\pm$ SD	30.1 $\pm$ 5.1	32.1 $\pm$ 5.8	0.068
Advanced maternal age ($\geq$ 35 years), pregnancies (%)	10/105 (17.1%)	19/51 (37.3%)	0.009
Pregnancies after disease onset, n (patients)	72 (44 patients)	46 (25 patients)	
Disease onset during pregnancy, n (patients)	2/12 patients	1/11 patients	
Disease onset after end of pregnancy, n (patients)	31 (31 patients)	7 (7 patients)	
Time from end of pregnancy to disease onset among post-pregnancy-onset cases (months), mean $\pm$ SD	4.42 $\pm$ 2.93 (n = 31)	3.97 $\pm$ 3.43 (n = 7)	0.821
Termination during pregnancy, (toposed pregnancies) (%)	4/72 (5.5%)	24/93 (25.8%)	
Oral prednisolone alone	1	5	
Azathioprine alone	2	8	
Azathioprine + oral prednisolone	6	30	
Rituximab	1	6	
Rituximab + oral prednisolone	0	1	
Annualized relapse rate (ARR) by period relative to pregnancy			
2 years before pregnancy	0.21 $\pm$ 0.30 (reference)	0.26 $\pm$ 0.47 (reference)	
First trimester of pregnancy	0.86 $\pm$ 0.47 (p < 0.001)	0.59 $\pm$ 0.35 (p = 0.008)	
Second trimester of pregnancy	0.80 $\pm$ 0.80 (p < 0.001)	0.27 $\pm$ 1.10 (p = 0.188)	
Third trimester of pregnancy	0.80 $\pm$ 0.80 (p < 0.001)	0.80 $\pm$ 0.80 (p < 0.001)	
Post-pregnancy, 0–3 months	0.39 $\pm$ 1.19 (p = 0.115)	0.74 $\pm$ 1.80 (p = 0.725)	
Post-pregnancy, 3–6 months	0.31 $\pm$ 1.11 (p = 0.092)	0.63 $\pm$ 1.49 (p = 0.427)	
Post-pregnancy, 6–12 months	0.18 $\pm$ 0.61 (p = 0.005)	0.23 $\pm$ 0.65 (p = 0.175)	

received conference and travel support for scientific meetings and courses from Roche, Novartis, Biogen, BMS, and Merck. Anna Francis received a travel grant from Alexion in 2024. Fiona Chan, Yvonne Sharawakanda, and Madalina Miron: No disclosures to declare.

EPO-0626 | Language and voice analysis as multidimensional biomarkers of silent progression in relapsing–remitting multiple sclerosis

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Background and aims: Progression independent of relapse activity (PIRA) is a major contributor to disability accumulation in MS but is difficult to detect in patients with low EDSS scores. Language and speech impairments are common yet underrecognized in MS and may reflect early cognitive and motor network dysfunction not captured by conventional disability measures. This study investigated whether multidimensional language and voice assessments could serve as sensitive functional biomarkers of subclinical progression in relapsing–remitting MS (RRMS).

TABLE 1 Demographic and clinical characteristics of the study population.

	IRMS (n=28)	Healthy controls (n=6)	P
Age (mean $\pm$ SD), years	41 $\pm$ 13	35 $\pm$ 14	ns
Sex, female, n (%)	20 (71%)	5 (83%)	ns
Education (mean $\pm$ SD), years	18 $\pm$ 3	17 $\pm$ 3	> .0001
Age at onset (mean $\pm$ SD), years	34 $\pm$ 10	/	
Disease duration (mean $\pm$ SD), years	6.7 $\pm$ 8	/	
EDSS (mean $\pm$ SD)	1.7 $\pm$ 0.9	/	
MS-D (mean $\pm$ SD)	3.5 $\pm$ 2.7	2 $\pm$ 0.8	ns

RRMS = Relapsing–Remitting Multiple Sclerosis; EDSS = Expanded Disability Status Scale; VAS-D = Visual Analog Scale for Depression; ‡ Student's *t* test; * Chi-square test.

TABLE 2 Language assessment of the study population (adjusted for age and education).

	IBS45 (n=28)	Healthy controls (n=8)	p
Token Test (mean $\pm$ SD)	32.5 $\pm$ 1.5	32.3 $\pm$ 0.5	ns
Phonemic Fluency (mean $\pm$ SD)	37.0 $\pm$ 11.1	48.0 $\pm$ 9.8	0.009 §
Semantic Fluency (mean $\pm$ SD)	40.2 $\pm$ 8.9	53.8 $\pm$ 5.7	0.004 §
Dysarthria assessment – Total score (median, IQR)	169.5 (166–175)	176 (176–180.5)	0.031
Respiration	19 (19–20)	20 (19–20)	0.04
Phonation	46 (43.5–47)	47 (46–48)	ns
Facial musculature	80 (76–80)	80 (80–80)	ns
Orofacial/neck	38 (36–40.5)	43 (38.5–43.5)	0.02
Reflexes	18 (17–18)	18 (18–18)	0.02
Articulation	30 (28–30)	30 (28–30)	ns
Intelligibility	24 (22.5–24)	24 (23–24)	ns
Prosody	19.5 (19–20)	20 (20.5–20)	ns
VHI – Total score (median, IQR)	6 (3–8)	1.5 (0–6)	0.03 *
Functional impact of voice problems	7.5 (1–7)	1.5 (0–4.5)	ns
Psychological impact	1 (0–2)	0 (0–1.5)	ns
Self-perceived voice quality	4.5 (3–5.5)	0 (0–4)	0.002 *

BR22 = Relaying-Restling Multiple Sclerosis, YH = Yusef Handicap Index
 χ^2 Student's t test for normally distributed variables, ^{2} Mann-Whitney U test for non-normal distributions
 CR = cerebrospinal fluid

Methods: Twenty-eight patients with RRMS and mild disability and eight age- and sex-matched healthy controls underwent comprehensive cognitive, linguistic, and speech evaluations. Cognitive assessment included MoCA and domain-specific neuropsychological tests. Language was examined using verbal fluency and comprehension tasks. Speech and voice were evaluated with a standardized dysarthria profile, the Voice Handicap Index, and automated acoustic analysis using VoxPlot software. Acoustic parameters included Cepstral Peak Prominence Smoothed (CPPS), Acoustic Voice Quality Index (AVQI), and Acoustic Breathiness Index (ABI).

Results: Compared with controls, patients with RRMS showed reduced phonemic and semantic verbal fluency with preserved language comprehension. Mild but significant dysarthria was observed, mainly affecting respiration, diadochokinesis, and reflexes. Acoustic analysis revealed reduced CPPS and increased AVOI and ABI, indicating impaired voice quality. While several

measures correlated with EDSS and disease duration, multivariate analyses showed that acoustic and dysarthric parameters were largely independent of global disability, age, and disease duration.

Conclusion: Language and voice assessments detect subtle multidimensional dysfunction in RRMS not captured by conventional disability scales, supporting their potential role as biomarkers of silent progression and PIRA particularly in patients with low physical disability.

Disclosure: Nothing to disclose.

EPO-0627 | Clinical relevance of evoked potentials for disease progression in early multiple sclerosis and clinically isolated syndrome in the ProVal-MS study

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Background and aims: The individual clinical course of multiple sclerosis (MS) is highly variable. Prospective studies evaluating the clinical relevance of evoked potentials (EP) in correlation with clinical and paraclinical parameters are scarce. This study investigates the association of somatosensory (SEP), visual (VEP), and motor evoked potentials (MEP) at diagnosis for clinical and paraclinical endpoints after 24 months in the ProVal-MS study (DRKS00014034), a multicentre prospective study at five German universities.

Methods: Assessments included VEP, tibial nerve SEP, and abductor hallucis MEP, clinical assessments (Expanded Disability Status Scale [EDSS], Symbol Digit Modalities Test [SDMT]), cerebral and spinal MRI, serum neurofilament light chain [sNfL], and serum GFAP [sGFAP]. Statistical analyses included Spearman correlations and multilevel linear regression.

Results: 367 patients (CIS, $n=27$, RRMS, $n=340$; median age 30.6 years; 64.9% female) were included. Spearman analyses revealed significant correlations between all baseline EPs and both EDSS and SDMT at baseline and after 24 months. Multilevel linear regression showed higher (=worse) VEP scores associated with poorer cognition ($\beta=-4.44$, 95% CI -7.68, -1.21, $p=0.007$), while even mildly abnormal VEP scores were already associated with higher disability (VEP score 1: $\beta=0.18$, 95% CI 0.02, 0.33, $p=0.023$). However, SEP showed no significant associations with cognition or disability. Further results, including associations with blood biomarkers, neuroimaging findings, and clinical scores will be presented at the meeting.

Conclusion: Baseline EPs correlate with disability and cognitive function over 24 months. Multilevel regression highlights VEP as associated with both cognitive impairment and disability, supporting it as a complementary tool for MS monitoring.

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EPO-0628 | Automated analysis of longitudinal language changes in multiple sclerosis

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Background and aims: Sensitive and objective biomarkers for monitoring cognitive decline in multiple sclerosis (MS) are critically needed. Language is often impaired in MS and may reflect underlying cognitive changes. Automated analysis of spontaneous speech offers a promising, non-invasive approach to capture longitudinal disease-related alterations. This study investigates whether longitudinal speech analysis can detect language changes in MS.

Methods: We analyzed 213 participants, including 97 patients with MS and 116 age-, sex-, and education-matched healthy controls. All participants completed a story-telling task, which was automatically transcribed using automatic speech recognition system. MS patients underwent follow-up assessment after two years. Six state-of-the-art linguistic features, three lexical and three syntactic, were extracted using natural language processing methods to quantify language performance. MS severity was ranked using the Expanded Disability Status Scale (EDSS).

Results: At two-year follow-up, MS patients demonstrated overall lexical deterioration, including reduced vocabulary diversity ($p=0.031$) and increased phrase repetition ($p=0.037$) (Figure 1). Among 97 MS patients, 24 exhibited clinical progression (EDSS change ≥ 0.5). This MS subgroup was associated with pronounced syntactic decline, including shorter sentences ($p=0.044$) and reduced sentence development ($p=0.038$). MS patients without clinical progression primarily showed lexical impairments, such as restricted vocabulary ($p=0.028$) and increased phrase repetition ($p=0.049$).

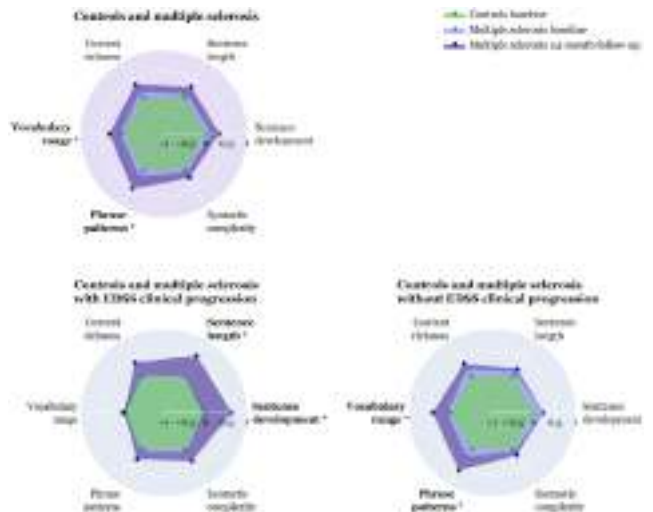


FIGURE 1 Longitudinal changes in language dimensions over 24 months. Spider plots show z-scored baseline and follow-up values; higher scores indicate greater severity. * p .

Conclusion: Automated speech analysis detects longitudinal language changes in MS, even in subgroup of patients without progression detected on gold-standard clinical scales. These findings highlight the potential of automated linguistic metrics as non-invasive biomarkers for disease progression, complementing traditional cognitive and clinical assessments.

Disclosure: This project was supported by the ERC-CZ program from the Ministry of Education Youth and Sports (project no. LL2504).

EPO-0629 | Stage-dependent relationship between retinal structure and visual function in radiologically isolated syndrome and early multiple sclerosis

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Background and aims: Radiologically isolated syndrome (RIS) represents a preclinical stage of multiple sclerosis (MS). In this context, structural and functional alterations of the anterior visual pathway remain poorly characterized. We aimed to evaluate retinal integrity and visual performance across the RIS-MS spectrum.

Methods: We conducted a cross-sectional study including healthy controls (HC), people with RIS (pwRIS) and people with early relapsing-remitting MS (pwMS), excluding subjects with prior optic neuritis or relevant ophthalmological comorbidities. Optical coherence tomography (OCT) was used to measure mean peripapillary retinal nerve fiber layer (pRNFL), ganglion cell-inner plexiform layer (GCIPL) and inner nuclear layer (INL) thickness, with age-/sex-adjusted z-scores from published normative data. An exploratory retinal neurodegeneration score

(RNS) was calculated as the mean of pRNFL and GCIPL z-scores. Visual function was evaluated using low-contrast letter acuity (LCLA) Sloan charts. Statistics included non-parametric tests and Spearman correlations.

Results: We included 123 subjects (50 HC, 23 pwRIS, 50 pwMS). pRNFL thickness, pRNFL z-scores and RNS differed significantly across groups (all $p < 0.05$), driven by significant reductions in pwMS versus HC, while pwRIS did not differ from either HC or pwMS. No significant differences were observed for GCIPL, INL and LCLA measures across all groups. While no structure-function correlations emerged in HC or pwRIS, pwMS showed significant positive correlations between LCLA and both pRNFL metrics and RNS (rho range = 0.38–0.53, $p < 0.05$).

Conclusion: Our findings support the concept of a pathological continuum; while pwRIS maintain a near-normal retinal profile, the transition to MS appears to be characterized by significant axonal loss and the emergence of structure-function coupling.

Disclosure: RMB, AdA, RC, MR, GR, MA, VR, MLF: nothing to disclose. RD: received honoraria as a speaker and member of advisory boards by: Merck-Serono, Roche, Novartis, BMS and travel funding from Almirall, Biogen, Novartis, Sanofi-Genzyme, Roche and Merck-Serono. AB: received speaker's honoraria and/or compensation for consulting services and for speaking activities from Biogen, Roche, Merck, Celgene, and Genzyme. AT: received honoraria as speaker or consultant from Lundbeck, AbbVie, UCB Pharma, Allergan, Almirall, and Bial. He is an editorial board member of European Journal of Neurology. AG: received speaker, consulting fees and travel support from Biogen, Genzyme, Merck Serono, Mylan, Novartis, Roche, and Teva, and receives research support from Fondazione Italiana Sclerosi Multipla and Italian Ministry of Health.

EPO-0630 | **Patient-reported outcomes after autologous haematopoietic stem cell transplantation compared with rituximab in multiple sclerosis**

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Background and aims: Autologous haematopoietic stem cell transplantation (AHSCT) has emerged as a highly-effective treatment for relapsing-remitting multiple sclerosis (RRMS). Patient-reported outcomes have been shown to improve following AHSCT, but how this compares to other established disease-modifying treatments is unclear. We aimed to describe the Multiple Sclerosis Impact Scale-29 (MSIS-29) questionnaire scores in comparable patients treated with AHSCT and rituximab.

Methods: This was a retrospective observational study using the Swedish MS registry. Eligibility for inclusion was on the basis of: a diagnosis of RRMS; treatment in Sweden with either AHSCT or rituximab between 2008 and 2020; a minimum clinical, radiological and MSIS-29 dataset. Propensity scores were derived from prespecified confounders and used for 1:4 matching. Linear mixed-effect models assessed differences in scaled scores between the matched groups.

Results: Of the eligible 95 AHSCT and 2196 rituximab patients, 74 AHSCT were matched to 281 rituximab patients with good

TABLE 1 Baseline Characteristics of included participants. Participants matched on propensity score between rituximab- and AHSCT-treated groups.

Table 1: Baseline Characteristics			
Continuous numerical data presented as 'mean (standard deviation)', ordinal numerical data as 'median (interquartile range)' and categorical data as 'count (percentage)'; d, standardized mean differences.			
	AHSCT-treated	Rituximab-treated	d
n	74	281	
Age, y	33.0 (7.4)	33.4 (8.5)	0.064
Sex, male	32.0 (29.7)	83.7 (29.6)	0.021
Disease duration, y	4.8 (4.3)	4.9 (4.4)	0.073
EDSS score	2.9 (1.4)	2.9 (1.4)	0.013
Annualized relapse rate	0.02 (0.05)	0.09 (0.13)	0.058
MRI brain T2-weighted lesions			
<20 lesions	22.0 (29.7)	65.4 (24.4)	0.13
≥20 lesions	52.0 (70.3)	215.6 (79.0)	
MRI T1-weighted gadolinium-enhancing lesions			
Present	9.0 (13.5)	32.1 (30.5)	0.181
Absent	52.0 (70.3)	190.1 (57.7)	0.096
Not measured ^a	12.0 (16.2)	38.8 (13.8)	0.060
Last DMT prior to current therapy			
High-efficacy	26.0 (35.1)	65.7 (33.1)	0
Moderate-efficacy	43.0 (58.1)	163.3 (50.0)	0
Treatment-naïve	5.0 (6.8)	19.0 (6.8)	0
Total DMT's prior to current therapy	2.2 (1.4)	2.1 (1.5)	0.041
MSIS-29 baseline scores			
Physical score	32.1 (23.4)	32.2 (23.1)	0.004
Psychological score	42.0 (25.5)	41.3 (25.7)	0.090

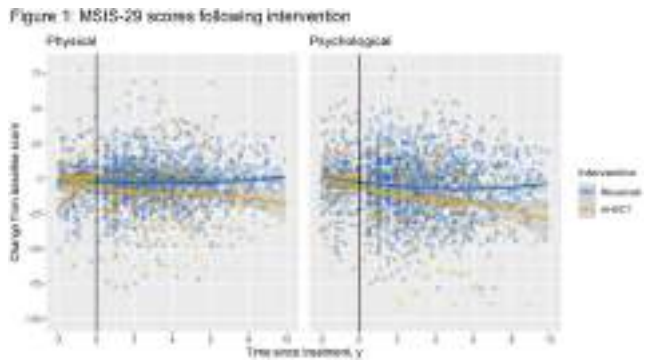


FIGURE 1 Trends in MSIS-29 scores in participants following intervention with either rituximab or AHSCT. Scores are presented relative to each individuals pre-treatment baseline.

covariate balance (Table 1, standardised mean differences <0.1). Changes from baseline MSIS-29 scores are shown in Figure 1 Both treatments were associated with a decrease in physical MSIS-29 scores over time, with AHSCT associated with a greater decrease over time (−0.90 points-per-year, $p = 0.019$, Table 2). With regards to psychological MSIS scores, both treatments were again associated with a decrease in scores over time, with AHSCT again associated with greater decreases over time (−0.99 points-per-year, $p = 0.022$, Table 2).

Conclusion: In propensity-matched RRMS patients, AHSCT was associated with greater improvement in both physical and psychological MSIS-29 scores than rituximab, supporting benefits on symp-toms and wellbeing.

TABLE 2 Linear mixed-effects model estimates for change in physical and psychological MSIS-29 scores with respect to time and intervention.

Table 2: Linear mixed-effects model for physical and psychological MSIS-29 scores

Fixed effects estimates of change in MSIS-29 score relative to baseline and / illustrative treatment. SE, standard error.

Physical MSIS-29 scores				Psychological MSIS-29 scores			
Term	Estimate	SE	P value	Term	Estimate	SE	P value
Reference: At baseline, baseline	27.5	1.55	-	Reference: At baseline, baseline	27.2	1.44	-
Time, y	-0.93	0.26	0.002	Time	-1.67	0.29	<0.001
Quadratic time, y ²	0.11	0.05	<0.001	Quadratic time, y ²	0.15	0.03	<0.001
AHSC1	-0.71	2.10	0.735	AHSC1	-2.18	2.43	0.373
Time : AHSC1	-0.93	0.38	0.019	Time : AHSC1	-0.88	0.43	0.023

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EPO-0631 | Dysphagia in neuromuscular disorders: A histopathological study of the cricopharyngeal muscle

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Background and aims: Dysphagia is a debilitating condition with complex etiology. Potential therapeutic interventions include cricopharyngeal myotomy. This study aims to investigate the histopathological characteristics of the cricopharyngeal muscle (CM) in patients with neuromuscular disorders (NMDs), to evaluate its diagnostic utility.

Methods: CM tissue was collected from 10 patients that underwent cricopharyngeal myotomy, including 4 patients with Oculopharyngeal Muscular Dystrophy, 1 with Oculopharyngeal Distal Myopathy, 3 with Inclusion Body Myositis (IBM) and 1 with idiopathic achalasia. The specimens were processed for routine histoenzymology and immunohistochemistry (antibodies against PABPN1, MHC class 1/2, p62, LC3). Seven samples were also analyzed using ultrastructural analysis.

Results: Common histological findings in CM include horizontal and transverse fiber orientation, the presence of nemaline rods and mitochondrial abnormalities, type I fiber predominance, and increased connective tissue (Figure 1). Rimmed or non-rimmed vacuoles were identified in four patients. Scattered necrosis was observed in five. Inflammatory infiltrates were found in two patients within the IBM cohort. Immunohistochemical staining is the most reliable method for detecting PABPN1 aggregates in CM and is also simpler to perform than ultrastructural studies. No distinctive histological pattern was identified in idiopathic achalasia. Ultrastructural analyses revealed also abundant autophagic structures.

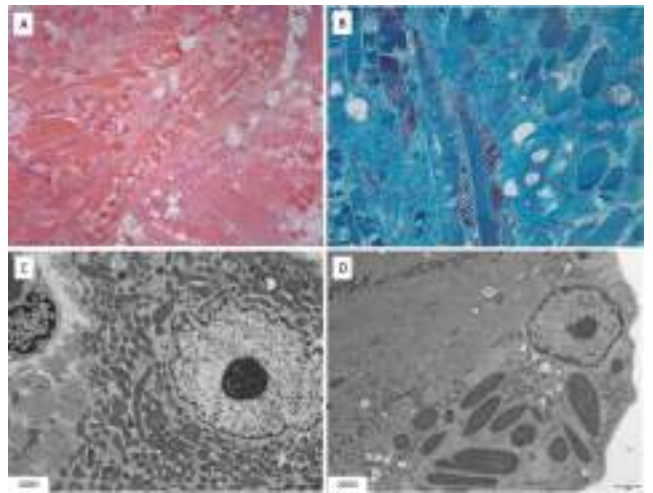


FIGURE 1 Histological and ultrastructural findings of the CM.

Conclusion: Histological examination of CM is rarely performed but still can provide critical insights for diagnosis. Our study emphasizes its potential value in the diagnostic process, particularly in patients whose clinical presentation is exclusively limited to dysphagia, thereby reinforce its role as complementary tool.

Disclosure: Nothing to disclosure.

EPO-0632 | MG patients facing heat waves. Heat waves exposure differentially worsens MG-ADL Scores in anti-acetylcholine receptor but not MuSK myasthenia gravis

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Background and aims: To assess the impact of heat wave exposure on symptom severity, as measured by the Myasthenia Gravis Activities of Daily Living (MG-ADL) scores in patients with autoimmune myasthenia gravis (MG), comparing anti-acetylcholine receptor (anti-AChR) and anti-MuSK subtypes.

Methods: We conducted a cross-sectional questionnaire-based study including 206 adult MG patients. Heat-related symptom worsening during heat waves, hot bath sensitivity, cooling strategies, and MG-ADL-derived heat-aggravation scores were analyzed according to antibody status.

Results: Heat wave exposure was associated with a significant worsening of MG-ADL scores in anti-AChR patients, whereas no significant effect was observed in MuSK patients. Anti-AChR patients frequently reported symptom exacerbation during days of heatwaves, after hot showers or baths and commonly required air conditioning. Cold exposure was associated with symptom improvement predominantly in the anti-AChR group.

TABLE 1 Composition of the heat related MG-ADL score.

MG-ADL domain	Heat-related question (Yes/No)	Score
Talking	Worsening of speech difficulties during heat exposure	1
Chewing	Increased difficulty chewing during heat exposure	1
Swallowing	Worsening of swallowing difficulties during heat exposure	1
Breathing	Increased shortness of breath during heat exposure	1
Brushing teeth / combing hair	Increased difficulty due to arm weakness during heat exposure	1
Arising from a chair	Increased difficulty standing up due to leg weakness during heat exposure	1
Double vision	Worsening or increased frequency during heat exposure	1
Eyelid droop	Increased ptosis during heat exposure	1
Total heat-related MG-ADL score range: 0-8		

TABLE 2 Baseline demographic and heat-related clinical characteristics of patients with myasthenia gravis according to antibody subtype. Values are presented as mean $\pm$ SD, median (interquartile range), or number (percentage), as appropriate. Heat-related MG-ADL sc.

Outcome	Anti-AChR MG (n = 185)	MuSK MG (n = 21)	p-value
Heat-related MG-ADL score (0-10), mean $\pm$ SD	7.0 $\pm$ 1.2	5.8 $\pm$ 1.8	<0.001
Symptoms worsening with heat, n (%)	167 (90%)	7 (33%)	<0.001
Worsening after hot shower or bath, n (%)	144 (87%)	4 (19%)	<0.001
Use of air conditioning during heat exposure, n (%)	133 (72%)	4 (19%)	<0.001
Improvement with cold exposure, n (%)	139 (75%)	6 (29%)	<0.01

TABLE 3 Heat waves -related clinical outcomes in myasthenia gravis according to antibody subtype. Values are presented as mean $\pm$ SD or number (percentage). Group comparisons were performed using Welch's t-test for continuous variables and χ^2 tests for cat.

Outcome	Anti-AChR MG (n = 185)	MuSK MG (n = 21)	p-value
Age (years), mean $\pm$ SD	51 $\pm$ 14	49 $\pm$ 12	0.08
Female sex, n (%)	93 (50%)	16 (76%)	0.06
Duration (years), median (IQR)	7 (3-18)	6 (2-11)	0.03
Heat-related MG-ADL score (0-10), mean $\pm$ SD	7.0 $\pm$ 1.2	5.8 $\pm$ 1.8	<0.001
Symptoms worsening with heat, n (%)	167 (90%)	7 (33%)	<0.001
Worsening after hot shower/bath, n (%)	144 (87%)	4 (19%)	<0.001
Use of air conditioning during heat, n (%)	133 (72%)	4 (19%)	<0.001
Improvement with cold exposure, n (%)	139 (75%)	6 (29%)	<0.01

Conclusion: Heat wave represents a clinically relevant aggravating factor in anti-AChR MG, supporting targeted environmental management strategies in the perspectives of climate change, in such a vulnerable population.

Disclosure: Disclosure interests Dr Lagrange.

EPO-0633 | Tanruprubart rapidly improves health-related quality of life in patients with Guillain-Barré syndrome: A randomised, placebo-controlled Ph3 study

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Background and aims: Guillain-Barré syndrome (GBS) is a rare, life-altering, neuromuscular emergency, causing long-lasting disabilities severely impacting quality of life. Tanruprubart (ANX005) inhibits complement component C1q. In a Ph3, double-blind, placebo-controlled study (GBS-02; NCT04701164), a single infusion of tanruprubart 30 mg/kg improved muscle strength and disability in patients with GBS at Day 8 and Week 8. This analysis presents patient-reported outcomes (PROs) with tanruprubart 30 mg/kg versus placebo.

Methods: Patients aged ≥ 16 years with a recent GBS diagnosis were randomised to tanruprubart 30 mg/kg ($n=79$), 75 mg/kg ($n=81$) or placebo ($n=81$). PROs included the EuroQol 5-Dimension 5-Level (EQ-5D-5L), Rasch-built Modified Fatigue Severity Scale (rFSS) and Patient Global Impression of Change (PGIC). Impact of treatment on HRQoL was evaluated using proportional odds logistic regression adjusted for baseline covariates.

Results: By Day 8 significant improvements in EQ-5D-5L occurred in mobility and self-care with tanruprubart 30 mg/kg versus placebo (Table 1). Rapid improvements were observed with tanruprubart versus placebo in all domains of the rFSS except for pain (Table 2). In the PGIC, eight (10.1%) patients reported being 'very much improved' with tanruprubart at Day 8, compared with three (3.7%) patients with placebo (aOR 2.5 [95% confidence interval (CI): 1.4, 4.6] $p=0.0028$). Tanruprubart-associated improvements in EQ-5D-5L mobility, self-care and usual activities domains continued to Week 8 (Tables 1, 2). PGIC improvements at Week 8 still favoured tanruprubart (aOR 1.4 [95% CI 0.8, 2.6, $p=0.2917$).

Conclusion: Tanruprubart demonstrated rapid and sustained improvements in HRQoL, consistent with observed objective clinical benefits, supporting its potential as a targeted immunotherapy for GBS.

Disclosure: The study was sponsored by Annexon Biosciences (Brisbane, CA, USA). MK, GM, JD, H-AK: Employment with and shareholder of Annexon Biosciences QDM: Consultancy/advisory role with Annexon Biosciences NP: Nothing to disclose

TABLE 1 Patients reporting no problems across EQ-5D-5L domains on Day 8 and Week 8.

Domain	Day 8, n (%)		aOR (95% CI) P-value ^a	Week 8, n (%)		aOR (95% CI) P-value ^a
	Tanruprubart 30 mg/kg	Placebo		Tanruprubart 30 mg/kg	Placebo	
Mobility	3 (3.3)	1 (1.2)	3.7 (0.6, 12.4) $p=0.0801$	25 (19.0)	8 (9.7)	2.3 (1.0, 5.4) $p=0.0424$
Self-care	9 (11.4)	3 (3.7)	3.5 (1.6, 7.5) $p=0.0007$	31 (26.4)	13 (16.8)	2.0 (1.1, 3.6) $p=0.0113$
Usual activities	7 (8.9)	1 (1.2)	7.4 (2.3, 18.9) $p=0.0001$	34 (27.2)	9 (11.1)	2.0 (1.1, 3.7) $p=0.0166$
Pain/discomfort	13 (16.3)	31 (14.4)	1.0 (0.6, 1.8) $p=0.9882$	31 (26.5)	18 (24.4)	1.3 (0.7, 2.4) $p=0.3735$
Sociality/depression	49 (31.8)	41 (14.8)	3.5 (2.0, 6.3) $p=0.0008$	42 (29.5)	34 (21.4)	1.5 (0.7, 3.4) $p=0.2820$

^aProportional odds ordinal regression model and a mixed model for repeated measures used to evaluate comparisons for tanruprubart versus placebo. EQ-5D-5L, EuroQol 5-Dimension 5-Level; aOR, adjusted odds ratio.

TABLE 2 Patients who answered “disagree” to statements across rFSS domains at day 8 and week 8.

Domain	Day 8, n (%)			Week 8, n (%)		
	Tamagrabant	Placebo	aOR (95% CI)	Tamagrabant	Placebo	aOR (95% CI)
	30 (30.3)	16 (16.6)	2.0 (1.1, 3.6)	38 (38.4)	33 (36.4)	1.1 (0.7, 2.3)
Fatigue causes frequent problems for me			p=0.0286			p=0.4073
Fatigue interferes with carrying out activities	36 (36.3)	18 (18.2)	1.8 (1.0, 3.3)	34 (38.4)	18 (23.5)	1.5 (0.8, 2.7)
			p=0.0609			p=0.2217
Exercise brings on my fatigue	35 (35.3)	18 (23.2)	1.2 (0.7, 1.7)	39 (34.3)	18 (22.3)	1.6 (0.5, 3.8)
			p=0.5150			p=0.9502
I am easily fatigued	34 (38.4)	16 (18.8)	1.9 (1.1, 3.5)	38 (33.8)	32 (27.3)	1.4 (0.8, 2.8)
			p=0.0206			p=0.2303
Fatigue interferes with physical function	35 (29.3)	13 (16.0)	1.7 (0.9, 3.3)	32 (27.8)	38 (29.8)	1.3 (0.7, 2.3)
			p=0.0882			p=0.4355
Fatigue prevents sustained physical function	38 (23.8)	14 (17.3)	1.3 (0.7, 1.8)	38 (23.8)	38 (37.8)	1.2 (0.7, 2.3)
			p=0.4039			p=0.5952
Fatigue interferes with work, family or social life	37 (34.2)	16 (18.8)	2.5 (1.2, 4.3)	33 (29.3)	37 (21.8)	1.4 (0.8, 2.8)
			p=0.0075			p=0.1808

^aProportional odds in final regression model and a robust model for repeated measures used to evaluate comparisons for tamagrabant versus placebo

CI, confidence interval; aOR, adjusted odds ratio; rFSS, Rasch-built Modified Fatigue Severity Scale

KCG: Consultancy/advisory role with Annexon Biosciences, argenx, Janssen, and Sanofi.

EPO-0634 | Late-onset TK2 deficiency in adults: Long-term clinical outcomes of deoxynucleoside therapy

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Background and aims: Thymidine kinase 2 deficiency is a mitochondrial myopathy causing progressive weakness, respiratory insufficiency, and early mortality. While nucleoside supplementation benefits pediatric cases, evidence in adults remains limited.

Methods: Six genetically confirmed adults with TK2d were treated under compassionate-use protocols in Türkiye, receiving escalating doses of oral or PEG-administered Doxycitine and Doxribtimine up to 800 mg/kg/day. Functional and clinical outcomes included the 6-minute walk test (6MWT), HFMSE, forced vital capacity (FVC), Fatigue Severity Scale (FSS), and BMI.

Results: Over a median follow-up of 24 (range 6–36) months, therapy was associated with sustained functional and respiratory improvements. Mean 6MWT increased from 152 m at baseline to 468 m at 24 months and exceeded 600 m in patients with 36-month data. The mean HFMSE increased from 20.5 at baseline to 24.5 at 3 months, 29 at 6 months. Patients with the longest follow-up reached a mean HFMSE of 58 at 36 months. FVC, which had declined before treatment, stabilized or improved in all patients (mean increase from 36% to 55.6% at 36 months). The youngest patient achieved near-normal respiratory function. Fatigue severity decreased by ~30%, and BMI improved notably in underweight individuals. Treatment was generally well tolerated; transient liver enzyme elevations and mild gastrointestinal side effects resolved spontaneously.

Conclusion: Deoxynucleoside therapy appears effective and well tolerated in adults with TK2d, producing sustained functional and respiratory gains. Early treatment initiation is crucial

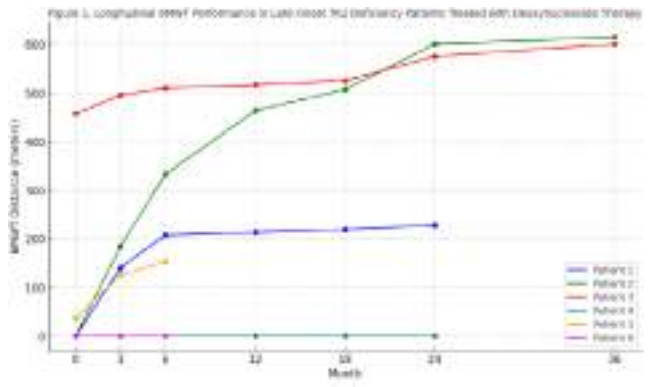


FIGURE 1 Longitudinal six-minute walk test performance in six adult patients with late-onset TK2 deficiency treated with deoxynucleoside therapy. Patient 1's 36-month measurement was unavailable due to surgery. Patients 5 and 6 were followed up to 6 mo.

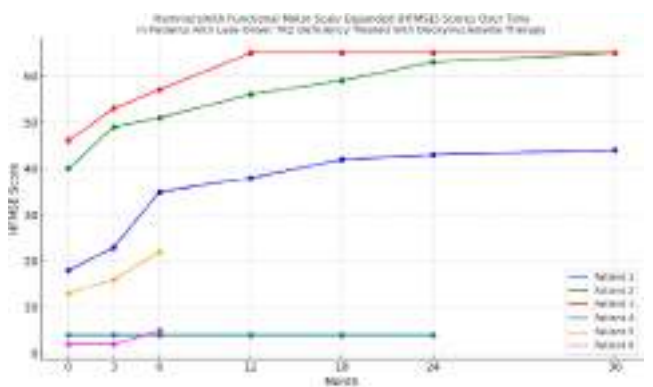


FIGURE 2 Hammersmith Functional Motor Scale scores over time in six adult patients with late-onset TK2 deficiency treated with deoxynucleoside therapy.

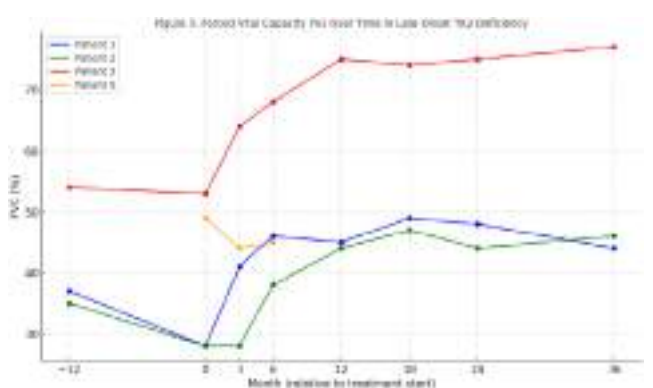


FIGURE 3 Forced vital capacity (%) over time in four adult patients with late-onset TK2 deficiency receiving deoxynucleoside therapy. Patients 4 and 6, who were tracheostomized and dependent on continuous ventilatory support, were excluded from this figure.

to maximize benefit; though partial improvements may occur even in advanced disease.

Disclosure: The authors have no conflicts of interest to declare.

EPO-0635 | **A novel Guillain–Barré syndrome clinical progression scale to characterise the patient’s care journey: Utilisation in a phase 3 trial of tanruprubart**

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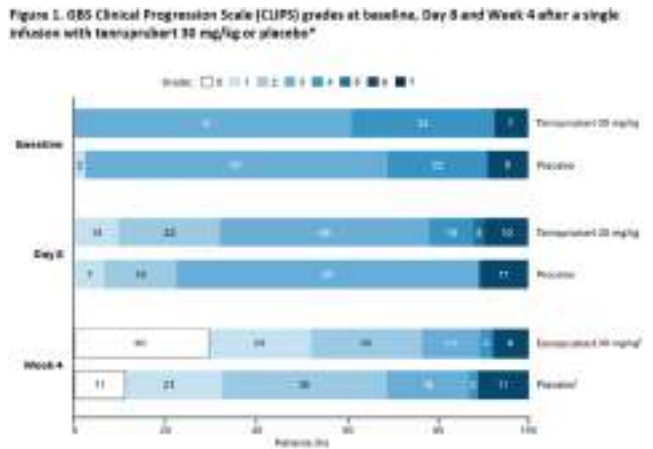
Background and aims: Guillain-Barré syndrome (GBS) is a rare, acute, life-altering autoimmune polyneuropathy with rapidly progressive weakness and prolonged recovery. In GBS-02, a Ph3, randomised, double-blind, placebo-controlled trial, tanruprubart (ANX005) 30 mg/kg led to rapid complement inhibition and clinical improvement with sustained clinical benefit over 6 months in patients with GBS (NCT04701164). We characterised the patient’s care journey using a novel GBS Clinical Progression Scale (CLIPS).

Methods: Patients aged ≥16years with a recent GBS diagnosis (n=241) were randomised to a single infusion of tanruprubart 30 mg/kg, 75 mg/kg or placebo. CLIPS was adapted from the WHO Covid-19 Clinical Progression Scale by the study sponsor and GBS experts. This ordinal measure is based on 8 levels of care (Table 1). During GBS-02, clinical status on the scale was physician recorded weekly for 4 weeks and at 2, 3, 4, and 6 months in a subset of patients (tanruprubart 30 mg/kg n=41, 75 mg/kg n=45, placebo n=45). Treatment impact on level of care was evaluated using a proportional odds logistic regression adjusted for baseline covariates.

Results: All patients were hospitalised and most required medical intervention (Grade ≥3; Figure 1). By Day 8, care requirements decreased. At Week 4 the odds of requiring less care favoured tanruprubart 30 mg/kg (aOR 2.3, 95% CI 0.99–5.39; p=0.054) over placebo, consistent with overall clinical outcome.

Conclusion: CLIPS captured patients’ transitions through levels of care, providing a pragmatic lens to interpret treatment benefit. Early separation at Week 4 favouring tanruprubart supports a clinically meaningful shift toward lower resource intensity during early GBS.

Disclosure: The study was sponsored by Annexon Biosciences (Brisbane, CA, USA). H-AK: Employment with and shareholder of Annexon Biosciences KCG: Consultancy/advisory role with Annexon Biosciences, argenx, Janssen, and Sanofi



*The CLIPS was included as a late amendment to the GBS-02 trial and assessed in a subset of patients tanruprubart 30 mg/kg n=41, tanruprubart 75 mg/kg n=45 (not shown here) and placebo n=45. At Week 4, data was missing for 2 patients in the tanruprubart 30 mg/kg group and 1 patient in the placebo group.

QDM: Consultancy/advisory role with Annexon Biosciences
TH: Received grant and honoraria from Dianthus Therapeutics, Sanofi, Janssen, Nuvig, Biocryst, Argenx, Abvie and Annexon.

EPO-0636 | **Validating the 2024 ENMC inclusion body myositis criteria: Diagnostic utility in early “Polymyositis-Mitochondrial” stages**

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Background and aims: The 2024 European Neuromuscular Centre (ENMC) criteria for Inclusion Body Myositis (IBM) integrates mitochondrial pathology as a key histological supportive feature. We evaluated their diagnostic capacity in a single-centre cohort, specifically focusing on the “Polymyositis-Mitochondrial” (PM-Mito) phenotype, an early recognised stage of IBM.

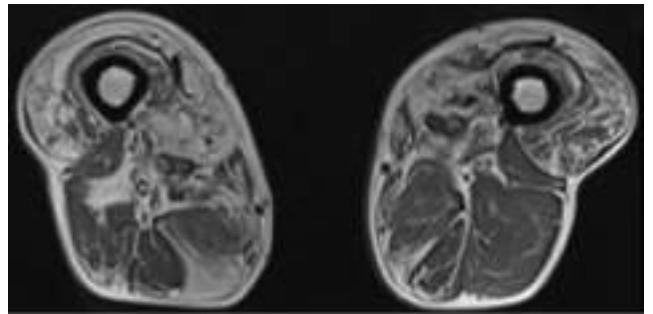
Methods: We retrospectively reassessed 15 IBM patients following the 2024 ENMC consensus and evaluated the clinical phenotype, anti-cN1A serology, muscle imaging and histological features.

Results: Mean onset age was 60.6 ± 9.5years with a diagnostic delay of 3.7 ± 2.4years. Mean Creatine kinase level was 711 ± 651 U/L. The “common” clinical phenotype (finger flexor/knee

TABLE 1 Eight grades of the ordinal GBS Clinical Progression Scale.

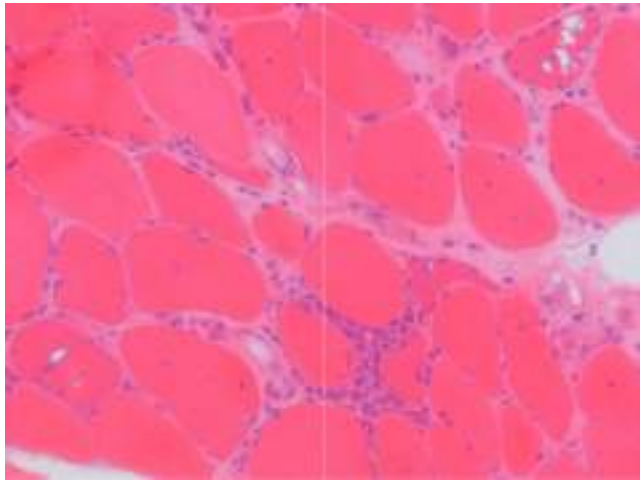
Grade	Description
0	Discharged home, independent ADLs, capable of self-care
1	Requiring ongoing nursing care or assistance by trained caregivers in ADLs
2	During recovery of GBS requiring intensive physical/occupational/speech therapy and/or general medical or nursing care
3	Hospitalised, requiring ongoing medical or nursing care during progression or recovery of GBS
4	Not mechanically ventilated, requiring intensive monitoring
5	Receiving non-invasive ventilation or high-flow oxygen devices
6	Receiving invasive mechanical ventilation
7	Death

ADLs, activities of daily living; GBS, Guillain-Barré Syndrome.

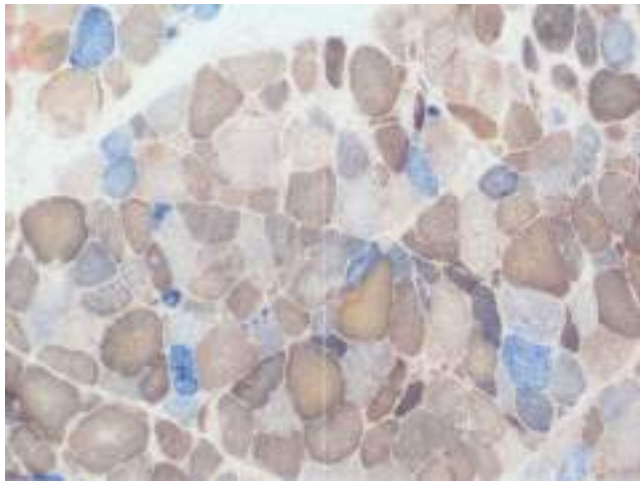


T1-weighted MRI image of both thighs showing anterior distal fatty infiltration.

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H&E muscle biopsy with endomysial lymphocyte infiltration.



Pathological increase of COX negative muscle fibers.

extensor weakness) was observed in 93% of patients and dysphagia in 67%. An inflammatory myopathic electromyogram pattern was found in 75% of analysed cases. Muscle magnetic resonance imaging revealed the classic distal/anterior thigh pattern in 70% (Figure 1). Anti-cN1A antibodies were positive in 62.5% of tested patients. Histologically, all biopsies showed endomysial lymphocyte infiltration (Figure 2), p62 aggregates were identified in 77%, whereas TDP-43 was rarely assessed. Two patients presented the PM-Mito pattern: endomysial inflammation with cyclooxygenase (COX)-negative fibres (Figure 3) exceeding the expected level for the patient's age, without additional supportive findings. Under previous criteria, this finding mimicked a polymyositis pattern. However, using the new criteria, the combination of “common” phenotype and mitochondrial pathology confirmed the IBM diagnosis.

Conclusion: The 2024 ENMC criteria enhance diagnostic sensitivity for early IBM. Elevating mitochondrial abnormalities to “supportive criterion” enables the definitive diagnosis of PM-Mito cases, preventing misdiagnosis and avoiding ineffective immunosuppressive therapies.

Disclosure: Nothing to disclose.

Background and aims: Eculizumab has demonstrated high efficacy in patients with anti-acetylcholine receptor (AChR) antibody-positive myasthenia gravis (MG). It prevents the cleavage of C5 into C5a and C5b, thereby blocking the formation of the membrane attack complex (MAC) in neuromuscular junction. However, the potential off-target effects of eculizumab on immune cell modulation—mediated via C5a-C5aR1 axis during long-term treatment—remain incompletely understood.

Methods: Patients with AChR Ab+ gMG receiving regular ecuzumab or prednisolone treatment were enrolled and divided into exploratory and validation cohorts. Bulk RNA-seq and flow cytometry were performed on peripheral immune cells. C5aR1 antagonist was administered to experimental autoimmune myasthenia gravis (EAMG) rats, and clinical scores, electromyograms, immunofluorescence staining of nicotinic AChR (nAChR), and flow cytometric analysis of immune cells were performed. B cells were co-cultured with monocytes for in vitro analysis.

Results: Bulk RNA sequencing revealed significant transcriptomic alterations in MG patients following ecuzumab treatment, enriched in B-cell-related pathways. Long-term ecuzumab markedly reduced peripheral CD19⁺ B cells and their subsets (including memory B cells and antibody-secreting cells).

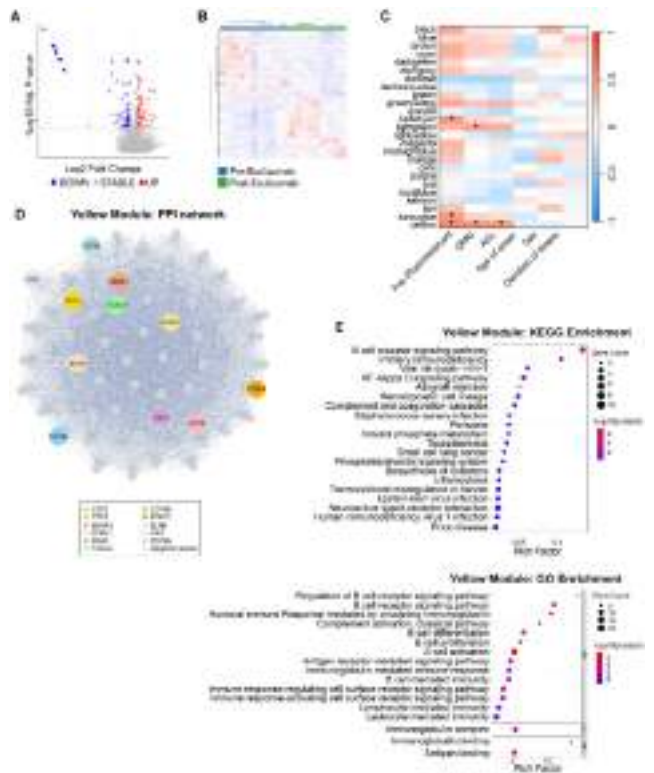


FIGURE 1 Bulk RNA-Seq analysis of differential gene expression and functional associations in AChR antibody-positive generalized myasthenia gravis patients before and after ecuzumab treatment.

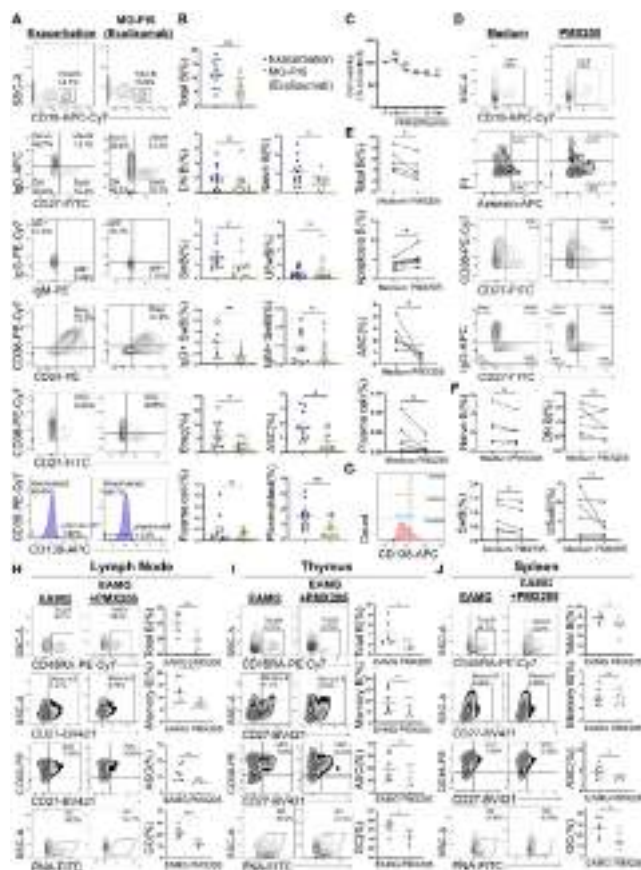


FIGURE 2 Modulation of B-cell subsets and viability by a C5aR1 antagonist in myasthenia gravis as shown by flow cytometry.

C5aR1 is predominantly expressed on myeloid cells and is up-regulated during acute exacerbations of MG. In vivo and in vitro co-culture studies indicated that blocking C5a-C5aR1 signaling pathway in monocytes inhibited B-cell differentiation, promoted B-cell apoptosis, and reduced circulating B-cell numbers, likely through decreased BAFF secretion.

Conclusion: In addition to blocking the terminal complement pathway, eculizumab modulates peripheral B-cell differentiation and survival by inhibiting the monocyte C5a-C5aR1-mediated BAFF secretion. This reveals its off-target immunomodulatory effects and the mechanism of systemic immunoregulation during long-term therapy.

Disclosure: This study was supported by the National Natural Science Foundation of China (82571535, 82171338, 82171277), and Young Scientific and Technological Talents (Level Three) in Tianjin.

EPO-0638 | Significant correlations between ME&MGTM digital biomarkers and equivalent MG-ADL and MG-QoL15r sub-items scores

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corporate member of Freie Universität Berlin and Humboldt-Universität zu Berlin, Department of Neurology with Experimental Neurology, Charitéplatz 1; 10117 Berlin, Germany; ³Ad Scientiam, Paris, France; ⁴Alexion, AstraZeneca Rare Disease, Boston, USA; ⁵University of North Carolina, Department of Neurology, Chapel Hill, USA

Background and aims: Symptoms of generalized Myasthenia Gravis (gMG) are routinely captured via patient-reported outcomes (PROs) questionnaires. MG-ADL and MG-QoL15r assess daily activities and quality of life; high scores indicate higher severity and worse quality of life. Digital biomarkers (dBMKs) provide objective measures for remote symptoms' self-assessment through smartphone-based active tests. ME&MGTM evaluates five gMG symptoms: ptosis, dysarthria, breathing, upper and lower limb weakness mirrored in 5 MG-ADL and 3 MG-QoL15r items. In the decentralized ME&MGopen study (NCT05566964), patients performed ME&MGTM tests for one year. Here, we describe the correlation between dBMKs and corresponding MG-ADL and MG-QoL15r sub-items.

Methods: Completed ME&MGTM tests were reviewed for quality: inaccurate dBMKs values and agreement with human measurements. The relationship between dBMKs and sub-items was determined using mixed models analyses. We expected negative associations with MyBreathing, MyEyelids, Mylegs and MyArms and positive correlations with MyVoice.

Results: 156 participants completed digital tests and questionnaires monthly (Figure 1). A total of 9106 evaluations were collected, of which 90.4% were of good quality. DBMKs scores for ptosis, breathing, and limb weakness decreased with increasing PRO assessed symptom severity and scores for dysarthria decreased with severity (Figure 2). Ptosis, dysarthria and limb weakness scores showed significant associations (all $p < 0.019$) with aforementioned sub-items of the MG-ADL and MG-QoL15r questionnaires.

Conclusion: Significant associations between clinical ME&MGTM dBMKs and clinical assessments support their potential use in clinical settings to track gMG manifestations. These findings highlight the importance of the ongoing DOMYA validation study (NCT05564936).

Disclosure: C. Barnett-Tapia: Ad Scientiam, Alexion, AstraZeneca rare Disease, Cartesian Therapeutics, US Department of Defence, Muscular Dystrophy Canada, MGNet, Grifols and Octapharma—research grants. Argenx, Alexion, AstraZeneca Rare Disease, Janssen and UCB—advisory board member. Argenx, Alexion, AstraZeneca Rare Disease, UCB, Novartis and Janssen—consulting fees. S. Lehnerer: Ad Scientiam, Alexion, AstraZeneca Rare Disease, argenx, Biogen, Hormosan, HUMA, Johnson & Johnson, Merck, UCB and Roche—speaker and consulting fees, research grants RareLink digital health GmbH—shareholder. S. Bieuvelet, L. Carment, C. Gorin, N. Pesic-Heuvrard, D. Ravindra, N. Sellami and S. Zinaï: employees of Ad Scientiam; may hold stock and/or stock options. B. Dutta, B. Yungher: employees of Alexion, AstraZeneca Rare Disease; may hold stock and/or stock options. J.F. Howard Jr : Ad Scientiam, Alexion AstraZeneca Rare Disease, argenx, Cartesian Therapeutics, Centers for Disease Control and Prevention, Merck EMD Serono, MGFA, Muscular Dystrophy Association, NIH, NMD Pharma, and UCB Pharma—research grants. AcademicCME, Alexion AstraZeneca Rare Disease, Amgen, argenx, Biohaven Ltd, Cartesian Therapeutics,

Table 1. Participants' baseline characteristics	
	N=156
Age, mean (SD)	58.7 (15.7)
Sex	
Female, n (%)	108 (67.9)
Male, n (%)	50 (32.1)
Country of residence	
Canada, n (%)	117 (93.6)
United States, n (%)	46.9 (21.0)
Age (y) at disease onset, mean (SD)	11.7 (13.0)
Disease duration, mean (SD)	11.2 (12.27)
Thymectomy	
Yes, n (%)	78 (50.0)
Need for caregivers, n (%)*	
Yes	37 (24.2)
MGFA disease class, n (%)	
Unreported	108 (69.2)
Class II	12 (7.7)
Class III	26 (16.7)
Class IV	10 (6.4)

*50 participants answered this query

FIGURE 1 Baseline characteristics of patients who completed all ME&MGTM during the ME&MGOpen study.

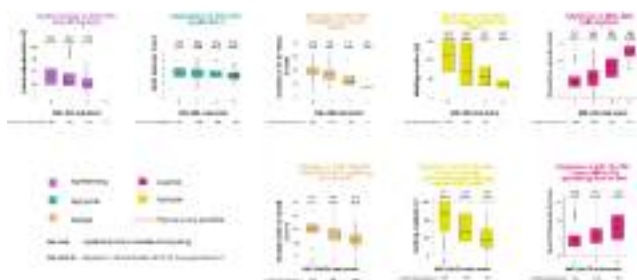


FIGURE 2 Correlations between ME&MGTM scores and MG-ADL or MG-QoL15r sub-items.

CheckRare CME, CoreEvitas, Curie.bio, H. Lundbeck A/S, Japan Tobacco Company, Kyverna Therapeutics, Merck EMD Serono, NMD Pharma, Novartis Pharma, PeerView CME, Physicians' Education Resource (PER) CME, PlatformQ CME, Regeneron Pharmaceuticals, Seismic Therapeutics, TG Therapeutics, Toleranzia AB, and UCB Pharma—consulting fees. Alexion AstraZeneca Rare Disease, argenx, Biohaven Ltd, Cartesian Therapeutics, Toleranzia AB, and UCB Pharma—non-financial support.

EPO-0639 | Minimal symptom expression with claseprubart, an active C1S inhibitor, in patients with generalized myasthenia gravis

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Background and aims: The classical complement pathway plays a significant role in generalized myasthenia gravis (gMG) pathology. Claseprubart is a potent monoclonal antibody that selectively targets the classical pathway by inhibiting active C1s (aC1s). Remission is a key goal in gMG and achieving minimal symptom expression (MSE) with claseprubart is a key treatment objective.

Methods: MaGic (NCT06282159), is a global Phase 2, randomized, double-blind, placebo-controlled trial. Patients treated with claseprubart (300mg, Q2W) were evaluated for MSE (MG-ADL ≤1 or QMG ≤3) compared with placebo. *p*-Values were one-sided and nominal significance was assessed at an alpha=0.1. Outcomes include proportion of patients achieving MSE at Week 13 and any-time during the study, earliest timepoint of MSE achievement, and durability defined as sustained MSE for at least 6 weeks.

Results: At Week 13, 37% of claseprubart treated patients achieved MG-ADL-MSE versus 14% with placebo (OR 3.81; *p*=0.0550). 43% achieved MG-ADL-MSE at least once during the study versus 14% of placebo (OR: 4.72; *p*=0.0231). This effect was observed as early as Week 1 (median time to response was Week 3). Sustained remission-like states (for at least 6 weeks) were seen in all patients who achieved MSE. Similar results were observed using QMG minimum symptom expression threshold. **Conclusion:** Claseprubart patients were more than four times more likely to achieve MSE than placebo patients and MSE was observed as early as week 1. Patients who achieved MSE maintained responses for at least 6 weeks. These results highlight the therapeutic potential of aC1s inhibition in AChR+ gMG.

Disclosure: MS nothing to declare. NG grants or honoraria from Dianthus, Amgen, Alexion, Argenx, UCB, Novartis, J&J, Medison, NMD Pharma, Lundbeck, and Merck. MA employee of Dianthus Therapeutics CB employee of Dianthus Therapeutics US employee of Dianthus Therapeutics LH employee of Dianthus Therapeutics ML employee of Dianthus Therapeutics SB nothing to declare.

EPO-0640 | Myasthenia Gravis Inebilizumab Trial (MINT): Adverse events of special interest during the randomized controlled period (RCP)

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Background and aims: B cells play a key upstream mechanistic role in autoimmune generalized myasthenia gravis (gMG) pathogenesis through the production of antibodies targeting the acetylcholine receptor (AChR+), muscle-specific kinase (MuSK+), or other related proteins. Inebilizumab is a monoclonal antibody targeting CD19+ B cells. B-cell-depleting therapies may impair humoral responses, increase the risk of infections and reduced response to vaccinations. Here we examine whether inebilizumab is associated with an increased risk of adverse events of special interest (AESIs) in gMG participants.

Methods: MINT (NCT04524273) was a phase 3 trial in adults with gMG. During the RCP (52-weeks for AChR+; 26-weeks for MuSK+), participants were randomized (1:1) to receive 300mg of inebilizumab or placebo on Day-1, Day-15, and Day-183 (AChR+ only). Subsets of adverse events were identified as AESIs due to their relevance to inebilizumab's mechanism of action.

Results: 238 patients were randomized: inebilizumab 119 (95 AChR+, 24 MuSK+), placebo 119 (95 AChR+, 24 MuSK+). Baseline demographics and disease characteristics were generally balanced between treatment groups. AESIs were infrequent, occurring in 10.1% ($n=12$) of the inebilizumab- and 16% ($n=19$) of the placebo-treated groups. Cytopenias occurred in 2.5% ($n=3$) of inebilizumab-treated and 9.2% ($n=11$) of placebo-treated participants. Serious and/or opportunistic infections were infrequent (inebilizumab: 3.4% [$n=4$]; placebo: 5.0% [$n=6$]), with COVID-19 being the most common (inebilizumab: 2/4; placebo: 3/6) and no instances of progressive multifocal leukoencephalopathy. Infusion related reactions occurred in 5.0% ($n=6$) of inebilizumab- and 2.5% ($n=3$) of placebo-treated participants (Table 1).

Conclusion: These results support a favorable safety profile of inebilizumab over a 26–52-week period.

Disclosure: RJN received research support from Alexion Pharmaceuticals, Amgen Inc, argenx, Grifols, Immunovant, Janssen, Myasthenia Gravis Foundation of America, the National Institutes of Health, and UCB and served as a consultant/adviser for Alexion Pharmaceuticals, Amgen Inc, argenx, Cabaletta Bio, Cour Pharmaceuticals, CSL Behring, Grifols, Immunovant, Janssen, and UCB. KU served as a paid consultant for argenx, Chugai Pharmaceutical, HanAll BioPharma, Janssen, Merck, Mitsubishi Tanabe Pharma, UCB, and Amgen Inc. and

received speaker honoraria from Alexion Pharmaceuticals, argenx, the Japan Blood Products Organization, and UCB. MB received grant support from the ALS Association, the ALS Recovery Fund, Eli Lilly, the Kimmelman estate, the Muscular Dystrophy Association, the National Institutes of Health, and Target ALS. He reports research support from Alexion Pharmaceuticals and Immunovant and served as an investigator for clinical trials funded by Biogen, Novartis, and Orphazyme. He received personal consulting fees from Alaunos Therapeutics, Alector Therapeutics, Alexion Pharmaceuticals, Annexon Biosciences, Arrowhead Pharmaceuticals, Canopy Immunotherapeutics, CorEvitas, Denali Therapeutics, Eli Lilly, Evox Therapeutics, Amgen Inc, Immunovant, Janssen, Merck, Novartis, Roche, Sanofi, Takeda, Trace Neuroscience, UCB, uniQure, VectorY Therapeutics, Voyager Therapeutics, and Woolsey Pharmaceuticals. EC has no disclosures. MIL received funding from the National Health Service (MRDS and NSCG for NMOSD) and the University of Oxford. She was awarded research grants from the UK association for patients with myasthenia (myaware) and the University of Oxford. She received speaker honoraria/travel grants from the Guthy-Jackson Charitable Foundation and UCB. She served on scientific/educational advisory boards for argenx, UCB, and Amgen Inc. JV has been a consultant for and/or received speaker honoraria from Alexion Pharmaceuticals, Amgen Inc, argenx, Dianthus Therapeutics, Johnson & Johnson, NMD Pharma, Novartis, Regeneron Pharmaceuticals, Roche, Toleranzia AB, and UCB. FT, BC, QL, and SC are employees of Amgen Inc. and own stock. JFH received research funding (paid to his institution) from Ad Scientiam, Alexion AstraZeneca Rare Disease, argenx, Cartesian Therapeutics, the Centers for Disease Control and Prevention, EMD Serono, Merck, the Muscular Dystrophy Association, Myasthenia Gravis Foundation of America, the National Institutes of Health, NMD Pharma, and UCB; honoraria/consulting fees from AcademicCME, Alexion AstraZeneca Rare Disease, Amgen Inc, Biohaven, CheckRare CME, CorEvitas, Curie.Bio, EMD Serono, Hansa Biopharma, Lundbeck, Merck, Novartis, PeerView CME, Physicians' Education Resource CME, PlatformQ CME, Regeneron Pharmaceuticals, Sanofi, Seismic Therapeutics, TG Therapeutics, Toleranzia AB, and UCB; and nonfinancial support from Alexion AstraZeneca Rare Disease, argenx, Biohaven, Cartesian Therapeutics, Toleranzia AB, and UCB.

TABLE 1 Treatment-emergent adverse events of special interest.

Adverse Events of Special Interest Category ^a Preferred Term	Placebo N = 119	Inebilizumab N = 119	Total N = 238	Inebilizumab Difference [95% CI]
Subjects with ≥1 AESI	19 (16.0%)	12 (10.1%)	31 (13.0%)	-5.8% (-14.7%, 2.8%)
CYTOPENIA	11 (9.2%)	3 (2.5%)	14 (5.7%)	-6.7% (-15.6%, 2.2%)
Anemia	2 (1.7%)	1 (0.8%)	3 (1.3%)	-0.8% (-5.2%, 3.1%)
Anemia megaklastic	1 (0.8%)	0	1 (0.4%)	-0.8% (-4.8%, 2.3%)
Cytopenia	1 (0.8%)	0	1 (0.4%)	-2.5% (-7.2%, 2.2%)
Leukopenia	1 (0.8%)	1 (0.8%)	2 (0.7%)	0.0% (-3.8%, 3.8%)
Lymphocyte count decreased	1 (0.8%)	0	1 (0.4%)	-0.8% (-4.4%, 2.8%)
Lymphopenia	1 (0.8%)	1 (0.8%)	2 (0.7%)	0.0% (-3.8%, 3.8%)
Neutropenia	2 (1.7%)	1 (0.8%)	3 (1.3%)	-0.8% (-5.2%, 3.1%)
Thrombocytopenia	2 (1.7%)	0	2 (0.8%)	-1.7% (-5.9%, 1.5%)
INFUSION RELATED REACTION	3 (2.5%)	6 (5.0%)	9 (3.8%)	2.5% (-2.8%, 8.4%)
Infusion related reaction	3 (2.5%)	6 (5.0%)	9 (3.8%)	2.5% (-2.8%, 8.4%)
SERIOUS AND/OR OPPORTUNISTIC INFECTIONS INCLUDING PNEUMONIA	6 (5.0%)	4 (3.4%)	10 (4.2%)	-1.7% (-7.6%, 3.0%)
COVID-19	3 (2.5%)	2 (1.7%)	5 (2.2%)	-0.8% (-5.2%, 3.7%)
COVID-19 pneumonia	1 (0.8%)	0	1 (0.4%)	-0.8% (-4.4%, 2.8%)
Hepatitis carrier	2 (1.7%)	0	2 (0.8%)	-1.7% (-5.9%, 1.5%)
Hepatitis carrier disseminated	0	1 (0.8%)	1 (0.4%)	0.8% (-2.3%, 4.8%)
Pneumonia	0	1 (0.8%)	1 (0.4%)	0.8% (-2.3%, 4.8%)
Sepsis shock	0	1 (0.8%)	1 (0.4%)	0.8% (-2.3%, 4.8%)
Urinary tract infection bacterial	0	1 (0.8%)	1 (0.4%)	0.8% (-2.3%, 4.8%)

^aSubjects are counted once for each category and preferred term regardless of the number of events

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Background and aims: Approximately 15%–20% of patients with generalized myasthenia gravis (gMG) are acetylcholine receptor antibody (AChR-Ab) seronegative. Efgartigimod is a human immunoglobulin G1 (IgG1) antibody Fc fragment that reduces IgG levels (including pathogenic autoantibodies) via neonatal Fc receptor blockade. ADAPT SERON is a Phase 3 trial investigating the efficacy and safety of intravenous (IV) efgartigimod in adults with AChR-Ab seronegative gMG.

Methods: Diagnosis of gMG was confirmed by an MG diagnostic adjudication committee. In the double-blinded, placebo-controlled Part A, adult participants were randomized 1:1 to receive 4 once-weekly infusions of 10 mg/kg efgartigimod IV or placebo followed by a 5-week follow-up period. Part B includes an ongoing open-label extension (≤2 years).

Results: Results included 119 participants ($n=40$, MuSK-Ab+; $n=6$, LRP4-Ab+; $n=73$, triple seronegative), 58 received efgartigimod IV, and 61 received placebo. Change in Myasthenia Gravis Activities of Daily Living (MG-ADL) total score from baseline to week 4 (primary endpoint) was significantly ($p=0.007$) different between efgartigimod IV and placebo groups (overall population), with least squares mean change from baseline (90% CI) of -3.35 (-3.98 to -2.72) and -1.90 (-2.51 to -1.28), respectively. Clinically meaningful improvement in mean MG-ADL total score with efgartigimod IV (overall population) was observed in Part A and continued over subsequent cycles in Part B. Efgartigimod IV was well tolerated, with no new safety signals observed. Results from additional analyses will be presented at EAN 2026.

Conclusion: Efgartigimod IV demonstrated statistically significant improvement in MG-ADL total score compared with placebo and was well tolerated in AChR-Ab seronegative gMG.

Disclosure: This study was sponsored by argenx. RHJ, IS, DM, JTG, and W-YH are employees of argenx. SH, JFH, TV, CZ, SL, KGC, LR, EC, AA, and AB have reported financial/nonfinancial relationships with argenx at the time of submission.

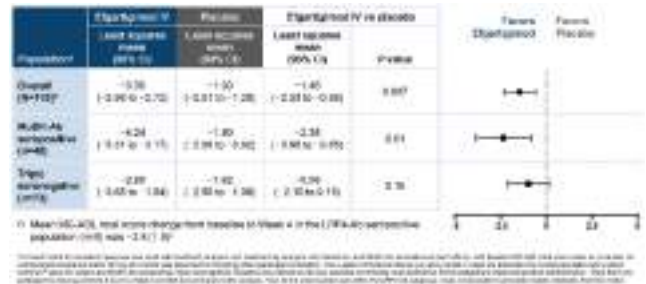


FIGURE 1 MG-ADL total score changes from baseline to Week 4.

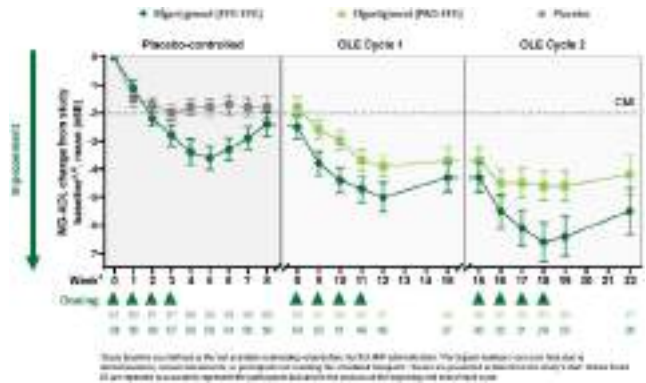


FIGURE 2 MG-ADL total score changes from study baseline over time—overall population.

TABLE 1 Summary of treatment emergent adverse events (TEAEs)—overall population.

	Efgartigimod IV (n=58; P101P102)		Placebo (n=61; P101P103)		Total Efg IV (n=119; P101P102)	
	n (%)	SD	n (%)	SD	n (%)	SD
≥1 TEAE	36 (62.1)	40.3	30 (49.2)	40.8	70 (58.1)	7.2
≥1 Serious TEAE	3 (5.2)	0.7	1 (1.6)	0.1	1 (0.8)	0.5
≥1 fatal TEAE	1 (1.7)	0.2	1 (1.6)	0.1	2 (1.7)	0.1
≥1 Grade ≥3 TEAE	3 (5.2)	0.7	1 (1.6)	0.1	1 (0.8)	0.5
≥1 life-threatening reaction event	1 (1.7)	0.1	3 (4.9)	0.3	3 (2.5)	0.3
Discontinuation in TEAEs	0	0	0	0	0 (0.0)	0.0
Most frequent TEAEs						
Upper respiratory tract infection	2 (3.4)	0.0	2 (3.3)	0.0	4 (3.3)	0.0
Headache	4 (6.8)	0.9	3 (4.9)	0.3	7 (5.8)	0.3
Nausea	2 (3.4)	0.0	7 (11.3)	1.3	13 (10.9)	0.4
Urinary tract infection	3 (5.2)	0.3	3 (4.9)	0.4	7 (5.8)	0.2
Fatigue	2 (3.4)	0.0	5 (8.2)	0.7	7 (5.8)	0.2

EPO-0642 | Effect of nivalocalimab on pathogenic IgG and complement markers in patients with seropositive gMG: Exploratory analysis in Vivacity-MG3 study

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Background and aims: Immunoglobulin (Ig)G class autoantibodies block and internalize the acetylcholine receptor (AChR) and activate complement at the neuromuscular junction, leading to symptoms of myasthenia gravis. Nivalocalimab, a fully human neonatal-Fc-receptor blocker, substantially reduced circulating IgG, including pathogenic subclasses, and demonstrated

sustained efficacy versus placebo during 24-week Vivacity-MG3 study (NCT04951622). This post-hoc analysis evaluated whether nivalimab reduces circulating complement components and activity indicative of complement activation, in Vivacity-MG3.

Methods: Available serum samples from seropositive (positive for AChR/muscle-specific receptor tyrosine kinase [MuSK]/Low-density Lipoprotein Receptor-related Protein-4 [LRP4]) participants were analysed at baseline and Week-24 and least square means were compared between placebo and nivalimab using linear mixed model. Complement markers measured were: C3d circulating immune complexes (C3d-CIC), C1q immune complex binding (C1q-ICB), and total complement activity (CH50). Parallel assays quantified total IgG, IgG1/2/3, and anti AChR titers, which informed a nonlinear mixed-model previously presented.

Results: Baseline levels of complement markers were comparable across nivalimab and placebo groups (Table-1). At Week-24, nivalimab was associated with substantial and sustained reductions in IgG1/2/3, and anti AChR titers (Table-2). Concomitant decreases were observed in selected immune complex-associated complement markers (C3d-CIC and C1q-ICB, $p < 0.05$); no significant change in CH50 was detected (Table 1).

Conclusion: Nivalimab led to sustained reductions in IgG subclasses and anti-AChR titers, accompanied by significant decreases in selected circulating immune complex-associated complement markers, while no significant change in total complement activity was observed. These data indicate changes in selected complement-related markers following nivalimab treatment; further exploration is needed to better understand the clinical relevance.

Disclosure: Sarah Hoffmann: Speakers' honoraria: Alexion, argenx, Grifols, Johnson & Johnson, Kyverna, Roche, UCB; honoraria/attendance at advisory boards: Alexion, argenx, Johnson & Johnson, Kyverna, Novartis, Roche; member of the medical advisory board: the German Myasthenia Society, DMG.

TABLE 1 Characteristics of complement protein measurements among seropositive patients.

Complement protein	Treatment	Time points		p-value	PDR ^{***}
		Baseline	Week 24		
CH50, activity units	Placebo	66 100.21 ± 5.65	58 101.22 ± 5.71	0.88	0.34
	Nivalimab	60 101.48 ± 5.97	54 99.34 ± 6.00		
C1q-ICB, µg	Placebo	66 6.35 ± 1.69	58 6.97 ± 1.71	0.81	0.03
	Nivalimab	60 7.40 ± 1.78	54 6.58 ± 1.79		
C3d-CIC, µg	Placebo	66 7.39 ± 0.96	58 7.23 ± 0.92	<0.001	<0.001
	Nivalimab	60 7.40 ± 0.94	54 7.64 ± 0.96		

Note: N indicates number of seropositive patients with measurable samples for complement proteins.
[†]LS mean adjusted values after accounting for confounding factors including age, sex, BMI, immunosuppression, time treatment arm, time point and treatment arm*time point.
^{**}PDR, multiple testing adjustment across the three complement proteins.
^{***}PDR, body mass index; C1q-ICB, circulating immune complex; C1q-ICB, immune complex binding; PDR, false discovery rate; LS, least square; SD, standard deviation.

TABLE 2 Relative change from baseline of IgG and anti-AChR antibodies in seropositive patients with gMG at week 24.

Titer	Placebo		Nivalimab	
	N	Observed mean % reduction [95% vpcPI]	N	Observed mean % reduction [95% vpcPI]
Total IgG	63	1.3 [-3.2; 3.3]	49	-67.9 [-69.9; -64.8]
IgG1	55	-2.0 [-4.0; 4.4]	54	-84.8 [-88.3; -62.4]
IgG2	55	1.8 [-4.7; 4.9]	54	-67.0 [-70.4; -63.1]
IgG3	55	3.1 [-5.4; 5.6]	54	-93.2 [-95.7; -89.9]
IgG4	55	-2.0 [-6.3; 6.3]	54	-34.0 [-62.0; -51.5]
Anti-AChR	46	-12.2 [-8.7; 6.6]	46	-69.1 [-72.9; -58.6]

Note: N indicates number of seropositive patients with samples available for modelling of antibody titer.
AChR, acetylcholine receptor; IgG, immunoglobulin G; vpcPI, visual predictive check prediction interval – a measure of model certainty in nonlinear mixed models.

Silke Miller, Yanfei Zhang, Jingyue Xi, Ruben Faelens, Marie Fitzgibbon, and Sindhu Ramchandren: Employees of Johnson & Johnson and may hold stock or stock options in the company.

Neurogenetics 4

EPO-0643 | Phenotypic and genotypic characterization of STUB1 variants in Portuguese pedigrees and analysis of TBP repeat expansions

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Background and aims: SCA48 (ATX-STUB1) is an adult-onset ataxia caused by heterozygous pathogenic variants in STUB1. Recently, families carrying pathogenic STUB1 variants and intermediate alleles in TBP gene have been described, raising the hypothesis of STUB1/TBP digenic inheritance or phenotypic modulation by TBP intermediate alleles. This study aimed to characterize the clinical, genetic and neuroimaging features of Portuguese families with SCA48 and analyze TBP repeat expansions.

Methods: SCA48 patients were identified through two sources: (a) Portuguese population-based survey on hereditary ataxias and (b) hospital-based cohorts from Northern Portugal. TBP repeat length was analyzed in all patients except one (deceased). Genetic, clinical and neuroimaging data were collected according to a standardized protocol.

Results: Seven pedigrees comprising 18 patients were identified, four from Northern Portugal and three from South. Five distinct STUB1 variants were detected: one in-frame deletion, one frameshift, two missense, and one nonsense variant. TBP intermediate alleles were present in 2 families. Mean age at onset was 41.5 years (range 28–64). Initial manifestations included gait ataxia ($n=12$), chorea ($n=1$), and diplopia ($n=1$). All patients developed cerebellar syndrome. Additional features included cognitive decline ($n=7$), depressive symptoms, chorea, and pyramidal signs ($n=6$, each). Neuroimaging revealed cerebellar vermal and hemispheric atrophy ($n=8$) and caudate nucleus atrophy ($n=3$). Twelve patients died after a mean disease duration of 17 ± 10.2 years.

Conclusion: Most Portuguese SCA48 pedigrees lacked TBP intermediate alleles, supporting a primary pathogenic role of heterozygous STUB1 variants. SCA48 presented as a complex adult-onset ataxia with prominent neuropsychiatric features and chorea.

Disclosure: Nothing to disclose.

Pedigree chart

I. II. III.

Subject Progress Reported age at onset		Exam 1 Age 1 History	Proband's mother Age 1 Admission 1	Proband's father Age 1 Birth history
First reported symptoms		Episodes: 10 minutes of brief and brief spells	Episodes: 10 minutes of brief and brief spells	Episodes: 10 minutes of brief and brief spells
Genetics	Gene	SCN1A	SCN1A	SCN1A
	Variant	SCN1A p.R1628C	SCN1A p.R1628C	SCN1A p.R1628C
	Pathogenic effect	Pathogenic	Pathogenic	Pathogenic
Neurological assessment	Seizures	Y	Y	Y
	Developmental delay	Y	Y	Y
	Intellectual disability	Y	Y	Y
	Autism	Y	Y	Y
	ADHD	Y	Y	Y
	Depression	Y	Y	Y
	Mania	Y	Y	Y
Psychiatric assessment	Psychiatric diagnosis	Psychiatric diagnosis	Psychiatric diagnosis	Psychiatric diagnosis
	Psychiatric treatment	Psychiatric treatment	Psychiatric treatment	Psychiatric treatment
	Psychiatric outcome	Psychiatric outcome	Psychiatric outcome	Psychiatric outcome
Genetic assessment	Genetic diagnosis	Genetic diagnosis	Genetic diagnosis	Genetic diagnosis
	Genetic treatment	Genetic treatment	Genetic treatment	Genetic treatment
	Genetic outcome	Genetic outcome	Genetic outcome	Genetic outcome
	Genetic diagnosis	Genetic diagnosis	Genetic diagnosis	Genetic diagnosis
	Genetic treatment	Genetic treatment	Genetic treatment	Genetic treatment
	Genetic outcome	Genetic outcome	Genetic outcome	Genetic outcome
	Genetic diagnosis	Genetic diagnosis	Genetic diagnosis	Genetic diagnosis
Neurophysiological assessment	Neurophysiological diagnosis	Neurophysiological diagnosis	Neurophysiological diagnosis	Neurophysiological diagnosis
	Neurophysiological treatment	Neurophysiological treatment	Neurophysiological treatment	Neurophysiological treatment
	Neurophysiological outcome	Neurophysiological outcome	Neurophysiological outcome	Neurophysiological outcome
Prognosis	Prognosis	Prognosis	Prognosis	Prognosis
	Prognosis	Prognosis	Prognosis	Prognosis
	Prognosis	Prognosis	Prognosis	Prognosis
Neuroimaging	Neuroimaging	Neuroimaging	Neuroimaging	Neuroimaging
	Neuroimaging	Neuroimaging	Neuroimaging	Neuroimaging
	Neuroimaging	Neuroimaging	Neuroimaging	Neuroimaging

Table 1: Demographic and clinical characteristics of three patients harboring a novel SCN1A mutation.

spectrum may vary from severe developmental delay, craniofacial dysmorphism and visceral tissue abnormalities to milder signs such as ataxia, nystagmus or strabismus. Defining non-canonical LOF phenotypes is critical to distinguish emerging multisystemic presentations from established LIWAS or epilepsy/PNKG spectra.

Results: We identified a novel heterozygous nonsense variant in *KCNMA1* (NM_001161352.2) [c.2005G>T p.Glu669Ter]. The phenotype manifests as upper limb hyperkinetic movements, psychiatric disturbances, and a shared metabolic phenotype characterized by chronic bronchial involvement (asthma/COPD), diabetes mellitus and liver disease, expanding the previous phenotype of *KCNMA1* LOF-related disorders.

Disclosure: Nothing to disclose.

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Background and aims: Omaveloxolone, an activator of Nrf2, has been approved by EMA for the treatment of Friedreich ataxia (FA) in patients aged ≥ 16 years. In Portugal, it was initially provided through a limited early access program from July 2024 to January 2025 (pending central approval), while reimbursement negotiations were ongoing. This study aimed to establish a national, prospective registry to systematically evaluate real-world effectiveness and safety in Portuguese FA patients receiving omaveloxolone.

Methods: Neurologists from 31 Portuguese hospitals were invited to participate. Study documentation was submitted for approval by ethics committees. Prospective assessments included mFARS, SARA, and FA-ADL at baseline, every 3 months during the first year, followed by 6-monthly assessments. Laboratory parameters and adverse events were monitored monthly during the first 3 months, quarterly until the end of the first year, and semiannually thereafter. Data were collected using REDCap.

Results: Fifteen hospitals (48.4%) agreed to participate, of which 13 actively followed FA patients. In Portugal, all genetically confirmed FA patients aged ≥ 16 years were eligible for treatment, irrespective of functional staging, provided no contraindications were present. A total of 67 patients were included: 39 (58.2%) receiving omaveloxolone, 27 (40.3%) waiting treatment initiation pending reimbursement negotiations, and 1 (1.5%) with cardiac contraindications to treatment. Two patients discontinued omaveloxolone during follow-up.

Conclusion: This registry demonstrates the feasibility of nationwide, prospective real-world monitoring of omaveloxolone in FA. While early access enabled treatment initiation in over half of eligible patients, a substantial proportion remained untreated. Continued follow-up will be essential to evaluate long-term safety and clinical outcomes as treatment availability expands.

Disclosure: Nothing to disclose.

EPO-0646 | Cortical metabolic signatures of visuospatial and gait dysfunction in GBA1-related Parkinson's disease

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Background and aims: GBA1 variants are the most common genetic risk factor for Parkinson's disease and are associated with an increased risk of cognitive decline. However, their impact on brain metabolism and related cognitive and motor features remains poorly understood: how visuospatial deficits reflect posterior cortical dysfunction and how these changes relate to other clinical manifestations? This study investigates cerebral metabolism and its association with clinical features in PD patients with and without GBA1 variants.

Methods: 38 GBA-PD and 46 non-GBA-PD underwent clinical, neuropsychological, and FDG-PET evaluation. Posterior cortical hypometabolism was quantified by single-subject SPM-analysis, and its association with visuospatial, motor, and non-motor features was assessed using Spearman correlations. Visuospatial function was assessed using a standardized posterior visuospatial composite score (PAROVIS).

Results: Compared with non-GBA-PD, GBA-PD patients were younger, had earlier disease onset, greater motor disability (including fluctuations and freezing of gait), higher depression, and worse quality of life, despite similar disease duration and education. FDG-PET revealed greater posterior cortical hypometabolism in GBA-PD. Cognitively, GBA-PD showed poorer visuospatial and executive performance, while other cognitive domains were comparable to non-GBA-PD. Within GBA-PD, the PAROVIS score correlated with posterior hypometabolism and freezing of gait severity, an association absent in non-GBA-PD ($\alpha = 0.77$).

Conclusion: Visuospatial dysfunction is a core cognitive feature of GBA-PD, associated with posterior cortical hypometabolism and gait impairment, even without global cognitive decline. These findings support a distinct posterior cortical-cognitive phenotype in GBA-PD, while longitudinal validation is needed to establish FDG-PET and visuospatial measures as reliable tools for monitoring disease progression and patient stratification.

Disclosure: Nothing to disclose.

EPO-0647 | Dystonia in Mowat-Wilson syndrome: A yet underrecognized neurological feature

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Background and aims: Mowat-Wilson syndrome (MOWS) is a rare neurodevelopmental disorder caused by heterozygous pathogenic variants in the ZEB2 gene. The neurological phenotype is characterized by intellectual disability, epilepsy, and characteristic craniofacial features. Based on the current literature, more than 300 individuals with MOWS have been reported. To date, only a single case report has documented dystonia in MOWS. The true frequency and clinical relevance of dystonia in MOWS therefore remain unclear.

Methods: We retrospectively identified three unrelated patients with genetically confirmed MOWS through a whole-exome sequencing (WES) project focused on individuals with dystonia. All patients were evaluated by a movement disorder specialist. Detailed clinical, genetic, and neuroimaging data were reviewed and compared with available literature.

Results: The case series included two females and one male, aged 5–21 years, all of whom exhibited dystonia. The onset of dystonia occurred between 3 and 18 years of age. Two patients developed generalized dystonia while one presented with a segmental type. WES identified heterozygous de novo ZEB2

variants in all cases: two missense variants classified as likely pathogenic and one frameshift variant classified as pathogenic. All patients showed a typical MOWS phenotype, including intellectual disability and distinctive craniofacial features. Epilepsy was present in one patient.

Conclusion: Our findings demonstrate that dystonia can occur in Mowat–Wilson syndrome and may represent an underrecognized feature of its neurological phenotype. Awareness of movement disorders in patients with ZEB2 mutations is important for accurate phenotyping and may facilitate earlier diagnosis, particularly in individuals with developmental delay and dysmorphic features.

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EPO-0648 | Therapeutic impact of iron chelation in PKAN and related NBIA subtypes: A systematic review of 552 cases

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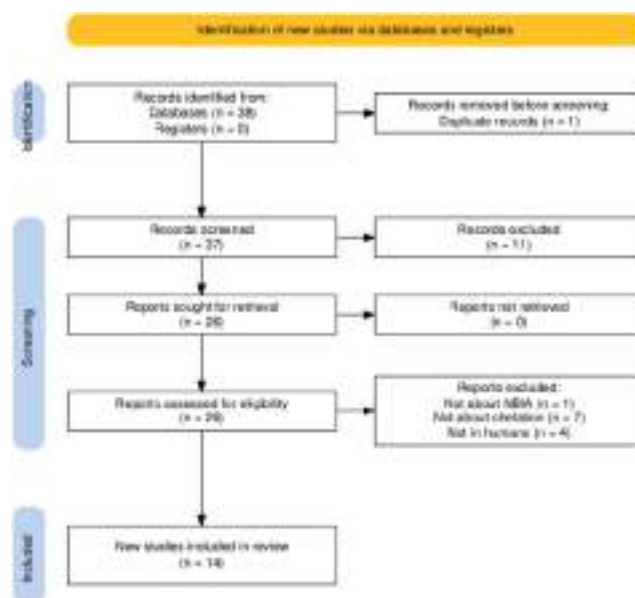
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Background and aims: Neurodegeneration with Brain Iron Accumulation type 1 (NBIA1), or Pantothenate Kinase-Associated Neurodegeneration, is a rare inherited neurodegenerative disorder characterized by iron deposition in the basal ganglia [1]. It affects fewer than one per million individuals. Iron chelation therapy has shown heterogeneous radiological and clinical outcomes [1].

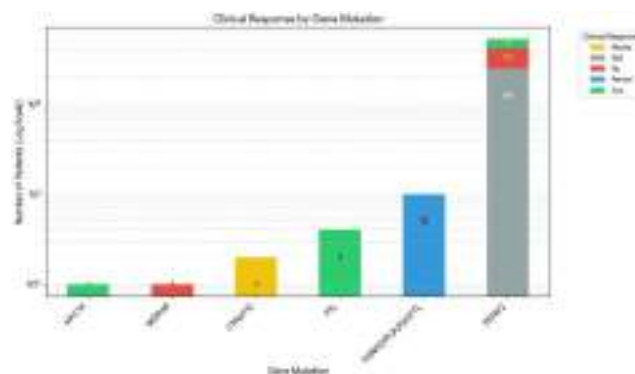
Methods: A systematic review of the literature was performed in accordance with PRISMA guidelines. PubMed, BVS and Google Scholar were searched using the keywords “Neurodegeneration with Brain Iron”, “Accumulation”, “NBIA1”, “Chelation Therapy”, “Deferiprone”.

Results: A total of 552 patients with NBIA with a mean age of 22.1 years were included. Pantothenate kinase-associated neurodegeneration (PKAN) was the predominant subtype accounting for 535 of the patients (96.9%); followed by other subtypes including MPAN ($n=2$), BPAN ($n=1$), neuroferritinopathy ($n=4$), VAC14-related NBIA ($n=1$) and idiopathic NBIA cases ($n=9$). 195 of the cases corresponded to males (53.6%) and 169 to females (46.4%). Defiprone was the most frequently used chelator, used at doses of 30 mg/kg/day varying in treatment duration from 17 weeks to 2 years. Reduction in brain iron was demonstrated radiologically in most cohorts leaning to stabilization and improvement with minimal reports of reversal. Hematologic events were reported as the main adverse effects accounting to 21% in the randomized data, the main cause of treatment discontinuation.

Conclusion: Iron chelation therapy with Deferiprone is associated with reduced brain iron and clinical stabilization in most



Prisma-P flowchart of the process of inclusion of studies in the systematic review.



Clinical response by gene mutation in iron chelation in PKAN and related NBIA subtypes.

NBIA1 patients, although neurological reversal is uncommon. Hematologic adverse effects limit treatment, emphasizing the need for safer long-term therapies and standardized outcome measures.

Disclosure: Nothing to disclose.

EPO-0649 | Clinical profile and treatment options in Nasu-Hakola syndrome: Systematic literature review

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Background and aims: Nasu-Hakola disease, or polycystic lipomembranous osteodysplasia with sclerosing leukoencephalopathy (PLOS), is a rare, autosomal-recessive, and fatal disorder caused by mutations in the TREM2 or TYROBP genes.



Clinical findings, diagnostic and treatment overview in Nasu-Hakola disease.

It is uniquely characterized by the co-occurrence of presenile dementia and cystic bone lesions, leading to death typically by the fifth decade [1–2]. This descriptive study aims to review its clinical manifestations, diagnostic findings and treatment.

Methods: A systematic review was performed following PRISMA guidelines. PubMed and BVS were searched using the keywords “Nasu-Hakola,” “PLOS,” and “brain-bone-fat disease”. Eligible studies were included and data were extracted into a standardized database for comparative analysis of clinical and treatment variables.

Results: Analysis of 12 patients (mean age 35.87 years; 33% familial) showed predominantly neurological onset (58%). Key findings included diffuse cerebral atrophy on MRI (91%), periventricular lesions (73%), basal ganglia abnormalities (64%), and bone cysts/lytic lesions (82%, 18% asymptomatic). Mutations were in TREM2 (58%) or TYROBP. Supportive treatments included antipsychotics/antiepileptics and fracture surgery; one case benefited from virtual reality neurorehabilitation.

Conclusion: This review confirms early neuropsychiatric presentation despite radiographic bone cysts. The diagnostic triad of frontal-predominant cerebral atrophy, characteristic bone lesions, and TREM2/TYROBP mutation is consistent. The universally fatal progression underscores the absence of disease-modifying therapies. Supportive care, including tailored neurorehabilitation, may ameliorate specific symptoms, but early genetic diagnosis remains crucial for definitive management and counseling.

Disclosure: Nothing to disclose.

EPO-0650 | Cerebrotendinous xanthomatosis mimicking or coexisting with juvenile ALS: A case with CYP27A1 and SETX mutations

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Background and aims: Cerebrotendinous xanthomatosis (CTX) is a rare autosomal recessive lipid storage disorder caused by CYP27A1 mutations, resulting in sterol 27-hydroxylase deficiency and systemic cholestanol accumulation. Amyotrophic lateral sclerosis (ALS) is the most common motor neuron disease; its juvenile form begins before age 25, and autosomal dominant ALS type 4 is associated with SETX mutations. Coexistence of CTX and ALS type 4 has not been previously described.

Methods: A 34-year-old male presented with progressive difficulty walking. Symptoms began 6 years earlier with gait imbalance, worsening weakness, and diffuse limb pain. His history included chronic diarrhea since infancy, febrile seizures in early childhood, bilateral cataract surgery at age 10, and hypophonia and dysarthria developing in his mid-20s. Neurological examination revealed severe tetraparesis with muscle atrophy, fasciculations, global hyperreflexia, bilateral clonus, and positive Babinski and Hoffman signs.

Results: Brain Magnetic Resonance Imaging showed bilateral cerebellar T2/FLAIR hyperintensities and periventricular white matter changes. Electromyography demonstrated widespread anterior horn cell involvement. Genetic testing identified a heterozygous SETX c.6625A>T (p.Lys2209Ter) mutation, confirming ALS type 4. Considering long-standing gastrointestinal, ophthalmologic, and neurological symptoms, CYP27A1 sequencing was performed and demonstrated a homozygous pathogenic variant (c.1263+1G>A), establishing CTX diagnosis. Treatment with riluzole and chenodeoxycholic acid was initiated.

Conclusion: This rare coexistence of CTX and ALS type 4 highlights the importance of recognizing metabolic warning signs such as chronic diarrhea and childhood cataract. Early metabolic and genetic evaluation is essential, as timely chenodeoxycholic acid therapy may slow neurological progression and improve long-term outcomes.

Disclosure: Nothing to disclose.

EPO-0651 | Beyond the classic phenotype: Optic neuropathy in a patient with Charcot-Marie-Tooth type 2Q

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Background and aims: Charcot-Marie-Tooth disease type 2 (CMT2) is a genetic disorder characterized by axonal involvement of the peripheral nerves, with a normally autosomal dominant inheritance pattern. There are multiple subtypes of CMT2, each defined by a different affected gene.

Methods: We present the case of a patient with optic neuropathy and axonal polyneuropathy caused by a mutation in the DHTKD1 gene.

Results: We have a 53-year-old male patient with no family history and a personal history of alcoholism who presents with sequential loss of visual acuity, as well as chronic paresthesia in all four limbs. Ophthalmological examination revealed bilateral papillary atrophy, and neurological examination revealed glove-and-stocking paresthesia, decreased vibratory sensitivity in all four extremities, and difficulty walking on heels and tiptoes. A blood test was performed with negative autoimmunity, normal cranial MRI, normal cerebrospinal fluid study, and neurophysiological study with data of bilateral axonal optic neuropathy and chronic axonal peripheral neuropathy. Genetic testing showed the c.1897-1G>A variant classified as pathogenic in heterozygosity in the DHTKD1 gene, associated with CMT2Q.

Conclusion: CMT2Q is a rare condition caused by mutations in the DHTKD1 gene, which usually presents with muscle weakness and progressive distal atrophy. The presence of optic neuropathy in our patient is an atypical finding, more commonly

described in the CMT2A subtype. We believe that the mitochondrial metabolism disorder caused by the DHTKD1 mutation, together with alcohol abuse, may have acted synergistically, amplifying the phenotype of this disorder.

Disclosure: Nothing to disclose.

EPO-0652 | NOTCH3 rs201118034 is not an independent risk factor of vascular Parkinsonism among Taiwanese population

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Background and aims: NOTCH3 p.R544C mutation is a common hotspot for developing monogenetic leukoencephalopathy among Taiwanese population. Clinically, this mutation can cause a variable clinical presentations, including vascular Parkinsonism (VaP). This study aimed to investigate the risk of VaP in the generalized Taiwanese population utilizing single nucleotide polymorphisms (SNPs).

Methods: Data were retrieved from the Taiwan Biobank Database (2008–2020) and the National Health Insurance Research Database (2000–2020), and they were linked using encrypted personal identification numbers. After excluding participants with a history of stroke, dementia, or Parkinson's disease other than VaP, a total of 147 patients with VaP and 98,699 control subjects were included in the analysis. In this study, VaP was defined specifically with ICD-10 of G21.4 and G21.8. rs201118034 was used to represented the NOTCH3 p.R544C mutation.

TABLE 1 Multivariable logistic regression analysis of vascular Parkinsonism focusing on NOTCH3 rs201118034.

	OR	95 % CI		p value
NOTCH3 rs201118034 (ref: GG)				
AG	2.72	0.86	8.59	0.089
AA	<0.001	<0.001	>999.999	0.989
Sex (ref: Female)				
Male	0.75	0.50	1.14	0.178
Age (ref: 30-40)				
41-50	1.35	0.59	3.11	0.480
51-60	1.83	0.85	3.94	0.125
61-70	6.56	3.10	13.87	<0.001
Smoke (ref: No)				
Yes	0.84	0.50	1.42	0.518
Alcohol drink (ref: No)				
Yes	1.12	0.60	2.08	0.729
Physical activity (ref: No)				
Yes	1.08	0.77	1.52	0.659
BMI (ref: 18.5-24)				
<18.5	2.32	0.99	5.43	0.053
24-27	1.57	1.08	2.28	0.019
≥27	0.81	0.50	1.33	0.401
Diabetes (ref: No)				
Yes	1.90	1.32	2.72	0.001
Hypertension (ref: No)				
Yes	2.15	1.46	3.15	<0.001
Hyperlipidemia (ref: No)				
Yes	1.73	1.13	2.66	0.012

TABLE 2 Multivariable logistic regression analysis of vascular Parkinsonism focusing on NOTCH3 rs201118034 (dominant model).

	OR	95 % CI		p value
NOTCH3 rs201118034 (ref: GG)				
AG+AA	2.71	0.86	8.56	0.090
Sex (ref: Female)				
Male	0.75	0.50	1.14	0.177
Age (ref: 30-40)				
41-50	1.35	0.59	3.11	0.481
51-60	1.83	0.85	3.94	0.126
61-70	6.56	3.10	13.87	<0.001
Smoke (ref: No)				
Yes	0.84	0.50	1.42	0.519
Alcohol drink (ref: No)				
Yes	1.12	0.60	2.08	0.728
Physical activity (ref: No)				
Yes	1.08	0.77	1.52	0.659
BMI (ref: 18.5-24)				
<18.5	2.32	0.99	5.43	0.053
24-27	1.57	1.08	2.28	0.019
≥27	0.81	0.5	1.33	0.402
Diabetes (ref: No)				
Yes	1.90	1.32	2.72	0.001
Hypertension (ref: No)				
Yes	2.15	1.46	3.16	<0.001
Hyperlipidemia (ref: No)				
Yes	1.73	1.13	2.66	0.012

Results: Among the multivariable logistic regression analysis, the AG+AA genotype on rs201118034 has a higher, but not significant, odds ratio of developing VaP (OR=2.71, $p=0.090$). However, in the regression model, a higher age group between 61 and 70years-old was a significantly independent risk factor of VaP (OR=6.56, $p<0.001$). Lastly, diabetes (OR=1.90, $p=0.001$), hypertension (OR=2.15, $p<0.001$), and hyperlipidemia (OR=1.73, $p=0.012$) still remained independent predicting factors of developing VaP in our population.

Conclusion: Our findings suggested that the risk of VaP of NOTCH3 p.R544C carrier is not determined independently via this mutation. An elder age, hypertension, diabetes and hyperlipidemia remained independent and critical factors in developing VaP.

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EPO-0653 | Depression and mood changes in Parkinson's disease over time. A 5-year follow-up study

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Background and aims: Depression is frequent in Parkinson's disease (PD), but it is unclear how mood changes and impacts patient's quality of life (QoL) over time. Our objective was to analyze the frequency of depression and mood changes in people with PD (PwP) over 5 years of follow-up, comparing it with a control group, as well as its relationship with the patients' QoL.

Methods: PwP and healthy controls (HC) recruited from the COPPADIS cohort from January/2016 to November/2017 were included in this 5-year follow-up study. Mood was assessed by the Beck Depression Inventory II (BDI-II), and participants were classified as having major depression, minor depression, subthreshold depression, or non-depression at baseline and at 2, 4, and 5 years of follow-up. Correlation analysis and linear regression models were conducted to determine how different factors contribute to mood changes.

Results: The BDI-II total score increased ($p < 0.0001$) from 8.1 ± 6.2 at baseline to 10.3 ± 8.1 at the 5-year follow-up visit in PwP but not in HC (from 4.1 ± 5.2 to 4.0 ± 5.7 [$p = 0.896$]). The prevalence of depression remained around 50% in the PwP group and 25% in the HC group throughout the follow-up period, but the mood state showed variability for each patient between visits (Figure 1). Patients' QoL was associated with the depressive state throughout the entire follow-up period (Figure 2). Worsening QoL, sleep, longer disease duration, and an increase in neuropsychiatric symptoms were identified as independent

TABLE 1 Factors associated with mood (BDI-II score) change from baseline (V0) to the 5-year follow-up visit (V5) (BDI-IIV5 – BDI-IIV0) in PD patients (N=346).

	β^a	95% CI ^b	p	Number of patients	Adjusted R ²	β^a	95% CI ^b	p ^c
Change in the BDI-II score (V5-V0)	-0.004	-0.008 - 0.000	<0.0001	346	0.00	-0.000	-0.013 - 0.011	0.984
Change in the NPI	0.000	0.000 - 0.000	<0.0001			0.000	0.000 - 0.000	0.997
Change in the PDSS	-0.014	-0.016 - -0.009	<0.0001			-0.001	-0.008 - 0.005	0.861
Change in the PDSS-19	0.012	0.009 - 0.014	<0.0001			0.000	0.000 - 0.001	0.999
Disease duration	0.000	-0.000 - 0.000	0.598			0.000	-0.001 - 0.001	0.999

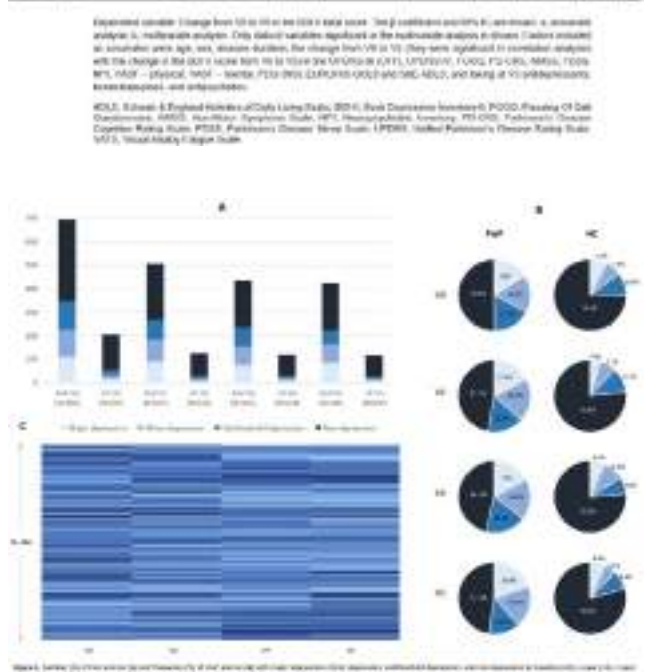


FIGURE 1 Number (N) (A) and frequency (%) (B) of PwP and HC with major, minor, subthreshold and no depression at baseline (V0), 2-year (V2), 4-year (V4) and 5-year (V5) follow-up. (C) Mood state for each patient from the PwP group at each visit.

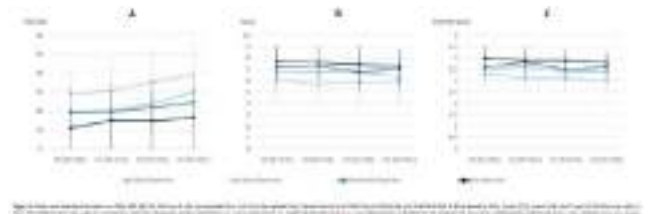


FIGURE 2 Mean and standard deviation of PDQ-39SI (A), PQ-10 (B) and EUROHIS-QOL8 (C) at baseline (V0), 2-year (V2), 4-year (V4) and 5-year (V5) follow-up in PwP. ANOVA comparisons between depression groups showed $p < 0.0001$.

factors associated with a worsening of mood over time in PwP (N=348) (Table 1).

Conclusion: Mood changes over time in PwP and are associated with QoL.

Disclosure: Nothing to disclose.

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Background and aims: Amygdala is crucial in emotional regulation and integrating interoceptive information, autonomic control, and motor behavior. The study aimed to assess amygdala volume and functional connectivity (FC) alterations in patients with Parkinson's disease with and without freezing of gait (PD-FoG, PD-NO-FoG).

Methods: Thirty PD-FoG patients, 30 PD-NO-FoG patients (with postural instability and gait disorders), and 30 matched healthy controls underwent neuropsychological, structural, and functional magnetic resonance imaging (MRI) assessments. Amygdala was segmented using FSL-FIRST. Based on volumetric findings, right amygdala was used as region of interest for seed-based FC analyses.

Results: PD-FoG and PD-NO-FoG patients showed comparable cognitive profiles, with impairments in memory, visuospatial, and executive-attentional domains, as well as increased depressive symptoms. MRI analyses revealed increased amygdala volume in PD-FoG compared with healthy controls. Resting-state fMRI analyses showed that, relative to controls, PD-FoG patients exhibited increased FC between the right amygdala and limbic-striatal regions, including the left thalamus, putamen, insula, and inferior frontal orbital cortex, along with reduced connectivity with the middle temporal gyrus and cerebellar regions (Crus I and lobules VI–VIII). Compared with PD-NO-FoG patients, PD-FoG patients showed reduced FC between the right amygdala and middle/superior temporal, inferior parietal, post-central gyri, insula, and fusiform cortex.

Conclusion: Increased amygdala–limbic-striatal connectivity in PD-FoG may reflect stronger emotional influence on motor circuits, contributing to impaired automatic gait control. Reduced connectivity with temporal, parietal, and cerebellar regions suggests disrupted integration across emotional,

cognitive, and motor networks, supporting the hypothesis that FoG arises from impaired network communication underlying gait automaticity.

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Background and aims: Gait disturbances are a core feature of people with Parkinson's disease (pwPD) and are influenced by motor, cognitive, and emotional factors, reflecting the reciprocal link between emotion and movement described by embodied emotion theories. This study examined whether observing emotional gait modulates gait performance and locomotor network activity in pwPD and healthy controls, focusing on the effects of positive and negative emotions on gait parameters and sensorimotor activation.

Methods: Thirty-one pwPD and twenty-six age-/sex-matched HC underwent gait analysis and task-based functional MRI (fMRI). Gait was assessed during spontaneous walking, top-down embodiment (self-evoked emotions), and bottom-up embodiment (imitation after observing emotional gait videos depicting happiness, sadness, anxiety). fMRI data were collected during emotional gait observation and analyzed using permutation-based nonparametric statistics focusing on sensorimotor regions.

Results: Both groups accurately recognized emotional gait and reported similar valence and intensity. Happiness improved gait in pwPD and HC, especially during bottom-up embodiment, enhancing stride length, arm kinematics, and turning velocity, whereas sadness and anxiety worsened gait performance, increasing variability and reducing pace. fMRI showed emotion-specific sensorimotor modulation, with happiness enhancing basal ganglia and sensorimotor activation and negative emotions reducing it. In pwPD, these neural patterns correlated with emotion-specific changes in gait during bottom-up embodiment.

Conclusion: Emotions modulate gait and its neural substrates in pwPD and HC. Positive emotional cues enhance motor system

engagement even in pwPD, whereas negative emotions exert detrimental effects. These findings support the integration of emotionally enriched action observation into gait rehabilitation and highlight the relevance of emotional states in clinical evaluation.

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Background and aims: We previously reported that patients receiving an optimized ND0612 regimen experienced a lower incidence of falls during the double-blind phase of the BouNDless trial, vs those treated with levodopa/carbidopa (IR-LD/CD) therapy (7% vs 12%, respectively).

Methods: We (a) examined the baseline characteristics of patients who experienced recurrent (>1) falls using an integrated safety database (ISD), and (b) explored potential factors contributing to the reduced fall rates. Postural instability and gait disorder (PIGD) and freezing of gait (FOG) were measured using MDS-UPDRS items; cognitive function via PDQ-39 cognitive scores; and dyskinesia using Hauser diaries.

Results: Patients with recurrent falls were older (62% aged ≥65 years vs 49% in the overall cohort), had lived with their PD for longer (time since diagnosis of 10.7 vs 9.4 years), and had higher (worse) motor severity (MDS-UPDRS Part III scores of 37.6 vs 34.5). In the BouNDless trial, ND0612 improved PIGD scores vs IR-LD/CD (improvement of +0.26 vs -0.02), reduced FOG (improvement of +0.33 vs +0.18) and enhanced cognition (improvement of +0.90 vs +0.31). Mean ON-time with troublesome dyskinesia decreased with ND0612 from 0.71 to 0.45 h.

Conclusion: Treatment with ND0612 has a positive effect on the various factors associated with falling in PD, and support the finding of reduced falls with optimized ND0612 therapy.

Disclosure: Nothing to disclose.

EPO-0658 | Long-term efficacy of ND0612 for motor fluctuations in Parkinson's disease

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Background and aims: Primary efficacy analyses from the double-blind phase of the BouNDless study demonstrated that treatment with ND0612 provided patients with Parkinson's

disease (PD) an additional 1.72 hours (h) of Good ON-time compared with levodopa/carbidopa (IR-LD/CD) ($p < 0.0001$). We describe here one-year efficacy outcomes from the ongoing open-label extension (OLE) phase.

Methods: Patients who completed the double-blind phase of the BouNDless study were eligible to enter the ongoing OLE phase (up to 54 months). All anti-PD medications were adjusted according to individual response. Efficacy was assessed with the Hauser diary at Months 6 and 12 of the OLE phase. Changes in ON-time with troublesome dyskinesia were analyzed for the subgroup of patients who had ≥1 h troublesome dyskinesia at treatment onset.

Results: Of 232 participants ($n = 113$ previously randomized to ND0612, $n = 119$ previously randomized to IR-LD/CD), 167 (72%) completed 1 year of ND0612 treatment in the OLE phase. By Month 6, patients had a mean ± SE change of -2.2 ± 0.2 h in OFF-time and $+2.4 \pm 0.2$ h in Good ON-time. Efficacy benefits were sustained at Month 12 with a change of -2.1 ± 0.2 h in OFF-time and $+2.1 \pm 0.2$ h in Good ON-time. The proportion of patients with ≥12 hours of Good ON-time increased from 16.4% before initiating ND0612 to 57.9% at Month 6 and 54.6% at Month 12. Patients who had ≥1 h of ON-time with troublesome dyskinesia at ND0612 initiation ($n = 45$) showed a change of -1.2 ± 0.3 h at Month 6 and -1.5 ± 0.3 h at Month 12.

Conclusion: These data support the long-term efficacy of ND0612 for PD patients experiencing motor fluctuations.

Disclosure: Nothing to disclose.

EPO-0659 | Efficacy of inhaled levodopa by age-based subgroups in patients with Parkinson disease: Post hoc analysis of pooled data from three phase III trials

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Background and aims: Many people with Parkinson disease (PwP) develop motor response fluctuations (OFF episodes) within 5 years of starting oral levodopa. CVT-301 is an orally inhaled formulation of levodopa developed for the treatment of episodic OFF periods. This analysis aimed to assess the efficacy of CVT-301 (84 mg) in reducing motor symptoms and improving patient-reported outcomes in PwP across age-based subgroups.

Methods: This descriptive post hoc analysis pooled data from three phase III clinical trials (CVT-301-004, CVT-301-004e, and CVT-301-005). Analyses were conducted across subgroups based on age at time of diagnosis, age at start of first OFF episode, and age at initiation of study treatment, defined as <55, 55–64, 65–74, and ≥75 years. Efficacy endpoints included Patient Global Impression of Change (PGIC) at Week 12 and change from baseline in Unified Parkinson's Disease Rating Scale Part III (UPDRS III) score from pre-dose to 30 minutes post-dose at Week 12.

Results: A total of 437 PwP were pooled. The mean age (range) was 63.5 (37–82) years; 64.3% were male and 96.3% were White. The mean (standard deviation [SD]) time since diagnosis was 8.7 (4.54) years. The mean (SD) time since first OFF episode was 3.8 (3.07) years. The mean change in UPDRS-III score from pre-dose to 30 minutes post-dose at Week 12 showed improvements across all age subgroups. Similarly, the PGIC consistently showed improvements at Week 12.

TABLE 1 Change in UPDRS-III score from pre-dose to 30 minutes post-dose and improvement on PGIC at Week 12 across age subgroups.

	Change in UPDRS-III score from pre-dose to 30 minutes post-dose at Week 12, N = 344 ^a		Improvement on PGIC at Week 12, N = 381 ^a	
	n	Mean (SD)	n improved/N	%
Age at first diagnosis				
<55 years	102	-15.6 (12.02)	139/170	81.8
55-64 years	145	-13.6 (8.86)	130/157	82.8
65-74 years	44	-15.6 (10.33)	46/51	90.2
≥75 years	3	-5.7 (8.99)	1/3	33.3
Age at first OFF episode				
<55 years	90	-15.2 (12.61)	82/102	80.4
55-64 years	104	-14.4 (9.77)	139/167	83.2
65-74 years	86	-15.1 (9.77)	83/96	86.5
≥75 years	14	-12.0 (10.32)	12/16	75.0
Age at study treatment initiation				
<55 years	55	-16.4 (13.84)	46/63	73.0
55-64 years	129	-14.2 (10.31)	116/136	84.8
65-74 years	131	-14.7 (9.94)	126/146	86.3
≥75 years	31	-13.6 (7.74)	27/34	79.4

^aObserved cases analysis

PGIC, Patient Global Impression of Change; SD, standard deviation; UPDRS-III, Unified Parkinson's Disease Rating Scale Part III.

Conclusion: Use of CVT-301 is an effective option for managing OFF episodes in PwP, with consistent motor symptom relief and patient-reported improvement across all ages.

Disclosure: This study was funded by Merz Therapeutics GmbH.

EPO-0660 | Familial tremor linked to a heterozygous variant in the ceruloplasmin gene

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Background and aims: Biallelic mutations in the ceruloplasmin (CP) gene cause aceruloplasminemia, a rare autosomal recessive disorder characterized by systemic and cerebral iron overload. Here we describe the clinical, genetic, and radiologic features of a family carrying a heterozygous variant in the CP gene.

Methods: The proband was an 18-year-old girl presenting action tremor in the upper limbs, which was also observed in her father and older sister. Serum ceruloplasmin levels and brain Magnetic Resonance Imaging was assessed in all three subjects. The proband was tested by New Generation Sequencing for a panel of genes associated with movement disorders. The results were validated and extended to the parents and the older sister by Sanger sequencing.

Results: The analysis revealed a heterozygous variant c.188T>C in the CP gene, introducing a missense change p.(Ile63Thr) in the proband. The variant was inherited from the father and shared with the affected sister.

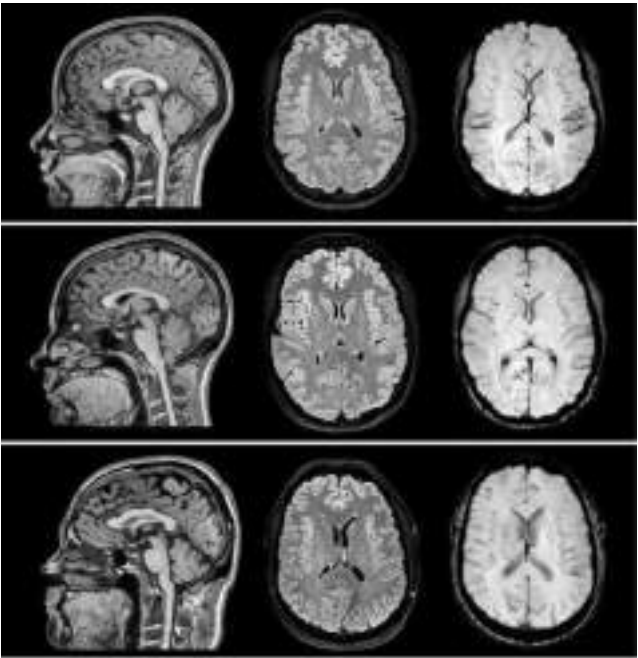


FIGURE 1 Brain MRI scans of the three subjects harboring the heterozygous CP gene variant: the proband, the older sister, and the father, from top to bottom. From left to right are shown a volumetric T1-weighted, a T2-weighted FLAIR and an SWI sequences.

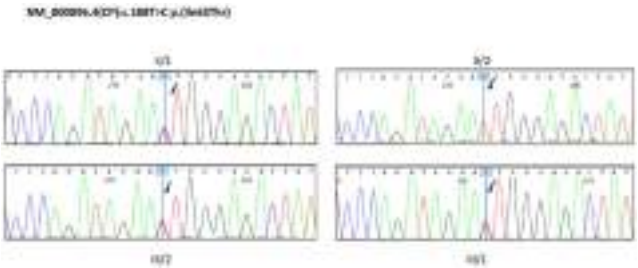


FIGURE 2 Sanger sequencing showing the CP mutation (c.188T>C, arrows) in the proband (III/2), her older sister (III/1) and her father (II/1), but not in the mother (II/2).

Conclusion: In our family the CP variant segregated with the clinical phenotype, characterized by action tremor, suggesting that this mild neurological manifestation may be likely attributable to the variant rather than representing an incidental finding. Neurological symptoms may occur in heterozygous carriers of CP gene variants, although they are usually believed to be asymptomatic.

Disclosure: Nothing to disclose.

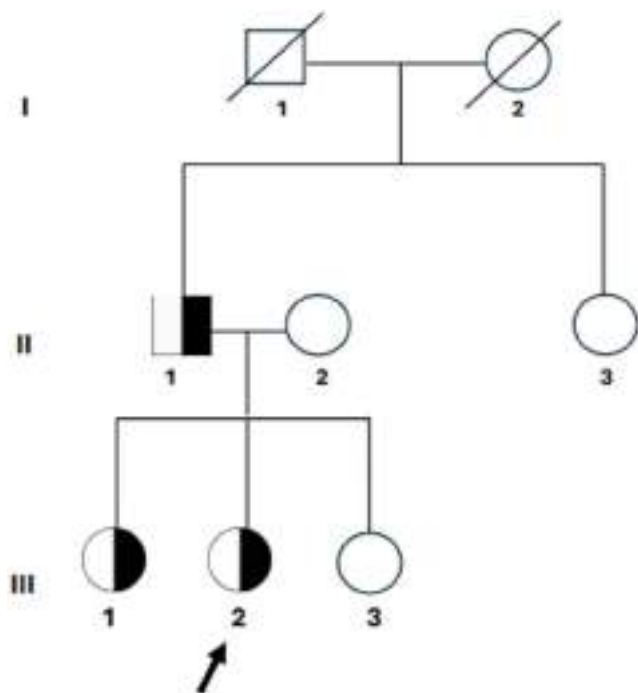


FIGURE 3 Family pedigree showing that the heterozygous variant, carried by the proband (arrow), her father and older sister, segregates with the phenotype. The younger sister of the proband has not been tested due to the minor age and absence of symptoms.

EPO-0661 | fMRI activity during working memory manipulation is associated with caudate integrity in early Parkinson's disease

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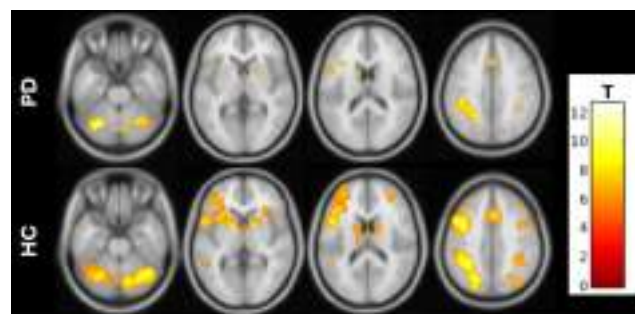
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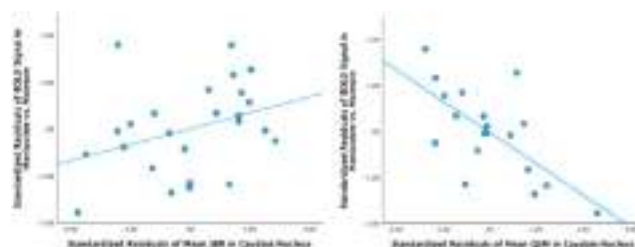
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Background and aims: Cognitive difficulties are common in Parkinson's disease (PD). Specifically, manipulation in working memory (WM) seems particularly vulnerable as it relies on frontostriatal networks. However, it is still relatively unclear how early alterations in nigrostriatal integrity – quantified as dopamine transporter (DaT) availability and iron depositions via Quantitative Susceptibility Mapping (QSM) – may shape cognitive neural responses. Thus, we aimed to investigate the relationship between caudate integrity and neural activity during WM manipulation in early PD.

Methods: 25 patients and 35 matched healthy controls (HC) performed a validated WM paradigm to identify neural activity specifically associated with WM manipulation. A two-sample t-test was used to test for group differences. Mean specific binding



T-maps for the WM manipulation contrast ($p < 0.05$, FWE-corrected). Upper row: PD, lower row: HC.



Scatterplots of the partial correlations between individual BOLD signal during WM manipulation and mean caudate DaT SBR (left) or mean caudate QSM (right) corrected for age and sex. For illustration, standardized residuals of BOLD activity and DaT SBR or.

ratios (SBRs) from DaT-SPECT and susceptibility values from QSM MRI were computed for the caudate. Mean BOLD activity was partially correlated with caudate SBR and QSM values.

Results: Both groups showed robust widespread activation in the frontoparietal, cerebellar and anterior striatal regions (Figure 1). There were no group differences in activation and task performance. Within the PD group, higher caudate DaT SBRs were trend associated with stronger BOLD responses (partial $r = 0.342$, one-sided $p = 0.055$), while higher caudate iron load as indexed via QSM correlated with reduced BOLD activity (partial $r = -0.681$, one-sided $p = 0.003$) during WM manipulation (Figure 2).

Conclusion: Patients show preserved large-scale WM activation patterns. However, task-related BOLD modulation during WM manipulation relates to nigrostriatal integrity within the caudate nucleus. These results highlight caudate-dependent modulation of executive processes within WM in early PD.

Disclosure: This work was supported by the Deutsche Forschungsgemeinschaft (DFG, German Research Foundation)—Project-ID 431549029—SFB 1451/C03. H.T. was supported by the Cologne Clinician Scientist Program (CCSP)/ Faculty of Medicine/University of Cologne, funded by the DFG—Project ID 413543196. 1 Giehl K, Ophey A, Hammes J, Rehberg S, Lichtenstein T, Reker P, Eggers C, Kalbe E, van Eimeren T. Working memory training increases neural efficiency in Parkinson's disease: a randomized controlled trial. Brain communications. 2020;2(2):fcaa115.

EPO-0662 | Multisite ultrasound investigation of vagus nerve echostructure, regional substantia nigra, extraneural markers in Parkinson's disease: MULTIVERSE-PD.

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Background and aims: Ultrasound sonography (US) is a reliable cost-effective tool to investigate brain and nerve tissue features. Substantia Nigra (SN) US has been extensively evaluated in Parkinson's Disease (PD) [1]. Along with SN, recent studies also examined vagus nerve (VN) US features, with alternate results [2]. The MULTIVERSE-PD study aims at implementing the use of US biomarkers in PD. In this preliminary phase we aimed at implementing the use of SN and VN sonography in a well characterized sample of PD with mobility and fluid biomarkers.

Methods: VN cross sectional area (VN-CSA) and SN hyperechogenic area (SN-HA) were consecutively collected in mild-to-moderate PD patients. Participants were clinically assessed through MDS-UPDRS, SCOPA, PDSS2, ESS, PDQ8 and other scales. All subjects underwent real-world mobility evaluations through IMUs (McRoberts MoveMonitor) and plasmatic inflammatory and neurodegeneration biomarker dosing.

Results: Twenty patients were recruited (10 male, 10 female; age 66 ± 9 ; disease duration 7.3 ± 4 ; MDS UPDRS III 26 ± 12). Mean SN-HA was 0.4 ± 0.1 cm², VN-CSA was 1.4 ± 0.5 cm². SN-HA was significantly related ($p < 0.01$) to total scores of MDS-UPDRS part I and II, SCOPA, NMSS ($r = 0.5-0.8$), PDSS2 ($r = 0.7$) and PDQ8 ($r = 0.5$). VN-CSA inversely related ($p < 0.01$) to MoCA ($r = 0.7$) and directly to ESS ($r = 0.5$).

Conclusion: SN and VN echographic features are promising biomarkers in PD. The MULTIVERSE-PD study will collect more echographic biomarkers from neural and extraneural targets that will be harmonized with mobility and biochemical parameters through artificial intelligence. A multicenter effort is required.

Disclosure: Nothing to disclose.

EPO-0663 | Adult Tourette syndrome: Patient experience and clinical insights from a UK Regional Neurology Centre

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Background and aims: This study investigated tic phenomenology, psychiatric comorbidity and clinical pathways in adults with Tourette Syndrome (TS). Variation in access to appropriate services can prolong the diagnostic journey and pose challenges for management.

Methods: Retrospective analysis of electronic clinical records for adults with TS attending a regional neurology Centre, The Walton Centre NHS Foundation Trust (Liverpool, UK), between

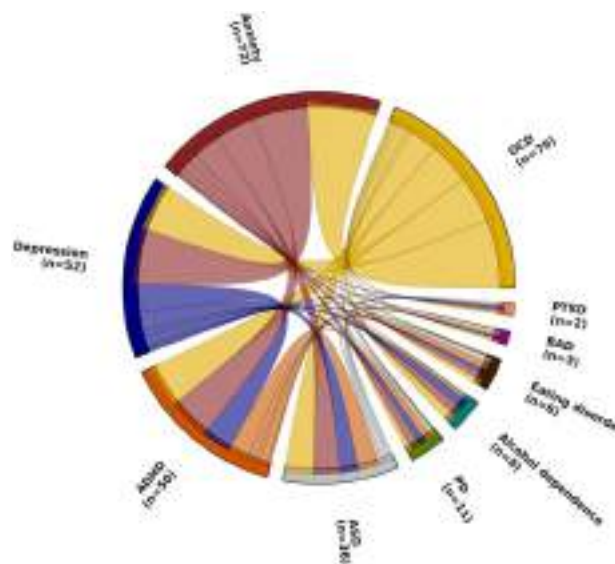


FIGURE 1 Co-existing mental health and neurodevelopmental disorders in adults with TS. Data shown for 118 patients (ten others did not have any mental health or neurodevelopmental diagnoses).

November 2020 and November 2025. Data collected included demographics, tic characteristics and medication use.

Results: Of 128 patients, 56 were women and the mean age was 30.6 years. The mean age of tic onset was 8.9 years and at TS diagnosis was 24 years. The estimated mean time from symptom-onset to diagnosis was 184 months. In childhood, 14 patients had contact with paediatric neurology services. The clinical course of TS worsened for 46.9%. Tic-related pain was experienced 26.6%. For tic management, 20.3% tried behavioural interventions and 66.4% tried medications (most commonly aripiprazole, clonidine and risperidone). Two patients were treated with deep brain stimulation at University College of London. Coexisting psychiatric disorders affected 118 (92.2%) individuals and, of these, 62 (52.5%) had three or more psychiatric diagnoses.

Conclusion: Most patients did not have access to appropriate TS services in childhood and faced a prolonged diagnostic delay. Only one-fifth of patients had access to gold standard tic treatment with behavioural interventions, as this is not funded by the National Health Service. The high rate of psychiatric comorbidity underlines the importance of multidisciplinary management.

Disclosure: Nothing to disclose.

Neuroimmunology 3

EPO-0664 | The invisible enemy : Shedding light on antibody-negative autoimmune encephalitis

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Background and aims: Despite diagnostic criteria proposed in 2016, antibody-negative autoimmune encephalitis (AE-) remain diagnostically elusive, often delaying access to effective treatments. While antibody-positive AE (AE+) has been extensively characterized, AE- remains underexplored.

Methods: We built a multicenter database from the three French AE reference centers (Lyon, Paris, Toulouse), including patients diagnosed between 2016 and 2023, and followed for 2 years. This preliminary analysis includes 139 patients from Paris and Toulouse.

Results: We identified 110 AE+ (79%) and 29 AE- (21%). Mean age (56 years old) and sex-ratio (1:1) were similar. AE- occurred more frequently in a post-infectious context (17% vs 3%, $p=0.01$) and showed less epilepsy (21% vs 43%, $p=0.03$), and a trend to more neuro-ophthalmologic involvement (31% vs 15%, $p=0.07$). Non-limbic presentations were also more frequent in AE- (45% vs 20%, $p=0.01$). Despite these differences, the clinical severity was comparable between AE- and AE+, with similar rates of intensive care unit admissions (30% and 28%, respectively) and clinical scores (median modified Rankin score = 3 and CASE = 5 in both groups). Nevertheless, the prognostic RAPID score was higher in AE- (3 vs 1, $p=0.0001$). On complementary investigations, AE- more frequently exhibited basal ganglia hypermetabolism on brain positron-emission tomography (100% vs 39%, $p=0.0169$), and diffuse or focal slowing on electroencephalography (63% vs 36%, $p=0.02$). CSF cell count and proteins were similar, but interleukin-6 levels were higher in AE- (21.5 pg/mL vs 3.4 pg/mL, $p=0.0265$).

Conclusion: These preliminary findings suggest that AE- follows a distinct clinico-radiological profile. Early identification may help guide more timely and targeted treatment strategies.

Disclosure: Nothing to disclose.

EPO-0665 | Real-world experience of transition from intravenous to subcutaneous Ocrelizumab in multiple sclerosis

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Background and aims: Subcutaneous (SC) Ocrelizumab has demonstrated non-inferiority to the intravenous (IV) formulation in Multiple Sclerosis (MS). However, real-world data on the transition from IV to SC remain limited. We aimed to evaluate patient-reported outcomes (PROM), safety and logistical impact associated with its transition in clinical practice.

Methods: We conducted a retrospective study including all patients treated with Ocrelizumab in 2025, at a tertiary MS center. Demographic and clinical data were collected. Adverse events (AEs) were graded according to CTCAE v5.0. Patient satisfaction and experience with SC administration were assessed using a modified version of Treatment Administration Satisfaction Questionnaire.

Results: Of 115 patients treated with Ocrelizumab, 61 (53%) received the SC formulation, and 51 of these completed PROMs. Thirty (58.8%) were men, with a mean age of 45.5 ± 13.0 years and a median disease duration of 9 (IQR 1–35) years. Forty-four (86.3%) patients previously received IV Ocrelizumab. The

median number of SC administrations was 1 (IQR 2). AEs occurred in 70.6% (36/51), predominantly mild (grade 1) local injection-site reactions. Overall, 82.3% of patients (42/51) were satisfied or very satisfied with the administration, 86.3% found it convenient, 81.8% (36/44) perceived time savings compared with IV infusion. Forty-eight percent preferred SC formulation, 40.9% were neutral. No clinical relapses were observed, CD19+ B-cell counts remained ≤ 5 cells/ μ L in 96% (24/25). The first SC administration reduced hospital time by 3h/patient (183h total); subsequent administration will reduce it 290h/session.

Conclusion: Transitioning from IV to SC Ocrelizumab was safe, well-tolerated, and associated with high patient satisfaction and substantial logistical benefits in real-world clinical practice.

Disclosure: Nothing to disclose.

EPO-0666 | Plasma neurofilament light chain, glial fibrillary acidic and Tau proteins dynamic in patients receiving CAR-T treatment

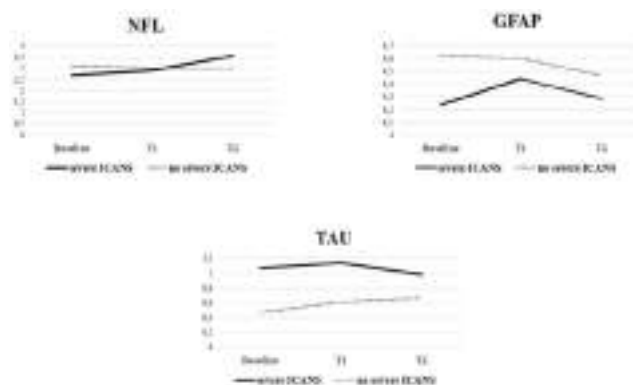
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Background and aims: ICANS is a severe adverse event of CAR-T cell therapy with limited predictive factors. Neuroaxonal and astroglial biomarkers may reflect pre-existing vulnerability and coexistent damage. This study evaluates the dynamic of neurofilament light chain (NFL), glial fibrillary acidic protein (GFAP), and Tau levels as predictors of ICANS.

Methods: Plasma NFL, GFAP, and Tau levels were measured at baseline (pre), day 7 (T1), and day 30 (T2) post-infusion. Their levels were analysed to assess associations with ICANS occurrence and severity.

Results: Among 20 patients (median age 56.6 years), moderate-severe ICANS occurred in 6/20 (30%), while 14/20 patients (70%) developed no or mild ICANS. Pre and T1 NFL, GFAP,



NFLs, GFAP and Tau dynamics in CAR-T treated patients.

and Tau levels did not differ between patients with moderate-severe ICANS and no or mild ICANS. NfL increased at T1 in moderate-severe ICANS ($p=0.021$). Δ NfL (T1-pre) predicted moderate-severe ICANS ($p=0.04$). NfL increased at T2 versus pre ($p=0.026$) and T1 ($p=0.027$) timepoints in moderate-severe ICANS. A trend toward higher GFAP levels was observed at T1 in moderate-severe ICANS ($p=0.054$) with a decrease at T2 in both groups. Tau showed no significant associations at any timepoint.

Conclusion: NfL, GFAP and Tau display distinct dynamics after CAR-T. An early increase of NfL levels after baseline predicts moderate-severe ICANS. Patients with moderate-severe ICANS also exhibit a higher late rise of NfL compared with baseline and early measurements. GFAP increase may play a role in the early phase but does not appear to be involved in established ICANS.

Disclosure: Nothing to disclose.

EPO-0667 | Persistent synapse-engulfing phagocytes shape the aftermath of encephalitis

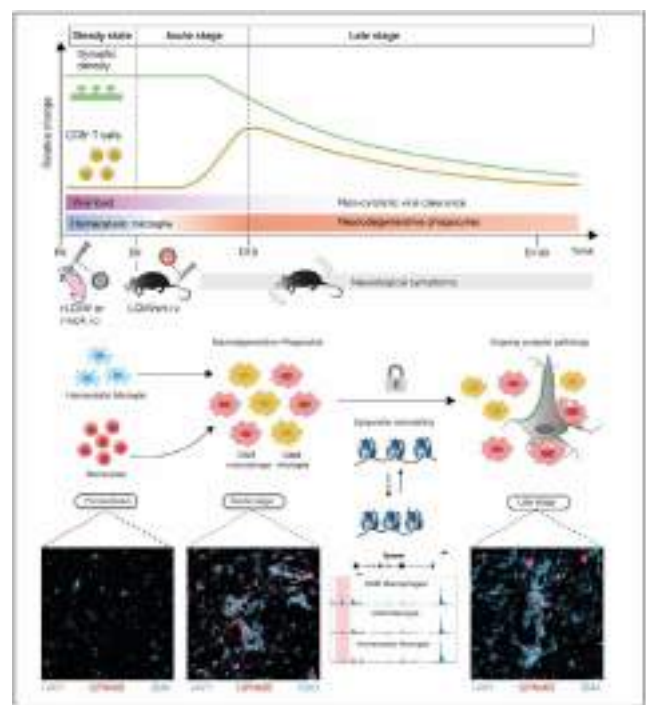
G. Di Liberto; A. Mariotte; I. Vincenti; N. Page; G. Shammass; M. Piccinno; K. Egervari; M. Kreutzfeldt; I. Wagner; S. Lemeille; D. Merkler

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Background and aims: T cell-mediated encephalitides often cause neurological deficits that persist after resolution of overt inflammation, suggesting that long-lived innate immune responses may sustain pathology beyond T cell contraction. While IFN- γ -producing T cells initiate disease, how IFN- γ enables phagocytes to acquire persistent pathogenic phenotypes remains unclear. As IFN- γ induces durable epigenetic remodeling of synaptic genes in neurons, we hypothesized that IFN- γ is required for acquisition of a persistent disease-associated phagocyte state driving ongoing synaptic pathology independent of continued T cell activity.

Methods: We used a murine viral déjà vu model of T cell-mediated encephalitis, combining CSF1R-mediated phagocyte depletion with genetic ablation of IFNGR2 or TREM2/APOE signaling. Lineage tracing tracked microglia and macrophages across disease stages. Sorted phagocytes were analyzed by single-cell RNA sequencing, bulk ATAC-seq, and SCENIC. Motor function and synaptic integrity were assessed behaviorally and histologically.

Results: CSF1R-mediated phagocyte depletion ameliorated neurological deficits. Microglia and macrophages persisted in the CNS during late disease stages, when T cell responses declined. Bulk ATAC-seq revealed sustained chromatin remodeling at phagocytosis-, antigen presentation-, and interferon-responsive loci shared by both cell types. During acute disease, phagocytes acquired a synapse-engulfing phenotype that persisted at lower levels later. Single-cell RNA sequencing showed early convergence toward a disease-associated state regulated by interferon-responsive transcription factors. Disruption of TREM2/APOE signaling was not protective, whereas IFNGR2 ablation significantly improved motor performance and synaptic pathology. Similar phagocyte signatures were observed in Rasmussen encephalitis.



Synapse-engulfing phagocytes persist after encephalitis. Acute CD8⁺ T-cell responses coincide with synaptic loss and phagocyte activation. Microglia and macrophages adopt durable DAM-like states with sustained chromatin remodeling and ongoing synaptic pat.

Conclusion: Persistent IFN- γ -dependent phagocyte reprogramming sustains synaptic pathology after encephalitis, identifying CNS phagocytes as key drivers of long-term neurological dysfunction.

Disclosure: Nothing to disclose.

EPO-0668 | The role of the C5a-C5aR1 axis in mediating peripheral immune activation and central nervous system demyelination in MOGAD

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Background and aims: Complement activation is involved in the pathogenesis of myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), but whether it acts mainly through complement-dependent cytotoxic effects or through bystander effects on the immune system—and to what extent it contributes—remains unclear.

Methods: Twenty-two patients with MOGAD (10 acute, 12 remission) were enrolled. Peripheral blood was collected for serum complement profiling, immune-cell analysis by flow cytometry, and peripheral blood mononuclear cell (PBMC) bulk RNA sequencing. The C5aR1 antagonist PMX205 was administered in a MOG-IgG-augmented MOG35–55 experimental autoimmune encephalomyelitis (EAE) model. Histopathological and immunophenotypic analyses were performed on the brain and spinal cord; additional co-culture experiments used naïve CD4⁺ T cells and myeloid cells.

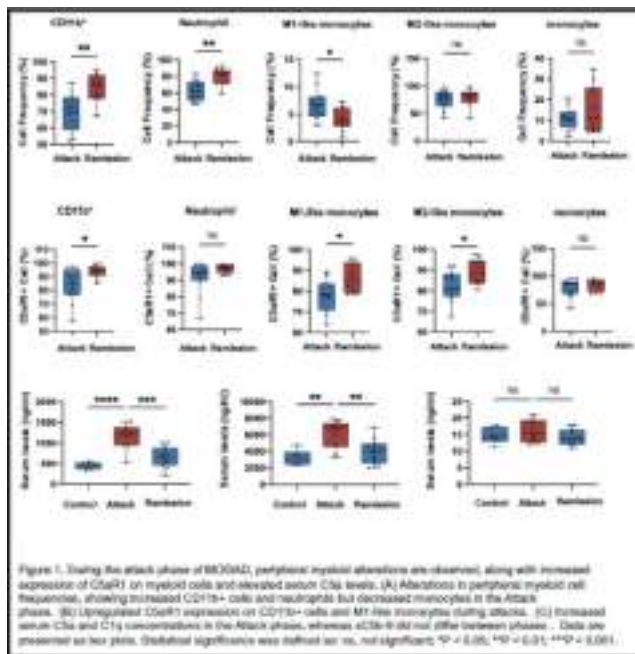


FIGURE 1 Acute-phase MOGAD exhibits a “low C5a-high C5aR1” imbalance.

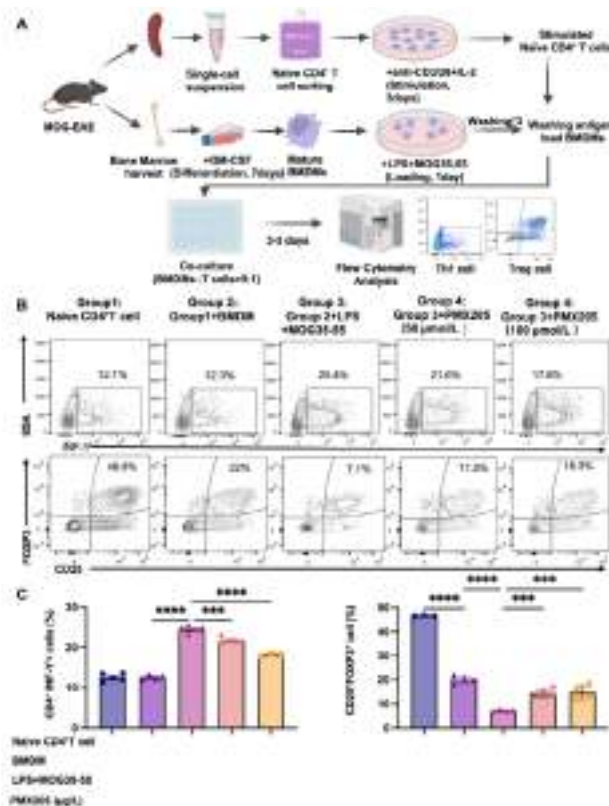


FIGURE 3 C5aR1 shapes macrophage–T cell crosstalk to bias Th1 polarization and Treg induction. (A) Schematic representation of the in vitro co-culture system involving naive CD4⁺ T cells and bone marrow-derived macrophages (BMDMs). (B) Representative flow cytometry plots showing gating of CD4⁺IFN- γ ⁺ cells (upper panels) and CD4⁺CD25⁺FOXP3⁺ regulatory T cells (lower panels). Percentages indicate the frequency within the CD4⁺ T cell gate. (C) Quantification of CD4⁺IFN- γ ⁺ cells and CD4⁺CD25⁺FOXP3⁺ Tregs. Bars represent mean $\pm$ SEM, and dots represent biological replicates. Statistical significance was assessed by one-way ANOVA with multiple-comparisons correction. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$, **** $P < 0.0001$.

FIGURE 3 C5aR1 shapes macrophage–T cell crosstalk to bias Th1 polarization and Treg induction.

Results: Acute MOGAD showed enrichment of inflammatory and chemotactic pathways, along with immune-cell redistribution. The complement axis was upregulated; C5aR1 correlated most strongly with the inflammatory module and was also associated with chemotaxis and antigen-presentation modules. Flow cytometry and enzyme-linked immunosorbent assay (ELISA) indicated increased circulating C5a with increased cellular C5aR1 expression. In augmented EAE, PMX205 improved neurological outcomes, reduced central nervous system (CNS) myeloid infiltration, shifted T-cell polarization ($\downarrow$ Th1, $\uparrow$ Treg), and mitigated demyelination; co-culture supported myeloid-driven T-cell differentiation.

Conclusion: The C5a–C5aR1 axis may contribute to peripheral immune activation in active MOGAD and, via myeloid recruitment and myeloid–T cell crosstalk, relate to CNS inflammation and demyelination.

Disclosure: This study was supported by the National Natural Science Foundation of China (82171277, 82571535, 82171338), and Young Scientific and Technological Talents (Level Three) in Tianjin.

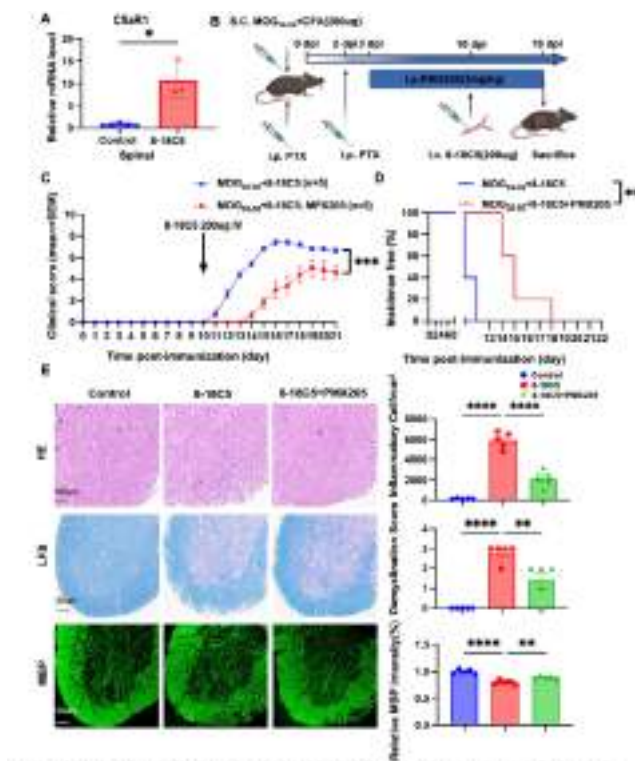


Figure 2. C5aR1 upregulation in the CNS and therapeutic impact of C5aR1 antagonism in a MOG-IgG-augmented MOG 35–55 EAE model. (A) Quantitative RT-PCR analysis of C5aR1 mRNA expression in spinal cord tissue comparing the Control and 8-18CS MOG-IgG groups. Expression was normalized to a housekeeping gene and presented as relative expression; each dot represents one mouse. (B) Schematic of the MOG-IgG-augmented MOG 35–55 EAE induction and treatment schedule. (C) Longitudinal clinical EAE scores assessed using a 10-point neurological disability scale for the indicated groups. Data are presented as mean $\pm$ SEM with individual animals shown. (D) Disease incidence/incubation analysis across the disease course shown as an “incidence-free” (Kaplan–Meier-style) curve comparing the indicated groups. (E) Representative spinal cord histopathology and quantification at day 15. H&E staining was used to evaluate inflammatory infiltration, Luxol fast blue (LFB) staining to assess demyelination, and MBP immunostaining to assess myelin integrity. Corresponding quantitative analysis is shown alongside representative images. Scale bars are indicated in the panels. Statistical tests are indicated in the figure; significance is denoted by asterisks (* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$, **** $P < 0.0001$).

FIGURE 2 C5aR1 upregulation in the CNS and therapeutic impact of C5aR1 antagonism in a MOG-IgG-augmented MOG 35–55 EAE model.

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Background and aims: Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), an immune-mediated neuropathy, causes progressive muscle weakness and sensory disturbance. Efgartigimod, a human IgG1 antibody Fc fragment, reduces IgG recycling by blocking FcRn, reducing impact on CIDP pathophysiology. The efficacy and safety of subcutaneous efgartigimod PH20 in patients with CIDP was demonstrated in the registrational ADHERE trial. ADHERE REAL will assess the real-world effectiveness of subcutaneous efgartigimod PH20 in patients with CIDP and describe their treatment journey and healthcare resource utilisation.

Methods: This real-world, prospective, multicentre study will assess adults with CIDP across the US and Germany. Patients will be efgartigimod PH20 naïve at screening and initiate treatment in routine clinical practice. Clinician-assessed and patient-reported outcomes will be recorded regularly, and treatment/dosage changes or relapses will be recorded.

Results: Change over time in scores, including adjusted Inflammatory Neuropathy Cause and Treatment and Inflammatory Rasch-built Overall Disability Score, will assess real-world effectiveness of efgartigimod PH20 and will be monitored via patient questionnaires, nurse assessments and medical record review. Proportions of participants with CIDP relapse and with evidence of clinical improvement will be assessed. Assessment of patient-treatment journeys will include disease course, comorbidities, CIDP treatment, modifications, and timing/rationale for these changes. Healthcare resource utilisation will be assessed by frequency, duration, and reasons for healthcare provider follow-ups, emergency room visits and hospitalisations. As of December 2025, 12 participants have been enrolled in Germany; enrolment has begun in the US.

Conclusion: This study will expand on the ADHERE trial and provide insights into the real-world usage of efgartigimod in patients with CIDP.

Disclosure: Jana Zschüntzsch: Alexion, Amicus, argenx, Biogen, CSL Behring, German Society for Muscle Diseases (DGM e.V.), German Society for Myasthenia Gravis (DMG), Hormosan, Janssen, Kedrion, Roche, Sanofi and UCB. Jeffrey A. Allen: Consultant fees: Akcea, Alexion, Alnylam, Annexon, argenx, CSL Behring, Grifols, Immunovant, ImmuPharma, Johnson & Johnson, Pfizer and Takeda. Jamie Aldridge, Arne De Roeck, Désirée Gneißl, Saman Haghighi, Geoffrey Istaş, Chris Pashos: Employees of argenx. Filip Eftimov: Received investigator-initiated research grants for CIDP from ZonMw, Prinses Beatrix

Spierfonds, the GBS-CIDP Foundation and Dianthus. As principal investigator of INCbase, reports investigator-initiated grants from Kedrion, Terumo BCT, CSL Behring, Grifols, argenx and Takeda. The institution has received fees from Hansa, Sanofi, Dianthus, CSL Behring, Grifols and Takeda for advisory board participation and/or lectures. All grants and fees were paid to the institution. Benjamin Knier: Alexion, argenx, CSL Behring, Heidelberg Engineering, Hexal, Merck, Novartis, Novo Nordisk, Roche, Teva, Deutsche Forschungsgemeinschaft (DFG). Rebecca Traub: Consultant: argenx, Intellia, Sanofi. Clinical trials/research funding: Alnylam, argenx, Immunovant, Ionis, Takeda. Chafic Karam: Alexion, Alnylam, Annexon, Alpine, argenx, AstraZeneca, Biogen, Corino, CSL Behring, Genentech, Ionis, Neuroderm, Novo Nordisk, Pfizer, Sanofi, UCB, Takeda and Zai Lab.

EPO-0670 | Clinical outcomes with exploratory biomarker analyses from phase 2 of KYSA-6, an open-label, single arm, multicenter study of miv-cel in gMG

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Background and aims: Generalized myasthenia gravis (gMG) is a B-cell and antibody-mediated neuromuscular autoimmune disease that can result in substantial disability. Novel therapies are needed to improve disease symptoms. Miv-cel (mivocabtagene autoleucel; KYV-101) is a fully-human, autologous CD19 CAR T-cell therapy with CD28 costimulation, designed for potency and tolerability. Updated data for miv-cel in gMG from KYSA-6 phase 2 (NCT06193889) are presented.

Methods: Adults (18–75y) with gMG (class IIb–IV), history of autoantibodies (AChR, MuSK, LRP4), MG activities of daily living (MG-ADL) score ≥ 6 , and ≥ 2 immunosuppressant/

immunomodulator failures, received lymphodepletion followed by 1×10⁸ CAR T cells.

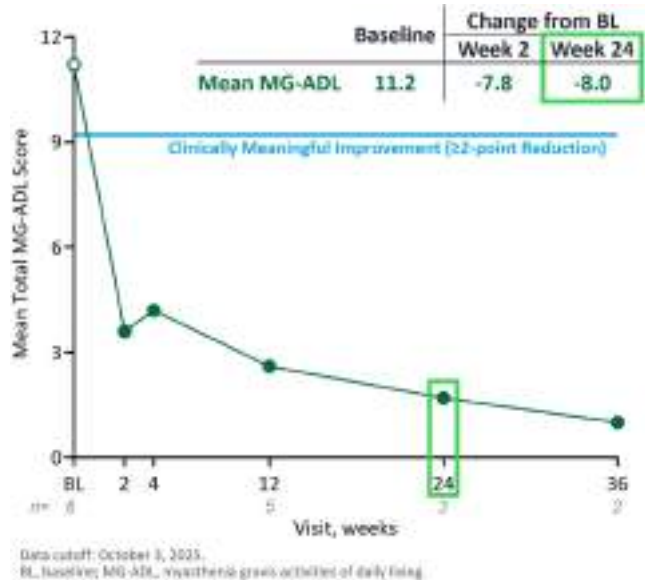
Results: As of October 3, 2025, 6 patients were dosed (Table). Follow-up was 28 days-9 months (≥24 weeks, n=3). Miv-cel rapidly expanded with CAR T-cell activation evidenced by deep B-cell depletion and transient immunomodulatory cytokine elevation. Of 3 patients with ≥24-weeks follow-up, mean reductions from baseline were 8.0 and 7.7 points for MG-ADL and QMG, respectively, at 24 weeks and 67% achieved minimal symptom expression (Figures 1 and 2). All 6 patients remained immunosuppressant therapy-free up to 24 weeks. No ICANS or high-grade CRS occurred. After miv-cel, alterations in T-cell subsets were observed in the 3 patients with ≥24-weeks follow-up and reconstituted B cells in 2 patients 3–4 months post-infusion were predominantly naïve, supporting immune reset.

Conclusion: With a single dose, miv-cel induced deep B-cell depletion with evidence of immune reset. Patients experienced rapid, robust, and sustained improvements in disease severity with acceptable safety. Miv-cel demonstrates the potential to deliver durable, drug-free, disease-free remission in patients with gMG. Phase 3 is ongoing.

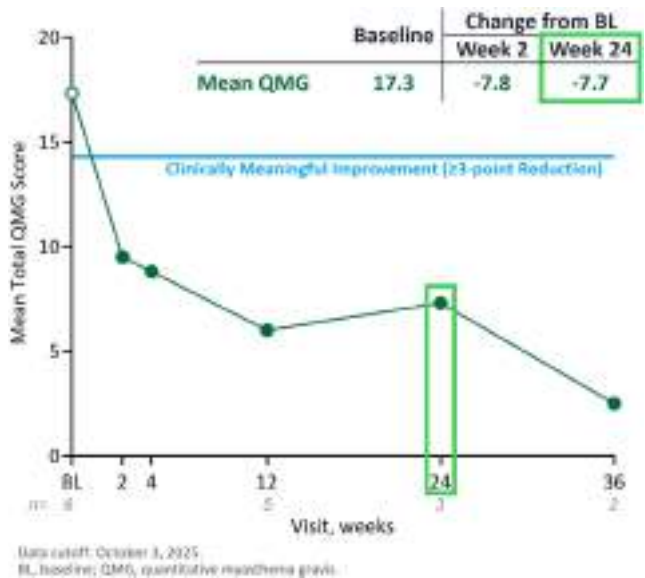
Baseline characteristics		N=6
Age, mean years (range)		45.5 (21–62)
Sex, female male, n (%)		5 (83) 1 (17)
Duration of MG, mean years (range)		5.3 (1.7–13.3)
Total outcome score, mean (range)		
MG-ADL		11.2 (7–16)
QMG		17.3 (9–28)
MGFA Class IIIb Class IV at screening, n (%)		4 (67) 2 (33)
AChR MuSK-positive at screening/historically, n (%)		5 (83) 1 (17)
Prior therapies, n (%)		
Acetylcholinesterase inhibitors		6 (100)
Nonsteroidal immunosuppressive therapy		6 (100)
FcRn and/or complement inhibitors		5 (83)
Steroids		6 (100)
Intravenous immunoglobulin and/or plasmapheresis		4 (67)
Rituximab		3 (50)

AChR, acetylcholine receptor; FcRn, neonatal fragment crystallizable receptor; MG, myasthenia gravis; MG-ADL, MG activities of daily living; MOC, MG composite; MGFA, Myasthenia Gravis Foundation of America; MuSK, muscle-specific kinase; QMG, quantitative MG.

Patient demographics.



Miv-cel showed rapid, robust, and sustained reduction in MG-ADL (primary endpoint).



Miv-cel showed rapid, robust, and sustained reduction in QMG scores.

Disclosure: JM reports research support from Novartis and Kyverna, consulting fees from Novartis, payment or honoraria from Grifols and Novartis, support for meetings and/or travel from Sanofi, Candit, Novartis, and Johnson & Johnson, stock/stock options for Sanofi, Bayer, and Amgen, and receipt of equipment, materials, drugs, medical writing, gifts or other services from Candit. SM has served on advisory boards for AstraZeneca, Argenx, J&J, UCB, Amgen, Kyverna, Arcellx, and Seismic Pharma, and DSMB for Dianthus Therapeutics. SH has received speaker honoraria from Alexion, Argenx BV, Grifols, Johnson&Johnson, Merck, Roche, and UCB, honoraria for attendance at advisory boards from Alexion, Argenx BV, Johnson&Johnson, Novartis and Roche, and participated in trials from Alexion, Argenx, Immunovant, Johnson&Johnson, Kyverna and Regeneron. RG has received research support and speaker's honoraria from Bayer Schering, Biogen-Idec, BMS, Chugai, Eisai, ELAN, Janssen, Kyverna, Merck Serono, Nikkiso Pharma, Novartis, Roche, Sanofi-Genzyme, and TEVA, and consulting honoraria from ZLB Behring, Baxter, Kyverna, and Talecris, and has private stock from Merck, Kyverna, Novartis, Sanofi and Roche. CS received honoraria from Alexion and Argenx. BDH reports a research grant from Johnson&Johnson, honoraria from Kite Pharma, Novartis, BMS, ADC Therapeutics, Johnson&Johnson, Genmab, Notable Labs, AbbVie, Genentech, Astellas, In8 Bio, Regeneron, Poseida Therapeutics, Caribou Biosciences, and stock holdings for Actinium Pharmaceuticals. EM has received research grant support from Kyverna and Orca Biosystems, has done consulting for AstraZeneca and has equity as consultant in TRACT therapeutics, Indee, Jura and GigaMune. MLH-K has received speakers' honoraria, and honoraria at advisory boards from Amgen, Grifols, Jazz, Sanofi, and Kyverna. CS-F has received honoraria from BMS, Johnson&Johnson, AbbVie, Kite Pharma, Mikrogen, Pierre Fabre and Roche. RS has served on an advisory board for Kyverna. FA has received honoraria from AbbVie, BMS, Kite/Gilead, Janssen, Therakos, Miltenyi Biomedicine, Novartis, Takeda, Medac, Neovii; and research funding from Therakos and Neovii. BX, JC, TVB, NG are employees of Kyverna Therapeutics and may have stock/stock options. AH reports honoraria from Merck Serono, Kyverna, BMS, Amgen, Neuraxpharm, Sanofi, and Galapagos. MCH and TH have nothing to disclose.

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Background and aims: NMOSD is a severe inflammatory demyelinating disease of the central nervous system, primarily affecting the optic nerves and spinal cord. Although NMOSD is classically considered to spare the brain, emerging evidence suggests functional cortical involvement. Altered cortical excitability may provide insights into the pathophysiology of NMOSD and its clinical manifestations.

Methods: A cross-sectional study was conducted on 30 NMOSD patients and 30 age- and sex-matched healthy controls. Transcranial magnetic stimulation (TMS) was used to assess key cortical excitability parameters, including resting motor threshold (RMT), short-interval intracortical inhibition (SICI), intracortical facilitation (ICF), and cortical silent period (CSP). Correlations between TMS measures and clinical parameters, including disease duration, EDSS scores, relapse history, and presence of brain lesions on MRI, were analyzed.

Results: NMOSD patients exhibited significant cortical excitability alterations compared to controls, including increased RMT ($p < 0.05$), reduced SICI ($p < 0.01$), prolonged CSP ($p < 0.05$), and enhanced ICF ($p < 0.05$). Reduced SICI correlated with higher EDSS scores ($r = -0.45$, $p < 0.01$) and greater relapse frequency ($r = -0.42$, $p < 0.05$), suggesting impaired GABAergic inhibition in patients with more severe disease. Patients with supratentorial MRI lesions showed greater cortical excitability changes than those without.

Conclusion: Our findings suggest that NMOSD is associated with significant alterations in cortical excitability. These changes correlate with clinical severity and may reflect underlying neurophysiological dysfunction beyond the spinal cord. TMS could serve as a useful biomarker for cortical involvement in NMOSD and a potential tool for monitoring disease progression and therapeutic response.

Disclosure: Nothing to disclose.

EPO-0672 | Phenotype-driven identification of autoimmune central vitamin B12 deficiency in idiopathic neuroinflammatory myelopathy

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Background and aims: The recent description of autoimmune vitamin B12 central deficiency mediated by antibodies against the transcobalamin receptor (CD320) provides a novel

pathophysiological framework for selected idiopathic neuroinflammatory phenotypes. We applied this mechanistic concept to idiopathic neuroimmunological disease with myelopathy resembling subacute combined degeneration.

Methods: Patients were identified from an institutional registry of idiopathic neuroimmunological disorders and screened for predominant spinal cord involvement. Based on the proposed pathophysiological model of autoimmune central vitamin B12 deficiency, patients with clinical and magnetic resonance imaging features suggestive of subacute combined degeneration were selected for targeted anti-CD320 testing in serum and cerebrospinal fluid at reference laboratories.

Results: Eighty-four patients were included in the registry, of whom 23 showed predominant spinal cord involvement (double-seronegative neuromyelitis optica spectrum disorder, $n = 8$; systemic autoimmune disease-associated neurological disease, $n = 5$; other idiopathic inflammatory phenotypes, $n = 10$). Six patients with posterior column-predominant myelopathy and compatible imaging features were prioritised; samples were available in all six (Table 1). Anti-CD320 antibodies were detected in four cases, including one with double-seronegative neuromyelitis optica spectrum disorder and three with other idiopathic inflammatory phenotypes. Seropositive patients presented with progressive myelopathy, posterior column dysfunction and non-inflammatory cerebrospinal fluid profiles. Following antibody identification, two patients initiated rituximab and high-dose intramuscular vitamin B12; follow-up is ongoing.

Conclusion: Anti-CD320 antibodies were identified in a subset of patients with idiopathic neuroinflammatory myelopathy characterised by posterior column-predominant, subacute combined degeneration-like phenotypes, supporting a phenotype-driven approach to targeted autoantibody screening with potential diagnostic and therapeutic relevance.

Disclosure: García-Sarreón, A.: has served as a speaker at events funded by Amgen and Sanofi; received travel grants from Roche, Novartis, and UCB Pharma; and completed an ECTRIMS Clinical Training Fellowship (2023–2024). Gifreu-Fraixinó, A.: has received academic support from UCB Pharma, Bial Pharmaceutical, Angelini Pharma, Merck, Bristol-Myers-Squibb, Biogen, Janssen, Novartis, Roche, Neuraxpharm, Sanofi, Amgen. Boix-Lago, A.: has received academic support from Merck, Bristol-Myers-Squibb, Biogen, Janssen. Gutiérrez Naranjo, J.: has received academic support from Merck, Lilly, Teva, Novartis, Abbvie, and speaking fees from Teva, Lilly, and Abbvie. Pluvinae, J. V.: has nothing to disclose. Álvarez-Bravo, G.: has received compensation for consulting services and speaking fees from Biogen, Novartis, Merck, Sanofi, Amgen, Roche, Bristol-Myers-Squibb, Teva.

TABLE 1 Clinical, cerebrospinal fluid, and MRI features of patients prioritised for anti-CD320 antibody testing.

Case	Age	Sex	Anti-CD320	Immunophenotype	CSF profile	MRI pattern	Treatment
Case 1 (BT)	35	F	Positive	Progressive myelopathy with extensive white matter involvement	Non-inflammatory	Longitudinal central cord lesions > 2 T2-FLAIR-enhanced	Rituximab + B12
Case 2 (BT)	50	F	Positive	Subacute-onset spinal myelopathy	Non-inflammatory	Longitudinal central cord lesions > 2 T2-FLAIR-enhanced	Observation
Case 3 (BT)	37	F	Positive	Subacute-onset spinal myelopathy	Non-inflammatory	Posterior column T2-FLAIR-enhanced	High-dose B12
Case 4 (BT)	55	M	Positive	Chronic progressive posterior column myelopathy	Non-inflammatory	Central upper thoracic cord lesions T2-FLAIR-enhanced	Observation
Case 5 (BT)	40	F	Negative	Spontaneous optic atrophy	Non-inflammatory	Posterior column lesions > 2 T2-FLAIR-enhanced	High-dose B12
Case 6 (BT)	41	F	Negative	Spontaneous optic atrophy	Non-inflammatory	Posterior column lesions > 2 T2-FLAIR-enhanced	High-dose B12

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Background and aims: Progression independent of relapse activity (PIRA) is increasingly recognised as a major driver of disability in multiple sclerosis (MS). Serum neurofilament light chain (NfL) is established for monitoring inflammatory activity, whilst glial fibrillary acidic protein (GFAP) has emerged as a potential marker of astrocytic damage and smouldering neurodegeneration. However, their comparative prognostic value for disability progression remains unclear. We aimed to compare serum GFAP and NfL as predictors of disability progression across MS subtypes.

Methods: We searched PubMed, Embase, Scopus, and Cochrane Library through December 2025. We included cohort studies and post-hoc trial analyses reporting hazard ratios (HRs) for PIRA or confirmed disability worsening (CDW). Random-effects meta-analyses with Hartung-Knapp adjustment were performed. Risk of bias was assessed using Newcastle-Ottawa Scale.

Results: Eighteen studies ($n=15,944$) were included. For PIRA, pooled HRs were 1.85 (95% CI 1.50–2.29; I-squared=0%) for GFAP and 1.51 (95% CI 1.23–1.85; I-squared=0%) for NfL. Overall pooled effects were HR 1.53 (1.28–1.82) for GFAP and HR 1.54 (1.41–1.67) for NfL. The formal GFAP/NfL ratio was 0.91 (0.80–1.04; $p=0.17$), indicating no significant difference between biomarkers. In mutual adjustment analyses, GFAP remained significant after NfL adjustment (HR 1.80; $p=0.008$), whilst NfL became non-significant after GFAP adjustment (HR 1.45; $p=0.09$).

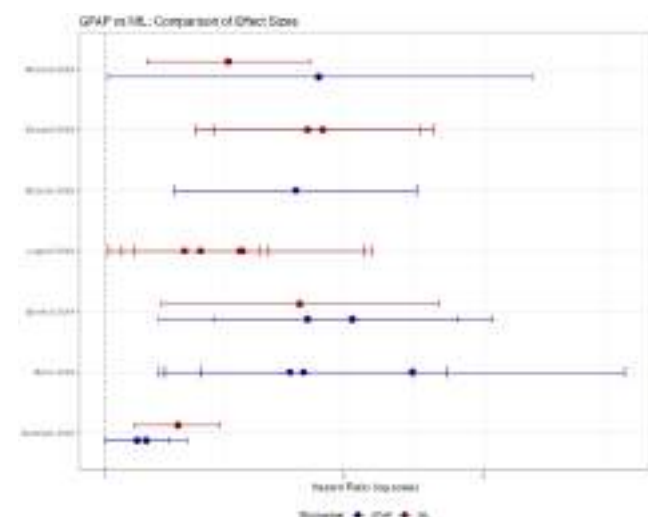


FIGURE 1

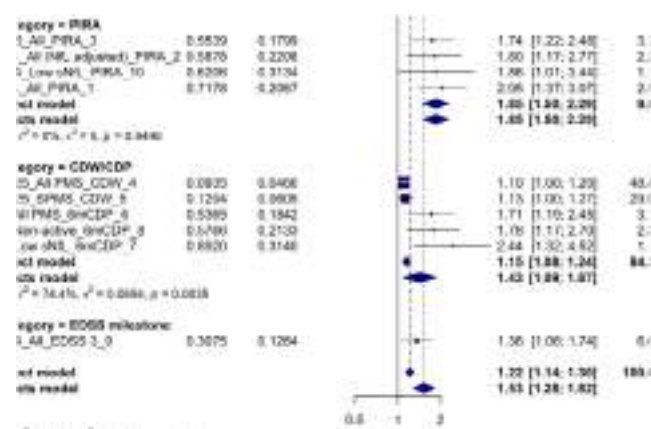


FIGURE 2

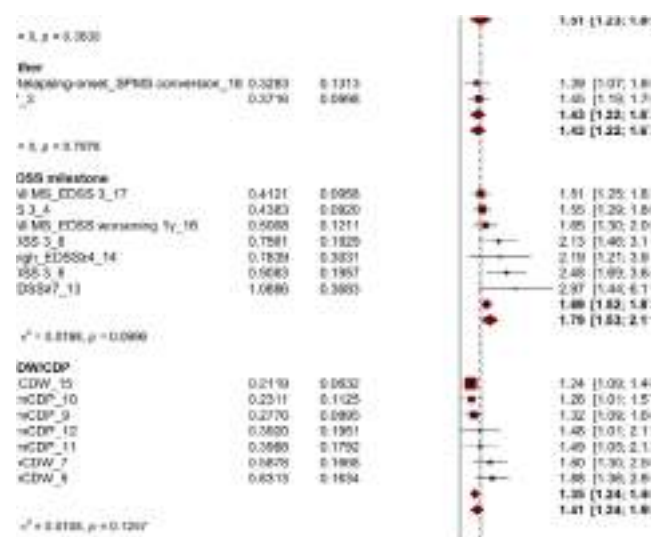


FIGURE 3

Conclusion: Both serum GFAP and NfL predict disability progression with similar overall effect sizes. GFAP shows stronger association with PIRA and provides independent prognostic information beyond NfL, supporting its complementary role in MS monitoring.

Disclosure: Nothing to disclose.

EPO-0674 | Early escalation of acute immunotherapy with plasma exchange and IVIG is associated with reduced relapse risk in pediatric MOGAD

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C. Hemingway¹; Y. Hacohen¹

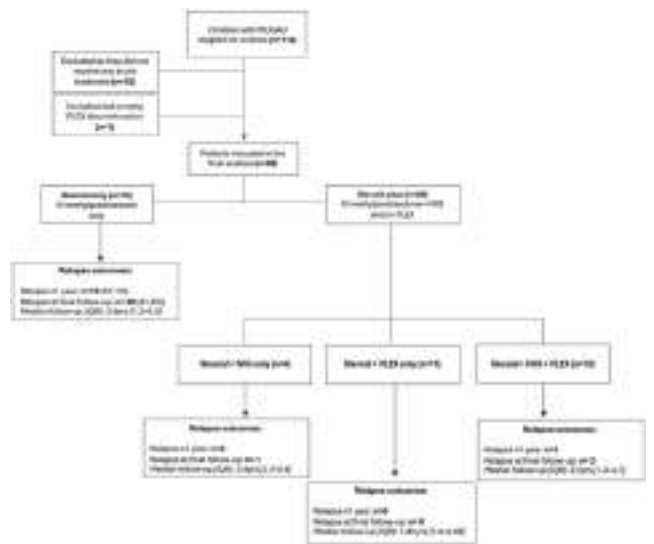
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Background and aims: Acute treatment of myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) includes high-dose intravenous corticosteroids (IVCS),

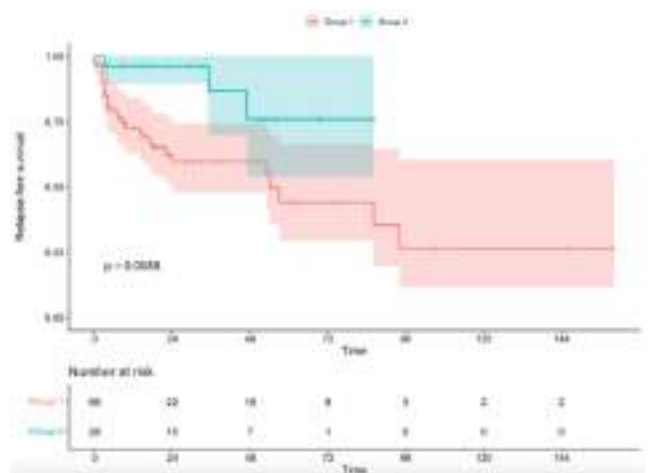
intravenous immunoglobulin (IVIG), and plasma exchange (PLEX), but the impact of different treatment strategies on long-term outcome remains unclear. We evaluated whether early escalation of acute immunotherapy at the first pediatric MOGAD attack reduces relapse risk.

Methods: We conducted a retrospective single-center cohort study of pediatric-onset MOGAD (<18 years) evaluated between 2015 and 2016 and followed from first attack to last visit (median 3.1 years). Of 114 screened patients, 98 were included. Patients received IV methylprednisolone (IVMP) alone or IVMP followed by escalation with IVIG, PLEX, or both based on disease severity or steroid response. Outcomes were early (≤ 1 year) and overall relapse. Cox regression assessed relapse risk.

Results: Among 98 patients (57.1% female; median age 6.3 years), onset phenotypes included optic neuritis (42.9%), acute disseminated encephalomyelitis (42.9%), cortical encephalitis (9.2%), and myelopathy (5.1%). Seventy received steroids alone and 28 received escalation therapy. Early relapse occurred in 27.1% of



Study flow-chart and relapse outcomes. Summarises the study flow-chart and reports relapse outcomes in paediatric MOGAD patients grouped according to acute treatment strategy.



Kaplan–Meier survival curves illustrating time to first relapse (months) by treatment group. Patients treated with intravenous steroids alone experienced earlier relapses, with a median time to relapse of less than one year, whereas those who underwent t.

Variable	Total cohort (n=98)	Steroid only (n=70)	Steroid Plus (n=28) (+ IVIG or PLEX)	p-value
Age at onset, [IQI], y	6.33 [4.37–11.12]	6.04 [4.02–9.43]	7.08 [4.88–12.02]	0.224
Male/female	42/56	31/39	11/17	0.822
Onset phenotype, n (%)				
Optic neuritis	42 (42.9%)	35 (50.0%)	7 (25.0%)	0.042
ADDEM	42 (42.9%)	29 (41.4%)	13 (46.4%)	0.658
Cortical encephalitis	9 (9.2%)	4 (5.7%)	5 (17.9%)	0.114
Myelopathy	5 (5.1%)	2 (2.9%)	3 (10.7%)	0.138
EDSS at nadir, [IQI]	1.0 [1.0–2.0]	1.0 [1.0–2.0]	2.0 [1.0–3.0]	0.095
Time to steroid, [IQI], days	7.0 [3.0–15.0]	8.0 [4.0–16.0]	7.0 [3.0–10.0]	0.248
Time to escalation, [IQI], days	10.8 [9–14]		10.0 [9–14]	
Follow-up duration, [IQI], y	3.13 [1.49–6.99]	3.58 [1.59–6.54]	2.58 [1.37–4.45]	0.110
EDSS at last follow-up, [IQI]	0.00 [0.00–1.00]	0.00 [0.00–1.00]	0.00 [0.00–1.00]	0.120
Relapse within 1 year, n (%)	20 (20.4%)	19 (27.1%)	1 (3.6%)	0.011
Relapse at final follow-up, n (%)	32 (32.7%)	25 (41.4%)	7 (25.0%)	0.004
Time to first relapse, median [IQI], (days)	184 (90–614)	183 (83–581)	1095 (561–1278)	0.317

Demographic and clinical characteristics of the cohort (N=98).

steroid-only patients versus 3.6% with escalation ($p=0.011$); overall relapse rates were 41.4% versus 10.7% ($p=0.004$). Escalation was associated with reduced relapse risk (adjusted HR0.24, 95% CI 0.07–0.82).

Conclusion: In pediatric MOGAD, early escalation with IVIG and/or PLEX following corticosteroids was associated with a substantially lower relapse risk compared with corticosteroids alone. These findings support the first clinical attack as a critical therapeutic window. Early aggressive therapy may reduce immune-mediated relapses and improve long-term disease trajectory. Prospective studies are needed to determine whether routine treatment escalation should be recommended to minimize relapse risk.

Disclosure: Bartolomeo S. received travel fundings from Biogen, Novartis, Sanofi, Bristol-Myers Squibb. O. Ciccarelli is an NIHR Research Professor (RP-2017-08-ST2-004); acts as a consultant for Biogen, Merck, Novartis, Roche, and Teva; and has received research grant support from the MS Society of Great Britain and Northern Ireland, the NIHR UCLH Biomedical Research Centre, the Rosetree Trust, the National MS Society, and the NIHR-HTA. Y. Hacohen received research grant support from the MS Society of Great Britain and Northern Ireland. Other authors have nothing to disclose.

EPO-0675 | Neuromyelitis optica spectrum disorder in Finland: A population-based study

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Background and aims: Neuromyelitis optica spectrum disorder (NMOSD) is a demyelinating autoimmune disease of the central nervous system. Epidemiology of NMOSD in Finland has not been previously investigated on a national level. We set out to determine the incidence, prevalence and mortality for patients with NMOSD (pwNMOSD) in Finland during the last decades.

Methods: National care registries were searched for all diagnosed pwNMOSD (ICD-10 code 36.0, ICD-9 341.0) during 1994–2023. To evaluate confounders, the Helsinki University Hospital cohort was collected in detail using patient medical records during 2005–2023 and using the 2015 International Panel for NMO Diagnosis (IPND) criteria. Standardized mortality ratio (SMR) and causes of death were assessed using national care registries for 2015–2023.

Results: We identified 128 pwNMOSD in Finland, of which 78% were female. During 2015–2023 standardized national incidence of NMOSD was 0.13/100,000 person-years (95% CI 0.10–0.17). National prevalence increased from 1.10/100,000 in 2014 to 2.05/100,000 in 2023, with no clear regional differences. Peak in incidence was seen for the ages 30–49. SMR was 2.36 (95% CI 1.23–4.53) for pwNMOSD and NMOSD was the underlying or contributing cause of death for 46.7% of all the deaths recorded ($n = 15$).

Conclusion: NMOSD incidence in Finland is somewhat higher compared to other Nordic countries, and unlike reported for multiple sclerosis incidence in Finland, shows no clear East-West gradient. NMOSD has a clear impact on mortality in this population.

Disclosure: TA: Nothing to disclose. MV: Nothing to disclose. SML: Lecture fees Alexion, Argenx, Jansen, Lundbeck, Merck, Novartis, Sanofi, Teva; congress and travel expenses Merck, Novartis, UCB Pharma; advisory fees Argenx, Johnson & Johnson, Novartis, Sanofi, UCB Pharma. Funding: Maire Taponen Foundation (to SML).

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EPO-0676 | Insight from Helsinki aneurysm database: Functional recovery in severely disabled survivors of aneurysmal subarachnoid hemorrhage

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Background and aims: Aneurysmal subarachnoid hemorrhage (aSAH) frequently results in severe neurological disability. With advances in neurocritical and neurosurgical care, prognostication and assessment of functional recovery in patients discharged with severe disability have become increasingly important.

Methods: We retrospectively reviewed our aneurysm database from 1995 to 2015. Patients admitted within 72 hours of aSAH onset who survived hospitalization and were discharged with a mRS score of 4 or 5 were included. The primary outcome was good functional recovery at 3 months (mRS 0–3), with additional assessment at 1 year.

Results: A total of 1253 patients were included; 315 were discharged with mRS 4. Mean age was 57.4 ± 12.7 years, and 65%



FIGURE 1

were women. Good functional recovery occurred in 730 patients (58.3%) at 3 months and in 266 (63.2%) at 1 year. In patients discharged with mRS 4, lower likelihood of recovery was associated with older age (OR 0.92), diabetes (OR 0.03), larger ICH volume (OR 0.96), and postoperative meningitis (OR 0.34). In patients discharged with mRS 5, reduced recovery was associated with older age (OR 0.95), Hunt and Hess grade 5 (OR 0.46), larger ICH volume (OR 0.98), cerebrospinal fluid diversion for hydrocephalus (OR 0.51), postoperative symptomatic brain infarction (OR 0.50), postoperative meningitis (OR 0.36), and pneumonia (OR 0.64).

Conclusion: More than half of patients discharged with severe disability after aSAH achieved good functional recovery within 3 months. Increasing age, severe hemorrhage, and postoperative complications were consistently associated with reduced recovery.

Disclosure: Behnam Rezai Jahromi and Mika Niemelä have patents pending on aneurysm imaging.

EPO-0677 | Endovascular treatment versus best medical treatment in ischemic stroke due to distal to middle vessel occlusions: A meta-analysis

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Background and aims: While mechanical thrombectomy (MT) is indicated in the treatment of large vessel occlusions, the consensus is still lacking regarding its superiority over the best medical treatment (BMT) in distal to middle vessel occlusions (DMVO). Aim of this pooled analysis was the investigation of the efficacy and safety of MT in DMVO compared to the BMT, including endovenous thrombolysis.

Methods: The study followed PRISMA guidelines. PubMed, EMBASE, and Cochrane Central were searched for studies comparing MT and BMT in adults with acute DMVO. The primary endpoint was good functional outcome at 180 days (mRS <3); secondary endpoints included excellent outcome at 3 months

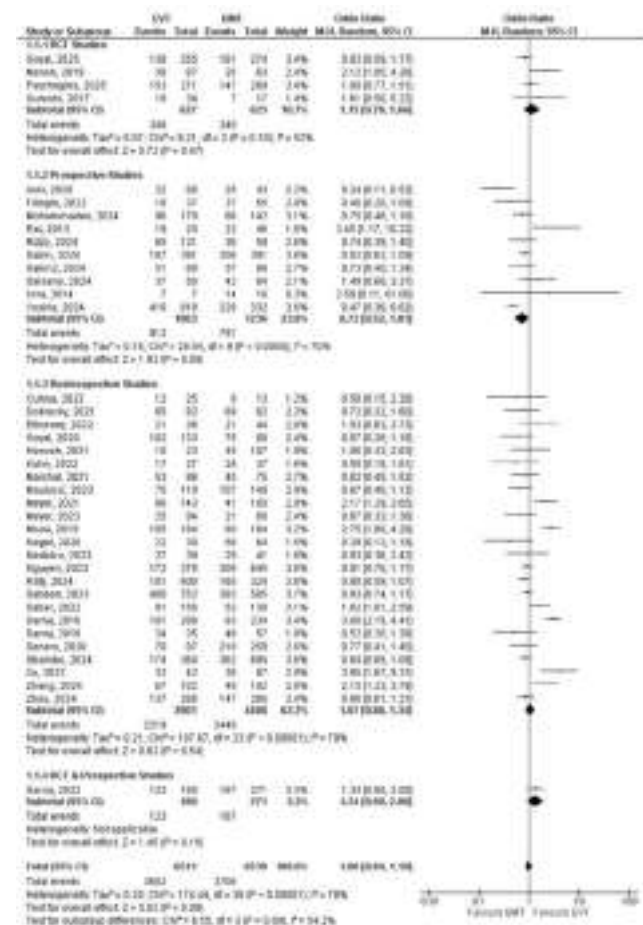


FIGURE 1 Forest plot of the primary endpoint with distribution by study type (EVT vs BMT).

Fig 3: Forest plots of the secondary endpoints of the pooled analysis EVT vs BMT.

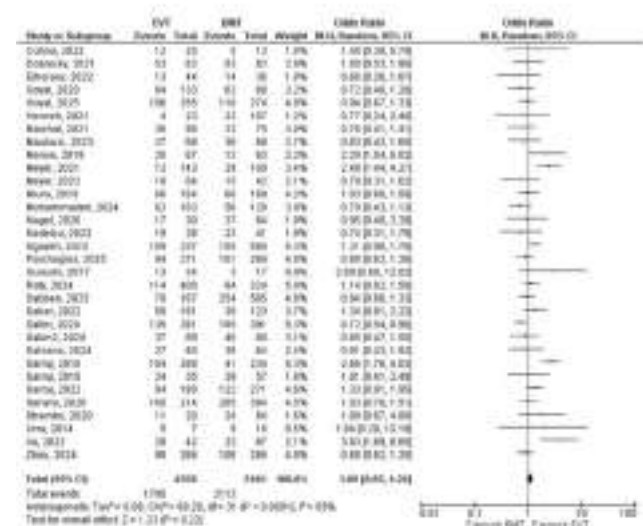


Fig 3A: Forest plot of excellent functional outcome.

FIGURE 2A Forest plot of excellent functional outcome.

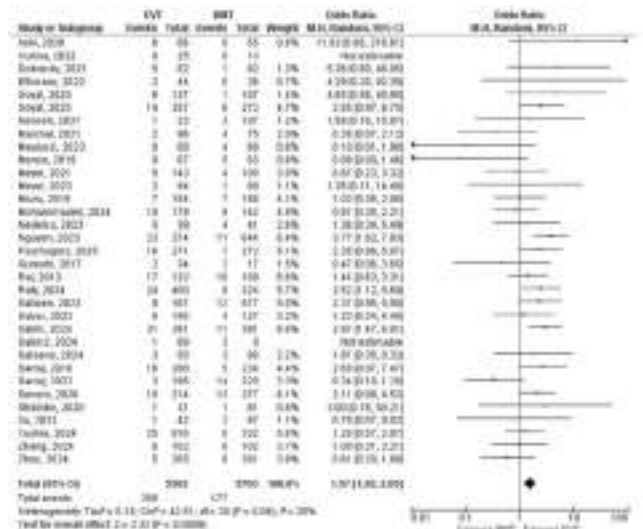


Fig 3B: Forest plot of death rate.

FIGURE 2B Forest plot of death rate.

(mRS <2), sICH, and 180-day mortality. Outcomes were pooled using meta-analysis.

Results: A total of 43 studies including 13,049 patients were analyzed. Good functional outcome at 180days was similar between MT and BMT (OR 1.00; 95% CI 0.84–1.19). The two treatment strategies had no differences in excellent outcome at 3 months (OR 1.09; 95% CI: 0.95–1.26; pheterogeneity <0.0001), as well as mortality (OR 1.09; 95% CI: 0.95–1.26; pheterogeneity <0.0001), while sICH rates were significantly higher in the MT group (OR=1.57; 95% CI: 1.20–2.05; pheterogeneity=0.06).

Conclusion: This meta-analysis shows similar functional outcomes between endovascular treatment and BMT in DMVO, with a higher rate of sICH in the MT group. These findings highlight the importance of patient selection based on occlusion site, collateral status, and clinical presentation, and support the need for further studies using stricter selection criteria.

Disclosure: Nothing to disclose.

EPO-0678 | Multiple fusiform cerebral aneurysms and inflammatory vasculopathy associated with cardiac myxoma

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Background and aims: Fusiform cerebral aneurysms are a rare complication of cardiac myxomas, possibly due to myxomatous cell embolization to the vasa vasorum. This phenomenon may lead to diagnostic uncertainty in acute cerebrovascular presentations.

Methods: We report a comprehensive diagnostic workup in a patient with recurrent ischemic events, including multimodal computed tomography (CT), digital subtraction angiography (DSA), brain MRI with vessel wall (VW) imaging, cerebrospinal fluid (CSF) analysis, autoimmune and infectious screening, and whole exome sequencing (WES).

Results: A 41-year-old woman with a previous left atrial myxoma resection and multiple cerebral aneurysms presented with acute headache and visual disturbances. Brain CT revealed a right occipital hypodensity of uncertain nature. DSA showed in situ thrombosis of a fusiform aneurysm of the right posterior cerebral artery. Acetylsalicylic acid was started. VW MRI demonstrated concentric wall thickening and enhancement of multiple aneurysms, with leptomeningeal enhancement in cerebellar and temporo-occipital regions. CSF analysis showed pleocytosis, elevated proteins, blood–brain barrier dysfunction, and oligoclonal band mirror pattern. Autoimmune, infectious, and genetic investigations were unremarkable. Whole-body CT angiography revealed an additional small splenic artery aneurysm. During follow-up, she experienced a recurrent ischemic event due to thrombosis of a fusiform aneurysm in the right middle cerebral artery territory. Six-month VW MRI showed persistent enhancement without size change.

Conclusion: This case highlights a dynamic inflammatory cerebral vasculopathy related to cardiac myxoma. Detailed

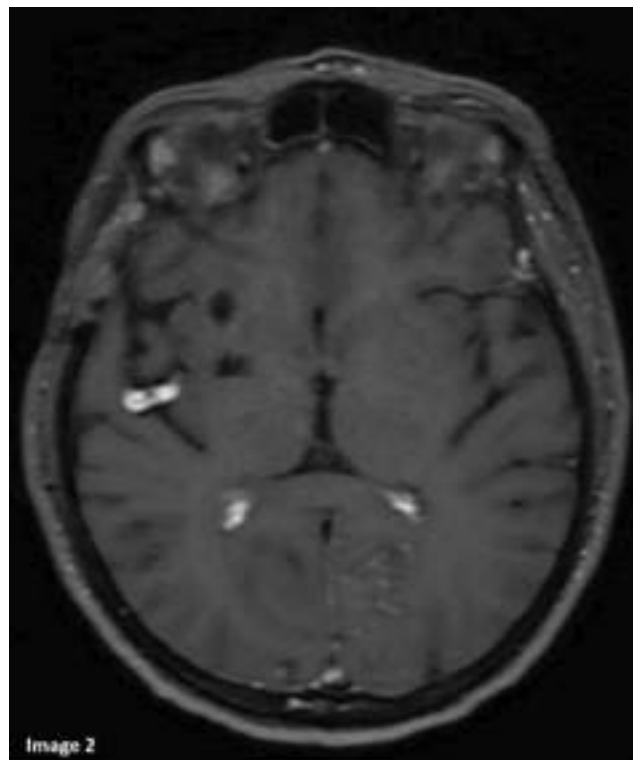


IMAGE 2 Axial T1-weighted VW MRI after contrast injection showing concentric thickening of one of the aneurysms located in the M2 segment of the right middle cerebral artery, with strong contrast enhancement.

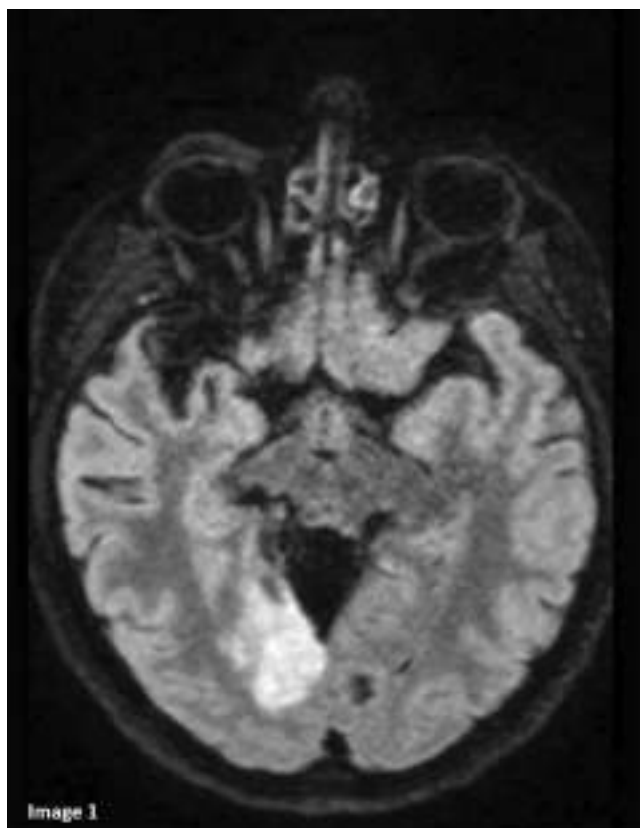


IMAGE 1 Axial FLAIR MRI showing hyperintense signal in the right occipital lobe.

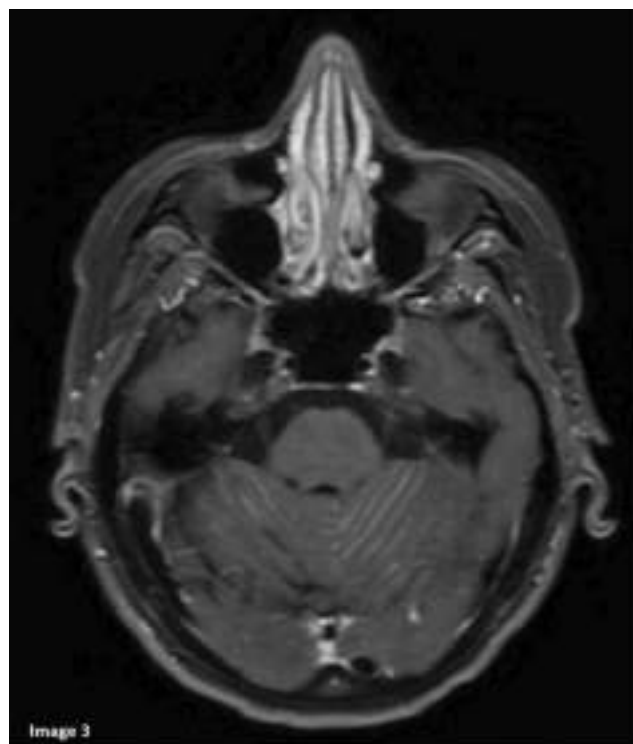


IMAGE 3 Axial T1-weighted MRI showing leptomeningeal contrast enhancement of the cerebellar folia.

vascular characterization is crucial to guide management and avoid misdiagnosis in the acute stroke setting.
Disclosure: Nothing to disclose.

EPO-0679 | **Determinants of acute symptomatic seizures and post-stroke epilepsy after reperfusion therapy: an 18-month Dual Center Cohort Study**

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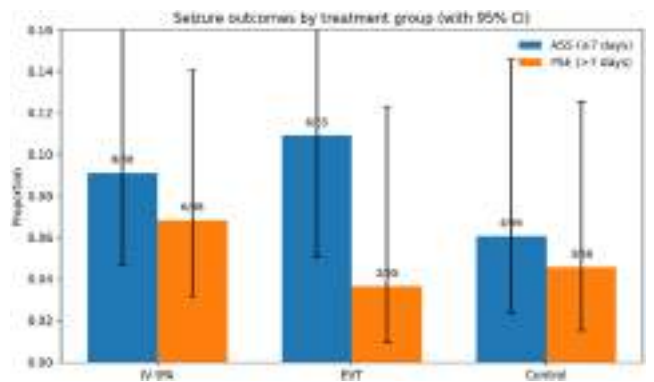
Background and aims: Seizures are a frequent complication of stroke and are associated with worse functional outcomes. With the widespread use of reperfusion therapies (RTs), including intravenous thrombolysis and mechanical thrombectomy, their potential impact on acute symptomatic seizures (ASS) and post-stroke epilepsy (PSE) requires clarification.

Methods: We conducted a retrospective cohort study over 18 months at the Neuro vascular Department of the University Hospital Centre “Mother Teresa” and Stroke Center American Hospital, Tirana. Consecutive patients with acute ischemic stroke were grouped as intravenous thrombolysis (IV tPA, $n=88$), endovascular thrombectomy with or without thrombolysis (EVT, $n=55$), and non-revascularized controls ($n=66$). ASS was defined as seizures within 7 days and PSE as unprovoked seizures beyond 7 days. Hemorrhagic transformation (HT) was assessed on follow-up imaging.

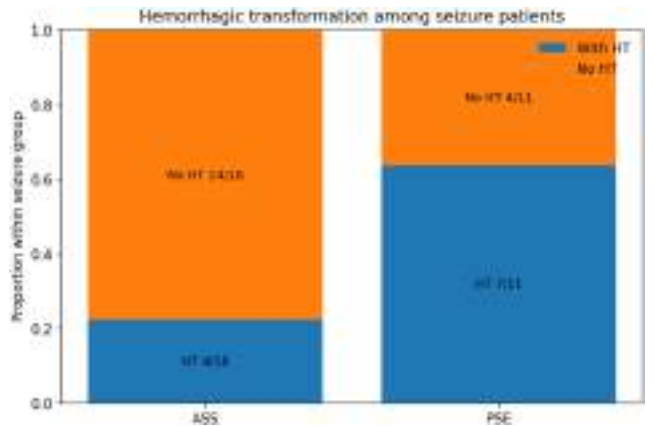
Results: ASS occurred in 8/88 (9.1%) IV tPA patients, 6/55 (10.9%) EVT patients, and 4/66 (6.1%) controls ($p>0.05$). PSE developed in 6/88 (6.8%), 2/55 (3.6%), and 3/66 (4.5%) patients, respectively ($p>0.05$). When reperfusion-treated patients were pooled, ASS occurred in 14/143 (9.8%) versus 4/66 (6.1%) in controls. HT was present in 4/18 (22.2%) patients with ASS and in 7/11 (63.6%) patients with PSE, indicating a strong association between HT and late epilepsy. Cortical involvement was frequent among seizure patients across all groups. Median NIHSS scores among patients with ASS were similar between groups.

Conclusion: Reperfusion therapies were not associated with an increased risk of ASS or PSE. Hemorrhagic transformation and cortical involvement appear to be the principal determinants of post-stroke epileptogenesis, particularly for late epilepsy.

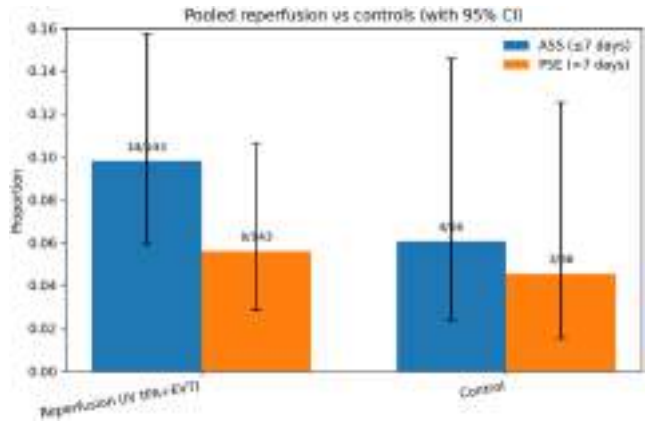
Disclosure: Nothing to disclose.



Seizure outcomes by treatment group with 95% confidence intervals.



Proportion of hemorrhagic transformation (HT) in patients with ASS and PSE.



Pooled reperfusion versus controls with 95% confidence intervals.

EPO-0680 | **Elevated cerebral venous pulsatility as an independent predictor of hemodynamic compromise and functional outcomes in atherosclerotic ischemic stroke**

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Background and aims: Venous pulsatility measurements provide insights into distal vascular resistance and intracranial hemodynamic burden, yet remain underinvestigated in cerebrovascular disease. We examined cerebral venous pulsatility as a potential biomarker for stroke severity and three-month functional recovery in patients with major artery atherosclerosis.

Methods: This observational cohort investigation enrolled 120 consecutive patients presenting with acute ischemic stroke attributable to $\geq 50\%$ stenosis of major cerebral vessels. Venous pulsatility index measurements were obtained from deep cerebral veins and principal venous sinuses using transcranial Doppler ultrasonography within 48 hours of symptom onset. Initial neurological impairment was quantified using the National Institutes of Health Stroke Scale, while 90-day functional status was determined through modified Rankin Scale assessment.

Results: Elevated venous pulsatility demonstrated strong associations with greater baseline neurological compromise (NIHSS ≥ 10 : venous pulsatility index 1.46 ± 0.18 versus 1.29 ± 0.16 , $p < 0.001$). Unfavorable three-month outcomes (mRS 3–6) occurred in 52 patients (43.3%) and showed significant

correlation with increased baseline venous pulsatility ($p < 0.001$). Multivariable logistic regression analysis, adjusting for patient age, lesion volume, stenosis severity, and collateral status, confirmed elevated venous pulsatility as an independent predictor of poor functional recovery (OR 2.7, 95% CI 1.5–5.0).

Conclusion: Cerebral venous pulsatility effectively identifies hemodynamic stress and independently forecasts functional outcomes following atherosclerotic ischemic stroke. Assessment of venous pulsatility may offer a valuable non-invasive prognostic instrument for stroke management.

Disclosure: Nothing to disclose.

EPO-0681 | Analysis of the racial differences in the prevalence of risk factors at stroke onset in Italy: a prospective, multicentric study on 2402 patients

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Background and aims: Risk factors for Ischemic Stroke (IS) and adherence to primary prevention strategies may differ substantially across racial and ethnic groups. However, available evidence derives from the USA, where the racial/ethnic composition differs markedly from that of Europe. The primary aim of our study was to investigate racial/ethnic differences in the prevalence of stroke risk factors and primary prevention therapies in Italy.

Methods: Consecutive adult patients with IS presenting to 14 Italian stroke centers between October 2024 and November 2025 were prospectively enrolled. Based on self-reported race/ethnicity, patients were classified as White or non-White. Outcomes were the prevalence of stroke risk factors and preventive therapies in use at time of stroke onset. The Mann-Whitney U -test and χ^2 -test were used for statistical comparisons. To identify independent associations between race/ethnicity and stroke risk factors, multivariable logistic regression models adjusted for sex and age were performed.

Results: A total of 2402 patients were enrolled in the study, of whom 2257 were Whites (94.0%). Compared with White

patients, non-White patients were younger (62.9 ± 16.7 vs 74.1 ± 13.9 years; $p < 0.001$) and less frequently women (33.8% vs 47.2%, $p = 0.002$). Cardiopathy ($p = 0.037$), dyslipidaemia ($p = 0.016$), and atrial fibrillation ($p = 0.001$) were more prevalent among White patients, whereas non-White patients more frequently reported no ongoing therapy at admission ($p < 0.001$). However, after adjustment for age and sex, White race was not independently associated with any of these factors.

Conclusion: Non-White patients experience ischemic stroke at a younger age than White patients, potentially reflecting earlier exposure to cardiovascular risk factors and lower awareness of them and their management.

Disclosure: Nothing to disclose.

EPO-0682 | Longer onset-to-door times in non-white patients in Italy: A prospective, multicentric study on 2402 patients

I. Scala¹; P. Rizzo²; M. Monforte³; A. Zini⁴; A. Falcou⁵; M. Del Sette⁶; M. Guarino⁷; A. Cavallini⁸; L. Chiapparini⁹; M. Bagnato¹⁰; V. Andreone¹¹; C. Rapillo¹²; L. Caputi¹³; M. Diomedì¹⁴; D. Centonze¹⁵; A. Morotti¹⁶; S. Sacco¹⁷; A. Bersano¹; P. Calabresi³; G. Frisullo³

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Background and aims: Studies from the USA have reported lower access to reperfusion treatments (RTs) for Ischemic Stroke (IS) among racial/ethnic minorities; however, evidence from Italy is lacking. We aimed to assess the impact of race/ethnicity on RTS administration and onset-to-door time (ODT).

Methods: Consecutive adult patients with IS presenting to 14 Italian stroke centers between October 2024 and November 2025 were prospectively enrolled. Based on self-reported race/ethnicity, patients were classified as White or non-White. Outcomes were Intravenous Thrombolysis (IVT), Endovascular Treatment (EVT), and ODT. Associations between ODT and race/ethnicity were assessed using linear regression adjusted for demographics, comorbidities, baseline NIHSS, premorbid mRS, education level, mode of arrival, occupational status, language barriers, and cohabitation status. Logistic regressions adjusted for the same variables, plus large vessel occlusion, anticoagulant use, and ASPECT score were used to identify predictors of IVT and EVT.

Results: In total 2402 patients were enrolled in the study, of whom 2257 were Whites (94.0%). Overall, 830 patients (35.7%) received IVT and 648 (27.9%) EVT. Compared with White patients, non-White patients were younger (62.9 ± 16.7 vs 74.1 ± 13.9 years; $p < 0.001$) and less frequently women (33.8% vs 47.2%, $p = 0.002$). White category was not associated with IVT and EVT administration (aOR 0.70 95% CI (0.34–1.67), $p = 0.497$ and aOR 2.50 95% CI (0.84–7.40), $p = 0.098$, respectively) but was independently associated with shorter ODT ($\alpha\beta -631.69$ 95% CI (–1100.34 to –163.03); $p = 0.008$).

Conclusion: Our study suggests that racial/ethnic disparities in timely access to stroke care exist even in countries with universal healthcare systems, highlighting the need for public health interventions.

Disclosure: Nothing to disclose.

EPO-0683 | Abstract withdrawn

EPO-0684 | Beyond objective measures: Perceived environment associates with post-stroke quality of life

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Background and aims: Stroke causes substantial disability and reduced health-related quality of life (HRQoL). Environmental and atmospheric factors are increasingly recognized as health predictors, yet current research predominantly relies on heterogeneous objective measurements (e.g. satellite-based green space indices, air quality monitors), while subjective environmental perception remains underexplored. We investigated associations between perceived environment quality and HRQoL after stroke.

Methods: In a prospective cohort study, stroke patients were surveyed during hospitalization and at 3- and 12-month follow-ups. HRQoL was measured using the Stroke Impact Scale (SIS). Subjective environmental ratings (air quality/noise, green spaces, thermal comfort) were assessed using the Perceived Residential Environment Quality questionnaire (PREQ) and Building Environmental Quality Questionnaire (BEQQ). Multivariable regression models adjusted for age, sex, baseline mRS, baseline NIHSS, and educational background were employed to analyse associations between subjective environmental perception and long-term HRQoL.

Results: Of 531 baseline participants (mean age 68.8 years, 39.4% female), 456 (85.9%) completed 12-month follow-up. First analyses revealed that thermal comfort at 3 months is significantly associated with multiple SIS domains at 12 months. Green spaces and air quality showed no significant associations after correction for multiple testing.

Conclusion: While prior research emphasizes objective environmental measures, our findings highlight subjective perception, particularly thermal comfort, as a potentially relevant factor for post-stroke HRQoL. Thus, a rather easily adaptable factor might already improve long-term outcomes of stroke survivors, if considered in rehabilitation and post-stroke care.

Disclosure: Nothing to disclose.

EPO-0685 | Association between cortical microinfarcts and cortical superficial siderosis in cerebral amyloid angiopathy: Evidence for a double-hit mechanism?

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Background and aims: Cerebral amyloid angiopathy (CAA) is characterized by hemorrhagic and non-hemorrhagic injuries. Cortical cerebral microinfarcts (CMI) have been associated with cortical superficial siderosis (cSS), although data are inconclusive. This study aims to determine the spatiotemporal relationship between CMI and cSS in CAA, with the hypothesis that cSS can promote local ischemic injury.

Methods: Probable CAA (Boston Criteria v2.0) were recruited from a prospective Massachusetts General Hospital cohort. CAA-related radiological manifestations—including CMI and cSS—were quantified at baseline and at 24 months. Multivariate logistic regression and generalized linear models were applied to assess associations between baseline radiological features and their predictive value on radiological progression. We compared the local density of CMIs—defined as CMI Density Index, CDI—in regions with and without cSS.

Results: Among 74 probable CAA (age: 73.0 [67.9–78.2] years), 26 (35%) had CMIs ($n = 135$, 39% underlying cSS). At baseline, CMI count demonstrated an exponential relationship with cSS volume ($\text{Exp}(B) = 1.12$ [95% CI 1.07–1.18]; $p < 0.001$) and with lobar lacunes count ($\text{Exp}(B) = 1.65$ [95% CI 1.52–1.80]; $p < 0.001$). The CDI was markedly higher in cSS-covered vs. non-cSS regions (median: 20.84 [0.00–56.95] vs. 0.49 [0.11–0.89]; $p = 0.010$). Follow-up scans ($n = 36$) revealed 23 incident CMIs (43% underlying cSS). Baseline cSS volume predicted CMI occurrence (OR: 1.41 [95% CI 1.02–1.95]; $p = 0.036$), whereas baseline CMI burden did not predict cSS progression.

Conclusion: Our findings demonstrate a CMI/cSS interplay, with cSS predicting incident CMI but not vice versa. Higher CDI in cSS-covered regions suggests local ischemia synergistically worsened by global ischemic burden. Further studies should identify the driving pathophysiology, a possible therapeutic target for preventing cSS-induced injury.

Disclosure: Nothing to disclose.

EPO-0686 | Stress hyperglycemia ratio and risk of cerebral edema in acute ischemic stroke: A systematic review and meta-analysis

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Faculty of Medicine, Mansoura University, Egypt

Background and aims: Stress hyperglycemia is common following acute ischemic stroke (AIS) and correlates with adverse outcomes. While absolute hyperglycemia is a known risk factor, the Stress Hyperglycemia Ratio (SHR) offers a more physiological metric of relative hyperglycemic stress. However, its specific relationship with life-threatening complications like cerebral edema remains incompletely defined. We aimed to evaluate the association between high SHR and the development of cerebral edema in patients with AIS.

Methods: A systematic review and meta-analysis were conducted in accordance with PRISMA guidelines. We searched major electronic databases including PubMed, Scopus, Web of Science and Cochrane Library up to December 2025 for observational studies that evaluate the predictive impact of SHR on cerebral edema formation in adult AIS patients. The primary objective was to quantify the risk magnitude associated with elevated SHR levels. Pooled odds ratios (ORs) with 95% confidence intervals (CIs) were calculated using a random-effects model.

Results: Five studies comprising a total of 2634 patients were included in the meta-analysis. The pooled analysis demonstrated that high SHR was significantly associated with an increased risk of cerebral edema following ischemic stroke (OR 2.56, 95% CI 1.32–4.96, $p=0.006$). This indicates more than a twofold higher odds of cerebral edema among patients with elevated SHR.

Conclusion: Stress hyperglycemia ratio is a significant predictor of cerebral edema after ischemic stroke. Early calculation of SHR may facilitate the rapid identification of high-risk patients, informing closer neurological monitoring and targeted therapeutic strategies to mitigate edema progression.

Disclosure: Mohamed Serageldin, Amir Hegazi, Hussein o. Hussein, Ahmed Hafez, Mohamed Elfadali, Mahmoud Abd EL-Hamied and Sherif Abouelenein: Nothing to disclose.

EPO-0687 | A decade of audio-vestibular presentations to TIA service: A high-risk cerebrovascular presentation

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Background and aims: Vestibular symptoms are common in emergency settings but are perceived as low risk for cerebrovascular disease. Sudden-onset hearing loss and pulsatile tinnitus, whilst recognised as necessitating urgent input, are commonly seen through ENT pathways. We aimed to quantify the burden of respectively vestibular and auditory presentations to a large tertiary transient ischaemic attack (TIA) service and determine associated stroke and TIA diagnoses.

Methods: Prospectively collated consecutive referrals to a central London TIA service between June 2015 and November 2025 were analysed. Referral reasons were categorised into neurological symptom clusters. Vestibular presentations were defined using Bárány Society-informed descriptors, including vertigo, dizziness, imbalance, unsteadiness, and giddiness. Auditory symptoms were defined as hearing loss and tinnitus. We calculated the proportion of referrals for these symptom clusters and subsequent stroke/TIA diagnoses, comparing outcomes using logistic regression.

Results: Vestibular symptoms accounted for 14% ($n=1783$) of 12,760 new referrals. Patients referred with isolated vestibular symptoms had a definite or probable stroke/ TIA diagnosis rate of 24% ($n=241/994$), which significantly increased to 39% ($n=306/789$) for vestibular presentations referred with additional neurological symptoms (OR 1.98, 95% CI 1.62–2.43; $p<0.001$). By contrast, just 46 patients (<0.5%) presented with auditory symptoms, but 5/46 were diagnosed with definite or probable stroke/TIA and 2/46 with cervical artery dissection. One dissection presented with isolated sudden-onset pulsatile tinnitus and headache without other neurological features.

Conclusion: Auditory and vestibular presentations to a specialist TIA clinic have high cerebrovascular diagnostic rates. Findings support low thresholds for specialist assessment to reduce missed neurovascular events.

Disclosure: Nothing to disclose.

Neuro-Ophthalmology/Neuro-Otology 1

EPO-0688 | Comparative evaluation of large language models in differentiating grades of papilledema using optical coherence tomography

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Background and aims: Accurate grading of papilledema is essential for monitoring disease progression and guiding management in patients with intracranial hypertension. Large language models (LLMs) have emerged as potential clinical decision-support tools; however, their performance in differentiating papilledema grades using Optical Coherence Tomography (OCT) -derived features remains unclear.

Methods: This observational comparative study included 15 de-identified OCT datasets from patients with idiopathic intracranial hypertension, stratified by papilledema severity: mild (Frisén grades I–II, $n=5$), moderate (grade III, $n=5$), and severe (Frisén grades IV–V, $n=5$). Structured OCT descriptions, including peripapillary retinal nerve fiber layer (RNFL) thickness, optic nerve head contour, and macular changes, were

independently analyzed by ChatGPT, Google Gemini, and Microsoft Bing. Model-assigned severity grades were compared with clinician-determined Frisén grades.

Results: Overall accuracy for correct papilledema severity classification was highest for ChatGPT (77%), followed by Google Gemini (69%) and Microsoft Bing (55%). For mild papilledema (grades I–II), ChatGPT correctly classified 80% of cases, Google Gemini 72%, and Microsoft Bing 60%. In moderate papilledema (grade III), accuracy declined across all models, with correct classification rates of 68% for ChatGPT, 70% for Google Gemini, and 48% for Microsoft Bing. For severe papilledema (grades IV–V), ChatGPT demonstrated the highest accuracy (88%), followed by Google Gemini (75%) and Microsoft Bing (58%). Misclassification most frequently occurred between adjacent grades, particularly between mild and moderate papilledema, and moderate and severe papilledema.

Conclusion: Large language models demonstrate moderate accuracy in differentiating papilledema severity using OCT-derived features, with more reliable performance at the extremes of disease severity and reduced accuracy for intermediate grades.

Disclosure: Nothing to disclose.

EPO-0689 | Vestibular agnosia in elderly subjects with small vessel disease and imbalance – preliminary results from an ongoing prospective study

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Background and aims: Vestibular agnosia (VA) is a brain disconnection syndrome that manifests with imbalance and an altered sensation of self-motion perception despite an intact peripheral vestibular system. VA can be measured and quantified employing vestibular psychophysics. Only recently VA was described for the first time in acute traumatic brain injury. VA has been reported to occur in elderly patients in whom there is disconnection within the brain due to small vessel disease (SVD). We assess vestibular perception and its relation to balance control in elderly patients with SVD and imbalance, dizziness and falls.

Methods: Ongoing exploratory prospective two-centre study. 9 patients with SVD (Fazekas 2–3) and imbalance/ falls were assessed, 2 males, 7 females (age 78–93, average 82.8 years). 4 elderly controls were assessed, 1 male, 3 females (age 78, 72, 74, 89 years). Vestibular perceptual thresholds (VPTs) were determined for yaw-plane rotations with randomly presented half cosine stimuli (0.1 Hz). Peripheral vestibular function, balance control, reaction times, cognition and self-reported symptoms of dizziness, anxiety and depression were assessed.

Results: In those subjects with SVD elevated VPTs (overall range 1.1–11.4 deg/sec, mean 6.1 deg/sec) were correlated with impaired balance control and lower subjective symptom scores of dizziness. VPTs in elderly control subjects were 2.4 deg/sec (1.7–3.1 deg/sec) which is within limits of normal subject's performances aged <65 years (cut-off 5.0 deg/sec).

Conclusion: VPTs in elderly subjects >70 years might be within a normal range. VPTs might serve as a biomarker for imbalance and risk of falls in elderly people.

Disclosure: Flurin Honegger has worked as a consultant for the company which made the equipment used in this study to assess balance control. This work was supported by Freiwillige Akademische Gesellschaft (FAG) Basel.

EPO-0690 | Square wave jerk in de novo idiopathic Parkinson's disease: Insights into the dynamic overshoot subtype

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Background and aims: Square wave jerks (SWJs) are pairs of small saccades that takes the gaze away from the target and then return it, commonly observed in neurodegenerative disorders. Although prominent in progressive supranuclear palsy, their clinical significance in Parkinson's disease (PD) is not well defined.

Methods: We retrospectively reviewed 145 de novo PD patients who underwent video-oculography, UPDRS part III, Hoehn and Yahr staging, cognitive testing, and assessments for orthostatic hypotension (OH) and probable REM sleep behavior disorder (RBD). SWJs were categorized as macro ($\geq 5^\circ$), staircase, or dynamic overshoot. Associations with motor severity, cognition, non-motor features, and PD motor subtypes (tremor-dominant [TD], postural instability/gait difficulty [PIGD], indeterminate) were analyzed.

Results: Among 67 TD, 41 PIGD, and 37 indeterminate patients, RBD (47.6%) and OH (29.7%) were most frequent in PIGD, though without statistical significance. Overall SWJ frequency did not differ across motor subtypes. Macro SWJs were rare (3.4%). Staircase SWJs (49.7%) correlated with higher SWJ counts ($p < 0.001$) but no with clinical parameters. Dynamic overshoot SWJs (42.1%) were associated with higher SWJ frequency ($p < 0.001$), worse motor severity (UPDRS part III, $p < 0.001$), and higher PIGD scores ($p = 0.032$). No associations were observed with cognition, OH, or RBD.

Conclusion: Dynamic overshoot SWJs, unlike other subtypes, were linked to more severe motor impairment in de novo PD. These findings suggest that dynamic SWJs may reflect cortical inhibitory dysfunction, in which diminished inhibitory control from the frontal eye field results in disinhibition and subsequent hyperactivation of the superior colliculus.

Disclosure: Nothing to disclose.

EPO-0691 | Magnetic resonance imaging of the visual pathway in Leber's hereditary optic neuropathy: A systematic review

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Background and aims: Leber's hereditary optic neuropathy (LHON) is a rare but important cause of blindness in young adults. Similarly to what is observed in other acquired optic neuropathies, paraclinical tests such as MRI could aid in LHON diagnosis; however, the typical MRI features of the visual pathway in LHON remain largely unknown. We conducted a systematic review on brain and/or orbit MRI findings in LHON.

Methods: We performed a search of published articles between 2008 and 2024 using databases from PubMed, EMBASE, SCOPUS and Web of Science Core Collection in accordance with PRISMA guidelines.

Results: We included 128 studies with 423 patients, plus 5 from our own series. The mean age at presentation was 34.3 years (SD 17.0). MRI abnormalities in patients presenting with simultaneous or unilateral vision loss, were observed in 48% of eyes. The most frequent pattern was a combination of intraorbital and intracranial/prechiasmal involvement of the optic nerve (19.5%), followed by isolated intraorbital involvement (18.2%), intraorbital, intracranial, and intracranial/prechiasmal involvement (19.5%), and intraorbital with intracranial involvement (6.1%). MRI abnormalities included T2 hyperintensity (77.4%), atrophy (7.5%), T1 gadolinium enhancement (3%), and combined T2 hypersignal with T1 gadolinium enhancement (11.3%).

Conclusion: Optic nerve MRI abnormalities are present in half of LHON patients and become less frequent by the time of second eye involvement. Importantly, these may mimic MRI patterns commonly seen in non-genetic acquired optic neuropathies such as aquaporin-4 and MOG-related diseases and multiple sclerosis. Awareness of MRI patterns in LHON is crucial, prompting early diagnosis and better treatment outcomes.

Disclosure: Nothing to disclose.

EPO-0692 | Effects of noisy galvanic vestibular stimulation on gait and balance in bilateral vestibulopathy: A randomised sham-controlled crossover pilot study

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Background and aims: Noisy galvanic vestibular stimulation (nGVS) has been suggested as a tool to improve gait and balance function in bilateral vestibulopathy (BVP). We conducted a pilot study to assess the effect of nGVS on gait and balance in BVP.

Methods: Subjects performed gait and balance tasks under stimulation and sham conditions: (1) standing on firm ground/foam with/without vision; (2) the timed-up-and-go (TUG) test; (3) 6m walk at a self-selected comfortable speed and slow speed

(50% of self-selected comfortable speed). The order of stimulation and sham was randomised and counterbalanced. A consistent nGVS stimulus (white noise, 0–100Hz, peak-to-peak 0.4mA, ramped up and down in 5 seconds) was used in all conditions. Gait and balance behaviour was quantified using a force plate, optical and video-based motion capture systems. Primary outcome measures were: postural sway during standing; TUG completion time and turn speed; gait pace, variability, rhythm, asymmetry, and postural control metrics during walking.

Results: Ten subjects (72yr median) with BVP participated in the study. No subject was able to distinguish stimulation from sham, confirming double-blinding efficacy and that nGVS stimulation was sub-perceptual. We did not find evidence of a difference between stimulation and sham conditions in gait or balance function during the standing, TUG, normal or slow speed walking tasks.

Conclusion: At the group level, we did not find evidence that nGVS improved gait and balance in patients with BVP. Individual improvements across some parameters were observed with nGVS and further studies may seek to characterise these further to develop more tailored treatment approaches.

Disclosure: Cochlear Technology Centre Belgium financially supported this study.

EPO-0693 | Bilateral vestibulopathy: Strong association with the ZNF91/LINC01224 locus

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Background and aims: Bilateral vestibulopathy (BVP) is a chronic disabling vestibular disorder. Known etiologies are, e.g. ototoxic exposure or genetic syndromes including RFC1-related disorders and FGF14-GAA-Ataxia (SCA27b). They explain only a minority of cases, leaving a substantial proportion idiopathic. To address this gap, we conducted a genome-wide association study (GWAS) in patients with idiopathic BVP.

Methods: We analyzed a European cohort of 132 individuals diagnosed with idiopathic BVP and 3410 unaffected controls. Diagnosis was based on the current diagnostic criteria of the Bárány Society. Following quality control and removal of outliers, GWA was performed on approximately 7.5 million variants, complemented by gene-based analysis with MAGMA.

Results: GWAS identified two loci reaching genome-wide significance. The strongest signal mapped to chromosome 19 (ZNF91/LINC01224), with rs12185481 ($p = 3.60E-16$) as the lead variant. A second, more tentative signal emerged on chromosome 2 represented by rs73006457 ($p = 4.46E-08$) with GPC1 and OTOS as the nearest candidate genes. Gene-based analysis revealed six significant loci, including ZNF91, highlighting novel candidate genes with potential relevance to BVP pathogenesis.

Conclusion: GWAS points to ZNF91/LINC01224 as a major contributor with a functional link between transcriptional regulation, retrotransposon silencing, and vestibular pathology with age-related decline in ZNF91 expression potentially amplifying genetic risk. The second locus near OTOS on chromosome

2 represents a less robust plausible candidate, as OTOS is expressed in the inner ear, previously associated with susceptibility to ototoxic-induced hearing loss. Together, these findings highlight novel pathways in BVP pathogenesis and merit replication in larger, independent cohorts.

Disclosure: Nothing to disclose.

EPO-0694 | Head circumduction paradigm as a model to stimulate the vestibulo-spinal activity

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Background and aims: Vestibulospinal reflex (VSR) mechanisms are difficult to isolate. Here we applied the head circumduction paradigm (Corrado et al., 2025), which induces a transient vertical semicircular canal asymmetry, to assess the VSR and compare responses with those of the vestibulo-ocular reflex (VOR)

Methods: We asked nine healthy participants to perform circumduction (circular) head rotations (0.75 Hz, i.e. a full head rotation every 1.33s) while standing on a force plate. We recorded baseline and post-rotational (“stopping”) posturography [centre of pressure (CoP) deviation and trunk kinematics with infrared motion markers, focusing primarily on post-rotational CoP shift. 3D video-oculography was simultaneously obtained. We assessed whether head reorientation in pitch could influence the post-rotational sway.

Results: On stopping head rotation 6 (66.7%) subjects’ trunk and CoP deviated towards the less active vestibular side (see Figure 1) by a mean of 13.4 ± 10.1 mm (CoP). The time constant of this direction-specific sway response was 12.9 ± 13.8 s. The “stopping” eye nystagmus was prominently torsional as in (Corrado et al., 2025). Head reorientation in pitch did not influence the time constant of the postural response.

Conclusion: The head circumduction paradigm can isolate the vertical canal vestibulospinal response. The direction-specificity of the postural response makes it potentially useful for clinical

purposes and not necessarily requiring complex or expensive equipment.

Disclosure: Nothing to disclose.

EPO-0695 | Vestibular migraine – Characterization of the long-term clinical course

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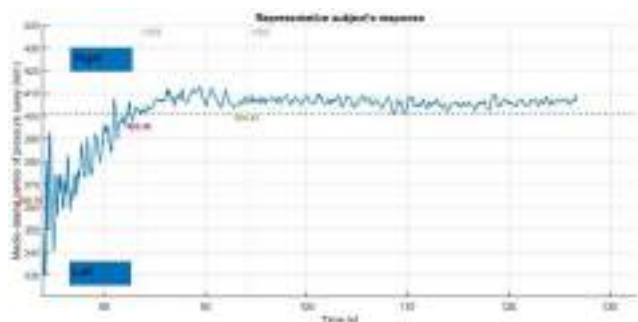
Background and aims: Vestibular migraine (VM) is the most common cause of episodic vertigo attacks with considerable clinical variability. Data on its natural course are sparse, as on effectiveness of treatment. Aim of the study was to evaluate its clinical presentation in the long-term course.

Methods: Data of 459 adult patients (~70% females, mean 42.5 years) with newly diagnosed VM according to Bárány Society’s diagnostic criteria at a tertiary vertigo centre (2010–2020) were retrospectively analyzed. All patients underwent comprehensive clinical and instrumental neuro-ophthalmological/otological examination. Long-term course of VM symptoms including therapeutic interventions was systematically explored using standardized questionnaires.

Results: At first presentation 51% of VM patients presented at least one ocular motor impairment, while extended vestibular testing was usually unremarkable. The questionnaires were completed by 111 patients after a mean of 9.7 years: 63.1% reported continued episodic vertigo, mostly improved in frequency (91%), duration (89%) and/or intensity (90%). Vertigo-related headaches decreased from 73% to 56%. Conservative migraine prophylaxis was carried out in 62% of patients, rated as helpful more for headaches (38%) than vestibular symptoms (34%). Pharmacological prophylaxis was used, at least temporarily, by 43%, rated as similarly effective for headaches and dizziness (34% vs. 37%).

Conclusion: After 10 years, approximately one third of VM patients became symptom-free, while the other two-thirds experienced less frequent and/or milder vertigo attacks. Conservative migraine prophylactic treatments were preferred, however, both conservative and pharmacological approaches were only partially effective. These results highlight the critical need for thorough patient education and further research regarding the effectiveness of various treatment strategies.

Disclosure: The study was supported by the Deutsche Stiftung Neurologie (project 80766125 and 80721017) and German Research Foundation (DFG) under Germany’s Excellence Strategy (Munich Cluster for Systems Neurology: EXC 2145 SyNergy) to MD. All other authors reported no disclosures.



Representative subject’s centre of pressure shift immediately on stopping after a right circumduction. The stopping response elicits a right vertical canals hyperactivity, whereas the subject’s COP shifts towards the left (less active) side.

EPO-0696 | Strategic postural control for differential diagnosis of persistent postural-perceptual dizziness (PPPD) and vestibular migraine (VM)

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Background and aims: Persistent postural-perceptual dizziness (PPPD) is a frequent cause of chronic dizziness/vertigo with subjective unsteadiness, whereas Vestibular Migraine (VM) is characterized by episodic vertigo attacks. However, some VM patients report persistent symptoms resembling PPPD. Clinical balance examination usually reveals no abnormalities in both. Aim of the current study was to determine whether PPPD and VM can be differentiated using posturographic stance parameters.

Methods: A retrospective analysis of a well-characterised cohort recruited at a tertiary vertigo centre included 51 PPPD and 86 VM patients. Forty-one of the 86 VM patients also presented with secondary persistent symptoms. Beside comprehensive clinical and instrumental neuro-ophthalmological/otological examination all patients underwent testing of postural control under simple and challenging sensory conditions on a stabilometer platform. Sixty-one age-matched healthy subjects served as controls.

Results: Posturographic analysis revealed distinct patterns of postural regulation in VM and PPPD: The sway variability was significantly higher in VM compared to PPPD and controls in all conditions with eyes closed. On the other hand, PPPD patients showed a stereotypical sway pattern restricted almost exclusively to the fore-aft axis. VM patients with secondary persistent symptoms presented the same postural regulation as those without PPPD-like symptoms.

Conclusion: VM and PPPD showed different postural control mechanisms providing quantitative parameters to enrich differential diagnosis. PPPD patients developed highly regulated postural control, whereas VM patients with secondary persisting symptoms maintained stance pattern of episodic VM patients with higher variability. Further analysis is still needed to understand the nature of the chronic component of this VM phenotype.

Disclosure: The study was supported by the Deutsche Stiftung Neurologie (project 80766125 and 80721017) and German Research Foundation (DFG) under Germany's Excellence Strategy (Munich Cluster for Systems Neurology: EXC 2145 SyNergy) to MD. All other authors reported no disclosures.

EPO-0697 | High-frequency vestibulo-ocular reflex in spinocerebellar ataxias with slow saccades: SHIMP to unmask saccadic dysfunction

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Background and aims: The vestibulo-cerebellum modulates the vestibulo-ocular reflex (VOR) and saccadic eye movements.

High-frequency VOR can be quantified with video head impulse testing (vHIT) using the head impulse paradigm (HIMP) and the suppression head impulse paradigm (SHIMP). Spinocerebellar ataxias (SCAs), particularly SCA2 and SCA7, often show slow saccades, but the value of SHIMP in this setting is unclear. We aimed to determine whether SHIMP can reveal saccadic dysfunction in SCA patients with slow saccades but normal high-frequency VOR gains.

Methods: We retrospectively analyzed five patients with genetically confirmed SCA (SCA2, $n = 3$; SCA7, $n = 2$). All showed horizontal saccades slowing on neurological and video-oculographic examination. VOR was assessed with an ICS Impulse vHIT system using horizontal HIMP and SHIMP, and we measured horizontal canal gains and the presence and pattern of compensatory (HIMP) and anti-compensatory (SHIMP) saccades.

Results: In all patients, horizontal canal VOR gains on HIMP were within the normal range (mean right 0.97, left 0.97). SHIMP gains were similarly normal (mean right 0.98, left 1.02), but the expected anti-compensatory saccades were absent in two patients and small-amplitude and scattered in three. Thus, despite preserved gains, SHIMP unmasked a failure to generate robust, time-locked anti-compensatory saccades in all cases, consistent with saccadic dysfunction.

Conclusion: In SCAs, SHIMP is a useful alternative VOR paradigm to unmask saccadic dysfunction that may be missed by conventional head impulse testing alone. The dissociation between normal gains and abnormal or absent SHIMP saccades suggests impaired VOR-saccade coupling and may help refine phenotyping in cerebellar ataxias.

Disclosure: Nothing to disclose.

Neuroimaging and Neurosonology

EPO-0698 | Towards metabolic biomarkers of aging

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Background and aims: Ageing is influenced by lifestyle and diseases. Multiple Sclerosis (MS) contributes to chronic low-grade inflammation ("inflammaging") and neurodegeneration, thereby affecting ageing processes. To date, brain ageing in MS has predominantly been studied using volumetric MRI approaches.

Methods: This interim analysis of an ongoing investigator-initiated trial (NCT06221631). We evaluated age-related variations in CEST pools in 7T MRI of healthy individuals ($n = 47$, aged 18–65 years) as potential biomarkers of ageing, and compared them to MS ($n = 22$, 18–63 years). CEST contrast was quantified using Lorentzian fitting of denoised, normalised, and B0- and B1-corrected z-spectra (55 offsets). Associations between age and CEST pool amplitudes were analysed for white matter (WM) and grey matter (GM), using Pearson correlations and a physics-informed deep learning model (PICA). Due to the interim nature, no covariate adjustment was applied. CEST

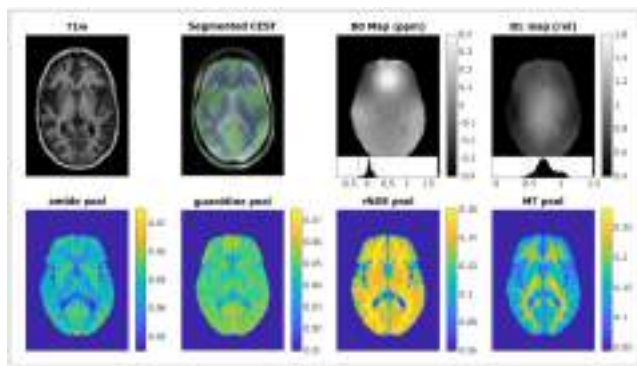


FIGURE 1 Representative MS patient included in the analysis (female, 23 years old). High-resolution anatomical image, CEST, B0 and B1 maps, and CEST contrast maps are displayed.

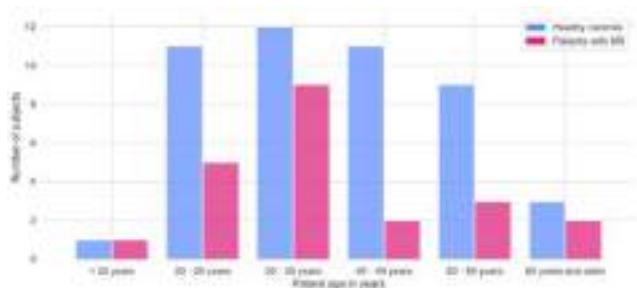


FIGURE 2 Age group distribution of the data set.

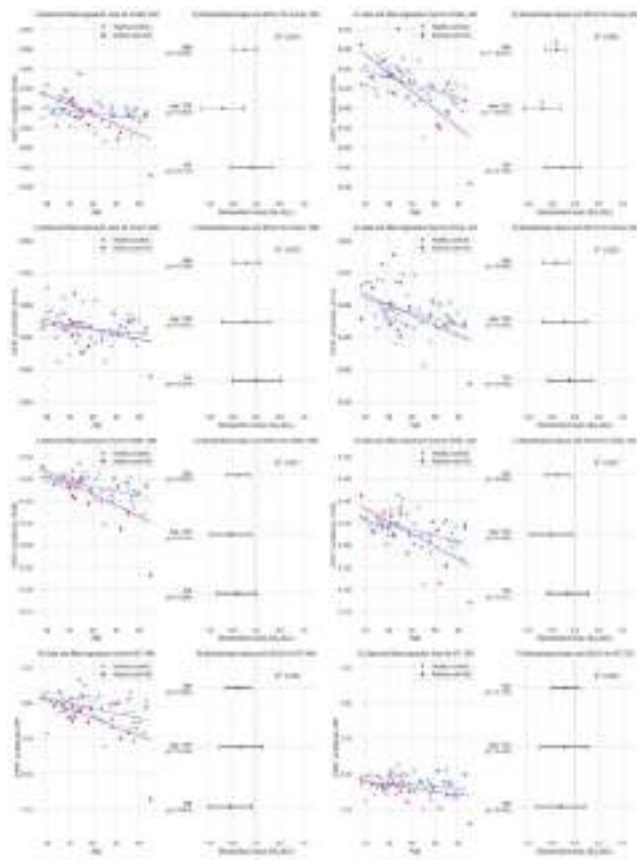


FIGURE 3 Linear regression results for amide, amine, rNOE, and MT in white matter and grey matter. Panels (A), (C), (E), (G), (I), (K), (M), (O): Measured CEST amplitudes and fitted CEST amplitude for healthy controls and patients with MS.

pool maps (amide, amine, magnetisation transfer [MT], and relayed Nuclear Overhauser Effect [rNOE]) of MS were compared to age-matched controls.

Results: We observed a consistently negative correlation of different pools with age, post pronounced in GM. A statistically significant effect of age and an MS-associated age dependency was detected especially on amides in GM (Holm corrected $p=0.013$ and $p=0.014$, respectively).

Conclusion: This preliminary analysis shows a decrease of most CEST pools with age, making them potential metabolic biomarkers of biological brain aging. After recruitment finalization (foreseen 100 HC and 100 MS) we will look more in detail into these age-associated changes overall. Additionally we will look at metabolic markers in MS lesions.

Disclosure: Betancourt A has no conflict of interests related to this work. Hoepner R has no conflict of interests related to this work. Marti S has no conflict of interests Capigliani M has no conflict of interests Radojewski P has no conflict of interests.

EPO-0699 | Evaluation of cerebral circulatory regulation using blood flow velocity spectrum analysis

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Background and aims: Near-constant global cerebral blood flow (CBF) is ensured by the simultaneous regulation of perfusion pressure and cerebral flow resistance (CVR). The CVR of a cerebral proximal vascular segment can be estimated from arterial blood pressure CBF velocity relationships during the cardiac cycle, particularly in diastole. We hypothesized that mathematical analysis of the diastolic phase of the flow velocity spectrum provides a novel and reliable method for assessing CVR. Our objective was to define a novel diastolic parameter that characterizes changes in CVR to a vasoactive stimulus and to compare its sensitivity with that of established indices.

Methods: 35 healthy volunteers' CBF velocity was measured in the middle cerebral artery using transcranial Doppler ultrasound, with noninvasive arterial blood pressure monitoring. Orthostatic stress induced by a tilt table was applied, with measurements performed at 0°, 30°, 60°, and 90°. In addition to the pulsatility index (PI) calculated from velocity ratios and the cerebral arterial resistance (CAR) expressed as the blood pressure-velocity ratio, the diastolic time constant (Tau) was introduced. Tau represents the time required for the initial diastolic flow velocity to decrease to 1/e of its value, determined by the decay constant (λ).

Results: Tau was significantly more sensitive than PI in detecting CVR changes (0–30°: $p=0.044$, $r=0.75$; 0–60°: $p=0.025$, $r=0.76$; 0–90°: $p=0.043$, $r=0.81$). Tau also showed greater sensitivity than CAR (0–30°: $p=0.047$, $r=0.754$; 0–60°: $p=0.017$, $r=0.907$; 0–90°: $p=0.043$, $r=0.504$).

Conclusion: The diastolic flow time constant (Tau) is a more sensitive marker of CVR changes than the PI or CAR.

Disclosure: Nothing to disclose.

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Background and aims: Neuronal processes underlying perception of self-produced naturalistic continuous speech remain unclear. While prior research has used temporal response function (TRF) analyses to investigate passive listening, this study established a novel MEG-based paradigm comparing passive speech perception with self-produced speech in an ecologically valid experimental setting.

Methods: Participants completed an experimental study alternating between 10-second passive listening and active speaking sections across 100 trials. They heard audiobook segments and repeated what they had just heard as closely as possible. Audio was recorded during active speaking trials. TRF analyses were computed using the first derivative of the speech envelope, marking syllable onset, as predictor of MEG measurements. Source localization extracted neuronal activation within the ROI of the early auditory cortex.

Results: An N1 component centered at 90–100 ms was visually recovered in both conditions across both hemispheres. P1 and P2 components emerged with much smaller amplitudes. Statistical analyses of defined time frames (10–50 ms for P1; 70–110 ms for N1; 140–180 ms for P2) revealed no overall differences between conditions or hemispheres. A significant N1 was found in both hemispheres of the passive condition and the right hemisphere of the active condition. P2 reached significance only in the left hemisphere of the passive condition. A glossokinetic artifact synchronous with syllable onset emerged specifically in the active speaking condition. Signal Subspace Projection (SSP) successfully corrected for this artifact.

Conclusion: This research successfully established a paradigm to measure neuronal responses to self-produced speech, demonstrating that measuring active speech in MEG using TRF requires specific artifact correction techniques.

Disclosure: Nothing to disclose.

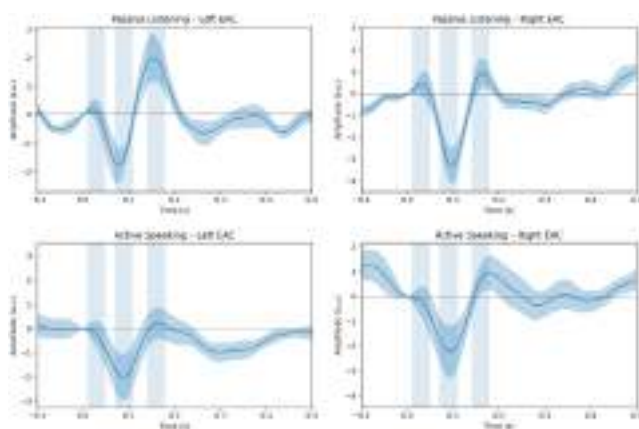


FIGURE 1 TRF wave forms extracted from the Early Auditory Cortex ROI in the passive (top row) and active condition (bottom row) in the left and right hemispheres.

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Background and aims: Although large artery atherosclerosis represents a leading cause of ischemic stroke, the contribution of venous circulation abnormalities to stroke pathophysiology has received limited attention. We investigated cerebral venous hemodynamic alterations in acute stroke patients with atherosclerotic arterial disease and examined their relationship with initial stroke severity and early clinical worsening.

Methods: This prospective investigation enrolled 120 consecutive acute ischemic stroke patients presenting with $\geq 50\%$ stenosis affecting extracranial or intracranial major arteries. Clinical assessment utilized the National Institutes of Health Stroke Scale at hospital admission and on day seven. Neuroimaging evaluation within 72 hours comprised brain MRI incorporating diffusion-weighted sequences and MR venography. Hemodynamic measurements included superior sagittal sinus flow velocity, venous pulsatility index, and venous sinus caliber, obtained through transcranial Doppler ultrasonography and MR venography.

Results: Study participants exhibited substantially diminished superior sagittal sinus flow velocity (21.9 ± 4.6 cm/s) alongside elevated venous pulsatility index values (1.38 ± 0.17). Statistical analysis revealed significant correlations between impaired venous flow and admission NIHSS scores ($r = -0.42$, $p < 0.001$) as well as ischemic lesion volume ($r = -0.39$, $p = 0.002$). Twenty-six patients (21.7%) experienced early neurological deterioration, which occurred predominantly among those demonstrating marked venous drainage impairment ($p = 0.004$). Multivariable regression modeling identified venous flow velocity as an independent determinant of early clinical worsening.

Conclusion: Disturbed cerebral venous drainage patterns occur frequently in atherosclerotic stroke and independently correlate with initial neurological severity and subsequent early deterioration. Integration of venous hemodynamic evaluation into acute stroke assessment could strengthen early prognostic stratification.

Disclosure: Nothing to disclose.

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Background and aims: Parkinson's disease (PD) is associated with neurovascular unit dysfunction and reduced cerebrovascular reactivity (CVR). Levodopa may affect cerebral hemodynamics, but its acute effect on CVR remains unclear.

TABLE 1 Demographic and clinical data.

Variation	Value
Age (years)	67.8 ± 7.0 (range 52–80)
Sex	Male 14 (70.0%)
Disease duration	8.1 ± 5.0 (range 2–21)
Hoehn & Yahr	2.10 ± 0.79 (range 1.5–4.5)
Most affected side	Left 11 (55%)
MoCA	26.9 ± 3.5 (range 16–19)
WDQ-19	1.6 ± 2.4 (range 0–8)
SCOPA-AUT	10.4 ± 6.8 (range 0–26)

Table 1: demographic and clinical data.

MoCA: Montreal Cognitive Assessment; WDQ-19: 19-item Wearing-Off Questionnaire; SCOPA-AUT: Scales for Outcomes in Parkinson's Disease – Autonomic Dysfunction.

Methods: Twenty people with PD with Hoehn and Yahr stage below 4 were enrolled. CVR was assessed using transcranial Doppler Breath Holding Index (BHI) at the middle cerebral arteries in OFF state after medication withdrawal and in ON state one hour after Levodopa intake. Sixteen patients completed Doppler assessment. Clinical evaluation included MDS UPDRS part III. Autonomic evaluation included SCOPA AUT and orthostatic blood pressure testing. Cognitive evaluation included MoCA, Stroop test and verbal fluency. Patients with cerebrovascular stenosis, MMSE below 18 or incomplete data were excluded.

Results: Mean BHI was reduced compared with normative values (0.757 vs 1.29). BHI showed a trend toward reduction in ON compared with OFF ($p=0.054$). In ON state, BHI was lower in the hemisphere contralateral to the more affected motor side ($p=0.035$), while within the more affected hemisphere BHI was higher in OFF than ON ($p=0.030$). Basal flow velocity and pulsatility index did not differ between states. Change in BHI correlated inversely with OFF BHI ($r = -0.668$, $p=0.003$). Four patients had neurogenic orthostatic hypotension without BHI differences. Levodopa improved UPDRS III scores ($p < 0.001$) and Stroop interference performance ($p=0.016$) without increasing CVR.

Conclusion: CVR is reduced in PD. Acute Levodopa administration does not improve vasodilatory reserve and may further compress CVR, supporting a vascular ceiling effect.

Disclosure: Nothing to disclose.

EPO-0703 | Non-invasive quantification of motor unit recruitment using ultrasound optical flow

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Background and aims: Motor unit assessment is commonly performed using electromyography (EMG), which records muscle fibre electrical activity but cannot directly measure muscle contraction or force generation—the processes most relevant to clinical weakness. EMG therefore provides limited insight into how neuronal activation translates into movement, including excitation–contraction coupling. As a result, surface and needle

EMG incompletely reflect motor unit output. This study assesses whether clinical ultrasound with optical flow analysis can non-invasively quantify motor unit recruitment and contractile behaviour during peripheral nerve stimulation.

Methods: Twenty-two healthy participants underwent peripheral nerve stimulation of the abductor digiti minimi, abductor pollicis brevis, and tibialis anterior muscles with simultaneous surface EMG and high-frame-rate ultrasound (100–150 frames/s). Stimulation current was reduced from supramaximal levels to generate recruitment curves. Stimulus–response relationships were modelled using four-parameter logistic regression, with EC50 defined as the current producing 50% of the maximal response.

Results: 11 male and 11 female participants took part; mean age 28 ± 9.16 . Forty-six paired recordings were analysed. Ultrasound detected motor units at lower stimulation thresholds (0.71 ± 0.82 mA earlier, $p < 0.0001$) and reached saturation earlier (2.6 ± 5.3 mA lower, $p < 0.0001$) than surface EMG. EC50 occurred at lower currents for ultrasound than EMG (7.89 ± 3.37 mA vs 8.94 ± 3.90 mA, $p = 2.35 \times 10^{-11}$).

Conclusion: Ultrasound with optical flow analysis enables non-invasive measurement of motor unit recruitment by directly quantifying muscle contraction, including deep motor units not detected by surface EMG, with potential application in neuromuscular disease assessment.

Disclosure: Nothing to disclose.

EPO-0704 | Cortical damage and tracts disconnection as predictors of 3-month aphasia severity after left ischemic stroke

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Background and aims: Tract-based disconnection metrics are increasingly used to predict aphasia, but their value beyond lesion burden remains debated. We compared atlas-based cortical lesion overlap and tract disconnection as predictors of aphasia severity at 3 months.

Methods: Fifty patients with left ischemic stroke (mean age 73.2 ± 12.8 years; 44% female) were included. Aphasia severity was assessed using NIHSS language subitem 9 at 3 months. Lesion masks were registered to MNI space. Using Tractotron, we computed the mean and maximum proportion of disconnection over the dorsal (gathering the arcuate fasciculus, the SLF and the frontal aslant tracts), ventral pathways (gathering the IFOF, ILF, uncinate tracts) and combination of both. Language cortical regions and lesion overlap percentages were computed using the Harvard–Oxford atlas. Correlations between cortical overlaps, tracts disconnection and aphasia severity were computed using the Spearman correlation test. Each feature was volume corrected by linearly regressing it out. Ordinal regression models were adjusted for lesion volume, baseline total NIHSS, age, education, and reperfusion status.

Results: Composite language-network disconnection was associated with aphasia severity at 3 months (Spearman $\rho \approx 0.55$, $p < 0.001$). Single-tract analyses showed strongest associations for dorsal pathways (arcuate long segment: $\rho = 0.51$, $p = 0.00018$; SLF III: $\rho = 0.50$, $p = 0.00026$). After lesion-volume adjustment, composite and dorsal residualized indices were not associated with outcome ($\rho = 0.17$, $p = 0.24$; $\rho = 0.16$, $p = 0.26$). Central Opercular Cortex overlap independently predicted aphasia severity ($\beta = 0.077$, $p = 0.003$; OR ≈ 2.16 per 10% overlap).

Conclusion: In this cohort, in this cohort, cortical involvement showed the most robust volume-independent association with 3-month outcome.

Disclosure: Nothing to disclose.

EPO-0705 | Carbon dots conjugated with procyanidin B2 function as diamagnetic CEST MRI theranostic agents for Alzheimer's disease

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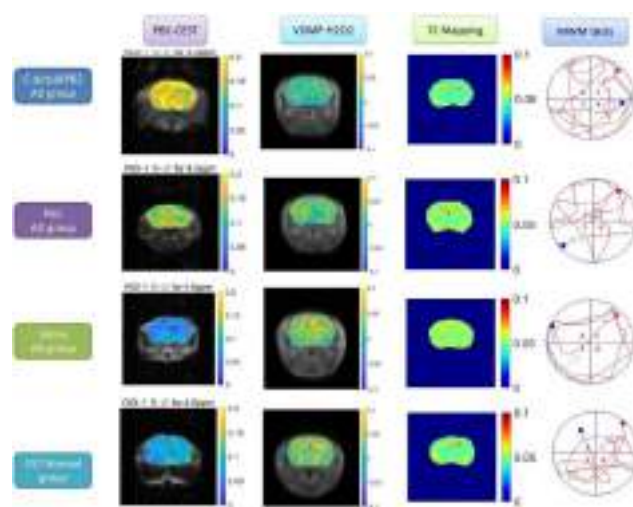
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Background and aims: Oxidative stress and neuroinflammation are recognized as central pathological mechanisms underlying Alzheimer's disease (AD). While natural procyanidins exhibit therapeutic potential by reducing reactive oxygen species (ROS) accumulation and neuroinflammation, their efficacy is limited by poor blood-brain barrier (BBB) penetration. Recent advances highlight arginine-derived carbon dots (C-dots) as biocompatible nanocarriers for brain-targeted delivery, owing to their small size, modifiable surface, and multifunctional properties, including optoelectronic behavior and size-dependent fluorescence.

Methods: We synthesized a novel MRI contrast agent C-dots@PB2. The agent has chemical exchange saturation transfer (CEST), which enabled the use of CEST imaging for monitoring c-dots distribution and provided the information for ROS and neuroinflammation accumulation.

Results: Based on in vitro and in vivo studies, we demonstrate that C-dots@PB2 exhibits CEST effects. Moreover, C-dots@PB2 efficiently mitigates ROS levels. Furthermore, the agent rapidly



In 8-month-old APP/PS1 mice and C57 mice, therapeutic efficacy was assessed by dividing subjects into C-dots@PB2 AD, PB2 AD, saline AD, and C57 normal control groups.



Histology and molecular analysis confirmed that PB2 treatment improved neuronal health, reduced amyloid plaques, and lowered neuroinflammation.

accumulates in the brains of AD mice and alleviate pathological features including A β plaque deposition, neuronal loss, and neuroinflammation. Additionally, they significantly enhance learning ability and memory function in AD mice.

Conclusion: This study demonstrates that C-dots@PB2 can attenuate the progression of AD by reducing levels of ROS and neuroinflammation accumulation. Additionally, its CEST effects offer a novel approach for developing theranostic agents targeting AD.

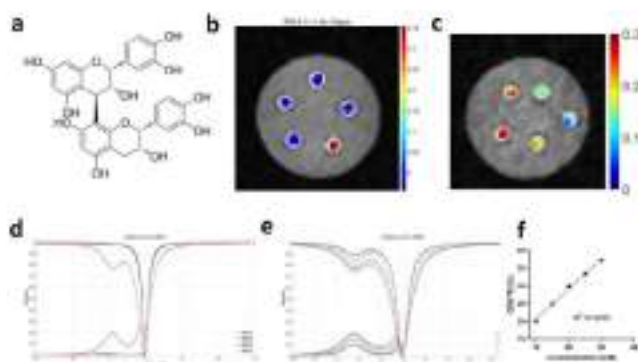
Disclosure: Nothing to disclose.

EPO-0706 | Abstract withdrawn

EPO-0707 | The importance of neuroimaging methods in assessing the aggressiveness of glioblastomas of cerebral hemispheres

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The chemical exchange saturation transfer (CEST) of PB2.

Background and aims: This study evaluates the clinical value of a newly developed MRI-based phenotypic aggressiveness scale for supratentorial glioblastomas and examines how phenotype-guided surgical strategy, including selective internal decompression, influences resectability, neurological outcomes, and early postoperative radiological dynamics.

Methods: A total of 155 patients with newly diagnosed supratentorial glioblastoma were retrospectively analyzed. Preoperative MRI findings (tumor volume, peritumoral edema, midline shift, and infiltration pattern) were used to classify tumors into three phenotypes: low-risk (A), intermediate-risk (B), and high-risk (C). Surgical outcomes, extent of resection, postoperative NANO scores, and early MRI changes were compared among groups. Selective internal decompression techniques were used selectively in B and C phenotypes.

Results: Phenotypic groups demonstrated distinct preoperative clinical and radiographic characteristics. Postoperative improvements were significant across all groups but varied by phenotype. Group A showed the most rapid recovery, with substantial reduction in edema and near-complete normalization of midline shift. Group B demonstrated moderate but significant improvement. Group C—initially the most compromised—achieved markedly better resectability (up to 80–89%) and neurological recovery when internal decompression was applied, compared with standard resection alone ($p < 0.01$). All key radiological and clinical parameters showed statistically significant differences between phenotypes ($p < 0.05$).

Conclusion: The MRI-based phenotypic aggressiveness scale reliably characterizes glioblastoma behavior and predicts surgical complexity. Phenotype-guided decision-making, particularly the use of selective internal decompression in high-risk tumors, improves extent of resection, reduces mass effect, and enhances early postoperative neurological outcomes. This combined radiologic–surgical approach represents a practical framework for optimizing treatment in supratentorial glioblastoma.

Disclosure: Nothing to disclose.

EPO-0708 | **BioSerenity-E1: A unique EEG foundation model for rapid AI development in neurological diagnostics, including Alzheimer's, sleep, or EEG abnormality**

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Background and aims: The development of AI algorithms for EEG-based diagnostics is hindered by limited annotated data, and the complexity of neurophysiological signals. BioSerenity-E1 is a unique general-purpose EEG foundation model that addresses these challenges by learning universal representations, enabling rapid fine-tuning for diverse clinical applications.

Methods: BioSerenity-E1 is a transformer-based foundation model pretrained on unlabeled EEG. The model employs intra- and inter-attention mechanisms to capture spatiotemporal dependencies. We showcase its adaptability through three key applications: Normal/abnormal EEG classification on internal multicentric ($n=4480$) and public (TUAB, $n=276$) datasets. Alzheimer's disease detection using an independent external

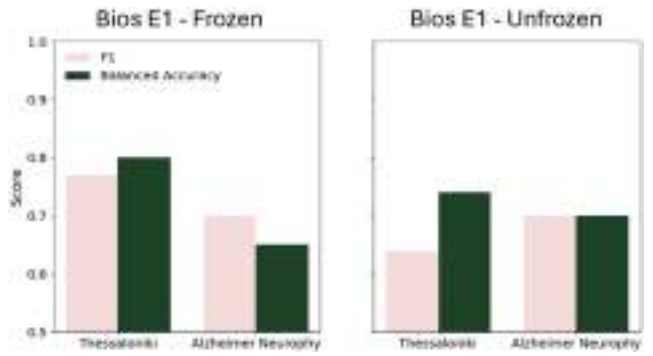


FIGURE 1 Balanced accuracy and F1-score for Bioserenity-E1 Unfrozen and Bioserenity-E1 Frozen models evaluated on internal (Neurophy) and external (Thessaloniki) test datasets.



FIGURE 2 Pediatrics sleep stages confusion matrices for internal (A, $n = 53$) and external (B, $n = 49$) datasets and macro F1 score boxplots for both datasets (C).

TABLE 1 Normal/abnormal classification performances with 95% CI of Bioserenity-E1 derived algorithm VS autoSCORE algorithm.

Algorithm	Normality (%)	Abnormality (%)	Normal accuracy (%)	Positive predictive value (%)	Negative predictive value (%)
BIOsERENITY E1	83.8	88.8	88.1	88.8	80.1
	[82.8, 84.78]	[88.1, 89.52]	[86.3, 90.1]	[87.8, 89.6]	[81.4, 82.1]
autoSCORE	80.5	84.9	87.81	85.1	88.1
	[77.6, 83.2]	[82.1, 87.8]		[81.5, 88.6]	[87.6, 90.6]

cohort ($n=65$) and an internal test set ($n=779$). Pediatric sleep stage classification on internal ($n=262$) and external datasets ($n=52$).

Results: Normal/abnormal classification: BioSerenity-E1 achieves a balanced accuracy of 93.1 [90.2 – 95.3] on a rigorously annotated test set ($n = 198$), outperforming the commercial auto-SCORE algorithm. On the unseen public TUAB benchmark, it reaches 82.25 [78.27–87.48] accuracy. Alzheimer's disease detection: The model demonstrates balanced accuracies of 0.74–0.78 on an independent external dataset and 0.69–0.70 on the internal test set, confirming its ability to generalize. Pediatric sleep stage classification: BioSerenity-E1 achieves a macro-F1 score of 0.746 on the internal test set and 0.65 on an external dataset, proving generalist viability without sleep pretraining.

Conclusion: BioSerenity-E1 accelerates the development of AI-driven EEG diagnostics, delivering state-of-the-art performance across multiple tasks. Its adaptability to heterogeneous EEG setups, and high specificity make it a powerful tool for developing additional specialized algorithms, addressing rare conditions and expanding the scope of EEG-based diagnostics.

Disclosure: Nothing to disclose.

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Background and aims: Eligibility for amyloid-targeting monoclonal antibody treatments in Alzheimer's disease requires fewer than four microhemorrhages (MH) and no superficial siderosis (SS) on baseline T2*GRE MRI. icobrain is a deep learning-based system designed to detect MH and SS. The latest version, icobrain 5.17, was trained on additional clinical trial and real-world data. This study compares its performance with the prior version, icobrain 5.16.

Methods: icobrain 5.16 was trained on 247 clinical trial cases. icobrain 5.17 incorporated an additional 356 cases (85 clinical trial, 271 real-world), totaling 603 datasets. Evaluation was performed using subsets of the Alzheimer's disease neuroimaging initiative dataset across four experiments: MH detection in MH/SS-positive cases ($n=300$); MH detection in MH-negative cases ($n=300$); SS detection in MH/SS-positive cases ($n=54$); and SS detection error in MH/SS-negative cases ($n=54$). Performance was assessed using the 90th percentile of lesion quantification error (p90) of MH and SS across cases.

Results: p90 for SS detection in SS-positive cases decreased from 3 (icobrain 5.16) to 2 (icobrain 5.17). p90 for SS in SS-negative cases decreased from 1 to 0. p90 for MH detection in MH-positive cases was 3 for both versions; and in MH-negative cases was 1 for both versions.

Conclusion: icobrain 5.17 demonstrated reduced SS quantification error compared with icobrain 5.16. Given that any SS finding may render patients ineligible for amyloid-targeting therapy, this improvement supports more accurate treatment eligibility assessment. MH error rates remained stable and below clinically relevant ineligibility thresholds.

Disclosure: All authors are employees of icometrix.

TABLE 1 90th percentile of lesion quantification error (p90) of MH and SS for icobrain 5.16 and 5.17.

icobrain version	MH+ p90	MH- p90	SS+ p90	SS- p90
5.16	3	1	3	1
5.17	3	1	2	0

p90: 90th percentile of lesion quantification error; MH+: positive microhemorrhage case, MH-: negative microhemorrhage case, SS+: positive superficial siderosis case, SS-: negative superficial siderosis case

EPO-0710 | How brain morphology predicts pain quality in trigeminal neuralgia patients: A neuroradiological regularized multivariate approach

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Background and aims: Trigeminal neuralgia (TN) is a facial pain condition characterized by unilateral, very short-lasting pain paroxysms. In up to 50% of patients, it can be associated with a distinct, more prolonged type of pain, which is commonly referred to as concomitant continuous pain. We aimed to elucidate the contribution of brain morphology into the development of this condition.

Methods: We collected 66 subjects with a definite diagnosis of classical or idiopathic right TN and acquired their brain magnetic resonance imaging (MRI) scans. We constructed a predictive model of concomitant continuous pain based on MRI-based data of cortical thickness and gray matter volume of 74 cerebral regions of interest (ROIs).

Results: According to our final model, we estimated that the most robust predictors of concomitant continuous pain in TN patients are contralateral orbital, precentral, superior frontal, superior and inferior temporal gyri, and temporal pole.

Conclusion: Our clinical and neuroradiological study can be considered a proof-of-concept for the capacity of brain morphology to predict concomitant continuous pain in TN patients. In particular, it could provide an innovative insight into the role of brain plasticity in the pathophysiology of TN.

Disclosure: The authors have no potential conflict of interest to disclose.

EPO-0711 | Small fiber dysfunction in fibromyalgia: A multimodal study

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Background and aims: Fibromyalgia (FMS) is characterized by widespread musculoskeletal pain associated with symptoms like depression, migraine and fatigue. A subset of FMS patients presents small fiber pathology. This study compares small fiber dysfunction in FMS patients with neuropathic pain and autonomic symptoms (FMS-SFN) versus pure small fiber neuropathy (SFN) patients.

Methods: 30 FMS-SFN patients and 67 SFN patients underwent sensory and autonomic evaluation through the "Small-Fiber-Neuropathy-Symptoms-Inventory-Questionnaire" (SFN-SiQ), "Composite-Autonomic-Symptoms-Score"(COMPASS-31),

Quantitative-Sensory-Testing (QST), cardiovascular reflexes, Sympathetic-Skin-Response (SSR) and Dynamic Sweat Test (DST). Cutaneous innervation was analysed on punch biopsies from three body sites applying indirect Immunofluorescence procedures.

Results: Compared to SFN patients (46 female, age 46 ± 16), FMS-SFN (27 female, age 41 ± 13) more frequently reported endometriosis (13% vs 1%), headaches (77% vs 16%) and depression (47% vs 7%). Dysimmune causes affected 47% of FMS-SFN and 36% of SFN patients, who had more glucose metabolism disorders (3% vs. 15%). FMS-SFN patients complained diffuse neuropathic pain, alongside more severe sensory and autonomic symptoms. Abnormal sensory thresholds were present in both groups with higher frequency of heat hyperalgesia in FMS-SFN (17% vs 1%) and higher thermal hypoesthesia at distal sites in SFN. FMS-SFN patients presented a lower RR variability at rest and non-length-dependent hypohidrosis with lower sweat volume per gland at proximal sites. Cutaneous sensory and autonomic innervation did not differ between the two groups.

Conclusion: FMS-SFN patients had a non-length-dependent SFN with a more diffuse neuropathic pain and more severe autonomic symptoms. They, also, presented a mild cardiovascular parasympathetic impairment and a proximal lower sweat production per gland. Skin biopsies did not differentiate the two groups.

Disclosure: Nothing to disclose.

EPO-0712 | Electrophysiological evidence of systemic neural hyperexcitability in trigeminal neuralgia: A nerve excitability study

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Background and aims: Primary trigeminal neuralgia (TN) is a representative neuropathic facial pain disorder and is classified into classical TN, which is associated with neurovascular compression, and idiopathic TN, in which no clear etiology is identified. Increasing evidence, including the identification of pathogenic genetic variants within the sensory electrogenesisome, supports a multifactorial pathophysiology that converges on neuronal hyperexcitability. This hyperexcitability may extend beyond the trigeminal nerve, reflecting a more generalized axonal predisposition. Aim: to determine whether patients with TN exhibit generalized axonal hyperexcitability detectable in peripheral motor nerves and to assess the effects of sodium channel-blocking therapy on axonal excitability.

Methods: Motor nerve excitability testing of the median nerve was performed in 35 patients with classical or idiopathic TN and compared with healthy controls. The primary outcome measure was the strength-duration time constant (SDTC), an index of persistent sodium conductance. Secondary measures included recovery cycle and threshold electrotonus parameters. Subgroup analyses compared treated and untreated patients and classical versus idiopathic TN.

Results: TN patients showed a significant prolongation of SDTC compared with controls, independent of treatment status. Superexcitability was significantly reduced in patients receiving

Na-blockers compared with untreated patients. No differences in excitability parameters were observed between classical and idiopathic TN.

Conclusion: These findings demonstrate generalized axonal hyperexcitability in TN, characterized by increased persistent sodium conductance and detectable in peripheral motor nerves. This supports the hypothesis of an intrinsic predisposition to neuronal hyperexcitability in TN. Na-blocking therapy modulates superexcitability but does not normalize SDTC, indicating distinct pharmacodynamic effects and a shared pathophysiological substrate across TN subtypes

Disclosure: Nothing to disclose.

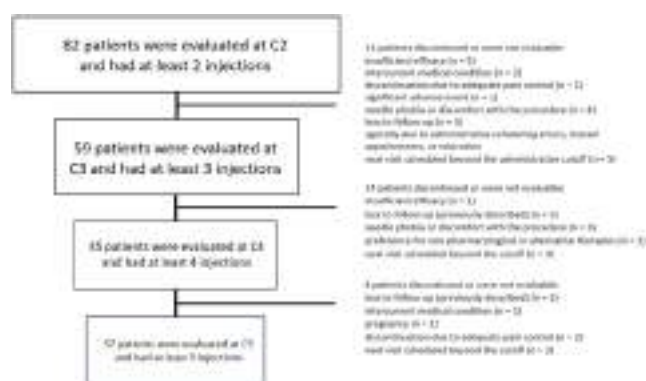
EPO-0713 | Real-world evaluation of botulinum toxin A in focal peripheral neuropathic pain: Longitudinal outcomes

E. Sole Cruz¹; E. Van Obberghen²; C. Hirtz³; I. Bennour²; M. Lanteri-Minet²; A. Donnet³

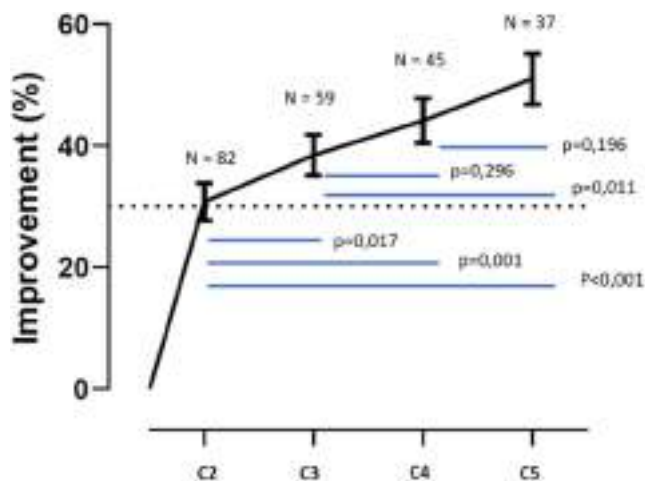
¹Reference Center for Neuromuscular Diseases and ALS, CHU Timone, Assistance Publique des Hopitaux de Marseille, Marseille, France; ²Pain treatment and evaluation Department, CHU de Nice, Nice, France; ³Pain treatment and evaluation center, CHU Timone, Assistance Publique des Hôpitaux de Marseille, Marseille, France

Background and aims: Neuropathic pain (NP) is frequently resistant to conventional treatments. Botulinum toxin type A (BT-A) is an authorized option for refractory focal peripheral NP, but the dynamics of its effect outside clinical trials in real-life conditions remain poorly characterized. The objective was to assess BT-A efficacy in a real-world study of patients with focal peripheral NP, over a one-year follow-up period.

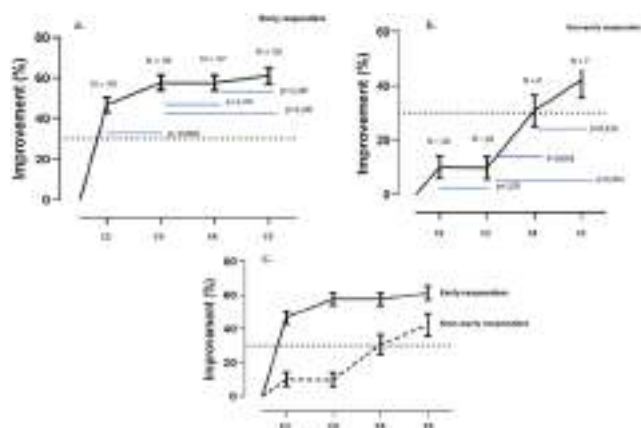
Methods: In this prospective, observational study, adult patients with chronic focal peripheral NP refractory to standard therapies were treated with BT-A in two French pain centers. Injections were individualized and administered at ~3-month intervals. The primary outcome was the patient-reported percentage of improvement since the first injection, assessed at each follow-up visit (Cycles 2 to 5). Secondary outcomes included the Patient Global Impression of Change (PGIC), average pain intensity over the previous 8 days, and exploratory analysis



Patient flow from C2 to C5. Reasons for discontinuation include insufficient efficacy, sufficient pain relief, needle phobia, loss to follow-up, and scheduling beyond study cutoff.



Mean patient-reported percentage of improvement from baseline across treatment cycles C2 to C5 in the overall study population. The dotted horizontal line represents the threshold for clinically meaningful improvement (30%). (Bonferroni-adjusted p -values).



Improvement (%) from C2 to C5 in early responders (a), non-early responders (b), and direct comparison (c). Early responders improved rapidly then plateaued, while non-early responders showed delayed but progressive gains, crossing the 30% threshold by C4.

of response trajectories distinguishing early ($\geq 30\%$ improvement at Cycle 3) from late responders.

Results: 82 patients received ≥ 2 BT-A cycles and were included in the analysis. Mean self-reported improvement increased from 30.7% at Cycle 2 to 51.0% at Cycle 5 ($p < 0.001$). PGIC and pain intensity also showed significant improvements. Among 59 patients evaluable at Cycle 3, 66% were early responders; late responders showed significant benefit after the third injection. BT-A was well tolerated: 63.6% reported end-of-dose effects, and 25.8% experienced only mild, transient adverse events.

Conclusion: This real-life study over one year suggests that BT-A provides progressive and sustained benefit in focal peripheral NP, supporting continued treatment beyond two cycles.

Disclosure: Nothing to disclose.

EPO-0714 | Repeated capsaicin patch therapy in elderly neuropathic pain: Progressive real-world improvements

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Background and aims: Localized peripheral neuropathic pain (PNP) is frequent in older adults. Systemic treatments often show limited effectiveness and are associated with relevant safety risks. Long-term real-world data on topical high-concentration capsaicin patch (HCCP) therapy in geriatric patients are limited. To assess real-world effectiveness, treatment progression, and safety of repeated HCCP applications in patients aged ≥ 75 years with localized PNP.

Methods: This retrospective cohort analysis used depersonalized 12-month routine-care data from the German Pain e-Registry (CASPAGE subgroup of CASPAR). Elderly patients receiving one to four consecutive HCCP applications were evaluated. Patient-reported outcomes included pain intensities, neuropathic pain phenotype, pain-related disability, sleep disturbance, quality of life, mood, and systemic analgesic use. The primary endpoint was a composite response defined by clinically meaningful improvement in highest pain intensity, pain-related disability, and neuropathic pain symptoms.

Results: Data from 576 patients (mean age 81.4 years) and 1576 HCCP applications were analyzed. Pain and functional outcomes improved after the first treatment and showed progressive benefits with additional applications. The composite response increased from 0.9% after one treatment to 27.5% after four. Patients receiving ≥ 3 treatments demonstrated large reductions in pain, disability, and systemic analgesic use. Adverse drug reactions were local, transient, and decreased with repeated use.

Conclusion: Repeated HCCP therapy is associated with progressive, clinically meaningful improvements and good tolerability in very elderly patients with localized PNP, supporting its role as a long-term topical treatment option.

Disclosure: The parent CASPAR registry study was supported by Grünenthal GmbH. The present CASPAGE subgroup analysis was conducted independently, without external financial support. Grünenthal had no influence on data selection, analysis, interpretation, or manuscript preparation. M.A. Überall holds leadership roles at IFNAP and O.Meany MDPM, which operate the German Pain e-Registry. Other authors declare no conflicts of interest.

EPO-0715 | Efficacy & safety of IncobotulinumtoxinA in patients with postherpetic neuralgia: Results from the PaiNT proof-of-concept phase 2 trial

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Background and aims: Postherpetic neuralgia (PHN) is a common peripheral neuropathic pain (PNP) and remains a major clinical challenge. Therapies often provide insufficient relief or cause systemic side effects. IncobotulinumtoxinA (incoBoNT-A, Xeomin®) may offer a promising local treatment. The PaiNT Phase 2 proof-of-concept trial evaluated incoBoNT-A in PHN and post-surgical/post-traumatic neuropathic pain (PS/PTNP).

Methods: The trial enrolled adults with moderate to severe PNP. The present results focus on PHN. Patients received subcutaneous injections of incoBoNT-A (≤ 300 U in ≤ 60 sites into painful area) or placebo and were followed for 20 weeks. The primary endpoint was the change from baseline in average daily pain (ADP) intensity (0–10 NRS) at weeks 2–12. Secondary endpoints included the change in Neuropathic Pain Symptom Inventory (NPSI, 0–100 points) total score and dimensions at weeks 2–12 and safety.

Results: 123 PNP patients including 63 with PHN were randomised. The endpoints did not significantly separate from placebo in the combined set. However, the PHN group showed a numerically greater reduction in ADP intensity from baseline throughout weeks 2–12 compared to placebo (LSM difference: -0.56 points) and statistically significantly greater reduction at week 6 (-1.14 points; $p=0.0488$). LSM difference to placebo in NPSI (total) change was -3.14. 50% of PHN patients received the maximum dose of 300 U. No new safety concerns were detected for incoBoNT-A.

Conclusion: The PaiNT proof-of-concept study demonstrated clinically meaningful results with a favourable tolerability profile in treatment of PHN with incoBoNT-A, supporting further research potentially with higher dosages in this PNP indication.

Disclosure: This study was funded by Merz Therapeutics GmbH. IB, IP, GK, TLG, and UM are employees of Merz Therapeutics NA has served on the advisory boards or speakers panels of Grünenthal, Merz therapeutics, Medtronic, Viatrix, Noema pharma and Pfizer. RB has received grant/research support from Pfizer, Genzyme, Grünenthal, German Federal Ministry of Education and Research (BMBF): German Research Network on Neuropathic Pain, NoPain system biology and German Research Foundation (DFG). He has received speaker honorarium from Pfizer, Genzyme, Grünenthal, Mundipharma, Sanofi Pasteur, Medtronic, Eisai, Lilly, Boehringer Ingelheim, Astellas, Desitin, Teva Pharma, Bayer-Schering, MSD and served as consultant for Pfizer, Genzyme, Grünenthal, Mundipharma, Allergan, Sanofi Pasteur, Medtronic, Eisai, Lilly, Boehringer Ingelheim, Astellas, Novartis, Bristol-Myers Squibb, Biogenidec, AstraZeneca, Merck, Abbvie. NBF has been a consultant for Mitsubishi Tanabe Pharma, Merck, Almirall, and NeuroPN. EV declares no competing interests RB has received grant/research support from EU Projects: „Europain“ (115007). DOLORisk (633491). IMI Paincare (777500). BMBF Projects: NoChro (13GW0338C), DFNS (01EM0903), NoPain, ERA-NET (IM-PAIN) Industry: Pfizer, Grünenthal, Mundipharma, Alnylam, Zambon, Sanofi Aventis, Viatrix Trademarks: „fiberASSESS®“ EU No. 018527050, „FabryScan®“ EU No. 012094959, „AmyloScan®“ EU No. 018188607, US No. 6168141. He has received Consultant /speaker honorarium from: Pfizer, Sanofi Aventis, Grünenthal, Eli Lilly, Desitin, Bayer, AbbVie, Janssen, Amicus, Novo Nordisk, Chiesi, Takeda, Stada, Ever, Unilab, Hexal, Viatrix, AstraZeneca, Aristo, Esanum, Docflix, Vertanical, Denk, Teva, Glenmark, Novartis, Biotest, Desitin, Lateral, Alnylam, Confo, EVA, Merz, Neumentum, Algo, Nanobiotix, Stada, Heat2Move, Hoba, BioNTech, Esteve, SIMR,

Desitin, SelectION, Max-Delbrück-C NBF has been a consultant for Merz, NeuroPN, Saniona, PharmNovo, Tris Pharma, AKIGAI, and Zyneyro. EV has served on the advisory boards or speakers panels of Grünenthal, Merz therapeutics, Ehyparm and Viatrix; and received research grants from Grünenthal, Merz therapeutics and Vertex.

EPO-0716 | Understanding patient diversity & placebo response in post-surgical/traumatic neuropathic pain: Results from the PaiNT proof-of-concept phase 2 trial

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Background and aims: Post-surgical/post-traumatic neuropathic pain (PS/PTNP) is characterised by diverse etiologies and pain therapies are often insufficient and cause systemic side effects. IncobotulinumtoxinA (incoBoNT-A, Xeomin®) may provide a promising local treatment of peripheral NP (PNP). The proof-of-concept PaiNT trial evaluated incoBoNT-A in postherpetic neuralgia (PHN) and PS/PTNP.

Methods: These results focus on the PS/PTNP subgroup. Patients received subcutaneous injections of incoBoNT-A (≤ 300 U in ≤ 60 sites into painful area) or placebo and followed for 20 weeks. The primary endpoint was the change from baseline in average daily pain (ADP) intensity (0–10 NRS) at weeks 2–12 (combined PHN & PS/PTNP). Secondary endpoints included the change in neuropathic pain symptom inventory (NPSI, 0–100 points) total score at weeks 2–12 and safety.

Results: The 60 PS/PTNP patients (28 incoBoNT-A, 31 placebo) were highly heterogeneous, with 48 distinct diagnoses (many potentially indicating mixed pain). Groups differed in diagnoses, PNP duration, pre-treatment history, and baseline characteristics. The endpoints did not significantly separate from placebo in the combined set. For PS/PTNP, LSM changes from baseline of ADP intensity and NPSI at weeks 2–12 for incoBoNT-A were -0.13 and -22.15. Placebo response was high (ADP intensity: -0.64; NPSI: -22.84), exceeding the changes for placebo observed in the PHN group (ADP intensity: -0.16; NPSI: -5.63). No new safety concerns were detected.

Conclusion: Improvements in ADP/NPSI in the PS/PTNP population were masked by very high placebo response. Treatment with incoBoNT-A is safe, but group heterogeneities make the comparison to placebo inconclusive. Careful patient selection is essential for future research.

Disclosure: This study was funded by Merz Therapeutics GmbH. IB, IP, GK, TLG, and UM are employees of Merz Therapeutics NA has served on the advisory boards or speakers panels of Grünenthal, Merz therapeutics, Medtronic, Viatrix, Noema pharma and Pfizer. RB has received grant/research support from Pfizer, Genzyme, Grünenthal, German Federal Ministry of Education and Research (BMBF): German Research Network on Neuropathic Pain, NoPain system biology and German Research Foundation (DFG). He has received speaker

honorarium from Pfizer, Genzyme, Grünenthal, Mundipharma, Sanofi Pasteur, Medtronic, Eisai, Lilly, Boehringer Ingelheim, Astellas, Desitin, Teva Pharma, Bayer-Schering, MSD and served as consultant for Pfizer, Genzyme, Grünenthal, Mundipharma, Allergan, Sanofi Pasteur, Medtronic, Eisai, Lilly, Boehringer Ingelheim, Astellas, Novartis, Bristol-Myers Squibb, Biogenidec, AstraZeneca, Merck, Abbvie. NBF has been a consultant for Mitsubishi Tanabe Pharma, Merck, Almirall, and NeuroPN. EV declares no competing interests RB has received grant/research support from EU Projects: “Europain” (115007). DOLORisk (633491). IMI Paincare (777500). BMBF Projects: NoChro (13GW0338C), DFNS (01EM0903), NoPain, ERA-NET (IM-PAIN) Industry: Pfizer, Grünenthal, Mundipharma, Alnylam, Zambon, Sanofi Aventis, Viatris Trademarks: “fiberASSESS®” EU No. 018527050, “FabryScan®” EU No. 012094959, “AmyloScan®” EU No. 018188607, US No. 6168141. He has received Consultant /speaker honorarium from: Pfizer, Sanofi Aventis, Grünenthal, Eli Lilly, Desitin, Bayer, AbbVie, Janssen, Amicus, Novo Nordisk, Chiesi, Takeda, Stada, Ever, Unilab, Hexal, Viatris, AstraZeneca, Aristo, Esanum, Docflix, Vertanical, Denk, Teva, Glenmark, Novartis, Biotest, Desitin, Lateral, Alnylam, Confo, EVA, Merz, Neumentum, Algo, Nanobiotix, Stada, Heat2Move, Hoba, BioNTech, Esteve, SIMR, Desitin, SelectION, Max-Delbrück-C NBF has been a consultant for Merz, NeuroPN, Saniona, PharmNovo, Tris Pharma, AKIGAI, and Zyneyro. EV has served on the advisory boards or speakers panels of Grünenthal, Merz therapeutics, Ehyparm and Viatris; and received research grants from Grünenthal, Merz therapeutics and Vertex.

EPO-0717 | Neuropathic pain in multiple sclerosis: Discrepancies between DN4 screening and neurophysiological measures

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Background and aims: Pain is a common and disabling symptom in multiple sclerosis (MS), yet its pathophysiological mechanisms remain poorly understood. Clinical assessment of neuropathic pain relies largely on subjective screening tools such as the DN4 questionnaire. This study aimed to characterize pain phenotype in MS using the DN4 score and to determine its relationship with objective neurophysiological markers of nociceptive and non-nociceptive pathway function assessed by laser-evoked (LEPs) and somatosensory evoked potentials (SEPs).

Methods: Fifty MS patients reporting pain underwent standardized clinical evaluation, including DN4, pain intensity (VAS), fatigue (FSMC), anxiety and depression (HADS), and disability (EDSS). LEPs were recorded after laser stimulation of the hand and foot dorsum, and SEPs following median and tibial nerve stimulation. Neurophysiological parameters were compared between patients with DN4 <4 and ≥4. Logistic regression, non-parametric group comparisons, and correlation analyses were performed.

Results: Neuropathic pain phenotype (DN4 ≥4) was identified in 56% of patients. DN4 showed moderate correlation with pain intensity and fatigue, and weakly with anxiety, but showed no

significant association with LEPs or SEPs. Logistic regression confirmed that no neurophysiological measure independently predicted DN4 ≥4 status. LEP and SEP abnormalities were not significantly correlated, suggesting dissociated involvement of nociceptive and large-fiber pathways.

Conclusion: In MS, DN4-defined pain phenotype reflects subjective symptom burden rather than objective sensory pathway dysfunction. Its limited correlation with neurophysiological measures highlights pain's multidimensional nature in MS. LEPs and SEPs provide complementary mechanistic insights, supporting a multimodal, mechanism-oriented approach to pain assessment and management.

Disclosure: Natalia Drobinska, Eglė Sukockiene, Marjolaine Uginet, Patrice H. Lalive have nothing to disclose. Agustina M. Lascano received honoraria for advisory from ATC Therapeutics, Biogen, Johnson and Johnson, Roche, Argenx, Amgen and Alexion, and speakers' fees from Sanofi, CSL-Behring, UCB, Takeda non-related to the current study.

EPO-0718 | Sexually dimorphic neuroimmune pathways in chronic pain: A comprehensive systematic review of cellular and molecular mechanisms

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Background and aims: Chronic pain shows marked sex differences in prevalence, severity, and persistence, suggesting underlying biological mechanisms beyond psychosocial factors. Increasing evidence indicates that sexually dimorphic neuroimmune pathways play a central role in chronic pain pathophysiology.

Methods: This systematic review was conducted according to PRISMA 2020 guidelines and synthesized human clinical and translational studies examining sex-specific neuroimmune and glial mechanisms in adult chronic pain. Multiple biomedical databases were searched from inception to December 2025, and studies reporting sex-stratified neuroimmune outcomes were included.

Results: Across pain conditions, males predominantly exhibited microglia-driven mechanisms, particularly P2X4 receptor-mediated signaling and BDNF-dependent neuronal disinhibition. In contrast, females showed greater involvement of adaptive immune pathways, including CD4⁺ and CD8⁺ T cell signaling, chemokine cascades, and astrocyte-mediated neuroimmune interactions. Cytokine profiles, immune cell phenotypes, and neuroimaging markers further distinguished sex-specific pain mechanisms. Heterogeneity and limited sex-stratified power constrained quantitative synthesis.

Conclusion: Chronic pain comprises biologically distinct, sex-dependent neuroimmune mechanisms, supporting the need for sex-informed biomarkers and precision medicine approaches in pain management.

Disclosure: Nothing to disclose

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Background and aims: Pain is highly prevalent in older adults. However, it may be overlooked when cognitive deficits limit self-report. Our study aimed to determine whether demographic factors and cognitive status are associated with pain.

Methods: We recruited individuals who completed two pain questionnaires (Doloplus-2 and NOPPAIN) and a cognitive evaluation. Descriptive statistics were performed, and group comparisons between cognitively impaired and cognitively preserved individuals were conducted using t-tests. Pain presence was defined as any non-zero subjective pain rating (VAS ≥ 1 , range 0–6). Factors associated with pain presence were examined using multivariable logistic regression.

Results: A total of 153 patients were included; 48 (31%) had cognitive impairment. The prevalence of pain was similar in cognitively impaired and unimpaired patients (86.0% vs 83.5%). Patients with cognitive impairment were significantly older (76.8 SD 9.5 vs 61.5 SD 15.4 years, $p < 0.001$), with higher subjective pain intensity (VAS 2.54 SD 1.47 vs 1.94 SD 1.24, $p = 0.010$), higher NOPPAIN total scores (2.54 SD 1.47 vs 1.95 SD 1.23, $p = 0.011$) and substantially higher Doloplus-2 total scores (13.6 SD 6.5 vs 7.8 SD 5.1, $p < 0.001$) compared with cognitively preserved patients. In adjusted logistic regression analysis, higher age was independently associated with pain presence (OR 1.04 per year, 95% CI 1.00–1.07, $p = 0.026$), while cognitive impairment and sex were not.

Conclusion: Pain is common across cognitive groups and increases with age. Cognitive impairment doesn't independently influence pain presence and shouldn't reduce clinical awareness of pain detection.

Disclosure: Nothing to disclose.

EPO-0720 | Restoring circadian rhythms with a citrus flavonoid to improve sleep and reduce neuropathic pain

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Background and aims: Neuropathic pain (NP) disrupts sleep architecture and circadian regulation, impairing quality of life. Nobiletin, a polymethoxylated citrus flavonoid, amplifies molecular clock oscillations and may stabilise sleep–wake dynamics. We hypothesised that nobiletin alleviates NP by restoring circadian rhythmicity and improving sleep in mice after spared nerve injury (SNI).

Methods: Adult male and female PER2::LUC C57BL/6 mice underwent SNI. Nobiletin (10 mg/kg/day) or vehicle was continuously delivered via osmotic mini-pumps. Telemetric EEG/EMG, body temperature and locomotor activity were recorded at baseline and post-injury. Mechanical allodynia was assessed with von Frey filaments and dynamic brush testing. Ex vivo bioluminescence from DRG, spinal cord and prefrontal cortex explants was monitored to quantify Per2-driven

rhythmicity; circadian metrics were analysed using cosinor and CircaCompare. Procedures complied with institutional animal-care guidelines.

Results: SNI induced robust ipsilateral mechanical hypersensitivity. Continuous nobiletin significantly attenuated dynamic brush allodynia ($p < 0.01$) but did not alter von Frey thresholds. At day 7 post-SNI, REM sleep was reduced in both sexes, while NREM exhibited phase-dependent changes (increase during dark phase; reduction in males during light phase). In female DRG, Per2::Luc bioluminescence rhythms showed altered mesor, reduced amplitude and shifted acrophase versus sham (all $p < 0.001$). Ongoing analyses test whether nobiletin rescues REM sleep and tissue-clock amplitude.

Conclusion: Preliminary data support nobiletin as a chronotherapeutic candidate that selectively mitigates NP-related behavioural hypersensitivity and stabilises sleep and circadian oscillations. If confirmed, this non-opioid, clock-targeting approach could inform timed interventions to improve sleep and reduce NP.

Disclosure: Nothing to disclose.

EPO-0721 | Headache during IVF treatment: A comparative evaluation of GnRH agonist and antagonist protocols

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Background and aims: Controlled ovarian hyperstimulation during in vitro fertilization (IVF) involves marked hormonal changes. GnRH agonist and antagonist protocols differ in the timing and dynamics of hypothalamic–pituitary axis suppression, yet headache during IVF treatment remains poorly investigated. This study aimed to compare headache frequency between GnRH agonist and antagonist protocols and to assess the temporal association between headache occurrence and antagonist administration.

Methods: Infertile women undergoing IVF/ICSI were retrospectively evaluated by protocol, with recombinant FSH stimulation and recorded headache reports, including post-cetorelix events

Results: A total of 46 women undergoing IVF were included in the analysis (GnRH agonist: $n = 18$; GnRH antagonist: $n = 28$). Headache was reported more frequently in patients treated with the GnRH antagonist protocol compared with those receiving the agonist protocol (53.6% vs. 22.2%). In the GnRH antagonist group, headache symptoms were most commonly reported within the first 24 hours following cetorelix administration, with 9 of 15 patients (60.0%) experiencing headache during this period. The temporal distribution of headache episodes was evaluated descriptively. Headache frequency appeared to increase across rising estradiol categories, being reported in 25.0% of patients at 250–1000 pg/mL, 43.8% at 1001–2000 pg/mL, 64.3% at 2001–3000 pg/mL, and 87.5% at levels above 3000 pg/mL; this pattern may reflect a descriptive trend rather than a causal association.

TABLE 1 Clinical characteristics and headache features according to ovarian stimulation protocol.

Parameter	GnRH Agonist (n = 18)	GnRH Antagonist (n = 28)
Age (years), mean ± SD	31.4 ± 4.8	32.1 ± 5.2
Duration of infertility (years), median (range)	4 (1–9)	5 (1–11)
Primary infertility, n (%)	11 (61.1)	18 (64.3)
Secondary infertility, n (%)	7 (38.9)	10 (35.7)
Any headache during IVF, n (%)	4 (22.2)	15 (53.6)
Migraine-like headache, n (%)	3 (16.7)	8 (28.6)
Tension-type headache, n (%)	1 (5.6)	5 (17.9)
Non-specific headache, n (%)	0 (0)	2 (7.1)
Headache severity (VAS), median (IQR)	4 (3–5)	6 (5–7)
Headache onset within 24 h after Cetrotide®, n (%)	—	9/15 (60.0)
Nausea, n (%)	1 (5.6)	7 (25.0)
Photophobia, n (%)	0 (0)	1 (3.6)
Dizziness, n (%)	1 (5.6)	6 (21.4)
Paresthesia, n (%)	0 (0)	2 (7.1)
Persistent neurological deficit	None	None

TABLE 2 Temporal distribution of headache occurrence in the GnRH antagonist protocol.

Headache Type	Before Cetrotide®	Within 24 Hours After Cetrotide®	24–48 Hours After Cetrotide®
Migraine-like	1	5	5
Tension-type	2	3	3
Non-specific	1	1	1

Headache timing was evaluated descriptively in relation to cetrotide administration. More than one headache episode could be reported by the same patient.

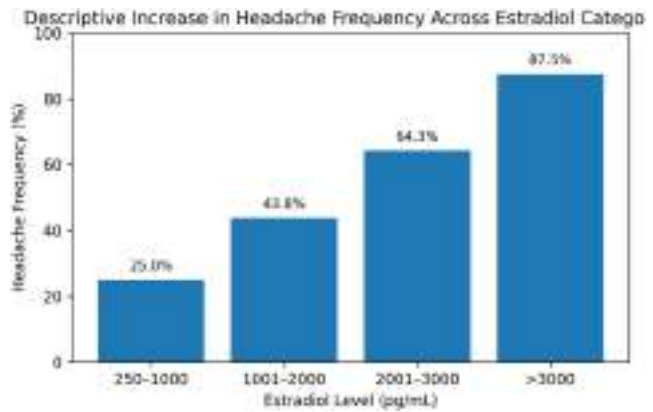


FIGURE 1 Descriptive distribution of headache frequency across estradiol level categories.

Conclusion: Headache is more frequently reported during IVF cycles using GnRH antagonist protocols compared with agonist protocols. The early onset of symptoms following antagonist administration may be related to acute hormonal changes, rather than the ovarian stimulation process itself.

Disclosure: The authors declare no financial or non-financial conflicts of interest related to this study.

Peripheral Nerve Disorders 1

EPO-0722 | Reliability of predicted outcomes by modified Erasmus Guillain Barre outcome score mEGOS versus followup outcomes in Guillain Barré syndrome patients

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Background and aims: Neuroprognostication in GBS relies on clinical assessments and neurophysiological evaluations. The mEGOS score, determined at hospital admission, predicts the likelihood of GBS patients achieving independent ambulation during follow-up.

Methods: This study aimed to evaluate the reliability of predicted outcomes based on the mEGOS score at hospital admission by comparing them to actual follow-up outcomes assessed using the Hughes Disability Scale at three months. A Hughes Disability Scale score of 0–2 was stratified as a good outcome and 3–6 as a poor outcome. A mEGOS score of 0–3 indicated a good outcome and 4–9 indicated a poor outcome.

Results: Among the 113 patients analysed from records of a GBS cohort admitted to our tertiary hospital, 64% were male, and 72% presented within one week of symptom onset. Nineteen per cent required mechanical ventilation. Nerve conduction studies at admission were demyelinating in 21%, axonal in 14%, equivocal in 44%, and normal in 13%. Among the 88 patients with all parameters, the mEGOS score at admission had a sensitivity of 45.5% and a specificity of 50.6% for predicting a poor outcome at three months. The positive predictive value was 11.6% while the negative predictive value was 86.7%.

Conclusion: The mEGOS score, therefore, is primarily an early triage and reassurance tool rather than a robust predictor of disability. Its high negative predictive value emphasises its clinical utility in identifying patients who are unlikely to experience poor outcomes, but since it tends to overestimate the risk of poor outcome it has a low positive predictive value.

Disclosure: Nothing to disclose.

EPO-0723 | Safety profile of tanruprubart (ANX005) in patients with Guillain-Barré syndrome: A randomised, placebo-controlled phase 3 study

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Background and aims: Guillain-Barré syndrome (GBS) is a rare, life-altering, neuromuscular emergency, where classical complement activation contributes to progressive muscle weakness. Tanruprubart (ANX005), a humanised monoclonal IgG4 antibody, selectively inhibits complement component C1q. Infections with encapsulated bacteria and systemic autoimmunity were events of interest.

TABLE 1 Summary of TEAEs.

TEAE, n (%)	Tanruprubart 30 mg/kg (N=79)	Tanruprubart 75 mg/kg (N=81)	Placebo (N=81)
TEAEs	79 (100)	80 (98.8)	79 (97.5)
Related to study drug	24 (30.4)	34 (42.0)	5 (6.2)
Grade ≥3 TEAEs*	32 (40.5)	36 (44.4)	35 (43.3)
SAEs	15 (19.0)	9 (11.1)	15 (18.5)
Related to study drug	1 (1.3)	5 (6.2)	1 (1.3)
TEAEs of special interest	30 (38.0)	36 (44.4)	15 (18.5)
IRRs	24 (30.4)	32 (39.5)	4 (4.9)
Severe (Grade 3)†	3 (3.8)	4 (4.9)	0
Life-threatening (Grade 4)†	1 (1.3)	5 (6.2)	1 (1.3)
Fatal (Grade 5)†	0	0	0
Rash	18 (22.8)	12 (14.9)	2 (2.5)
Infection with encapsulated bacteria	13 (16.5)	10 (12.3)	12 (14.8)

*Severity based on National Cancer Institute Common Terminology Criteria for Adverse Events Version 5.0

†After unblinding of data, it was found that 1 patient in the placebo group had apparently received tanruprubart and had treatment-related SAEs of Grade 3 myocardial infarction and Grade 4 IRR.

GBS, Guillain-Barré Syndrome; IRR, infusion-related reaction; SAE, serious adverse event; TEAE, treatment-emergent adverse event.

Methods: In GBS-02 (Phase 3), participants ≥16years with a recent GBS diagnosis without planned plasma exchange or intravenous immunoglobulins were randomised to receive a single intravenous dose of tanruprubart 30 or 75 mg/kg or placebo. Antihistamine and acetaminophen were administered pre-infusion to mitigate for infusion related reactions (IRRs). Vaccinations and prophylactic antibiotics were not mandated. Safety over 6 months included treatment-emergent adverse events (TEAEs), serious adverse events (SAEs), investigator-assessed treatment-related TEAEs, and events of special interest (IRRs; encapsulated-bacteria infections; systemic immune disorders other than GBS).

Results: The safety set comprised 241 participants; 95% did not receive prophylactic antibiotics. 98.8% reported ≥1 TEAE (Table 1). Treatment-related TEAEs were mostly IRRs (rash), which were generally mild/moderate and transient without infusion interruption. One participant per group discontinued due to an IRR; none withdrew due to TEAEs. Encapsulated-bacteria infection rates were similar across groups. TEAEs and SAEs were consistent with GBS morbidity. No systemic autoimmune diseases other than GBS occurred. Three non-treatment related deaths occurred in each group.

Conclusion: Single-dose tanruprubart was generally well-tolerated. Common TEAEs were associated with disease course. Tanruprubart did not increase risk of infections with encapsulated bacteria, despite most participants not receiving prophylactic antibiotics. Findings support the promising potential of tanruprubart in GBS.

Disclosure: The study was sponsored by Annexon Biosciences (Brisbane, CA, USA). EM, H-AK, GM, EP, AJ: Employment with and shareholder of Annexon Biosciences.

EPO-0724 | Tanruprubart, anti-C1q therapy, accelerates recovery and reduces ventilation in severe Guillain-Barré Syndrome: Results from the phase 3 GBS-02 trial

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Background and aims: Guillain-Barré syndrome (GBS) is a severe, autoimmune, life-altering disorder mediated by classical

complement affecting peripheral nerves, resulting in muscle weakness and often prolonged recovery. The safety and efficacy of tanruprubart, a C1q inhibitor designed to treat patients with GBS, was evaluated in the randomised placebo-controlled Phase 3 trial GBS-02 (NCT04701164). Here, we report on prespecified measures of clinical benefit associated with patient recovery time and resource utilisation.

Methods: Patients with GBS aged ≥16years and a baseline GBS disability score (GBS-DS) of 3, 4, or 5 were randomised 1:1:1 to a single intravenous (IV) infusion of tanruprubart (30 or 75 mg/kg) or placebo. Neither IV immunoglobulin or plasma exchange was given. Time to walking independently (GBS disability score ≤2) was analysed using time-to-event methods. Duration of mechanical ventilation and intensive care unit (ICU) stay were assessed using the zero-inflated negative binomial (ZINB) model (imputed for death).

Results: A total of 241 patients were randomised. Patients who received tanruprubart 30 mg/kg ($n=79$) could walk independently a median 31 days earlier ($p=0.0211$) compared with placebo ($n=81$). The duration of mechanical ventilation was a median 28 days less ($p=0.0356$) compared with placebo. The median ICU stay for the tanruprubart group was 7 days less than placebo ($p=0.5306$).

Conclusion: In this Phase 3 study of severe GBS, tanruprubart improved recovery-related outcomes, including earlier independent walking and shorter ventilation duration, with the possibility of shorter time in the ICU, consistent with clinically meaningful benefit.

Disclosure: The study was sponsored by Annexon Biosciences (Brisbane, CA, USA). HAK, GM, EP: Employment with and shareholder of Annexon Biosciences QDM: Consultancy/advisory role with Annexon Biosciences KCG: Consultancy/advisory role with Annexon Biosciences, argenx, Janssen, and Sanofi.

EPO-0725 | Targeted immunotherapy with Tanruprubart (ANX005) reduces ventilation requirements in Guillain-Barré syndrome: A phase 3 subgroup analysis

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Background and aims: Guillain-Barré syndrome (GBS) is a rare, life-altering neuromuscular emergency requiring hospitalisation, and mechanical ventilation in severe cases. Tanruprubart (ANX005) is a targeted, humanised monoclonal IgG4 antibody that rapidly and selectively inhibits classical complement component C1q. In GBS-02, a Phase 3, randomised, double-blind, placebo-controlled trial of tanruprubart 30 and 75 mg/kg (NCT04701164), a single infusion of tanruprubart 30 mg/kg led to early and sustained clinical benefit, and was well tolerated. This analysis evaluated the effect of tanruprubart on the duration of mechanical ventilation in GBS-02.

TABLE 1 Duration of ventilation by baseline disease characteristics.

	Tanruprubart 30mg/kg (N=79)		Placebo (N=82)	
	Participants ventilated, n/N	Median duration (IQI)	Participants ventilated, n/N	Median duration (IQI)
Overall	13/79	27.0 (15, 42)	14/82	48.9 (30, 220)
Baseline MRCss				
0–20	13/38	27.0 (15, 47)	9/38	125.0 (46, 181)
21–40	0/17	180.0 (35, 181)	6/33	51.4 (30, 100)
41–60	1/4	13.0 (–)	0/7	–
Baseline GBS-DI				
1	0/12	–	0/30	–
2	9/36	28.0 (15, 47)	9/36	142.0 (31, 181)
3	13/31	22.0 (14, 114)	10/33	45.5 (26, 220)
Baseline NfL (pg/mL)				
<352.64	0/31	40.5 (34, 114)	0/39	137.0 (34, 181)
≥352.64	0/47	25.0 (18, 18)	1/40	48.0 (30, 242)

IQI, Interquartile range; MRCss, Medical Research Council sum score; N, total number of participants with the given characteristic; n, participants with the given characteristic who received mechanical ventilation; NfL, neurofilament light

Methods: Individuals aged ≥16 years with a recent GBS diagnosis and a GBS-disability score (GBS-DS) of 3 (can walk 10 metres with help) to 5 (requiring ventilation) were enrolled. Ventilation duration was analysed using a zero-inflated negative binomial model; participants who died were imputed to 26 weeks (trial end). Ventilation duration was assessed overall and by baseline disease characteristics assessing muscle weakness (Medical Research Council sumscore [MRCss], 0=tetraplegic, 60=normal), a stratification factor (GBS-DS) and axonal damage (neurofilament-light [NfL] levels).

Results: Mechanical ventilation requirements were similar across treatment groups (Table 1). Among ventilated patients, approximately 75% were already ventilated at randomisation. Duration of ventilation was median 27 days shorter with tanruprubart 30 mg/kg versus placebo (Table 1). This difference was particularly notable in participants with greater muscle weakness (lowest MRCss tertile 0–20) and worse axonal damage (median NfL ≥352.64) at baseline compared with their healthier counterparts (Table 1).

Conclusion: Tanruprubart reduced the duration of mechanical ventilation in individuals with GBS, especially among those with more severe disease.

Disclosure: The study was sponsored by Annexon Biosciences (Brisbane, CA, USA). H-AK, GM, JD, RG: Employment with and shareholder of Annexon Biosciences QDM: Consultancy/advisory role with Annexon Biosciences JN: Consultancy role with Annexon Biosciences KAKA: No relevant disclosures ZI: Research funding from Fogarty International Center, National Institute of Neurological Disorders and Stroke of the National Institutes of Health, USA, and Annexon Biosciences KCG: Consultancy/advisory role with Annexon Biosciences, argenx, Janssen, and Sanofi.

EPO-0726 | Comparison of anti-MAG antibody-associated neuropathy and distal or predominantly sensory CIDP: An analysis of the Portuguese CIDP registry

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Background and aims: Distal or predominantly sensory chronic inflammatory demyelinating polyneuropathy (CIDP) and anti-myelin-associated glycoprotein (anti-MAG) neuropathy share overlapping clinical features but differ in prognosis and treatment response. Early differentiation is important, particularly when anti-MAG antibody testing is unavailable.

Methods: Patients from the Portuguese CIDP registry were retrospectively analyzed. Diagnoses were reviewed according to the 2021 EAN/PNS criteria. Anti-MAG neuropathy was defined by antibody titers >7000 BTU. Demographic data, disease duration, initial symptoms and signs, cerebrospinal fluid (CSF) findings, nerve conduction studies, and response to therapy were evaluated.

Results: Sixty patients were included: 48 with distal/sensory CIDP and 12 with anti-MAG neuropathy. Time to diagnosis was significantly longer in the anti-MAG group ($p=0.013$), while age at onset was similar. Albuminocytologic dissociation and higher CSF protein levels were more frequent in CIDP ($p<0.001$ and $p=0.014$, respectively). Muscle atrophy at presentation was more common in CIDP ($p=0.016$). Neurophysiological studies showed abnormalities predominantly restricted to the lower limbs in anti-MAG neuropathy ($p<0.001$), whereas sural sparing was exclusive to CIDP ($p=0.003$). CIDP patients also exhibited higher peroneal and tibial CMAP amplitudes and shorter distal peroneal latency. Improvement with intravenous immunoglobulin (IVIg) was observed in 45.8% of CIDP patients compared with 8.3% of anti-MAG patients ($p=0.044$). In multivariate analysis, sural sparing and response to IVIg remained independent predictors of CIDP.

Conclusion: Differentiating distal/sensory CIDP from anti-MAG neuropathy remains challenging. However, sural sparing, albuminocytologic dissociation, and particularly response to IVIg support a diagnosis of CIDP and may guide earlier therapeutic decisions.

Disclosure: Nothing to disclose.

EPO-0727 | Sensory recovery following digital nerve repair using conduits versus autologous nerve grafts: A systematic review and meta-analysis

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Background and aims: Digital nerve injuries can impair sensory function and hand performance. Autologous nerve grafting (ANG) remains the standard for repairing nerve gaps but is associated with donor site morbidity. Nerve conduits—such as collagen-based, chitosan-based, and muscle-in-vein types—are emerging alternatives that avoid these complications. This systematic review and meta-analysis compared the effectiveness and safety of nerve conduits versus ANG for sensory recovery after digital nerve repair.

Methods: A systematic search of PubMed, Embase, Scopus, and Web of Science was conducted through July 2025 following PRISMA guidelines. Included studies were randomized controlled trials and observational studies comparing conduits with ANG. Primary outcomes were static and moving two-point discrimination (S2PD and M2PD); complication rates were secondary. Data were pooled using a random-effects model with

mean differences (MD) and risk ratios (RR) and 95% confidence intervals (CI).

Results: Five studies with 214 patients were included. Sensory recovery was similar between conduits and ANG for S2PD (MD = -0.21; 95% CI: -2.25 to 1.83; $p=0.84$) and M2PD (MD = -0.67; 95% CI: -2.00 to 0.66; $p=0.84$). Chitosan-based conduits showed significantly better S2PD outcomes than ANG (MD = -2.57; 95% CI: -3.40 to -1.74; $p<0.0001$). Complication rates were comparable overall (RR = 2.36; $p=0.08$), but RCTs showed fewer complications with conduits (RR = 3.32; $p=0.03$).

Conclusion: Nerve conduits provide similar sensory outcomes to ANG and may reduce complications, especially with chitosan-based designs. They represent a promising alternative for digital nerve repair.

Disclosure: Nothing to disclose.

EPO-0728 | Tanruprubarb in GBS: Targeting early complement-associated inflammation conserved across the clinical spectrum and regions

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⁶University Hospital Würzburg, Würzburg, Germany

Background and aims: Guillain-Barré syndrome (GBS) is an acute, immune-mediated polyradiculoneuropathy resulting in mild weakness to severe paralysis. Converging evidence supports an early pathogenic window where classical complement activation and innate immune effector mechanisms drive neuroinflammation and early nerve injury.

Methods: We conducted serum proteome-wide longitudinal profiling of acute inflammatory effectors using the Olink® Explore HT platform in GBS patients from a Ph3, randomised, placebo-controlled study of anti-C1q therapy tanruprubarb in Asia ($n=139$), an IVIg/PE-treated US cohort ($n=14$) and healthy controls ($n=10$). Prespecified prognostic and clinical strata were defined.

Results: Across the tanruprubarb Ph3 and IVIg/PE US cohorts, 352 proteins were elevated at baseline, including 81 proteins mapping to the top 3 Reactome inflammatory pathways, with ~2–4-fold higher levels versus healthy controls. In placebo-treated subjects, inflammatory signature rose/remained high, reaching a consistent peak at ~Day 8, then declining below baseline by Day 29, indicating a brief early inflammatory phase. In contrast, tanruprubarb produced rapid, broad suppression of these pathway-linked proteins by Day 8, confirmed in the top 12 marker panel spanning cytokine signaling, interferon signaling, innate immune pathways, and neutrophil associated markers. The IVIg/PE US cohort exhibited no early reduction in these markers.

Conclusion: Across geographically and clinically diverse cohorts, GBS demonstrated a conserved, complement-linked early inflammatory signature peaking at Day 8, defining a narrow window where short-duration upstream complement inhibition is most impactful. Tanruprubarb rapidly and durably suppressed this inflammatory cascade across strata and regions, highlighting C1q inhibition as a mechanism-targeted strategy for GBS

and supporting extrapolation of these biomarker effects to US patients.

Disclosure: The study was sponsored by Annexon Biosciences (Brisbane, CA, USA). PP, HAK, EC, TY, RA: Employees and shareholders of Annexon Biosciences ZI: Research funding from Fogarty International Center, National Institute of Neurological Disorders and Stroke of the National Institutes of Health, USA, and Annexon Biosciences DL: External contractor of Annexon Biosciences to perform data analysis YR: Nothing to disclose KS: Research funding from NINDS/NIH and DoD. Consultant Annexon Biosciences CS: Nothing to disclose.

EPO-0729 | Role of magnetic resonance neurography in CIDP: Experience from a single center

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Background and aims: According to the European Academy of Neurology/Peripheral Nerve Society guidelines, magnetic resonance (MR) imaging is a supportive diagnostic criterion for chronic inflammatory demyelinating polyneuropathy (CIDP). The aim of this study was to evaluate MR neurography findings in patients with suspected or confirmed CIDP and to compare them with patients with alternative diagnoses.

Methods: A longitudinal retrospective analysis included all patients who underwent MR neurography at Hospital Garcia de Orta between July 1, 2024, and June 31, 2025, excluding examinations for traumatic indications. Patients were stratified into two groups: those with suspected or confirmed CIDP and those with other diagnoses.

Results: Thirteen patients were included: eight with suspected or diagnosed CIDP and seven controls (83.3% vs. 57.1% male; median ages 64.5 and 56.7 years, respectively). Two patients with newly diagnosed CIDP/possible CIDP showed MR neurography abnormalities: a 65-year-old woman with suspected classic CIDP demonstrated a diffuse thickening of the bilateral cervical plexus; and a 79-year-old man with suspected right lower limb focal CIDP showed nerve thickening and post-gadolinium enhancement of the right lumbosacral plexus. No MR neurography



FIGURE 1 MR neurography.

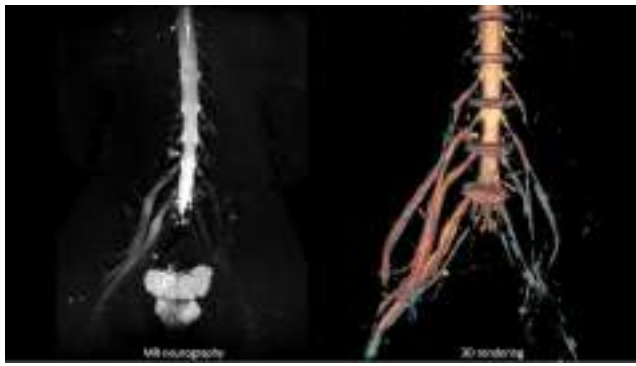


FIGURE 2 MR neurography.

abnormalities were observed in clinically stable CIDP patients receiving immunoglobulin therapy ($n = 2$), patients with possible CIDP without electrophysiological criteria ($n = 2$), or in controls. **Conclusion:** MR neurography revealed structural abnormalities exclusively in patients with a newly CIDP diagnosis, supporting its utility as a diagnostic tool. The absence of abnormalities in clinically stable, treated CIDP patients, suggests that MR neurography may also reflect treatment response and disease activity, underlying its potential value as a prognostic biomarker.

Disclosure: Nothing to disclose.

EPO-0730 | Early ultrasonographic swelling ratio of the facial nerve as a prognostic threshold for functional recovery in acute facial palsy

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Background and aims: Early prognostic evaluation in acute facial palsy is difficult because early clinical grading is subjective and electroneurography (ENoG) is unreliable during the first 1–2 weeks after onset. An objective marker applicable in the early stage is needed. We investigated whether early ultrasonographic measurement of facial nerve swelling predicts outcomes at 3 months.

Methods: A retrospective review was conducted in 22 patients with early peripheral facial palsy within 14 days of onset. Facial nerve ultrasonography was performed using a standardized protocol, with nerve diameter measured below the earlobe. The swelling ratio was defined as the affected-to-unaffected side diameter ratio. Outcomes at 3 months were assessed using the House–Brackmann (HB) grade and Sunnybrook Facial Grading System (SFGS). Poor outcome was defined as HB grade >1 or SFGS <90 . Prognostic performance was evaluated using receiver operating characteristic (ROC) analysis.

Results: ROC analysis showed the facial nerve swelling ratio predicted 3-month outcomes. A cut-off value of 1.27 discriminated incomplete recovery (HB grade >1 ; AUC 0.750, sensitivity 75.0%, specificity 100.0%). Functional impairment (SFGS <90) was predicted with an AUC of 0.886, sensitivity 85.7%, and specificity 100.0%. Patients with ratios ≤ 1.27 showed near-complete recovery (mean SFGS 93.2), whereas those with ratios >1.27 had poorer outcomes (mean SFGS 70.2; $p = 0.0182$). In contrast, ENoG thresholds ($\geq 85\%$ and $\geq 90\%$) showed low discriminative ability (AUC 0.21–0.33).

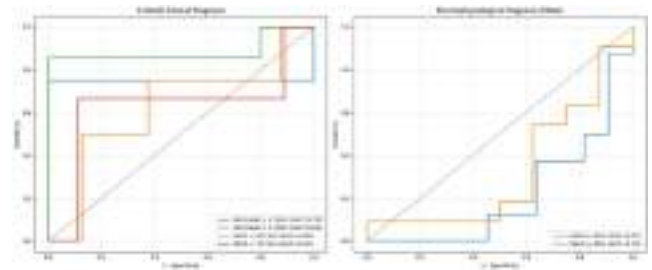


FIGURE 1 Receiver operating characteristic curves comparing early ultrasonographic facial nerve swelling ratio and electroneurography (ENoG) for prediction of 3-month outcomes in acute facial palsy.

Conclusion: An early facial nerve diameter ratio greater than 1.27 was associated with incomplete recovery and functional impairment at 3 months. Ultrasonography may provide a objective tool for early prognosis in facial palsy.

Disclosure: This research was supported by a grant of the Korea Health Technology R&D Project through the Korea Health Industry Development Institute (KHIDI), funded by the Ministry of Health & Welfare, Republic of Korea (RS-2022-KH127692).

EPO-0731 | Defining a standardized facial movement protocol for AI-based video analysis in facial nerve palsy: A Delphi study

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Background and aims: Reliable facial nerve palsy assessment is hindered by rater dependency and the limited precision of gross grading scales like House–Brackmann. Although our static image-based artificial intelligence (AI) model achieved high accuracy (Quadratic Weighted Kappa: \$0.7594\$), static analysis cannot evaluate essential dynamic features such as movement trajectories and velocity. This study aimed to define a standardized dynamic assessment framework via expert consensus, serving as a reference foundation for next-generation video-based AI applications.

Methods: A Delphi study was conducted with 10 specialists (> 3 years of clinical experience). Twenty-eight video-based assessment items—encompassing basic movements, contracture, synkinesis, and functional metrics—were derived from clinical practice and literature. Items were rated on a 9-point Likert scale. Consensus was predefined as a Median (Mdn) ≥ 7.0 , Interquartile Range (IQR) ≤ 2.0 , Content Validity Ratio (CVR) ≥ 0.62 , and an agreement rate $\geq 70\%$.

Results: In the first round, consensus was achieved for 20 of 28 proposed items by meeting all predefined statistical thresholds. Core facial movements (eye closure, smiling, and lip puckering) and primary metrics (symmetry and range of motion) demonstrated the highest consensus (Mdn: 9.0, IQR: 0.0, CVR ≥ 0.80). In contrast, items involving natural speech tasks and speech-rate variability showed lower agreement and were identified for further refinement in subsequent validation steps.

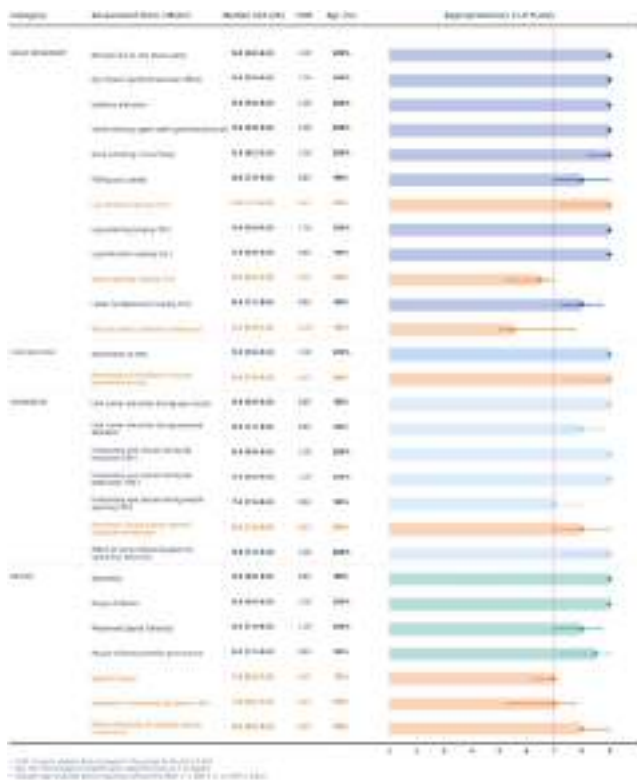


FIGURE 1 Main results of the first Delphi round for video-based facial nerve palsy assessment. Median scores and interquartile ranges are shown. Items in orange did not meet consensus criteria and require further refinement.

Conclusion: This study identifies expert-validated components for video-based assessment, addressing the functional gap between static image evidence and dynamic clinical requirements. The ongoing second Delphi round will finalize this framework, providing a critical foundation for high-precision, objective digital monitoring of facial nerve palsy in clinical and research settings.

Disclosure: This research was supported by a grant of the Korea Health Technology R&D Project through the Korea Health Industry Development Institute (KHIDI), funded by the Ministry of Health & Welfare, Republic of Korea (RS-2025-02220534). The authors declare no other conflicts of interest.

Headache 5

EPO-0732 | Epidemiology and clinical characteristics of nummular headache in observational studies: A systematic review and meta-analysis

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Background and aims: Nummular headache (NH) is a rare primary headache disorder with unknown epidemiology and prevalence of the most common clinical characteristics - this study aimed to establish all of the crucial aspects of NH.

Methods: Major databases were thoroughly searched for observational studies reporting the relevant data regarding NH.

Results: Out of initial 2441 records, 17 studies met all of the inclusion criteria. The pooled mean age of onset of NH was 46.91 (95% CI: 43.85–49.96). The PPE of NH in adult patients evaluated for a headache in a clinic-based setting was 0.7% (95% CI: 0.2–2.4), with slight female predominance (females=0.5%, 95% CI: 0.2–1.4 vs males=0.3%, 95% CI: 0.1–0.8). The majority of patients (69.4%, 95% CI: 58.1–78.8) experience chronic course of NH. The shape of the headache was round/circular in 78.4% (95% CI: 71.9–83.7) and oval/elliptical in 21.6% (95% CI: 16.3–28.1) of patients. In 7.5% (95% CI: 2.7–19.0) of individuals pain



FIGURE 1 Round/elliptical.

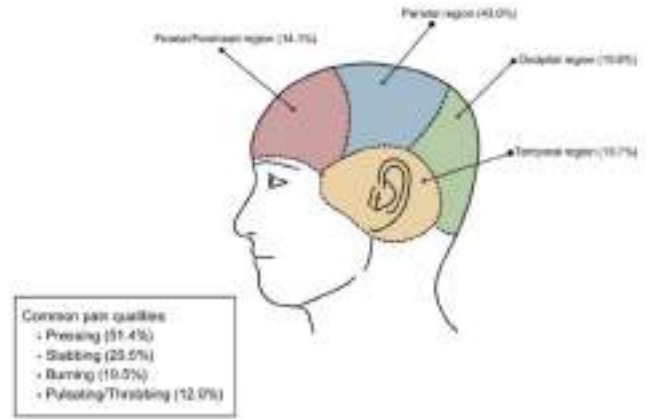


FIGURE 2 Pain qualities and location.

had multifocal location and 59.1% (95% CI: 49.7–68.0) of NH patients experienced pain exacerbations. The pain was most prevalent in the strictly parietal region (43.0%, 95% CI: 37.4–48.7) of the cranium and had pressing quality (51.4%, 95% CI: 41.6–61.1). **Conclusion:** The results of our study showed that NH is a very distinct and relatively rare to encounter headache disorder. Due to its unique clinical phenotype, physicians need to be aware when a patient presents with a small, well-localized round/oval headache in the cranium region. **Disclosure:** Nothing to disclose.

EPO-0733 | **Headache as the most common manifestation of giant cell arteritis? A systematic review with meta-analysis**

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Background and aims: This study aimed to assess the relative frequency of clinical features of giant cell arteritis (GCA) and to investigate the predictors of temporal artery biopsy (TAB) outcomes. **Methods:** A literature search of major electronic databases was conducted for observational studies that reported original data on clinical features of patients with GCA. A random-effects meta-analysis was performed to determine the pooled prevalence estimates. The study's design adhered closely to the MOOSE standards. The JBI appraisal tool was used to evaluate the risk of bias. The study's protocol was pre-registered on PROSPERO (ID: CRD42024584763). **Results:** Out of initial 12,628 records, 62 articles (9971 patients) met all of the eligibility criteria. Mean patients' age upon diagnosis was 74.33 years (95% CI: 74.12–74.54 years). The most prevalent clinical feature of GCA was new-onset headache (75.7%; 95% CI: 72.2–79.0; 95% PI: 0.47–0.92). Other common symptoms

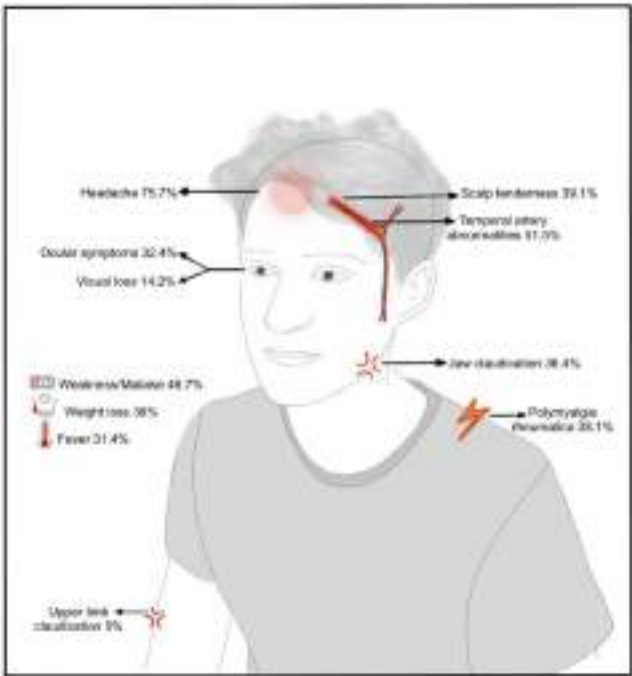


FIGURE 1 Presentation of headache as the most common manifestation of GCA with comparison to other symptoms.

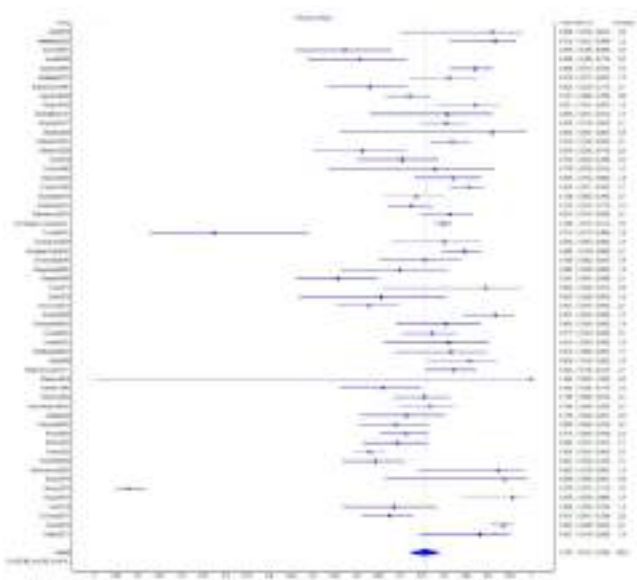


FIGURE 2 Forest plot presenting pooled prevalence estimate of headache in GCA.

of GCA were temporal artery abnormalities (51.5%; 95% CI: 45.2–57.7; 95% PI: 0.25–0.77), weakness/malaise (46.7%; 95% CI: 35.4–58.4; 95% PI: 0.09–0.88), and scalp tenderness (39.1; 95% CI: 35.3–43.1; 95% PI: 0.22–0.59). Positive TAB results were present in 73.8% of patients (95% CI: 68.1–78.8%; 95% PI: 0.35–0.94). The presence of headache (LogOR= −1.11; 95% CI: −1.92 to −0.29) or PMR (−0.71; 95% CI: −1.09 to −0.32) significantly decreases the chance of receiving positive TAB results. **Conclusion:** Since there is a greater likelihood of obtaining negative biopsy results, the TAB may not be required when a patient exhibits a headache along with other clinical symptoms that enable them to be diagnosed with GCA. **Disclosure:** Nothing to disclose.

EPO-0734 | **Glial neurovascular unit protein alterations and risk of idiopathic intracranial hypertension: A systematic review and meta-analysis**

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Background and aims: Idiopathic intracranial hypertension (IIH) is characterized by raised intracranial pressure with incompletely understood pathophysiology. Increasing evidence implicates dysfunction of the glial neurovascular unit, including astrocytic, vascular, and neuroaxonal components, in disease development. This systematic review and meta-analysis evaluated the association between glial neurovascular unit-related protein alterations and the risk of IIH. **Methods:** A systematic search of PubMed, EMBASE, Cochrane Library, Scopus, and Web of Science was conducted up to August 2025. Studies comparing IIH patients with non-IIH controls and reporting levels of aquaporin-4, glial fibrillary acidic protein, fibrinogen, or neurofilament light chain were included. Data extraction and quality assessment using the Newcastle–Ottawa Scale were performed independently. Random-effects models

were used to calculate pooled standardized mean differences (SMDs) with 95% confidence intervals (CIs). BMI was analyzed as a secondary outcome.

Results: Ten studies comprising 327 IIH cases and 216 controls were identified, of which seven were included in quantitative synthesis. Cerebrospinal fluid NfL levels were significantly higher in IIH patients compared with controls (SMD 0.78, 95% CI 0.51–1.05), indicating neuroaxonal injury. Plasma fibrinogen levels were also elevated in IIH (SMD 0.66, 95% CI 0.08–1.23), suggesting a hypercoagulable and inflammatory state. Due to methodological heterogeneity, data on AQP4 and GFAP could not be pooled. Higher BMI was significantly associated with increased IIH risk (SMD 0.80, 95% CI 0.17–1.44).

Conclusion: Elevated NfL and fibrinogen levels are associated with IIH, supporting roles for neuroaxonal damage and

vascular–inflammatory mechanisms in disease pathophysiology. Well-designed multicentre studies are needed to validate glial neurovascular unit biomarkers and improve risk stratification.

Disclosure: Nothing to disclose.

EPO-0735 | People with migraine and two years treatment with anti-CGRP therapies. Second report from the prospective national registry “Hercules”

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Background and aims: To record the long-term efficacy and safety of anti-CGRP treatments in people with migraine in Hellas.

Methods: HERCULES is a 2-year, patient-centered, national, multicenter, longitudinal (5 total assessments every 6 months), prospective, observational study enrolling individuals with migraine treated with anti-CGRP agents. The primary endpoints are discontinuation of treatment due to adverse events and reduction in monthly migraine days (MDM).

Results: 425 subjects with migraine and prior failure to at least 3 repurposed anti-migraine prophylactic treatments attending 8 headache centers in Greece have been recorded so far [erenumab 41 (9.6%), galcanezumab 57 (13.4%), fremnezumab 287 (67.4%), eptinezumab 39 (9.2%) and rimegepant 2 (0.5%)], of whom 180 (42.3%) completed the 2-year treatment with anti-CGRP agents [29/180 (16.1%) subjects with erenumab; 22/180 (12.2%) with galcanezumab; and 129/180 (71.7%) with fremanezumab]. 57/425 (13.4%) participants discontinued treatment [fremanezumab 28/287 (9.7%); galcanezumab 17/57 (29.8%); erenumab 10/41 (24.4%); eptinezumab 2/39 (5.1%)], due to patient's decision [29/57 (50.9%)], failure [14/57 (24.6%)], adverse events [2/57 (3.5%)], complete remission of migraine [6/57 (10.5%)], or pregnancy [1/57 (1.7%)]. The most common AE recorded were constipation [47/425 (11%)] and injection site reactions [57/332 (13.4%)]. Only one severe AE has been recorded [1/425 (0.2%)] during infusion of eptinezumab (allergic reaction). After 24 doses a $\geq 50\%$, $\geq 75\%$, or 100% reduction in MDM compared to pretreatment was recorded in 147/180 (81.6%), 81/425 (19%), and 13/180 (7.2%) subjects, respectively.

Conclusion: Anti-CGRP mAbs are effective, safe, and well tolerated after long-term administration and should be considered as a first-line prophylactic treatment for migraine.

Disclosure: Hercules is supported by grants from Pharmaserve Lilly SACI, Pfizer Hellas AE and Teva Hellas SA. D.D.M. has received fees for participation in advisory committees and

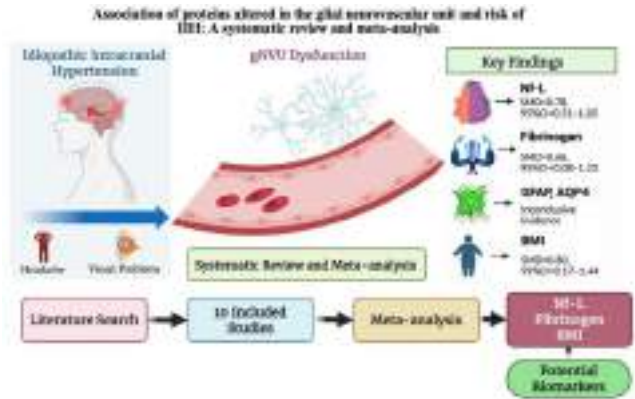


FIGURE 1 This systematic review and meta-analysis demonstrate that elevated cerebrospinal fluid neurofilament light chain and plasma fibrinogen levels are significantly associated with idiopathic intracranial hypertension, indicating underlying neuroaxonal injury.

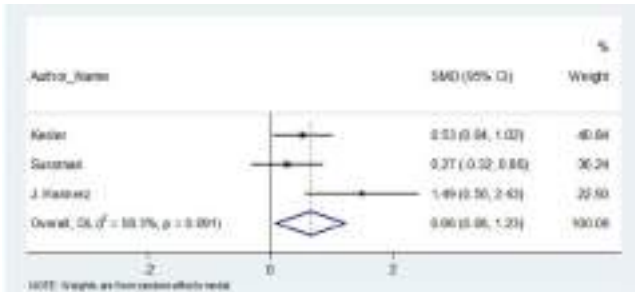


FIGURE 2 Forest plot showing the association between Fibrinogen and the risk of IIH.

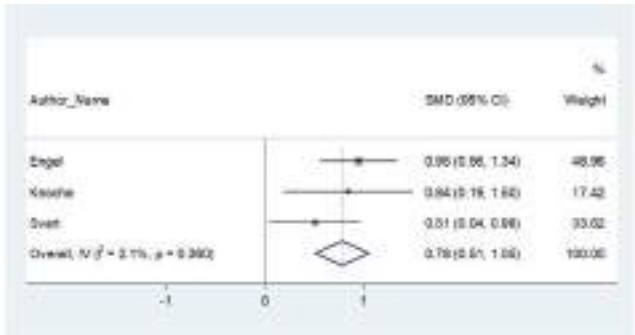


FIGURE 3 Forest plot showing the association between Neurofilament – Light and risk of IIH.

honoraria for lectures from AbbVie, Amgen, AstraZeneca, Bennett, Bristol-Meyers Squibb, Eli Lilly, Cross, ELPEN, Haleon, Intermed, Genesis Pharma, Lundbeck, Merck, Mylan, Orion Pharma, Novartis, Pfizer, Roche, and Teva Pharmaceuticals; has participated in clinical trials for Amgen, Eli Lilly, Lundbeck, Novartis, Pfizer, Swixx, and Teva Pharmaceuticals as principal investigator; is the president of the Hellenic Headache Society and a member of the Management Group of the Coordinating Panel for the Functional Neurological Disorders at the European Academy of Neurology. C.D. has received personal fees/ honoraria/ travel expenses from Pfizer, Merck, Astra-Zeneca, Teva, Orion Pharma, Haleon, Bennett. C.D. serves as member at large of European Headache Federation. E.G. has received travel grants, speaking fees and participated in advisory boards for AbbVie, Eli Lilly, Lundbeck, Novartis, Pfizer, and Teva/Specifar. E.K. has received travel grants and honoraria from Bristol-Meyers Squibb, Eli Lilly and Company, Innovis, Merck, Novartis, Lundbeck, Roche and Teva. I.S. has received travel grants from Lundbeck and Teva. T.S.C. received honoraria for participation in advisory boards, preparation and addressing of lectures in satellite symposia, as well as funding for congress participation from Abbvie, Teva, Sanofi, Cross, Elpen, Pharmathen-Innovis, Pharmaserv-Lilly, Novartis, Braintherapeutics Neuraxpharm, Merck, C.A. Papaellinas, Bayer, Swixx, Vianex, UCB and Genesis Pharma-Biogen. T.S. received honoraria from UCB. E. C. received travel funds and honoraria for participation in Advisory Meetings and Satellite Symposia from Merck, Sanofi Genzyme, Genesis Pharma, Teva Pharmaceuticals, UCB, ITF, Medison Pharma, Alexion, AstraZeneca, CSL Behring, and Takeda. Z. T. has received compensation for lecture honoraria and travel expenses, participation in scientific meetings, clinical trial membership, or clinical advisory board participation from Teva, Argenx, Sanofi, Novartis, Janssen, Immunovant, Roche, Lundbeck, Bayer, Shionogi, Merck, Genesis Pharma, Bristol-Myers Squibb, and UCB. E.D. has received compensation for lecture honoraria and travel expenses, participation in scientific meetings and advisory boards, clinical trial membership, from Abbvie, Astra-Zeneca, Bayer, Bristol-Myers Squibb, Elpen, Lundbeck, Merck, Mylan, Novartis, Sanofi, UCB, Teva, Tikun and Genesis. C.T. received honoraria for lecturing, travel grants for travel and participation in advisory meetings from Cross, Lundbeck and Teva. S.S.K. has received grants and honoraria from Abbvie, AstraZeneca, Bristol-Myers Squibb, Sanofi Aventis, Innovis, Ipsen, ITF Hellas, Genesis Pharma, ERKIM Pharma, Medison Pharma, Medtronic Hellas, Merck, Novartis, Pharmaserv, Rosche, Sandoz, Teva, Viatrix, and UCB.

EPO-0736 | Do European Headache Federation criteria reflect response to acute treatments in resistant and refractory migraine? The REFINE study

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Background and aims: The European Headache Federation (EHF) defines resistant and refractory migraine by failure of ≥ 3 (resistant) or all (refractory) pharmacological preventive treatment classes, without considering acute treatments. This study aimed to evaluate whether these definitions also correlate with response to acute treatment.

Methods: We conducted a multicenter, prospective, international study (REFINE) to test the EHF definitions in a real-life setting in 15 European headache centers. The Headache Under Response to Treatment (HURT) Questionnaire assessed the impact of migraine on daily activities and effectiveness of acute medications in patients with resistant, refractory, and non-resistant non-refractory (NRNR) migraine.

Results: We included 689 patients, of whom 261 (37.9%) had resistant, 73 (10.6%) refractory, and 355 (51.5%) NRNR migraine. Patients with refractory migraine experienced more significant impairment in daily activities (HURT-2) and social activities (HURT-3) versus those with resistant and NRNR migraine (46 [63.0%] vs 110 [42.2%] vs 51 [14.4%], $p < 0.001$; 37 [50.7%] vs 67 [25.7%] vs 25 [7.0%], $p < 0.001$). Regarding acute medication efficacy (HURT-5), more patients with refractory migraine stated that one dose “never” relieved their headache, and delayed or avoided taking acute medication due to concerns about adverse events (HURT-7), versus those with resistant and NRNR migraine (29 [39.7%] vs 28 [10.7%] vs 25 [7.0%], $p < 0.001$; 16 [21.9%] vs 7 [2.7%] vs 11 [3.1%], $p < 0.001$).

Conclusion: Patients with refractory and resistant migraine report poorer acute treatment response compared with those with NRNR migraine, which significantly affect daily lives. Management of difficult-to-treat migraine should focus on optimizing acute treatments, alongside preventive therapies.

Disclosure: Nothing to disclose.

EPO-0737 | Longitudinal effects of CGRP monoclonal antibodies for migraine prophylaxis: Impact of treatment type and age group

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Background and aims: Calcitonin gene-related peptide (CGRP) monoclonal antibodies (mAbs) are established preventive treatments for episodic and chronic migraine, but comparative longitudinal data across agents and age groups remain limited. This study aimed to evaluate long-term clinical responses to different CGRP mAbs, assess whether age or treatment type influences response trajectories, and characterize multidimensional patterns of treatment response.

Methods: We conducted a retrospective, single-centre observational study including 443 adults with high-frequency episodic or chronic migraine treated with erenumab, galcanezumab, fremanezumab, or eptinezumab between 2018 and 2025. Patients were stratified by age (< 50 vs ≥ 50 years). Longitudinal mixed-effects models assessed changes in monthly migraine days, acute medication use, pain intensity, disability, quality of life, and mood over 12 months. Generalized linear mixed models evaluated predictors of $\geq 50\%$ response. Principal component

analysis (PCA) was used to identify multidomain response patterns.

Results: All CGRP mAbs produced rapid and sustained improvements across migraine frequency, disability, and patient-reported outcomes over 12 months. Age did not significantly influence baseline severity, longitudinal trajectories, or responder status. Treatment differences were modest overall; however, at 12 months, fremanezumab and eptinezumab were associated with higher odds of response compared with erenumab, while galcanezumab showed consistently higher response odds

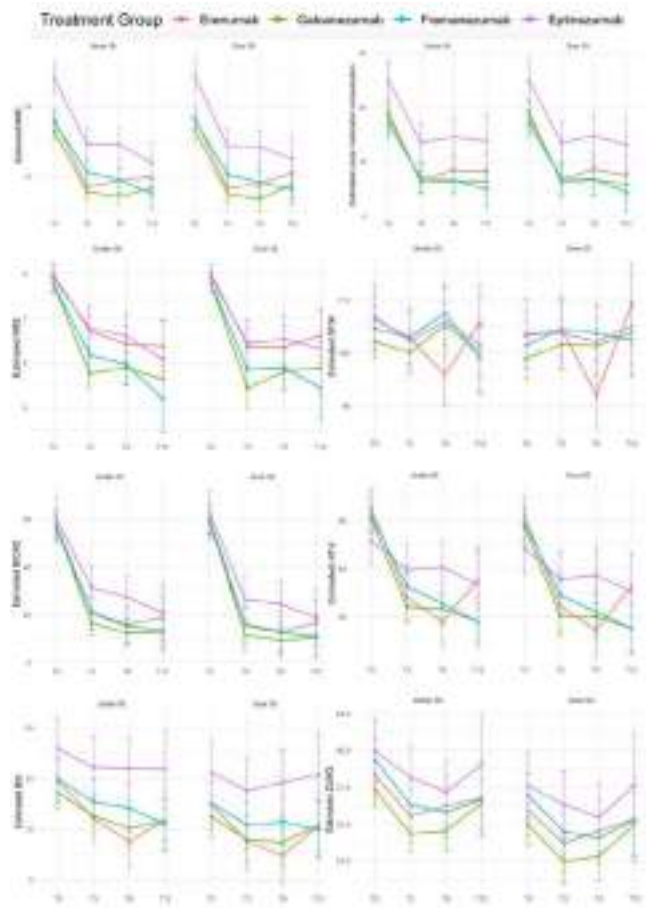


FIGURE 1 Clinical and migraine-related variables change over time across age and treatment groups.

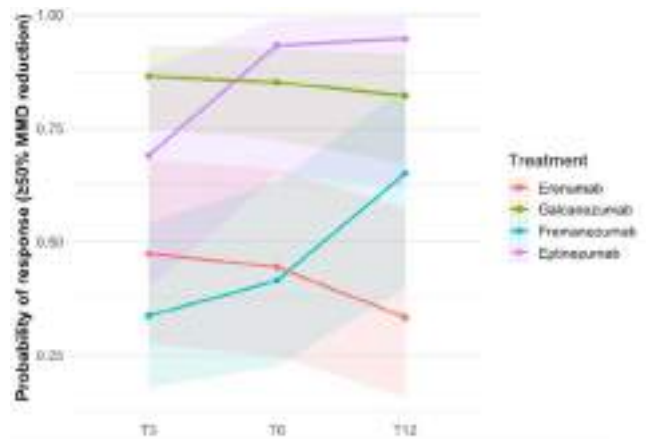


FIGURE 2 probability of response over time across treatment groups (MMD=monthly migraine days).

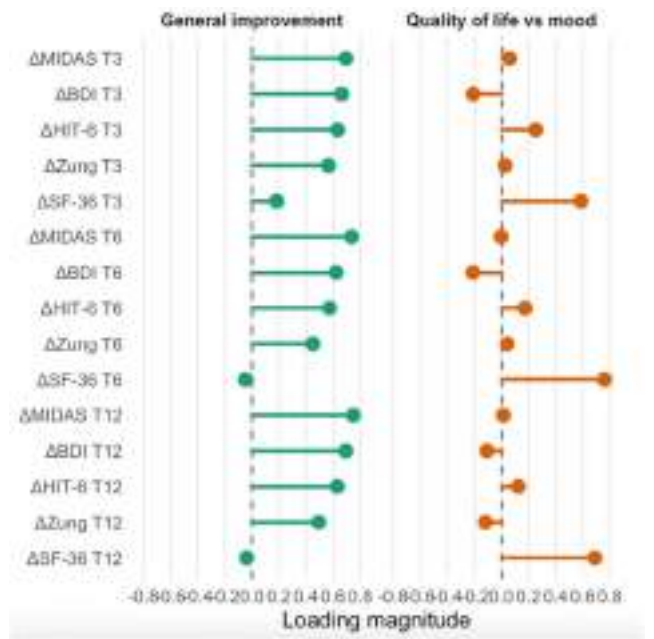


FIGURE 3 Component loadings from PCA.

independent of time. PCA identified a “General improvement” component capturing multidomain benefit, which was more pronounced in patients treated with eptinezumab.

Conclusion: In real-world clinical practice, CGRP mAbs demonstrate broadly comparable and durable effectiveness across age groups. Subtle long-term differences between agents may emerge, particularly for multidimensional clinical improvement, supporting individualized treatment selection.

Disclosure: IM has received support for conference participation from Allergan/AbbVie, Pfizer, and Lundbeck; has received funding for an advanced training course from Teva; and is currently enrolled in a DPhil program funded by GlaxoSmithKline (Oxford–GSK). GC and MDP has received support for conference participation from Allergan/AbbVie, Pfizer, Teva and Lundbeck. APa has served on the scientific advisory board of GE Healthcare, Eli-Lilly, and Actelion Ltd. Pharmaceuticals, and has received speaker honoraria from Nutricia, PIAM, Langstone Technology, GE Healthcare, Lilly, Lundbeck, UCB Pharma, and Chiesi Pharmaceuticals. RR has received speaker honoraria from Eli-Lilly, Novartis, Lundbeck, and Teva. All other authors declare no conflicts of interest.

EPO-0738 | Work productivity and indirect cost savings of atogepant versus topiramate: Results from the TEMPLE trial

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Background and aims: Using the Work Productivity and Activity Impairment (WPAI): Migraine, we contrasted presenteeism, absenteeism, overall work productivity, and indirect cost savings of participants who received atogepant versus topiramate for the preventive treatment of migraine.

Methods: TEMPLE was a phase 3b, randomized, multicenter, double-blind (DB), double-dummy, active-controlled trial to evaluate tolerability, safety, and efficacy of atogepant compared with topiramate for the preventive treatment of migraine. WPAI:Migraine was evaluated at each 4-week time point during the 24-week DB period. An excel-based cost calculator used WPAI data to calculate the per-patient-per-year (PPPY) work productivity cost by multiplying the percent overall work productivity loss, weeks per year, standard weekly full-time work hours (40), and the average hourly wage in Canada, Portugal, United Kingdom, and United States. Productivity benefit from using atogepant was calculated as the difference between atogepant and topiramate productivity loss PPPY.

Results: Based on WPAI data from TEMPLE, atogepant demonstrated greater improvements in presenteeism and overall work productivity (Figures 1 and 2), and lower absenteeism and activity impairment versus topiramate. The cost calculator using overall work productivity data demonstrated atogepant provided greater productivity benefits and indirect cost savings compared with topiramate across all 4 countries (Table 1).

Conclusion: Atogepant consistently demonstrated greater improvement compared to topiramate across all four WPAI domains. Using TEMPLE WPAI data, atogepant demonstrated work productivity benefit compared to topiramate in Canada, Portugal, UK, and United States.

Disclosure: Disclosures Javier Díaz de Terán declares public research funding from the Instituto de Salud Carlos III (Ministry of Health, Spain), project PI24/01254, outside the submitted work; and reports honoraria for consultant and/or advisory boards from AbbVie, Almirall, Lilly, Lundbeck, Novartis, Organon, Pfizer, Boston Scientific, and Teva. Jan Versijpt received personal fees and nonfinancial support from Teva, Pfizer and Medtronic, personal fees from Novartis and Lundbeck, and grants and non-financial support from Allergan/AbbVie. Raquel Gil-Gouveia reports honoraria for consultant and/or advisory boards from Allergan/AbbVie, Astra Zeneca, Almirall, Bial, Biogen, Bristol-Myers Squibb, CMBE, FLOAT, Lilly, Lundbeck, Merck, Novartis, Novo Nordisk, Organon, Pfizer, Roche, Sanofi, Tecnifar and Teva; research funding from Fundação Ciência e Tecnologia (Project 29675, MigN2Treat, 02/SAICT/2017); Learning-Health, Luz Saúde (LiON, Luz Innovation on Neurosciences); Novartis-Sociedade Portuguesa de Cefaleias; Bolsa-Proyecto Centro de Investigación Interdisciplinar em Saúde and UCP. Dawn Buse has received research funding from the US Food and Drug Administration, the National Headache Foundation and Amgen

TABLE 1 Work productivity benefit for Atogepant vs. Topiramate per patient per year.

	Canada	Portugal	United Kingdom	United States
Productivity Cost Benefit vs. Topiramate (PPPY)	CAD\$ 6,362	€ 2,195	£ 4,052	\$ 8,434
Productivity Cost Benefit including Treatment Costs vs. Topiramate (PPPY)	CAD\$ 419	€ 1,513	£ 1,728	\$ 2,271

Productivity cost benefit is the difference between atogepant and topiramate. Incremental values are calculated relative to topiramate. Productivity cost benefit is calculated as percent improvement in overall productivity from WPAI. Treatment costs represent the annual drug cost difference of atogepant compared with topiramate. CAD\$, Canadian dollars; PPPY, per-patient-per-year; WPAI, Work Productivity and Activity Impairment.

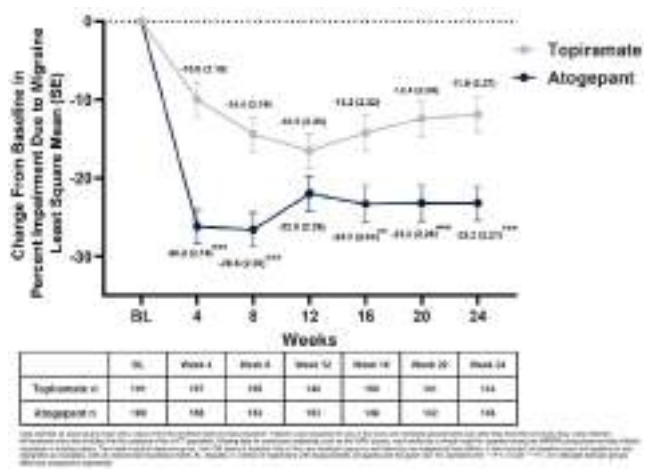


FIGURE 1 Change from baseline in overall work productivity loss.

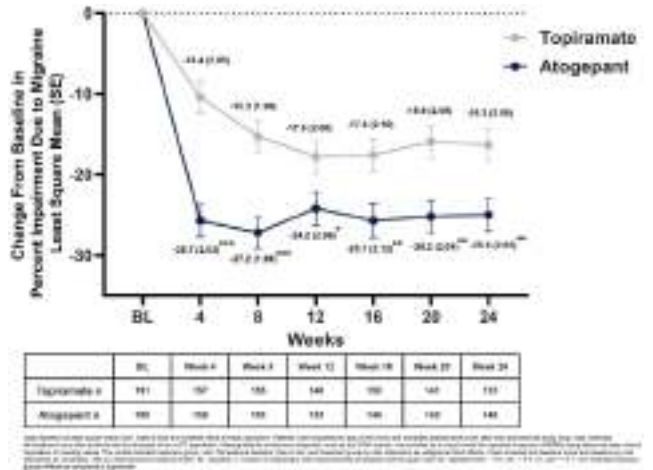


FIGURE 2 Change from baseline in presenteeism.

and received consulting fees from Amgen, Allergan/AbbVie, Eli Lilly, Lundbeck and Teva. Richard B. Lipton has received research support from the National Institutes of Health, the FDA, and the National Headache Foundation. He serves as consultant for, advisory board member of, or has received honoraria or research support from AbbVie/Allergan, Aeon, Amgen, Axsome, Biohaven, Dr. Reddy's Laboratories, electroCore, Eli Lilly, GlaxoSmithKline, Linpharma, Lundbeck, Pfizer, Merck, Novartis, Teva, and Vector. He receives royalties from Wolff's Headache, 8th edition (Oxford University Press, 2009), and Informa. He holds stock/options in Biohaven, CoolTech, and NuVieBio. PG, HG, FH, KN, and KC are employees of AbbVie and may hold AbbVie stock.

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Background and aims: Reversible cerebral vasoconstriction syndrome (RCVS) is a cerebrovascular disorder characterized by acute headaches and reversible cerebral arterial narrowing. While case series suggest an association between serotonergic antidepressant use and RCVS onset, clinical and long-term outcomes data in this population remain scarce. We aimed to characterize the presentation and prognosis of antidepressant-associated RCVS.

Methods: We conducted a retrospective analysis of adult patients with RCVS and concurrent antidepressant use from the Lariboisière Hospital RCVS cohort (2004–2023). Clinical and neuroimaging features were compared with those of non-exposed patients. Long-term outcomes were assessed in the exposed group through structured telephone interviews.

Results: Among 501 RCVS patients, 56 (11.2%) were using antidepressant at RCVS onset. Exposed patients were more often female (82.1% vs. 60.7%, $p=0.002$) and older (median age: 48 vs. 41 years, $p=0.001$), with acute emotional stress more frequently reported at onset (41.1% vs. 20.2%, $p=0.001$). Focal deficits (25.0% vs. 13.0%; adjusted OR=2.62, 95% CI [1.26–5.46]) and hemorrhagic complications (32.1% vs. 16.4%; OR=2.28, 95% CI [1.15–4.52]) were more frequent in antidepressant users. Antidepressants were reintroduced in 53.6% of cases. Among the 39 patients with long-term follow-up, RCVS recurrence was

TABLE 1 Multivariate analysis of RCVS severity outcomes associated with antidepressant use. Adjusted odds ratios (OR) with 95% confidence intervals are reported for neurological deficits, hospitalization, and hemorrhagic complications.

Variable, n(OR ^a , 95% CI)	Neurological deficit	p-value	Hospitalization	p-value	Hemorrhagic complication	p-value
Antidepressant use	2.62 (1.26–5.46)	0.008	1.77 (0.95–3.28)	0.070	2.28 (1.15–4.52)	0.019
Female sex	2.50 (1.17–5.33)	0.018	2.26 (1.01–5.05)	< 0.001	5.99 (2.48–14.39)	< 0.001
Age (per year)	1.01 (0.98–1.03)	0.614	1.03 (1.01–1.05)	0.004	1.03 (1.00–1.06)	0.037
Migraine	1.30 (0.61–2.98)	0.745	0.99 (0.64–1.53)	0.953	1.40 (0.83–2.37)	0.209
Hypertension	1.36 (0.86–2.08)	0.163	0.99 (0.51–1.90)	0.981	1.05 (0.43–2.57)	0.915
Smoking	1.96 (1.08–3.61)	0.021	1.10 (0.71–1.72)	0.664	0.82 (0.58–1.17)	0.794
Emotional stress	0.36 (0.28–1.12)	0.162	0.78 (0.49–1.22)	0.275	0.87 (0.38–1.23)	0.185
Vasomotor medication	0.65 (0.33–1.28)	0.218	1.02 (0.60–1.71)	0.954	1.05 (0.55–1.92)	0.902
Physical stress	3.47 (1.85–6.58)	0.001	2.40 (1.45–3.96)	0.001	2.37 (1.31–4.28)	0.004
Illicit drug use	0.97 (0.42–2.23)	0.951	2.08 (1.19–3.62)	0.010	1.38 (0.59–3.24)	0.468

TABLE 2 Psychiatric outcomes at long-term follow-up in 39 antidepressant-associated RCVS. Values are no./total no. (%) or mean (25th–75th percentiles). Percentages are based on the subgroup with complete follow-up ($n=39$).

Psychiatric symptom assessed by the Modified Mini Screen	n (%)
Depressive features	22 (56.4)
Current anhedonia	16 (41.0)
Current depressed mood	10 (25.6)
Persistent depressive mood	13 (33.3)
Passive death wishes	6 (15.4)
Mood and hypomanic features	3 (7.7)
History of elevated mood or increased goal-directed activity	3 (7.7)
History of irritable mood	2 (5.1)
Anxiety and obsessive-compulsive spectrum symptoms	34 (87.2)
Excessive anxiety lasting at least 6 months	31 (79.5)
Excessive and persistent daily anxiety for 6 months	19 (48.7)
History of panic attacks	17 (43.6)
Current agoraphobia	13 (33.3)
Current social anxiety	5 (12.8)
Current compulsive behaviour	3 (7.7)
Trauma- and stressor-related symptoms	22 (56.4)
History of exposure to a traumatic event	22 (56.4)
Current intrusive symptoms (e.g., flashbacks, nightmares)	6 (15.4)
Psychotic symptoms	5 (12.8)
Preservatory delusions	5 (12.8)
Thought broadcasting or thought insertion	1 (2.6)
Other psychotic symptoms*	0 (0.0)
Psychiatric symptoms assessed by the Hospital Anxiety and Depression Scale	n (%)
Anxiety Scale, mean (± SD)	9.7 (± 3.4)
≥ 8: suspected anxiety	28 (71.8)
≥ 11: definite anxiety	14 (35.9)
Depression Scale, mean (± SD)	8.3 (± 3.4)
≥ 8: suspected depression	11 (28.2)
≥ 11: definite depression	4 (10.3)

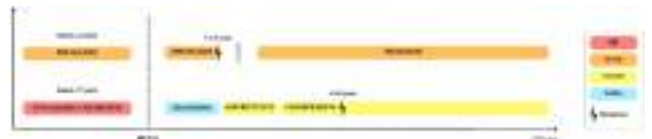


FIGURE 1 Two cases of long-term recurrence in antidepressant-associated RCVS.

rare (5.1%). Persistent psychiatric symptoms, particularly stress-related disorders, anxiety and depressive features, remained highly prevalent.

Conclusion: Antidepressant-associated RCVS is associated with increased neurological severity and hemorrhagic risk. Recent serotonergic exposure and emotional stress may act synergistically. Antidepressant delayed reintroduction appears feasible with individualized risk assessment and close neurological and psychiatric monitoring.

Disclosure: Nothing to disclose.

EPO-0740 | Epidemiology of primary exercise headache: A systematic review and meta-analysis with comparison between clinic- and population-based settings

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Background and aims: Primary exercise headache (PEH) is a rare primary headache disorder that occurs specifically during or right after physical exertion and lasts up to 48 hours. This study aimed to evaluate the epidemiology of PEH in clinic- and population-based settings.

Methods: Major databases were thoroughly searched for articles that presented original data on the epidemiology of PEH. Random-effects meta-analysis was performed in order to calculate the pooled prevalence estimates (PPE). The study's structure adhered closely to the MOOSE and PRISMA requirements. The protocol of the study was pre-registered on PROSPERO (CRD42024612283).

Results: Out of the original 2083 records, 12 articles satisfied all inclusion requirements. The majority of the studies showed a low risk of bias. The headache is a rare finding in clinical-based settings (PPE=0.46%, 95% CI: 0.18–1.20, 95% PI: 0.01%–14.29%, I²=94.86), more common in population-based surveys (PPE=4.92%, 95% CI: 1.84–12.49, I²=98.73), shows a female predominance, and, moreover, typically has onset in the late twenties (27.47 years).

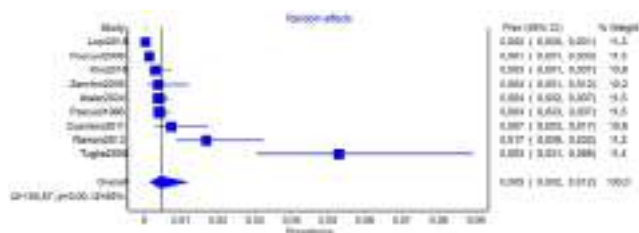


FIGURE 1 Forest plot presenting the prevalence of PEH in general adult population.

Conclusion: Our study showed that PEH is a rare primary headache disorder among adults evaluated for a headache in a clinic-based setting and is significantly more common in the general population. Furthermore, PEH is diagnosed within similar frequency among both sexes in headache-oriented clinics and moreover, predominantly late twenties. However, in the general population, there is a notable female preponderance.

Disclosure: Nothing to disclose.

EPO-0741 | Assessing physician and patient alignment in migraine severity: Evidence from real-world clinical practice

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Background and aims: Migraine is characterised by unilateral headache pain. The burden of migraine is well established,

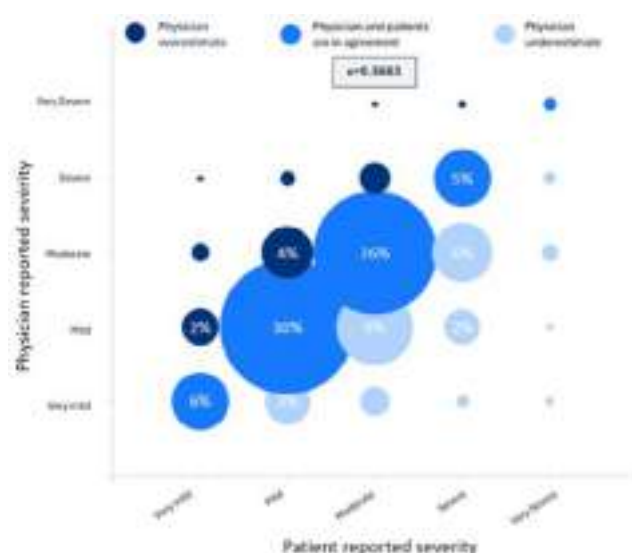


FIGURE 1 Physician and patient-reported migraine severity over the past 3 months (relative to time of data collection). Data are total percentages.

yet potentially equivocal across different stakeholders, such as patients with migraine (PwM) and their physicians. This study aimed to assess the alignment between physician and patient reported migraine severity in real world clinical practice.

Methods: Data were drawn from the 2022/23 Adelphi Migraine Disease Specific Programme™, a cross-sectional survey of physicians and their patients in France, Germany, Italy, Spain, the United Kingdom, China, Japan, and the United States. Physicians and their PwM reported average migraine severity over the past three months. The alignment between physician and patient reported severity was assessed by calculating Cohen's weighted kappa (κ) statistic. Interpretation of the κ value was based on Landis and Koch (1977).

Results: Overall, 938 physicians (391 PCPs, 527 neurologists, and 20 neurosurgeons) reported data for 7357 PwM, of which patient-reported data were available for 3125. Matched severity data were available for 3078 PwM. Physicians reported 55% of patients ($n=1688$) experienced very mild/mild migraine, 37% ($n=1137$) experienced moderate migraine, and 8% ($n=253$) experienced severe/very severe migraine. Conversely, 47% ($n=1443$) of PwM reported experiencing very mild/mild migraine, 38% ($n=1171$) reported moderate migraine, and 15% ($n=464$) reported severe/very severe migraine. A kappa value of 0.57 was observed, suggesting only moderate alignment between physicians and their patients (Figure 1).

Conclusion: This study indicates physicians may under-recognise the severity of migraine, highlighting the importance of open dialogue to support a shared understanding of migraine severity and guide optimal management decisions.

Disclosure: William Whitton, Lucy Hargreaves, Sarah Cotton, and Sophie Barlow are all employees of Adelphi Real World.

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Background and aims: Migraine is a leading cause of disability, yet substantial gaps in diagnosis, documentation, and treatment continue to impact care quality. Applying natural language processing (NLP) to real-world electronic medical record (EMR) data provides an opportunity to reveal these systemic challenges and pinpoint critical patient care gaps.

Methods: We performed a descriptive retrospective analysis of Accuro EMR data from one general practice (GP) (2012–2023) and one neurology clinic (2022–2023) in Ontario using the DARWEN NLP engine. Extracted variables included demographics, monthly migraine days (MMDs), migraine classification, comorbidities, acute and preventive therapies, and medication overuse headache (MOH).

Results: The cohort comprised 967 GP and 149 neurology patients, predominantly female, with mean ages 41–42 years. Psychiatric and sleep comorbidities were common in both groups - anxiety (28.9%; 27.5%), depression (11.7%; 4.0%), and insomnia (40.1%; 26.8%), respectively—underscoring migraine complexity. Migraine classification was rarely documented in GP, with 72.7% of patients having an unknown classification compared with 22.1% in neurology; MMDs were likewise underreported. Treatment patterns diverged, GP relying heavily on NSAIDs and opioids, while neurology commonly used triptans and a broader, though still limited, use of preventive therapies. MOH was rarely documented in GP (0.6%) but frequently identified in neurology (42.3%).

Conclusion: Findings highlight system level barriers—poor documentation, underdiagnosis, and inconsistent access to migraine-specific therapy—which fragment migraine care. Enhancing EMR systems, integrating AI-enabled decision support, and improving equitable access to evidence-based treatments are essential to advancing migraine care in Canada.

Disclosure: This study was sponsored by Pfizer.

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Background and aims: Cluster headache (CH) is a primary headache disorder historically considered more prevalent in men. As a chronobiological condition, it has been hypothesised CH may be influenced by seasonality or sunlight-related factors. The French open healthcare databases offer an opportunity to analyse real-world dispensing patterns of CH treatments. The aim of this study was to provide an innovative interpretation of the clinical expression of this condition through healthcare data, and to highlight the evolving economic burden of its treatments.

Methods: We extracted consumption data for the two validated attacks treatments for CH: 6 mg subcutaneous sumatriptan (SS) and oxygen therapy (OT). Treatments patterns were analysed by sex, age group, and French administrative region, and compared with climatic, demographic and economic variables.

Results: Between 2014 and 2024, the total amount for OT in CH tripled, while SS costs increased by only 1.6-fold. A consistent decrease in the male-to-female sex ratio was observed across all age group for both treatments. A distinct peak in SS dispensing was observed in July. However, no statistically significant associations were found between SS dispensing and sunshine or rainfall. Younger men exhibited significantly higher consumption than older men, and overall consumption was higher in men than in women.

Conclusion: Our findings highlight evolving treatment patterns and shifting sex ratio in the delivery of CH therapies in France. While no clear environmental drivers of treatment used were identified, the study underscores the value of open health data in monitoring prescribing trends and evaluation the economic impact of CH therapies.

Disclosure: Nothing to disclose.

Movement Disorders 8

EPO-0744 | Movement disorders in multisystem proteinopathies: A systematic review

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Background and aims: Multisystem proteinopathies (MSP) represent a group of genetic disorders classically manifesting



FIGURE 1 Abstract infographic.

[illegible]

Methods: The search was conducted in the Medline and Scopus databases by the following terms: “Multisystem proteinopathy”; “MSP”; “valosin containing protein”; “VCP”; “MSP1”; “heterogeneous nuclear ribonucleoprotein A2B1”; “hnRNPA2B1”; “MSP2”; “heterogeneous nuclear ribonucleoprotein A1”; “hnRNPA1”; “MSP3”; “Sequestosome 1”; “SQSTM1”; “MSP4”; “Matrin 3”; “MATR3”; “MSP5”; “T-cell restricted intracellular antigen 1”; “TIA1”; “optineurin”; “OPTN”; “annexin A11”; “ANXA11”. All kind of studies reporting single cases or case series of MSP-MD published up to 02/01/2026 were included.

Conclusion: The spectrum of MSP-MD was summarized suggesting possible clinical scenarios for a proper MSP syndromic diagnosis.

EPO-0745 | Incidence of calvarial bone infarction after focused ultrasound thalamotomy for refractory tremor: Associated factors

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Disclosure: Nothing to disclose.

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Methods: A prospective cohort was conducted with adult CD patients attending treatment at Antwerp University Hospital, Belgium (UZA) and a specialized private physiotherapy practice. Depression and Anxiety were measured using the Beck Depression inventory (BDI-II) and the Beck Anxiety Inventory (BAI-II), respectively. Sleep quality was measured using Pittsburgh Sleep Quality Index (PSQI); and QoL was measured using EuroQoL-5 dimensions (EQ-5D) at baseline and 6-weeks

post treatment. Paired T-test and Wilcoxon signed rank was used for comparison.

Results: A total of 29 CD patients were included. The mean age was 61.6 ± 9.25 years. The mean $\pm$ standard deviation (SD) score of the global PSQI score decreased from 10.1 ± 4.6 at baseline to 7.41 ± 4.7 post treatment. The mean (SD) BAI-II score decreased slightly from 13.5 ± 9.43 at baseline to 12.52 ± 10.2 post treatment. The median (IQR) of BDI-II score changed from 7 (5–19) to 8 (2.5–20). There was significant improvement of PSQI following BoNT treatment ($MD = 2.65$, $p < 0.001$).

Conclusion: BoNT injection significantly improved sleep quality. However, no statistical difference was observed in depressive and anxiety symptoms. The persistence of psychiatric symptoms underscores the necessity of comprehensive bio-psychosocial interventions. Considering this, future studies targeting development of intervention modality is needed.

Disclosure: Nothing to disclose.

EPO-0747 | The immediate effect of peripheral stimulation on cervical dystonia disease outcomes: Single-case experimental design

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Background and aims: Limited Cervical Range of Motion (CROM) and pain are among the most common symptoms of Cervical Dystonia (CD) which are treated with Botulinum NeuroToxin injections. However, adjuvant interventions may offer additional benefit. Therefore, this study evaluated the immediate effects of peripheral stimulation comprising conventional transcutaneous electrical nerve stimulation (TENS), muscle stimulation (NMES), and muscle vibration on CROM and pain in patients with CD.

Methods: A Single-Case Experiment Design (SCED), alternating treatment design was conducted with 19 CD patients, recruited at the Antwerp University Hospital (UZA). Participants randomly received conventional TENS, NMES and muscle vibration on three different days. Main outcome measures were CROM, measured using a 3D-stereophotogrammetric scanning system (3dMD Inc, GA, USA) and pain, quantified by Visual Analogue Scale (VAS).

Results: Mean age of participants was 59.87 ± 8.9 years. Baseline CROM for rotation ranged from 87° to 137.6° and from 79° to 132.4° for flexion/extension. Improvements in neck rotation were observed in 5 (33.3%) participants after conventional TENS, 8 (42.1%) after NMES, and 8 (42.1%) after muscle vibration. Similarly, improvement in flexion and extension was observed in 10 (55.6%) after conventional TENS, 8 (53.3%) after NMES, and 7 (46.7%) after muscle vibration. Significant reductions in pain shortly after the three interventions were observed in patients experiencing pain.

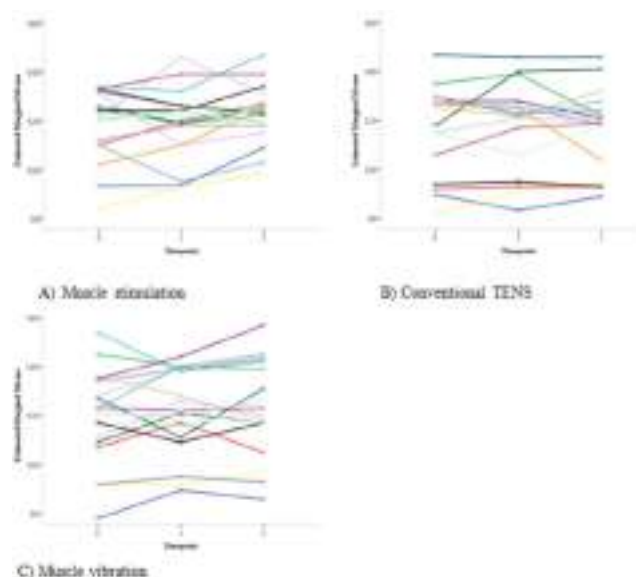


FIGURE 1 Individual mean score of full range of neck rotation at baseline (0), immediately post stimulation (1), 10 min. post stimulation (2) for the different interventions.

Conclusion: These findings suggest that peripheral stimulation may provide immediate symptomatic relief in CD, particularly for improving cervical range of motion and reducing pain. While promising, these results should be considered as exploratory and warrant further investigation in larger, controlled studies.

Disclosure: Nothing to disclose.

EPO-0748 | Divergent clinical phenotypes in siblings with a shared GBA1 mutation and posterior cortical hypometabolism

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Background and aims: Variants in the glucocerebrosidase gene (GBA1) are among the strongest genetic risk factors for Parkinson's disease (PD), yet disease penetrance is incomplete and marked intrafamilial heterogeneity is observed. Neuroimaging biomarkers may capture early neurobiological changes that are not reflected by clinical severity. The objective of this study was to describe the dissociation between clinical phenotype and brain metabolic patterns in siblings carrying a GBA1 mutation.

Methods: Two siblings carrying the same severe GBA1 mutation (p.H255Q) underwent a comprehensive cross-sectional evaluation. Assessments included motor and non-motor examination using the Movement Disorder Society Unified Parkinson's Disease Rating Scale, standardized cognitive testing, and brain metabolic imaging with fluorodeoxyglucose positron emission tomography (FDG-PET). Peripheral biomarkers included glucocerebrosidase enzymatic activity and monomeric

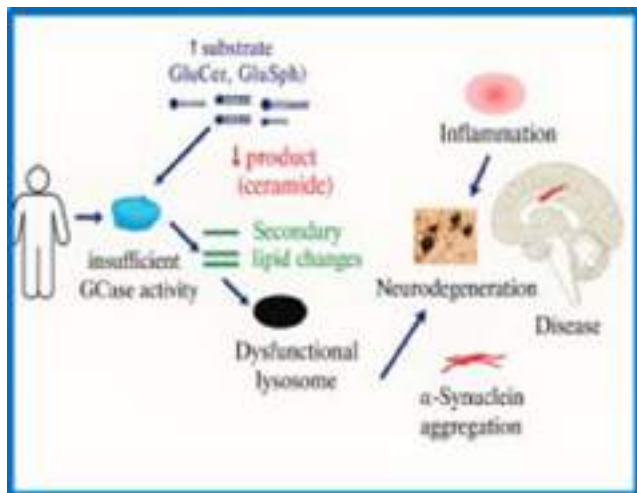


FIGURE 1 Schematic representation of the proposed pathogenic pathway linking GBA1 mutations to reduced glucocerebrosidase activity, lysosomal dysfunction, lipid accumulation, and alpha-synuclein aggregation in Parkinson's disease.

TABLE 1 Comparison of cognitive test performance between the sibling with GBA1-associated Parkinson's disease (GBA-PD) and the non-affected GBA1 carrier (GBA-nonPD) across global cognition, memory, and visuospatial domains.

Evaluation	GBA-PD	GBA-nonPD
Scholarita	17	17
MoCA	23,52	25,52
TMT A	28	28
TMT B	122	75
TMT B-A	94	47
ROCF copy	29	34
ROCF recall	9,75	15
Degraded figure test (judgment)	0	1
Superimposed figure test	48,94	71
Cognitive Profile (0=normal 1=MCI 2=PD)	1	0

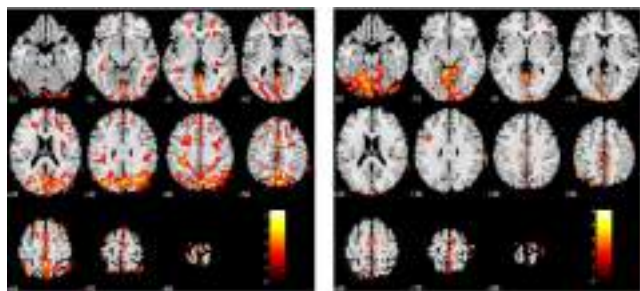


FIGURE 2 FDG-PET hypometabolism patterns in a GBA1-associated Parkinson's disease patient (59-year-old male) and an unaffected GBA1 carrier (56-year-old female), obtained using single-subject SPM analysis versus 125 age-corrected healthy controls ($p < 0.05$).

alpha-synuclein levels measured in peripheral blood mononuclear cells.

Results: The proband, a 59-year-old man with Parkinson's disease, showed motor impairment, multidomain cognitive deficits consistent with Parkinson's disease-mild cognitive impairment, and a high non-motor symptom burden. His 56-year-old sister exhibited only minimal motor signs, preserved cognition, and negligible non-motor symptoms. Despite the marked

clinical divergence, FDG-PET demonstrated a similar pattern of bilateral posterior cortical hypometabolism in both siblings. Glucocerebrosidase activity was comparable, while monomeric alpha-synuclein levels were higher in the affected sibling.

Conclusion: This intrafamilial observation highlights a dissociation between cortical hypometabolism and clinical expression in carriers of a severe GBA1 mutation. These findings suggest that metabolic brain changes may precede or evolve independently of overt clinical manifestations, supporting the potential role of FDG-PET in the phenotypic characterization of genetic Parkinson's disease.

Disclosure: Nothing to disclose.

EPO-0749 | Intrafamilial biochemical correlates of disease expression in GBA1 mutation carriers discordant for Parkinson's disease

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Background and aims: Pathogenic variants of the glucocerebrosidase gene (GBA1) are a major genetic risk factor for Parkinson's disease, yet disease penetrance is incomplete. Discordant sibling designs allow intrafamilial comparisons that control for shared genetic background and early-life environment. The objective of this study was to investigate intrafamilial differences in peripheral glucocerebrosidase activity and total alpha-synuclein levels in siblings carrying the same GBA1 variant but discordant for Parkinson's disease.

Methods: We conducted a family-based cross-sectional study including sibling pairs carrying identical GBA1 variants and discordant for Parkinson's disease. The most recent peripheral blood mononuclear cell sample with available biochemical data was selected for each subject. Glucocerebrosidase activity and total alpha-synuclein levels were quantified and normalized to total protein content. Intrafamilial paired differences were calculated as values in the Parkinson's disease-affected sibling minus those in the non-manifesting carrier. Analyses focused on paired descriptive statistics and resampling approaches.

Results: Five discordant sibling pairs were available for analysis. Glucocerebrosidase activity showed heterogeneous intrafamilial differences without a consistent directional pattern. In contrast, total alpha-synuclein levels were higher in the Parkinson's disease-affected sibling in most pairs. Resampling analyses supported a positive intrafamilial difference for alpha-synuclein despite limited statistical power. Larger intrafamilial

TABLE 1 Demographic characteristics of discordant sibling pairs carrying the same GBA1 variant. Each couple includes one Parkinson's disease-affected sibling (GBA-PD) and one non-manifesting GBA1 carrier.

Couple	Group	Age (years)	Sex	Education (years)	GBA1 variant
Couple 1	GBA-PD	61	M	11	L447P
Couple 1	GBA-NC	61	M	11	L447P
Couple 2	GBA-PD	61	M	15	N370S
Couple 2	GBA-NC	61	F	15	N370S
Couple 3	GBA-PD	61	F	17	R449G
Couple 3	GBA-NC	61	M	17	R449G
Couple 4	GBA-PD	61	M	18	L447P
Couple 4	GBA-NC	61	F	17	L447P
Couple 5	GBA-PD	61	F	17	T369M
Couple 5	GBA-NC	61	M	17	T369M

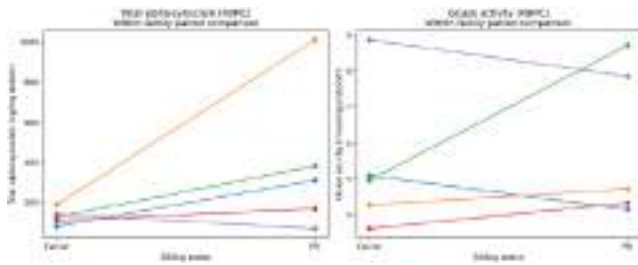


FIGURE 1 Within-family paired comparison of total alpha-synuclein and glucocerebrosidase activity in peripheral blood mononuclear cells from GBA1 discordant sibling pairs. Each line represents one family, comparing non-manifesting carriers and Parkinson.

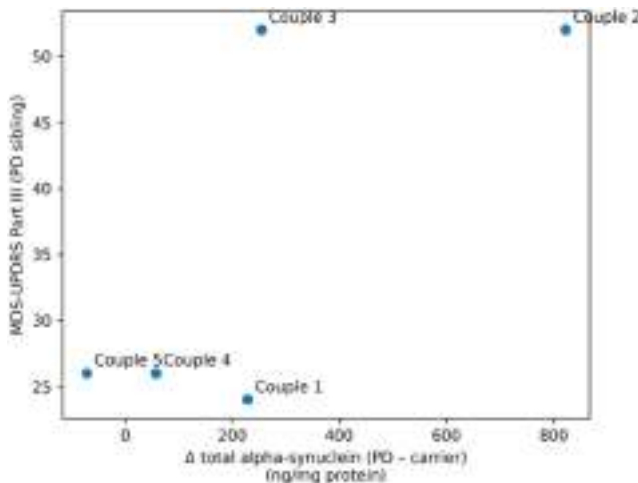


FIGURE 2 Exploratory association between intrafamilial differences in total alpha-synuclein (Parkinson's disease minus carrier) in peripheral blood mononuclear cells and motor severity, assessed by MDS-UPDRS Part III, in GBA1 discordant sibling pairs.

differences in alpha-synuclein tended to associate with greater motor and non-motor burden.

Conclusion: In this discordant sibling design, the Parkinson's disease-carrier difference captures biological processes associated with disease expression beyond shared genetic risk. While peripheral glucocerebrosidase activity appears variable within families, total alpha-synuclein emerges as a more consistent downstream readout associated with Parkinson's disease manifestation in GBA1 mutation carriers.

Disclosure: Nothing to disclose.

EPO-0750 | RTA-408 enhances Nrf2 activation and mitochondrial function to improve SCA3 cell and drosophila models

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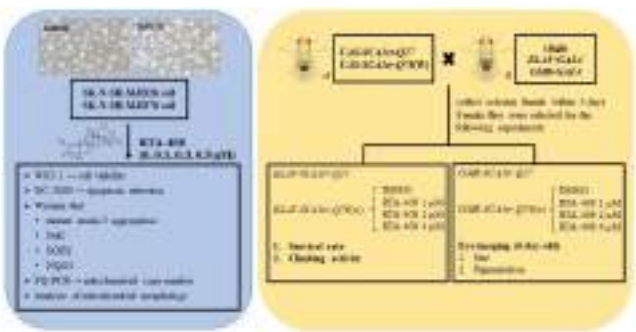


FIGURE 3 Material and methods.

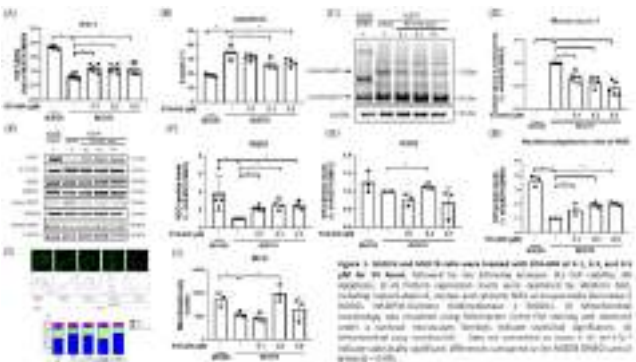


FIGURE 2 MJD26 and MJD78 cells were treated with RTA-408 at 0.1, 0.3, and 0.5 μ M for 24 hours.

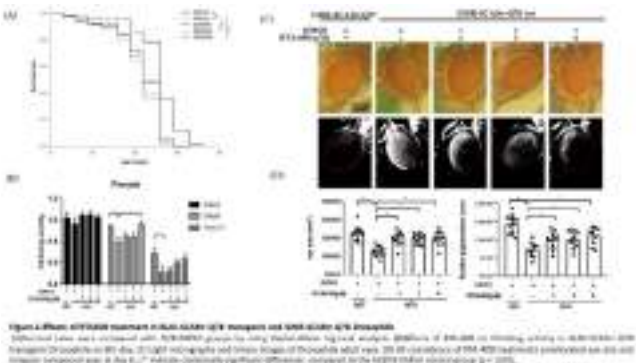


FIGURE 3 Effects of RTA408 treatment in ELAV-SCA3tr-Q78 and GMR-SCA3tr-Q78 Drosophila.

Background and aims: Spinocerebellar ataxia type 3 (SCA3) is a polyglutamine neurodegenerative disorder characterized by mutant ataxin-3 aggregation, oxidative stress, mitochondrial dysfunction, and progressive neuronal degeneration. Dysregulation of the Nrf2 antioxidant pathway contributes to SCA3 pathogenesis. RTA-408 (omaveloxolone), a potent Nrf2 activator, has therapeutic potential in Friedreich's Ataxia. This study evaluated the effects of RTA-408 on Nrf2 signaling, mitochondrial integrity, and disease-related phenotypes in SCA3 cellular and Drosophila models.

Methods: Human SK-N-SH neuroblastoma cells expressing normal (MJD26) or expanded polyglutamine (MJD78) ataxin-3 were treated with RTA-408 (0.1–0.5 μ M) for 24 hours. Cell viability, apoptosis, Nrf2 nuclear translocation, antioxidant enzyme

expression, mutant ataxin-3 protein levels, mitochondrial morphology, and mitochondrial copy number were assessed. In vivo efficacy was examined in ELAV- and GMR-driven SCA3 transgenic Drosophila treated with RTA-408 (1–4 μ M), assessing survival, locomotor performance, and retinal degeneration.

Results: MJD78 cells showed reduced viability, increased apoptosis, impaired Nrf2 nuclear localization, elevated mutant ataxin-3 levels, and fragmented mitochondria. RTA-408 significantly improved cell viability, enhanced Nrf2 nuclear translocation, increased antioxidant enzyme expression, reduced mutant ataxin-3 protein levels, and partially restored mitochondrial morphology and copy number. In SCA3 transgenic Drosophila, RTA-408 significantly prolonged survival, improved climbing activity, and ameliorated retinal degeneration.

Conclusion: RTA-408 confers neuroprotection in SCA3 models by activating Nrf2 signaling and improving mitochondrial function. The reduction of mutant ataxin-3 likely reflects decreased protein accumulation or enhanced clearance secondary to improved cellular redox homeostasis, supporting Nrf2-targeted mitochondrial modulation as a potential disease-modifying strategy for SCA3.

Disclosure: Nothing to disclose.

EPO-0751 | Sensor-based extended timed up and go parameters and cognitive performance in idiopathic Parkinson's disease

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Background and aims: This study investigated whether Extended Timed Up and Go (eTUG) parameters capture global and composite cognitive impairment in Parkinson's disease (PD).

Methods: Sixty-two PD patients (Hoehn-Yahr 2–3, age 55–74, MMSE >24) underwent sensor-based eTUG assessment using a single lumbar inertial measurement unit. Temporal (duration, turning time) and kinematic (velocity, trunk acceleration) parameters were extracted. Cognitive assessment included Montreal Cognitive Assessment (MoCA), MMSE, Frontal Assessment Battery (FAB) and composite scores reflecting attentive-executive functions. Motor severity was evaluated using Unified Parkinson's Disease Rating Scale Part III (UPDRS-III) and Postural Instability and Gait Difficulty (PIGD scores), and functional endurance was assessed with the six-minute walk test (6MWT). Spearman's correlations examined associations among eTUG, cognitive, motor, and 6MWT measures, considering $|r| \geq 0.3$ and $p < 0.05$ as significant.

Results: Turning time showed moderate correlations with global cognition (MoCA: $r = -0.39$) and attentive-executive function ($r = -0.38$), $p < 0.01$ (Figure 1). eTUG duration correlated with motor severity (UPDRS-III: $r = 0.51$, and PIGD: $r = 0.37$, $p < 0.01$) and 6MWT parameters (distance: $r = -0.76$; gait speed: $r = -0.40$, $p < 0.01$). Kinematic parameters of eTUG showed

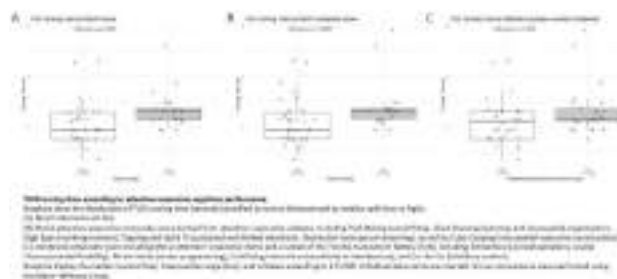


FIGURE 1 TUG turning time according to attentive-executive cognitive performance.

weaker cognitive associations but correlated moderately-to-strongly with motor impairment and sustained walking performance (6MWT).

Conclusion: Sensor-based eTUG turning metrics showed greater sensitivity to global and attentive-executive cognitive performance compared to straight-walking measures, highlighting the added value of turning analysis. Total eTUG duration captured both motor impairment and functional endurance. Overall, these findings support eTUG as a feasible motor-cognitive marker for clinical stratification and research applications in PD.

Disclosure: Nothing to disclose.

EPO-0752 | Switching to CREXONT/IPX203 improves good on time and reduces motor fluctuations: Interim real-world results from the first 100 patients in ELEVATE-PD

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Background and aims: CREXONT (IPX203), a novel extended-release carbidopa-levodopa formulation containing a mucoadhesive polymer, is designed to improve levodopa delivery and absorption. Here, we evaluate interim outcomes from the real-world study of CREXONT, assessing its impact to extend "Good On" time and reduce "Off" time in PD patients after switching from other levodopa-based therapies.

Methods: ELEVATE-PD is an ongoing, open-label phase 4 study where PD patients are converted from prior levodopa-based therapies (IR CD/LD, IR+COMT inhibitors, Rytary) to CREXONT. Following a 5-week dose-optimization period and a 1-week stable dosing, patients are followed for 12 months. Eligible participants had ≥ 2.5 hours of daily "Off" time at baseline. The primary endpoint is change in daily "Good On" time from baseline/Visit 1 to Day 42/Visit 4. This interim analysis includes 111 patients who completed Day 42.

Results: At baseline, mean daily "Good On" time was 9.39 ± 2.53 hours, "Off" time was 6.21 ± 2.31 hours. • CREXONT treatment increased daily "Good On" time: +3.40 hours for patients switching from IR CD/LD ($n = 74$), +2.91 hours from IR+COMT inhibitors ($n = 9$), +3.07 hours from Rytary ($n = 22$).

Corresponding reductions in “Off” time were: -3.18, -3.09, -2.90 hours, respectively. CREXONT increased “Good On” time per dose by: +2.09, +0.91, +0.98 hours, respectively. CREXONT also improved MDS-UPDRS total scores: -15.3, -10.0, -10.3 points, respectively. Majority of TEAEs were mild-to-moderate; most common ($\geq 3\%$) were dizziness (9.0%), nausea (7.2%), fall (7.2%), dyskinesia (4.5%), hallucination (3.6%), headache (3.6%).

Conclusion: Switching from other levodopa-based therapies to CREXONT significantly improved daily symptom control by increasing “Good On” time, decreasing “Off” time, and enhancing motor function across all treatment groups.

Disclosure: Supported by Amneal Pharmaceuticals.

EPO-0753 | Effect of cognitive and motor dual-task activities on balance in patients with PPPD, FGD, and healthy controls

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Background and aims: Balance control relies on integrating sensory inputs and motor responses and can be challenged by simultaneous cognitive or motor tasks, known as the dual-task effect. This study investigated balance under cognitive and motor dual-task conditions in individuals with FGD, PPPD, and healthy controls.

Methods: In this observational cross-sectional study, 15 participants with PPPD (61.7 ± 12.5 years [mean $\pm$ SD]), 16 with FGD (56.1 ± 15.5 years), and 15 healthy controls (55.3 ± 12.5 years) were assessed. Diagnoses followed Bárány Society criteria (PPPD) and Gupta and Lang criteria (FGD). Postural sway was measured using force plate during three 20-second conditions: eyes closed, eyes closed with a motor dual task (dominant-hand

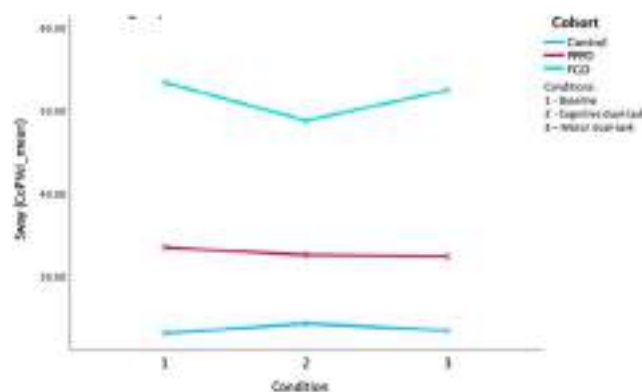


FIGURE 2 Sway across conditions in FGD, PPPD, and healthy control groups.

prono-supination), and eyes closed with a cognitive dual task (listing months backward) (Figure 1).

Results: FGD participants showed significantly higher sway than PPPD ($p=0.035$) and healthy controls ($p<0.001$) across all conditions. Healthy controls and PPPD did not show changes in sway during the dual-task. A small, non-significant reduction in sway was observed in the FGD group during the cognitive dual-task. (Figure2) Dual-task performance, measured by task completion time ($p=0.996$) and months-backward errors ($p=0.491$), did not differ between groups. No learning or adaptation effects were detected.

Conclusion: FGD and PPPD demonstrate distinct postural control profiles, with higher sway in FGD under all conditions. Dual-tasking did not significantly influence performance, although a trend toward reduced sway during cognitive dual-tasking was noted in FGD. Additional analyses will examine balance changes across monthly intervals during the cognitive task.

Disclosure: Nothing to disclose.

EPO-0754 | Features of the cerebellar fMRI connectivity in Parkinson's disease patients with cognitive decline, REM sleep behavior disorder and freezing of gait

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Background and aims: Sleep disturbances like RBD, postural instability (PI), freezing of gait (FOG) and cognitive decline in Parkinson's disease (PD) are closely linked, and often untreatable. These symptoms are caused by disturbances in brain networks involving the cerebellum, which determines the nature and severity of the motor and non-motor PD manifestations. We investigated the altered cerebellar functional connections (FC) in relation to RBD, PI, FOG severity and cognitive impairment in PD.

Methods: We used questionnaires and tests for assessment sleep problems, gait disturbances and cognition. Rs-fMRI data were analyzed using the SPM12 with the CONN toolbox. The PD patients were divided into 2 groups according to REM Sleep Behavior Disorder scoring (Table 1).

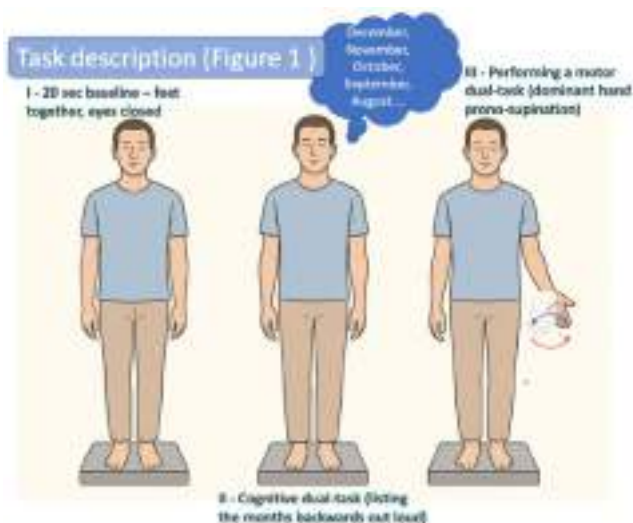


FIGURE 1 Task description.

TABLE 1 Comparative assessment of the PD patient's groups and the control group.

Parameters	PD Group 1 n = 19	PD Group 2* n = 14	Control group n = 80
	Median (IQR)		
Age, years	61 (55 – 68)	59 (52 – 68)	60 (55 – 70)
H & Y stage	1.5 (1–3)	1.5 (1–3)	–
Disease duration (years)	8 (6 – 15)	5 (4 – 13)*	–
UPDRS (part I)	21 (16 – 27)	10 (14 – 28)	–
UPDRS (part II)	17 (10 – 49)	17 (18 – 48)	–
Freezing of Gait Questionnaire	11 (5–29)	11 (13–18)	–
Postural instability (from 0 to 4, where 0 is the norm; 4 = cannot stand without help)	2 (1 – 3)	2 (1 – 3)	–
Parkinson's Disease Sleep Scale-2	28 (10 – 80)	25 (19 – 81)	–
REM Sleep Behavior Disorder rating scales (RBDs)*	8 (0 – 20)	2 (1–3)*	2 (1–8)
MMSE	28 (27 – 29)	28 (27 – 29)	29 (28 – 30)
MoCA	23 (22 – 26)	25 (22 – 28)	27 (26 – 28)

* Patients with a RBDs score of 5 or higher are included in Group 1. PD patients with an RBDs score of 4 or less are included in Group 2.
IQR = p < 0.05: Significant differences between Group 1 and Group 2 (Mann-Whitney test).
MMSE = Mini-mental State Examination
MoCA = Montreal Cognitive Assessment

Results: PD patients both groups demonstrated altered FC between cerebellum (lobule VI, Crus I-II, vermis) and Somatomotor, Default Mode (DMN), Central Executive Network (CEN) nodes. The 1stgroup had a longer PD duration and increased FC within the cerebellar network and between the cerebellum and the Salience Network (SN) compared to 2ndgroup. A significant negative correlation was found between the RBDs score and pedunculopontine nucleus (PPN)-cerebellum FC. In addition to reduced PPN-cerebellum FC, FOG and PI severity negatively correlated with cognitive impairment and reduced PPN – CEN FC.

Conclusion: The cerebellum is the key structures involved in the large-scale functional network, which is compromised by neurodegeneration, contributing to sleep problems, gait disturbances, and cognitive decline in PD. These clinical manifestations have their own specific patterns of the «brainstem (PPN)-cerebellum-cerebrum» FC alteration, what can be considered as a target for neuromodulation.

Disclosure: Nothing to disclose.

EPO-0755 | Clinical decision-making for patients with Friedreich ataxia: Real-world insights from a survey of health care providers in four European countries

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Background and aims: Friedreich ataxia (FA) is a rare genetic neurological disease. This study assessed knowledge of FA disease characteristics and clinical decision-making practices of European health care providers (HCPs) in real-world clinical settings.

Methods: Between May-October 2025, we conducted a cross-sectional survey of HCPs in France, Italy, Poland, and Spain to

assess disease knowledge and decision-making practices in the management of patients with FA. HCPs were asked about prior experience with diagnosing and managing patients with FA.

Results: The sample included 549 HCPs comprising 135 pediatricians, 155 primary care physicians, 216 adult neurologists, and 43 pediatric neurologists. 75% of HCPs reported experience managing ≥1 patient with FA and 36% of all HCPs indicated being confident or very confident with identifying the symptoms of FA, while 36% and 25% were somewhat or not at all confident, respectively. 48% of all HCPs estimated confirming an FA diagnosis takes a minimum of 7 months, and a majority of HCPs indicated delayed FA diagnosis has an above average or very high impact on worse quality of life (QoL, 69%), delays in treatment initiation (63%), worse survival outcomes (56%), and disease progression (53%). Improving QoL was the most important treatment objective (mean rank = 2.9 out of 6), followed closely by delaying loss of ambulation (3.2) and improving function (3.4).

Conclusion: In our sample of HCPs, confidence with FA disease characteristics and management was limited, potentially contributing to delayed diagnoses and treatment. Across all four European countries and HCP specialties, improving QoL was seen as the most important treatment objective.

Disclosure: Dr Pietro Guaraldi received speaker fees and honoraria from Alnylam, AstraZeneca, Akcea Therapeutic, Biogen, Chiesi, and Theravance Biopharma and received congress and travel accommodation expense compensations from AbbVie, Alnylam, Bial, and Zambon.

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EPO-0756 | Long-term global survival analysis from the Ferrara registry of device-aided therapies for Parkinson's disease (ReFerDAT study)

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Background and aims: Long-term follow-up data in advanced Parkinson's patients receiving Device-Aided Therapy (DAT) remain limited, our objective is to assess survival and determinants over up to 15 years of therapy.

Methods: Clinical and demographic data of all patients who underwent DAT implantation (Deep Brain Stimulation (DBS) or Levodopa-Carbidopa Intestinal Gel (LCIG)) at the Advanced Therapies for Movement Disorders Clinic, Neurology Department of Ferrara, from 2000 to 2023, were retrospectively analysed and compared.

Results: A total of 207 patients underwent DAT implantation during the observation period, 141 with DBS and 66 with LCIG. LCIG patients were older at implantation and had more advanced PD, with higher dropout at follow-up, whereas the DBS group showed a higher proportion of mortality from

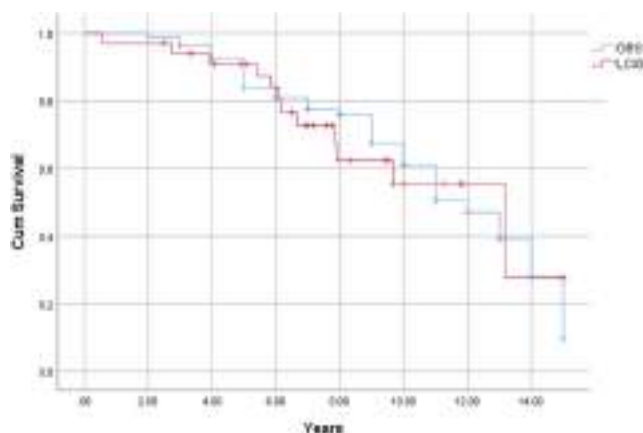


FIGURE 1 Kaplan–Meier survival analysis over a 15-year follow-up comparing patients receiving Deep Brain Stimulation (DBS) and Levodopa–Carbidopa Intestinal Gel (LCIG/Duodopa). Censored observations are indicated on the curves.

TABLE 1 Patients characteristics: deep brain stimulation (DBS) vs Levodopa–Carbidopa Intestinal Gel (LCIG). Data are reported as mean $\pm$ standard deviation or count (percentage). Patient characteristics and age refer to the time of implantation.

	DBS (n = 141)	LCIG (n = 66)	p-value
Sex (M/F)	68/53 (52.4/37.6)	37/29 (56.1/43.9)	0.384
Age (y)	66.4 $\pm$ 6.0	67.3 $\pm$ 6.6	<0.001
Years of disease (y)	12.6 $\pm$ 4.3	14.3 $\pm$ 6.1	0.003
MDS-UPDRS III	24.0 $\pm$ 9.7	31.3 $\pm$ 10.6	<0.001
H&Y	2.5 $\pm$ 0.5	2.8 $\pm$ 0.5	<0.001
LEDD (mg)	885.7 $\pm$ 481.5	1094.4 $\pm$ 342.3	0.141
Follow elsewhere after implant	15 (10.6)	1 (1.5)	0.022
Lost in follow-up (after at least 1 year)	19 (13.5)	1 (1.5)	0.006
Drop out	13 (9.2)	30 (45.5)	<0.001
Follow-up (m)	100.3 $\pm$ 65.3	73.3 $\pm$ 9.4	0.003
Alive/Deceased	55/39 (39.0/27.7)	22/12	0.627
Cause of Death			
Sudden Death	3 (7.7)	1 (8.3)	0.942
Pulmonary Complications	15 (38.5)	1 (8.3)	0.039
Malignant Disease	4 (10.3)	1 (8.3)	0.845
Suicide	3 (7.7)	0 (0)	0.999
Traumatic Accident	2 (5.1)	2 (16.7)	0.194
Terminal Stage of PD	4 (10.3)	2 (16.7)	0.547
Unknown	8 (20.5)	5 (41.7)	0.141

TABLE 2 Results of Cox proportional hazards models at 10- and 15-year follow-up, number of events (deaths) shown in parentheses.

10-year survival (35)	B	SE	P value	HR	95% CI
DAT (LCIG)	0.50	0.41	0.23	1.64	0.73–3.69
Age at implant	0.08	0.03	0.003*	1.08	1.03–1.15
Sex (Male)	-0.35	0.38	0.36	0.70	0.33–1.50
H&Y at implant	-0.31	0.39	0.43	0.73	0.34–1.59
Years of disease at implant	0.02	0.03	0.44	1.02	0.97–1.08
15-year survival (47)					
DAT (LCIG)	0.52	0.36	0.17	1.67	0.80–3.52
Age at implant	0.08	0.03	0.004*	1.08	1.02–1.14
Sex (Male)	0.32	0.33	0.34	1.37	0.72–2.62
H&Y at implant	0.05	0.37	0.90	1.05	0.51–2.15
Years of disease at implant	0.04	0.03	0.24	1.04	0.98–1.10

cardiopulmonary and infection-related complications. Fifteen-year Kaplan–Meier analysis showed similar mean and median survival (DBS: mean 10.99 years, median 12.0 years; Duodopa:

mean 10.62 years, median 13.17 years, $\chi^2 = 0.021$, $p = 0.884$). Age at implantation was the only independent predictor of mortality at 10- and 15-year follow-up (10-year: HR 1.09 per year, 95% CI 1.03–1.15, $p = 0.003$; 15-year: HR 1.08 per year, 95% CI 1.02–1.14, $p = 0.004$) by Cox models, while DAT type, sex, preoperative Hoehn–Yahr, and time from disease onset to implantation were not significantly associated with mortality.

Conclusion: Although causes of death differ between groups, overall long-term survival is comparable across DAT types. Age at implantation is the only factor exerting a stable long-term effect on survival, independent of device type.

Disclosure: Nothing to disclose.

EPO-0757 | Sensor-based kinematic markers of subthalamic deep brain stimulation efficacy in Parkinsonian tremor

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Background and aims: Subthalamic nucleus deep brain stimulation (STN-DBS) improves tremor in Parkinson's disease, but objective kinematic outcome markers remain limited. We quantified STN-DBS-related changes in tremor frequency, amplitude, and rhythmicity in tremor-dominant Parkinson's disease as objective outcome measures.

Methods: Thirty-one patients with tremor-dominant Parkinson's disease underwent bilateral STN-DBS. Resting and postural tremor of the more affected hand were recorded with a three-dimensional gyroscope preoperatively and at follow-up (≥ 6 months). Fourier-based analysis extracted peak tremor frequency, peak amplitude, rhythm (coefficient of variation), and tremor frequency range. Motor symptoms were assessed with the Movement Disorder Society Unified Parkinson's Disease Rating Scale (MDS-UPDRS) part III. Active contact locations were measured relative to the subthalamic nucleus center.

Results: Mean age was 63.2 years (standard deviation, SD 7.67). Disease duration at surgery was 8.1 years (SD 4.37). MDS-UPDRS part III scores improved from 15.8 (SD 10.85) to 8.3 (SD 7.07) with stimulation ON. Resting tremor scores decreased from 1.9 (SD 1.16) to 0.26 (SD 0.51). Postural tremor scores decreased from 1.3 (SD 1.14) to 0.2 (SD 0.42). Peak tremor frequency increased for resting (4.8–7.2 Hz) and postural tremor (5.3–8.0 Hz). Peak amplitude decreased by 56.2% for resting and 87.4% for postural tremor. Rhythmicity became less regular, with a 55% reduction for resting and 42% for postural tremor.

Conclusion: STN-DBS in Parkinson's disease shifts tremor toward higher-frequency, less rhythmic oscillations and improves tremor scores, especially for postural tremor. Quantitative kinematic measures provide objective markers of STN-DBS efficacy and may support sensor-based outcome assessment and adaptive DBS programming.

Disclosure: Nothing to disclose.

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Background and aims: Non-motor symptoms (NMS) in Parkinson's disease (PD), particularly those fluctuating with motor states (non-motor fluctuations, NMF), significantly impact patient's quality of life but are often underrecognized and undertreated. The aim of the study is to evaluate the effect of adjunctive therapy with the COMT inhibitor Opicapone (OPC) on static and fluctuating NMS in PD patients with motor fluctuations.

Methods: In this prospective observational study we enrolled twenty-one PD patients with motor fluctuations who initiated OPC 50 mg/day as add-on therapy to levodopa. Clinical evaluations at baseline (T0) and after 8–10 weeks (T1) included the MSD-UPDRS part III-IV and the Non-Motor Fluctuation Assessment (NoMoFA) Questionnaire, including the subscores pertaining to fluctuating symptoms (NoMoFA NMF), non-fluctuating symptoms (NoMoFA NoDif). Pre/post-treatment comparisons were analyzed using the Wilcoxon signed-rank test.

Results: We observed a significant reduction in the severity of nonmotor symptoms reported in OFF and in the severity of nonmotor symptom fluctuations (NoMoFA NMF) at T1 ($p < 0.001$). No significant changes were found in the 'static' non-motor symptoms (NoMoFA NoDif subscore; $p = 0.936$). The total NoMoFA score showed a trend toward improvement (median 18 to 14; $p = 0.083$).

Conclusion: Adjunctive OPC therapy was associated with a significant reduction in fluctuating NMS, particularly during OFF periods, in PD patients with motor fluctuations. These real-world findings support the hypothesis that improved dopaminergic

TABLE 2 NoMoFA scores at T0 and T1.

	T0	T1	P
NoMoFA ON	0 (0-0)	0 (0-0)	0.773
NoMoFA OFF	2 (0-4)	0 (0-3)	0.005**
NoMoFA NoDif	14 (9-19)	14 (8-21)	0.936
NoMoFA NMF	2 (0-3)	0 (0-3)	0.005**
NoMoFA TOT	18 (11-20)	14 (8-21)	0.083*

The p-values of the Wilcoxon test are presented and the level of significance is indicated by asterisks as follows: * = $p < 0.05$; ** = $p < 0.01$; *** = $p < 0.001$.
NoMoFA: Non-Motor Fluctuation Assessment Questionnaire; NoDif: No Difference; NMF: Non-motor fluctuations.

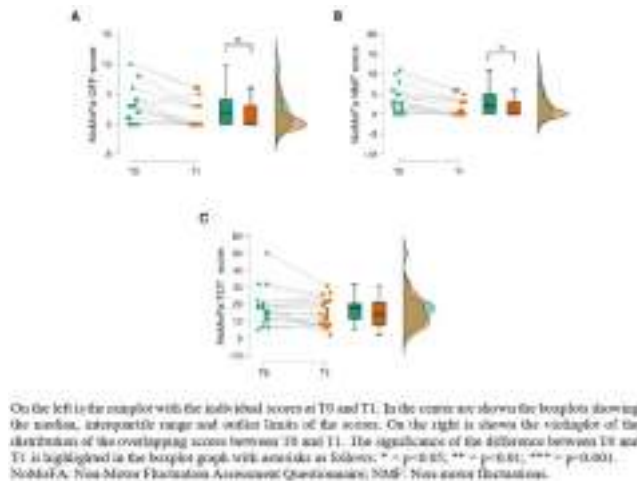


FIGURE 1 NoMoFA scores change between T0 and T1 in OFF (A), NMF (B) and Total (C) section.

stability may benefit the non-motor domain, reducing the burden of non-motor symptom fluctuations

Disclosure: Nothing to disclose.

TABLE 1 Demographic and clinical characteristics of included patients.

		PD (n = 21)
Age (years)		67.2 ± 8.4
Sex (F/M)		9/12
Disease duration (years)		7.2 ± 5.2
LEDD (mg)		684 ± 205
mHY		2 (2-2.5)
Phenotype	TD	0 (0)
	PGID	17 (81)
	IND	4 (19)
MDS-UPDRS I		8 (6-11)
MDS-UPDRS II		8 (5-11)
MDS-UPDRS III		32 (22-40)
MDS-UPDRS IV		2 (1-4)
MDS-UPDRS TOT		50 (38-65)
WOQ-19		3 (1-4)
MoCA		26 (24-26)

Results are reported as mean ± standard deviation or absolute values, as appropriate.
LEDD: Levodopa equivalent daily dose; mHY: Hoehn and Yahr; TD: Tremor dominant; PGID: Postural Instability Gait Disorder; IND: Indeterminate; MDS-UPDRS: Movement Disorders Unified Parkinson Disease Rating Scale; WOQ-19: Wearing off Questionnaire-19; MoCA: Montreal Cognitive Assessment.

EPO-0759 | Somatosensory timing captures early disease heterogeneity and predicts disease trajectory in Parkinson's disease

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Background and aims: The somatosensory temporal discrimination threshold (STDT) is a neurophysiological parameter reflecting the shortest time interval between two tactile stimuli required to perceive them as temporally separate. STDT is a stage-, severity- and disease duration-dependent parameter, possibly reflecting dopaminergic degeneration. However, its long-term evolution and predictive value remain underexplored. We aimed to investigate longitudinal STDT changes and its potential to predict clinical progression in PD and discriminate divergent disease trajectories.

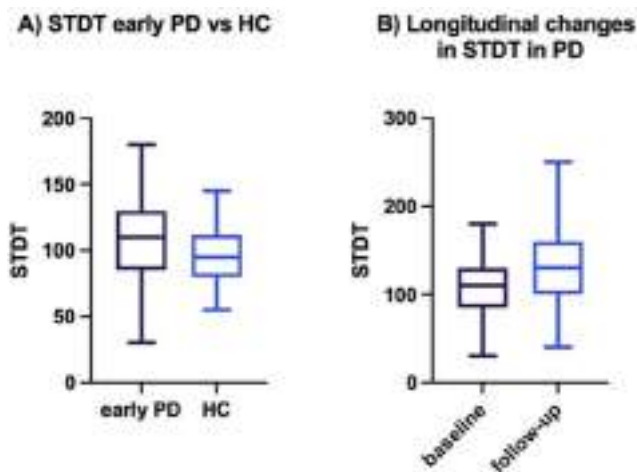


FIGURE 1 Differences in Somatosensory Discrimination Threshold among groups: (A) Comparison of STDT values between early PD patients and HC; (B) Longitudinal changes of STDT in PD patients.

Baseline STDT predicting motor worsening

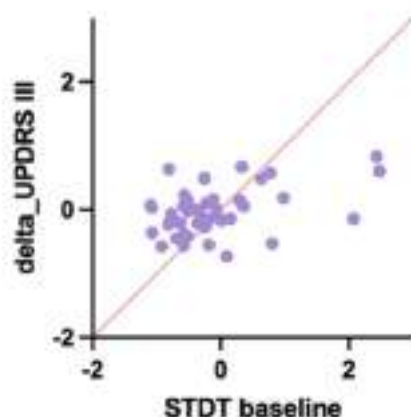


FIGURE 2 Baseline STDT values predict motor symptom progression in Parkinson's disease. Scatter plot showing the relationship between STDT z-scores at baseline and changes in UPDRS III scores (delta_UPDRS III) over a 4-year follow-up in PD patients.

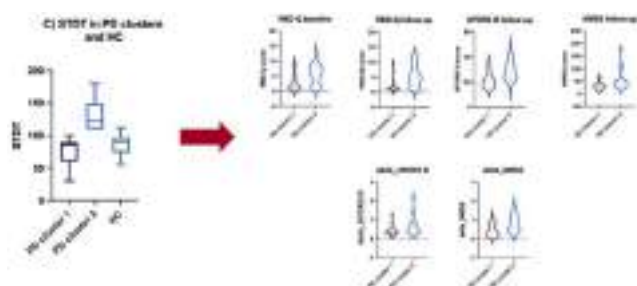


FIGURE 3 Significant clinical differences between PD clusters. Box and violin box illustrate significant differences between PD cluster 1 and cluster 2 patients in clinical scores at baseline, follow-up and in the progression rate.

Methods: We conducted a 4-year longitudinal study involving 60 early-stage PD patients and 50 healthy controls (HC). In PD patients, clinical and STDT assessments were performed at baseline and follow-up. STDT was measured at the distal phalanx of the index finger using an ascending stepwise paradigm. Longitudinal changes, correlations between STDT and clinical measures, regression analyses for progression prediction, and k-means clustering based on baseline STDT were performed.

Results: At baseline, STDT values were similar between patients and HC, but significantly worsened over time in PD. Initially associated with REM sleep behaviour disorder (RBD) symptoms, STDT later correlated with cognitive decline and predicted motor progression. STDT-based clustering identified two subtypes: cluster 1, defined by higher baseline STDT and more severe RBD, exhibited greater motor and non-motor burden and faster progression at 4-year follow-up than cluster 2.

Conclusion: Early STDT differentiates patients with distinct clinical profiles and divergent disease trajectories, capturing early inter-individual variability and predicting long-term motor outcomes in PD. STDT-based clustering aligns with brain-first vs body-first models, supporting its value in early PD stratification.

Disclosure: The authors declare no financial disclosures relevant to the present manuscript. Over the past twelve months, A.C. has received speaker honoraria from Roche, Biogen, Novartis, Almirall and Merck, all unrelated to the content of this manuscript.

EPO-0760 | Longitudinal assessment of smartphone-based motor and speech biomarkers in isolated REM sleep behaviour and Parkinson's disease

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Background and aims: The sensitivity of smartphone assessments to disease progression in isolated rapid eye movement sleep behaviour disorder (iRBD) and Parkinson's disease (PD) remains unexplored.

Methods: Participants completed a set of active smartphone tasks every 14 days over a two-year period in their home environments. The cohorts included 23 patients with iRBD, 24 patients with early PD, and 26 healthy controls. The tasks comprised (i) speech assessments (sustained vowel phonation, rapid syllable repetition, and reading a passage), (ii) psychomotor assessments (tapping speed, alternating tapping, and word typing), (iii) a tremor assessment (holding the phone with an outstretched arm), and (iv) a walking turnaround task.

Results: Most tasks, including sustained vowel phonation, rapid syllable repetition, tapping speed, alternating tapping, and the tremor task, demonstrated strong learning effects, limiting their ability to differentiate between groups or detect progression. Word typing with the dominant hand showed group

differentiation (iRBD and PD vs. controls; $p < 0.05$). Measures of monopitch during reading and turnaround walking performance exhibited significant longitudinal worsening in the iRBD group ($p < 0.05$).

Conclusion: Measures capturing monotone speech and walking turnaround performance show potential for inclusion in future clinical trials in iRBD. Most simple active tasks demonstrated limited sensitivity to disease progression due to pronounced learning effects.

Disclosure: This project was supported by the Czech Ministry of Health (grant no. NW24-04-00211) and the ERC-CZ program from the Ministry of Education Youth and Sports (project no. LL2504).

EPO-0761 | Assessing dysarthria severity via long-term average spectrum in neurological diseases

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Background and aims: Speech is one of the most complex quantitative markers of motor function and is often affected already in most progressive neurological diseases. This study aimed to evaluate sensitivity of long-term averaged spectrum (LTAS) descriptors computed for reading passage in a broad range of neurological diseases and various types and severities of dysarthria.

Methods: Four spectral moments (spectral mean, spectral standard deviation, spectral skewness and spectral kurtosis) based on LTAS were computed for reading passage collected from 462 speakers, including 306 healthy controls and 156 neurological patients secondary to multiple sclerosis, Parkinson's disease, supranuclear palsy, multiple-system atrophy, Huntington's disease, essential tremor, cerebellar ataxia, and amyotrophic lateral sclerosis. One-way analysis of covariance with post hoc Fisher's least significant difference test was applied.

Results: Compared to controls, Parkinson's disease showed lower mean ($p=0.015$) and higher skewness ($p=0.014$) and kurtosis ($p=0.01$). Among parkinsonian groups, multiple system atrophy also had lower mean ($p=0.042$). Conversely, mean was higher ($p=0.006$) and skewness lower ($p=0.036$) for cerebellar ataxia. Multiple sclerosis demonstrated only a lower mean ($p=0.023$). Among diseases, mean ($p<0.001$), skewness ($p<0.001$) and kurtosis ($p=0.008$) were able to separate cerebellar ataxia and multiple system atrophy.

Conclusion: Our results confirmed LTAS as a suitable approach to objectively describe speech changes due to neurological diseases. Furthermore, spectral moments were able to significantly separate diseases with similar clinical manifestations, which could have important implications for differential diagnosis.

Disclosure: The project was supported by the Czech Ministry of Health, project number NW24-04-00211 and by the ERC-CZ program from the Ministry of Education Youth and Sports, project number LL2504.

EPO-0762 | Artificial intelligence-driven identification of Parkinson's disease using oculometrics

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Background and aims: Parkinson's disease (PD) diagnosis remains largely clinical, motivating the search for objective biomarkers to support diagnosis, early detection, and disease monitoring. Eye movement and pupillary abnormalities in PD can be quantified as oculometrics and provide objective features for data-driven analysis. We aimed to develop and evaluate artificial intelligence (AI) models for PD detection using quantitative ocular motor and pupillary measurements acquired with a virtual reality (VR)-based medical eye-tracker.

Methods: Eighty-one individuals with idiopathic PD were recruited at clinical centres in Zurich (Switzerland) and Exeter (UK). Participants completed two study visits including standardized VR-based ocular motor assessments and routine clinical examinations. The analysis included 132 PD examinations and 148 examinations from an independent healthy control cohort. Each PD examination was matched to an age- and sex-matched control using a globally optimal matching algorithm. Group differences were assessed using standardized mean differences (SMDs) and Welch's t-tests. A supervised machine learning classifier was trained using the most discriminative oculometric features, with site-specific performance evaluation.

Results: The largest PD-control differences were observed in saccadic parameters (accuracy, peak velocity, and main sequence), vergence measures, ocular alignment, and pupillary dynamics (constriction and dilation velocity and latency). Classification performance was high across sites: Zurich—AUC 0.96 (sensitivity 0.96; specificity 0.89); Exeter—AUC 0.91 (F1-score 0.84; sensitivity 0.89; specificity 0.79).

Conclusion: VR-derived oculometrics can help differentiate individuals with PD from healthy controls, supporting the potential of an oculometrics-based framework as an objective biomarker approach for PD. Further work focuses on cohort expansion, cross-site data integration, and correlation with clinical disease measures.

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Background and aims: Parkinson's disease (PD) has a prodromal phase characterized by non-motor symptoms and clinical features that can be integrated into probabilistic risk estimates. Data on medical and neurological comorbidities across levels of estimated prodromal PD risk remain limited.

Methods: We conducted a cross-sectional analysis within the Healthy Brain Aging (HeBA) study, an online population-based cohort. Prodromal PD risk was estimated using the Movement Disorder Society probabilistic research criteria. Participants were classified as high (MDS score >30%) or low (MDS score <1%) risk and matched 1:1 by age, sex, and recruitment site ($n = 326$ total; 163 high-risk and 163 low-risk participants). Type II Diabetes mellitus and depression/anxiety were excluded from the risk calculation and analyzed separately. Medical and neurological conditions and clinical symptom scores were compared between high and low risk groups.

Results: High-risk participants had more medical conditions, including hypertension (58.9% vs. 30.1%), depression/anxiety (55.8% vs. 14.1%), hypercholesterolemia (52.1% vs. 30.1%), diabetes mellitus (20.2% vs. 8.0%), and peripheral vascular disease (14.1% vs. 3.1%) (all $p < 0.01$). Restless legs syndrome (17.8% vs. 0.6%) and essential tremor (8.6% vs. 1.2%) were also more frequent. Multimorbidity increased the odds of high-risk classification (odds ratios: 3.47 for two conditions, 12.38 for three, and 19.50 for >4). Clinical symptom scores including NMS total, GDS, UPDRS-II, and Quality of Life (VAS), differed significantly between groups (all $p < 0.001$).

Conclusion: Higher estimated prodromal PD risk was associated with increased multimorbidity and worse clinical symptom profiles, according to the scales used, in individuals without a PD diagnosis.

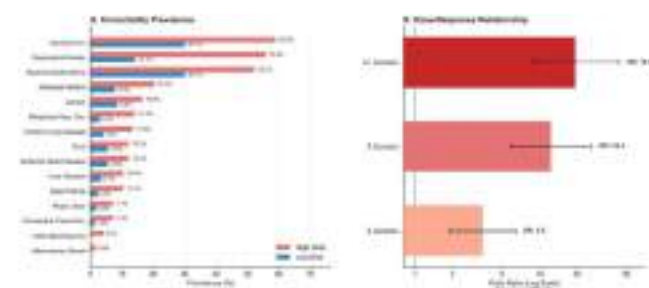


FIGURE 1 Morbidity phenotype associated with prodromal Parkinson's disease risk. (A) Prevalence of selected medical conditions by risk group. (B) Dose-response association between morbidity burden and High-Risk status, expressed as odds ratios.

Disclosure: The HeBA study is supported by a grant from The Michael J. Fox Foundation for Parkinson's Research (grant MJFF-PI047794). All authors report no disclosures related to this work.

EPO-0764 | Language as an early indicator of cognitive impairment in Parkinson's disease: Development and validation of an AI-based application

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Background and aims: Parkinson's disease (PD) is not only defined by motor symptoms but also by early changes in speech that affect linguistic, semantic and executive functions. Because speech production engages widespread neural networks, subtle alterations can signal early cognitive decline. With recent advances in artificial intelligence (AI), these patterns can now be captured and analyzed automatically, making speech a promising non-invasive marker of cognitive impairment in PD.

Methods: We developed an automated pipeline that records speech, transcribes it using Whisper AI, and analyzes linguistic and semantic features with spaCy and an OpenAI large language model. Extracted markers included word count, sentence structure, verb-noun ratios, semantic coherence and content complexity. These features were used to train three machine-learning models—Support Vector Classifier, Random Forest Classifier and Logistic Regression—to differentiate patients with cognitive impairment (MoCA <24) from those without impairment (MoCA ≥24). The entire workflow was implemented in a web-based application for standardized and scalable data processing.

Results: Automated linguistic measures showed a high degree of concordance with manually obtained reference values, confirming the validity of the pipeline. All machine-learning models demonstrated good discriminative performance. Logistic



FIGURE 1 Screenshot of the web-based application prototype showing audio input, automated transcription, feature extraction, and model output.

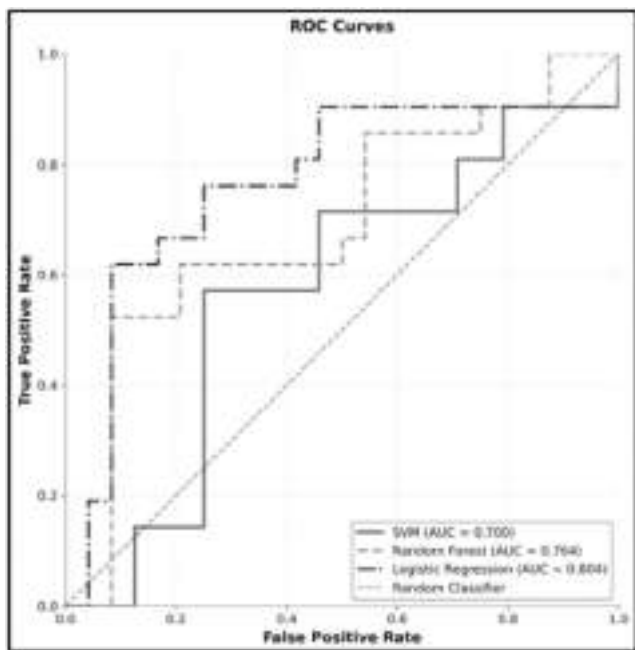


FIGURE 2 ROC curves for all ML models, with Logistic Regression achieving the highest AUC (0.804).

TABLE 1 Performance metrics of the trained Support Vector Machine, Random Forest, and Logistic Regression models. Shown are accuracy, precision, recall, specificity, F1-score, AUC-ROC, and the mean AUC values from the stratified 5-fold cross-validation.

Model	Performance Metrics					Cross-Validation AUC (M ± SD)
	Accuracy	Precision	Recall	Specificity	F1-Score	
SVM	0.705	0.708	0.871	0.792	0.832	0.709
RF	0.807	0.800	0.810	0.708	0.834	0.764
LR	0.798	0.727	0.762	0.790	0.744	0.804

Regression performed best, achieving an AUC-ROC of 0.804 and showing the most robust classification across repeated evaluations. The integrated web application processed recordings reliably and consistently.

Conclusion: Our study suggests that AI-based speech analysis can reliably detect speech alterations that indicate cognitive impairment in Parkinson's disease. The validated pipeline and its integration into a web application support its potential as a scalable, non-invasive screening tool.

Disclosure: Nothing to disclose.

EPO-0765 | Abstract withdrawn

EPO-0766 | Cortical oscillatory and connectivity profiles of stimulation-induced bradykinesia in dystonia

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Background and aims: Pallidal neuromodulation is an established treatment for dystonia, likely acting through modulation of distributed brain networks and their oscillatory dynamics. However, in some patients, pallidal deep brain stimulation (DBS) induces bradykinesia. While this adverse effect has been linked to local stimulation sites and oscillatory changes, its network-level correlates remain unclear.

Methods: We recorded whole-head magnetoencephalography (MEG) in 16 dystonia patients with pallidal DBS at rest with stimulation ON and after a ≥30-minute washout (DBS OFF). In both conditions, patients performed a video-recorded finger-tapping task. Kinematics were extracted using computer vision-based pose estimation. DBS electrodes were localized using Lead-DBS, and functional connectivity from active contacts was estimated using a normative fMRI connectome ($n=1000$). Connectivity profiles were correlated with DBS-induced changes in tapping frequency. To assess oscillatory network effects, we computed cortical spectral power (theta 4–8 Hz, alpha 8–12 Hz, beta 13–30 Hz, gamma 40–90 Hz) and related whole-cortex power changes to movement slowing.

Results: At the group level, DBS did not significantly alter tapping frequency, indicating marked interindividual variability. Across patients, however, the connectivity profile of the active contact predicted DBS-induced slowing ($R=0.45$, $p=0.04$): stronger connectivity to sensorimotor and supplementary motor areas was associated with greater bradykinesia. Cortical modulation in the alpha and gamma bands explained 50% ($R=0.77$, $p=0.001$) and 31% ($R=0.56$, $p=0.01$) of the variance, respectively. These signatures were spatially and spectrally distinct from those associated with therapeutic DBS effects, suggesting separable network mechanisms.

Conclusion: Integrating connectivity and oscillatory profiling may support individualized DBS programming to minimize motor side effects.

Disclosure: Nothing to disclose.

EPO-0767 | Abstract withdrawn

Movement Disorders 10

EPO-0768 | Efficacy and safety of magnetic resonance-guided focused ultrasound thalamotomy in essential tremor: A systematic review and meta-analysis

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Background and aims: Magnetic resonance-guided focused ultrasound is a surgical intervention for medication-refractory essential tremor, however, meta-analyses have been limited in their exploration of the durability of the treatment effect. This study aimed to assess the treatment effect and safety of this procedure over time. Different to other meta-analyses, this study assessed treatment effect durability over time from 1 month to 5 years follow-up, as well as tremor recurrence.

Methods: A systematic search of the literature using set criteria was conducted via PubMed, Scopus, Web of Science, and Cochrane library databases, in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. Data analysis was conducted in R, utilising a random-effects model for meta-analysis and a mixed-effects model for meta-regression.

Results: 45 studies met the inclusion criteria, with 42 included in analyses. Significant changes in hand tremor, total tremor, disability scores, and quality of life scores were demonstrated across the time points investigated, the pooled standardized mean differences were -2.36 ($p < 0.0001$), -2.08 ($p < 0.0001$), -2.85 ($p < 0.0001$), and -1.41 ($p < 0.0001$) 1 year post-operation. Sensory symptoms and unsteadiness adverse events were frequently observed, with pooled proportions of 22% (95% CI 15%; 31%) and 23% (95% CI 16%; 31%) 1 month post-operation.

Conclusion: Although the procedure demonstrated efficacy and safety across the studies evaluated, meta-regression analysis suggests a decrease in treatment effect over time that requires further investigation.

Disclosure: Nothing to disclose.

EPO-0769 | Multi-target deep brain stimulation for dystonia and complex tremor syndromes: A single-centre case series and literature review

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Background and aims: Deep Brain Stimulation (DBS) is an established neurosurgical procedure for pharmacologically refractory dystonia and complex tremor syndromes. Multiple target stimulation, such as internal globus pallidus (GPi) and ventral intermediate nucleus (VIM) stimulation, can be used to achieve the control of dystonic features as well as the severe tremor at the same time. This clinical study aims to highlight 10 cases treated at the Walton Centre NHS Foundation Trust (Liverpool, UK) wherein DBS has been implemented beyond unilateral and singular target procedures. It describes the immediate and long-term response and effects of multiple-target DBS in patients with dystonia and complex tremor syndromes (including Holmes tremor).

Methods: Case notes were reviewed retrospectively for the included patients. Data were collected from baseline and subsequent appointments, including treatment details, quantitative and qualitative findings, and side effects. Metrics included the cervical dystonia scale TWSTRS, Fahn-Marsden dystonia scale, FTM scale for tremor, EQ-5D for quality of life, and HADS Anxiety and HADS Depression scores.

Results: Our cohort experienced symptom relief following multi-target DBS, with varying levels of symptom suppression and stimulation-related side effects (i.e. slurred speech, gait disturbance); stimulation adjustments were made accordingly. Importantly, there were discrepancies in objective clinical improvement and perceived symptom relief by the patients.

Conclusion: Multi-target DBS can be useful in managing dystonia and complex tremor cases; it requires balancing symptoms improvement with side effects through continued follow-up and setting adjustments. Tremor suppression and side effect profiles are patient dependent, with varying levels of success demonstrated in our case series.

Disclosure: Nothing to disclose.

EPO-0770 | Early COMT inhibition improves motor stability and real-world mobility in Parkinson's disease: A randomized single-blind study with digital biomarkers

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Background and aims: Whether early COMT inhibition provides superior real-world mobility compared with levodopa dose escalation in patients with Parkinson's disease (PD) and emerging motor fluctuations remains unclear. This study compared the efficacy of early add-on therapy with Opicapone versus an additional levodopa dose, using clinician-rated outcomes (CROs), patient-reported outcome measures (PROMs), and digital biomarkers.

Methods: Forty-six PD patients were randomized into two clinically-matched groups ($n=23$ each) receiving either add-on Opicapone 50 mg or an additional levodopa/carbidopa dose (100/25 mg). Single-blind clinical evaluations were performed at baseline and after one month using CROs (MDS-UPDRS III, CGI-C) and PROMs (WOQ-19, PDQ-9, PGI-C). Also, home monitoring with a validated waist-worn wearable sensor provided digital biomarkers of daily steps, step length, gait velocity, cadence, their coefficients of variation (CV), and estimated ON/OFF time.

Results: While CROs did not show between-group differences after treatment, PROMs and digital biomarkers consistently indicated that patients receiving Opicapone had reduced fluctuation severity (WOQ-19), greater perceived improvement (PGI-C),

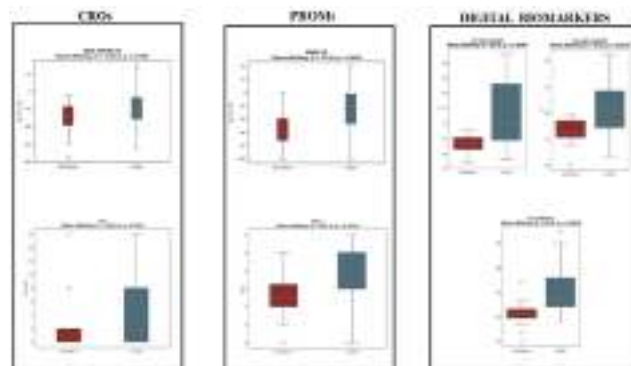


FIGURE 1 Main findings from clinician-rated outcomes (CROs), patient-reported outcome measures (PROMs), and digital biomarkers.

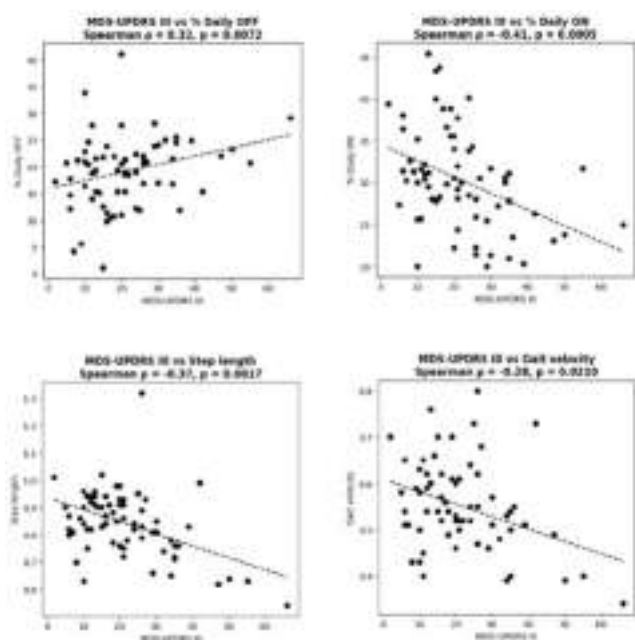


FIGURE 2 Main clinical-behavioural correlations.

longer objective daily ON time, and more stable gait performance (step length, gait velocity, and cadence CVs) than those taking an additional levodopa dose (Figure 1). MDS-UPDRS III scores correlated with daily ON/OFF time and objective gait measures, indicating worse real-world mobility with increasing motor severity (Figure 2).

Conclusion: Early add-on therapy with Opicapone appears more effective than an additional levodopa dose in improving motor stability and patient-perceived well-being in PD patients with emerging motor fluctuations. While CROs may fail to capture changes in long-term motor phenomena, digital biomarkers provide an ecologically valid assessment of real-world motor behaviours.

Disclosure: Nothing to disclose.

EPO-0771 | Cognitive performances are associated with subclinical gait alterations in drug-naïve cognitively unimpaired patients with Parkinson's disease

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Background and aims: Gait impairment is the most disabling symptom in Parkinson's disease (PD) patients and is associated with high falling risk, worse disease progression and reduced survival. Cognitive impairment has been consistently associated with impaired walking ability and higher fall risk in PD. Detection of subclinical gait alterations could identify early PD patients at risk to develop cognitive impairment.

Methods: Thirty-four drug-naïve cognitively unimpaired PD patients (disease duration ≤ 3 years, Hoehn & Yahr ≤ 2.5) and

20 age- and sex-matched healthy controls were consecutively enrolled. All patients underwent a II-level neuropsychological assessment to exclude cognitive impairment. Motor symptoms were evaluated using the Unified Parkinson's Disease Rating Scale part III (UPDRS-III). Gait and balance parameters were acquired using six Opal V2R wearable sensors when performing the Timed up and go test in single and dual task (ST/DT) conditions. Bivariate correlation analysis between clinical and wearable sensors metrics was performed.

Results: Worse gait and postural parameters detected by wearable sensors in ST/DT conditions significantly correlated with cognitive tests exploring visuo-spatial processing, psychomotor coordination and executive functions involved in retrieving words and information recalling. No correlations have been found between cognitive tests and UPDRS-III scores.

Conclusion: Subclinical gait and postural changes are already associated with cognitive performances in PD patients with normal cognition. Wearable sensors could provide useful information to characterize early PD patients, even when gait and postural disturbances are not clinically detectable. Integrating clinical, neuropsychological and digital data may identify patients more prone to develop clinical overt cognitive impairment useful for prevention strategies.

Disclosure: Nothing to disclose.

EPO-0772 | Neurophysiological markers of motor compensatory mechanisms in early Parkinson's disease: A 2-year longitudinal study

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Background and aims: Parkinson's disease (PD) often shows asymmetric motor onset despite bilateral nigrostriatal degeneration, suggesting cortical compensation. To investigate 24-month



FIGURE 1 Screening and selection of Parkinson's disease patients. The initial cohort of 82 subjects had already been screened for other clinical exclusion criteria.

TABLE 1 Neurophysiological data in patients with Parkinson's disease at baseline and follow-up of the no-progressors group.

Time	Baseline	Follow-up					
Hemisphere	Precomp	Symp	Precomp	Symp	p [*]	p ^{**}	p ^{***}
Number	8	8	8	8			
MDS-UPDRS III total	16	16	16	16			0.230
MDS-UPDRS III side	0 (0-2)	9 (5-14)	0 (0-2)	11 (8-22)	<0.001	<0.001	0.577
AIM	200 (142.875-200)	185.533 (152.941-200)	200	185.533			0.665
AMT	36 (27-38)	40 (32-48)	31 (22-41)	31 (22-38)	0.322	0.712	0.017
RMT	51 (35-78)	55 (35-78)	49.5 (30-48)	39 (25-43)	1	0.557	0.008
100% RMT	0.148 (0.089-0.245)	0.134 (0.0541-0.248)	0.064 (0.048-0.113)	0.089 (0.048-0.119)	0.834	0.618	0.002
120% RMT	1.207 (0.612-2.106)	0.778 (0.348-1.756)	1.057 (0.345-2.189)	0.882 (0.281-1.707)	0.659	0.680	0.779
140% RMT	2.042 (0.956-4.038)	1.179 (0.628-4.733)	1.641 (0.790-4.393)	1.905 (1.123-3.888)	0.674	0.680	0.674
I/O slope	0.938 (0.372-1.888)	0.543 (0.442-2.262)	0.777 (0.371-1.700)	0.508 (0.542-1.905)	0.674	0.636	0.889
SICI 2 ms	54.058 (11.847-59.469)	82.439 (33.693-103.440)	39.591 (10.732-44.870)	27.793 (8.365-145.797)	0.898	0.481	0.68
ICF 10 ms	88.823 (40.215-188.221)	104.719 (71.893-143.480)	37.578 (21.338-112.562)	44.423 (18.839-277.892)	0.800	0.680	0.327
ppslope	43.179 (24.998-158.680)	23.482 (-27.247-75.580)	29.476 (-8.326-85.802)	19.527 (-13.778-112.085)	0.248	0.529	0.484
IHI 10 ms	77.133 (37.395-125.686)	193.488 (29.357-173.472)	80.326 (25.101-87.824)	48.243 (19.821-166.533)	0.248	0.674	0.327
IHI 40 ms	59.572 (27.151-121.816)	194.190 (76.826-355.665)	84.502 (35.956-112.631)	64.328 (37.588-166.411)	0.621	0.680	1.0
PAS 5 ms	128.890 (84.635-171.265)	94.527 (38.272-166.476)	152.168 (41.802-327.720)	137.451 (37.897-322.641)	0.884	0.508	0.283
PAS 15 ms	132.734 (84.635-188.772)	96.647 (73.576-187.067)	178.869 (51.048-229.629)	128.902 (52.813-304.040)	0.248	0.172	0.123
PAS 30 ms	124.810 (97.091-209.886)	93.954 (53.485-208.273)	155.043 (50.518-126.518)	137.172 (45.891-182.872)	0.059	0.148	0.263
PAS average	129.099 (97.091-171.589)	98.239 (64.613-184.634)	179.222 (51.829-327.222)	137.918 (38.837-298.192)	0.615	0.481	0.123

longitudinal clinical and TMS changes in early PD with unilateral motor signs, comparing stable versus progressive patients.

Methods: Following our previous study [1], 16 PD patients with unilateral motor signs and bilateral putaminal DAT-SPECT loss, and 28 healthy controls (HC), were assessed at baseline; 13 completed follow-up. Clinical scales and bilateral TMS measures of corticospinal excitability, intracortical excitability, interhemispheric inhibition, and paired associative stimulation were collected.

Results: Motor disability increased with reduced asymmetry [2]; 5 out of the original 13 patients with unilateral motor signs converted to bilateral involvement (defined as 'progressors'; presymptomatic-side MDS-UPDRS-III ≥ 3), while others remained unilateral ('non-progressors'). Compared with HC, PD showed increased corticospinal excitability in both hemispheres and reduced intracortical facilitation. In the total PD cohort, presymptomatic hemispheres showed longitudinal motor worsening, accompanied by increased corticospinal excitability (RMT reduction $p=0.003$) and reduced intracortical facilitation (ICF $p=0.013$; slope $p=0.039$). In progressors, presymptomatic hemispheres converting to symptomatic showed increased RMT ($p=0.042$), reduced ICF ($p=0.043$), flattening of the intracortical slope ($p=0.043$), and loss of long-interval IHI ($p=0.043$). In non-progressors, the presymptomatic hemisphere showed increased excitability (RMT reduction $p=0.018$) with SICI enhancement ($p=0.050$), while ICF, IHI, and motor scores remained stable.

TABLE 2 Neurophysiological data in patients with Parkinson's disease at baseline and follow-up of the progressors group.

Time	Baseline	Follow-up					
Hemisphere	Precomp	Symp	Precomp	Symp	p [*]	p ^{**}	p ^{***}
Number	3	3	3	3			
MDS-UPDRS III total	16 (14-18)	27 (23-32)					0.013
MDS-UPDRS III side	1 (0-3)	9 (5-20)	3 (4-9)	14 (11-18)	0.888	0.689	0.038
AIM	161.834 (148-200)	186 (20-111.111)					0.043
AMT	35 (24-38)	36 (25-45)	36 (24-35)	30 (24-34)	0.751	0.451	0.445
RMT	43 (38-48)	44 (40-58)	38 (34-38)	32 (28-40)	0.799	0.285	0.042
100% RMT	0.091 (0.009-0.107)	0.098 (0.061-0.200)	0.073 (0.044-0.166)	0.075 (0.038-0.083)	0.670	0.588	0.317
120% RMT	1.026 (0.448-2.247)	0.867 (0.313-1.718)	1.533 (1.126-2.968)	1.245 (0.984-4.926)	0.485	0.753	0.043
140% RMT	2.558 (0.648-8.876)	1.114 (0.900-2.507)	2.839 (1.432-6.688)	2.299 (1.444-7.088)	0.251	0.754	0.686
I/O slope	1.219 (0.188-4.393)	0.528 (0.434-1.152)	1.385 (0.689-2.323)	1.119 (0.682-3.518)	0.251	0.754	0.686
SICI 2 ms	28.947 (11.889-88.072)	51.389 (40.655-57.789)	34.690 (4.798-40.042)	28.091 (9.674-74.20178)	0.317	0.754	0.686
ICF 10 ms	157.184 (50.828-199.067)	62.735 (46.975-158.478)	36.270 (5.382-75.311)	33.228 (12.668-76.642)	0.251	0.682	0.043
ppslope	138.925 (26.387-157.621)	11.888 (-6.228-143.318)	7821 (-6.178-36.712)	34.386 (-13.075-57.686)	0.847	0.347	0.043
IHI 10 ms	44.413 (7.968-156.722)	56.842 (34.418-74.722)	72.875 (39.498-112.707)	75.742 (9.578-90.670)	0.834	0.754	0.225
IHI 40 ms	48.898 (38.984-72.978)	41.738 (22.081-104.811)	75.773 (62.903-96.388)	37.655 (14.339-81.040)	0.675	0.117	0.043
PAS 5 ms	98.829 (60.371-221.041)	97.620 (78.265-98.466)	128.280 (88.833-167.310)	175.852 (153.187-214.780)	0.682	0.686	0.889
PAS 15 ms	217.640 (78.237-264.288)	108.310 (78.089-117.173)	184.884 (128.007-232.580)	202.786 (128.351-418.381)	0.251	0.754	0.889
PAS 30 ms	158.927 (51.898-213.489)	99.581 (84.778-178.357)	168.233 (187.663-222.097)	147.342 (117.18-223.877)	0.917	0.567	0.138
PAS average	135.600 (64.542-213.788)	97.144 (91.906-125.027)	181.839 (122.538-188.913)	208.384 (139.858-288.295)	0.275	0.347	0.686

Conclusion: Corticospinal hyperexcitability reflects PD state and progression, whereas preserved intracortical and inter-hemispheric inhibition supports compensation. Loss of facilitation and interhemispheric control marks maladaptive changes linked to clinical conversion. TMS measures appear promising biomarkers of compensatory capacity and motor progression in early PD.

Disclosure: Nothing to disclose.

EPO-0773 | Real-world post-market outcomes of magnetic resonance-guided focused ultrasound in movement disorders

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Background and aims: MR-guided focused ultrasound (MRgFUS) is an established, CE-approved therapy for unilateral Parkinson's disease (PD), unilateral and staged-bilateral essential tremor (ET) and neuropathic pain (NP). Over 30,000 procedures, including 8000 in Europe, have been performed. This analysis reports updated safety outcomes based on accumulated clinical experience.

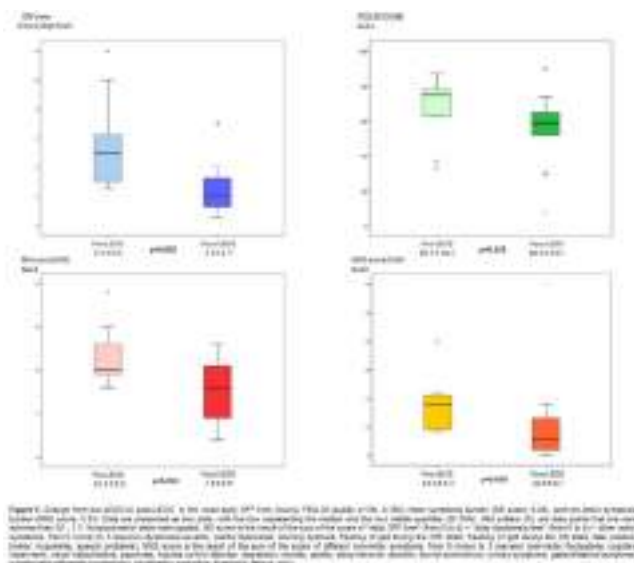


FIGURE 1

phases, respectively (Table 2). Daily OFF time was reduced from Vpre to Vpost in 3.2 ± 1.9 hours ($p=0.002$). PwP improved significantly from Vpre to Vpost in motor symptoms, whereas a trend of significance was found for non-motor symptoms burden and quality of life (Figure 1).

Conclusion: LECIG could be a good therapeutic option in PwP who failed fLD/fCD.

Disclosure: Nothing to disclose.

EPO-0775 | Low-income settings, low dyskinesia? A treatment paradox in Parkinson's disease care

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Background and aims: Levodopa-induced dyskinesia (LID) is a common complication of long-term dopaminergic therapy in Parkinson's disease (PD) in high-income countries. In contrast, low-resource settings report a paradoxically lower prevalence. This study evaluated whether limited access to optimized dopaminergic treatment and a conservative approach is linked to a distinct motor complication profile in PD patients from a low-income country.

Methods: We conducted an observational study of 113 adults with idiopathic PD at a tertiary neurological center in Moldova during 2025. Data collected included disease duration, diagnostic delay, Hoehn and Yahr stage, current dopaminergic therapy (dose, frequency, duration), use of adjunctive and anticholinergic medications, and dyskinesia presence. Access to advanced and dyskinesia-specific therapies was described.

Results: Despite a median disease duration of 3 years (IQR 1.55–6) and advanced stages at presentation (Hoehn & Yahr stage 2: 50%; stage 3: 36%; stage 4: 9%; stage 5: 1%), dyskinesia was rare (8%). Levodopa was often started late (median 1.775 years, IQR 0.6–3.5) and given at relatively low doses (median LEDD

600 mg, IQR 375–750), reflecting limited availability and concern over complications. Individual dose adjustment was restricted by the absence of alternative formulations and adjunct therapies. Effective dyskinesia treatments, such as amantadine (2 patients) or device-aided therapies, were seldom accessible. Anticholinergics were commonly used (17%), indicating compensatory strategies in a resource-limited setting.

Conclusion: In conclusion, the low prevalence of dyskinesia in this low-income setting likely reflects chronic underexposure to dopaminergic therapy rather than disease-related protection, highlighting the need for earlier diagnosis and improved access to diversified PD treatments.

Disclosure: This study was funded by the National Agency for Research and Development (NARD) of the Republic of Moldova, research project no. 25.80012.8007.04TC.

EPO-0776 | Brain-heart interaction in patients with Parkinson's disease treated by STN-DBS

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Background and aims: Parkinson's disease (PD) is recognized as a multisystem disorder with a variety of symptoms including autonomic dysfunctions. There is increasing evidence of cardiovascular symptoms including altered heart rate variability (HRV) compared to healthy controls. Deep brain stimulation of the subthalamic nucleus (STN-DBS) is an established therapy for motor symptoms. This study used simultaneous EEG and ECG recordings to examine the effects of STN-DBS on cortical-autonomic interactions.

Methods: EEG and ECG were recorded in 24 PD patients treated with STN-DBS at rest in DBS OFF and ON condition. For ECG, HRV metrics, SD1 and SD2 were computed from Poincaré plots. In EEG, spectral parameterization (FOOOF) was performed to extract oscillatory power (delta, theta, alpha, beta, low/high gamma) and aperiodic features (offset, exponent). DBS-induced change was defined as $\Delta = \text{ON} - \text{OFF}$. Associations between ΔEEG and ΔHRV were assessed using Spearman correlations.

Results: Short-term HRV (ΔSD1) was positively correlated with Δalpha power, strongest at F4 ($r=0.54$, $p=0.011$). Overall autonomic flexibility, reflected by ΔSD2 , was negatively associated with $\Delta\text{exponent}$. This coupling was most prominent in central and temporoparietal areas, with significant effects in C4 ($r=-0.59$, $p=0.005$), T8 ($r=-0.46$, $p=0.038$), and Cz ($r=-0.44$, $p=0.048$).

Conclusion: DBS exerts no coherent autonomic effect. However, our main findings reveal a structured pattern underlying this heterogeneity. Increases in SD2, reflecting increased autonomic flexibility considered beneficial, were associated

with shifts toward cortical excitation, while reductions in SD2 corresponded to increased cortical inhibition.
Disclosure: Nothing to disclose.

EPO-0777 | Interpretable digital gait biomarkers for multiclass screening of Parkinson's disease, Alzheimer's disease, and healthy controls

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Background and aims: Parkinson's disease (PD) and Alzheimer's disease (AD) are prevalent neurodegenerative disorders that present with overlapping gait impairments, limiting the utility of clinical observation for differential screening.
Methods: We investigated whether shoe-embedded inertial measurement units (IMUs) can support multiclass classification of PD, AD, and healthy controls while revealing interpretable, disease-specific digital gait biomarkers. Ninety-four participants (41 PD, 26 AD, 27 controls) performed 10-meter walking tasks (196 trials total). From bilateral tri-axial acceleration and angular velocity signals (100 Hz), 373 gait features spanning spatiotemporal, time-domain, frequency-domain, and entropy measures were extracted. To prevent information leakage from repeated trials, we applied nested cross-validation with GroupKFold at the participant level.
Results: Among 11 classifiers, Linear Discriminant Analysis achieved optimal performance (test macro-F1 0.840, 95% CI: 0.77–0.92; AUC 0.946; balanced accuracy 0.843) using only 20 features with the smallest generalization gap (0.040). Class-wise sensitivity was 0.99 for controls, 0.82 for PD, and 0.73 for AD. SHAP analysis revealed distinct pathophysiological signatures: PD classification was driven by increased variability in inversion-eversion angular velocity and double-support phase duration, consistent with basal ganglia-mediated motor control deficits; AD classification relied on entropy measures, gait velocity reduction, and timing alterations, reflecting impaired cognitive–motor integration.
Conclusion: These findings demonstrate that shoe-embedded IMU gait analysis combined with explainable machine learning can effectively differentiate neurodegenerative conditions while providing clinically interpretable biomarkers aligned with established disease mechanisms, supporting scalable screening and hypothesis-driven biomarker discovery.
Disclosure: Nothing to disclose.

EPO-0778 | Prevalence of nocturnal hypertension and pathological nocturnal dipping profile in patients with synucleinopathies in a tertiary care center

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Background and aims: Nocturnal hypertension and pathological nocturnal dipping profiles are adverse prognostic factors in α synucleinopathies (Parkinson's Disease and Multiple System Atrophy), yet they remain under diagnosed because they are usually asymptomatic. This study evaluated their prevalence in a cohort of synucleinopathy patients followed at a tertiary centre.
Methods: Forty adults (26M/14F) with synucleinopathies and history of orthostatic hypotension were recruited from the CHUV-Movement Disorders Unit. All underwent 24 hour Ambulatory Blood Pressure Monitoring (ABPM). Nocturnal hypertension was defined as mean nighttime SBP ≥ 120 mmHg or DBP ≥ 70 mmHg. Dipping status was classified as dippers (10–20% night time fall), non dippers (<10%) and risers (night time BP higher than daytime). Isolated nocturnal hypertension meant elevated night time BP with normal daytime BP (<135/85 mmHg). Association between variability indices and

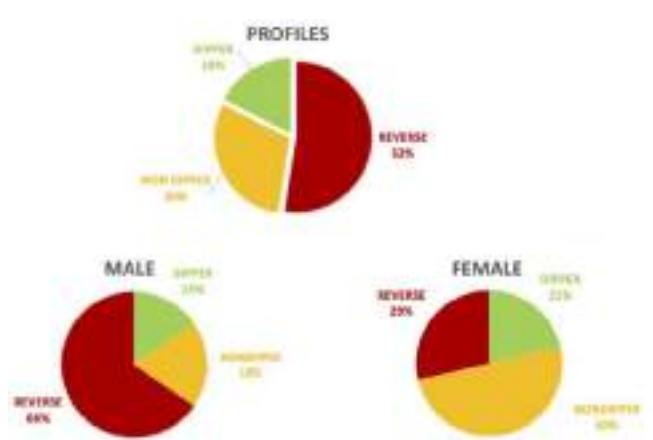


FIGURE 1 Dipping status classification and their prevalence.

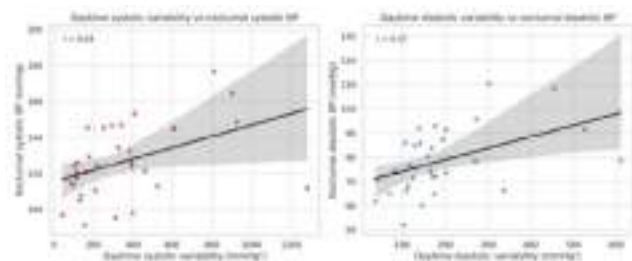


FIGURE 2 Association between daytime variability and nocturnal blood pressure levels.

mean nocturnal BP were assessed using Pearson correlation coefficients and linear regression.

Results: 80.0 % percent of participants had nocturnal hypertension, and 81.3% of those met criteria for isolated nocturnal hypertension. Pathological nocturnal BP profiles were present in 82.5% of the cohort (Figure 1), more severe in male than in female. Higher daytime BP variability was moderately associated with higher nocturnal mean SBP ($r=0.44$) and DBP ($r=0.47$) (Figure 2).

Conclusion: Nocturnal hypertension and abnormal dipping are highly prevalent in synucleinopathy patients, supporting routine 24 hour ABPM—especially in those with orthostatic hypotension complaints—to enable early cardiovascular management and improve quality of life. Larger prospective studies are needed to confirm these findings and assess their impact on disease morbidity and mortality.

Disclosure: Nothing to disclose.

Treatment in MS and Related Disorders 2

EPO-0779 | Efficacy and safety of fenebrutinib vs ocrelizumab in primary progressive multiple sclerosis: Primary results of the phase III FENTrepid study

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Background and aims: Bruton's tyrosine kinase inhibitors (BTKi) have the potential to meet the need for multiple sclerosis

(MS) treatments targeting both relapsing and progressive disease biologies. The Phase II FENopta study (NCT05119569) showed rapid reduction of acute inflammatory disease activity in relapsing MS with fenebrutinib, an oral, highly selective, non-covalent, reversible, central nervous system-penetrant BTKi. The effect of fenebrutinib on progressive MS subtypes has not been demonstrated. FENTrepid (NCT04544449) is the first clinical trial of fenebrutinib in primary progressive MS (PPMS), using ocrelizumab (the only approved treatment for PPMS) as an active comparator.

Methods: FENTrepid is a Phase III, multicentre, randomised, double-blind, double-dummy, parallel-group study evaluating the noninferiority of fenebrutinib vs ocrelizumab in people with PPMS (2017 McDonald criteria), age 18–65 years and Expanded Disability Status Scale [EDSS] score 3.0–6.5. Participants (randomised 1:1) received 200-mg oral fenebrutinib twice daily or 600-mg intravenous ocrelizumab every 24 weeks. The primary endpoint was time to onset of 12-week composite confirmed disability progression via worsening on EDSS, Timed 25-Foot Walk Test and 9-Hole Peg Test.

Results: PwPPMS ($N=985$) had a mean (SD) age of 48.9 (10.3) years and median baseline EDSS score of 5.0. Half were female (49.5%) and most (84%) had no recent exposure to prior disease-modifying therapies. Mean (SD) durations since MS symptom onset and diagnosis were 9.0 (6.7) and 4.7 (5.4) years, respectively. Primary efficacy and safety results will be presented.

Conclusion: FENTrepid will provide evidence on the benefits and risks of fenebrutinib vs ocrelizumab on disability progression in PPMS.

Disclosure: Sponsored by F. Hoffmann-La Roche Ltd. Writing and editorial assistance were provided by Nucleus Global and funded by F. Hoffmann-La Roche Ltd. A. Bar-Or, J. Oh, G. Giovannoni, M.P. Sormani, M.S. Weber, S. Stoll, J.A. Nicholas, H.-C. von Büdingen, J. Lopez, L. Roberts, M. Triyatni, Q. Qi, J.N. Ratchford, J. Napieralski, A. Goodyear, S.L. Hauser and L. Kappos have conflicts of interest to disclosure. These disclosures will be included in full at the time of presentation.

EPO-0780 | Long-term frexalimab treatment modulates the activity of immune networks

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Background and aims: Frexalimab, a second-generation anti-CD40L monoclonal antibody, inhibits the costimulatory CD40/CD40L pathway, and may recalibrate immune networks involving adaptive and innate immunity. During the double-blind-period of the phase-2 trial (NCT04879628) in participants with relapsing multiple sclerosis (pwRMS), frexalimab-1200mg/intravenous (IV) reduced new gadolinium-enhancing T1-lesions

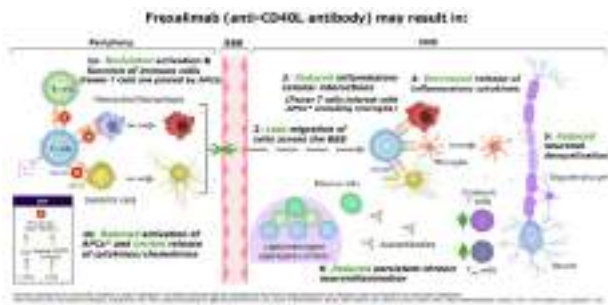


FIGURE 1 Proposed mechanism of action of frexalimab (anti-CD40L antibody) in multiple sclerosis.

by 89% vs placebo at Week (W) 12. Proteomic and transcriptomic analyses revealed modulation of B-cell activation and antibody production, and reduced plasma levels of innate immune cell-secreted chemokines and cytokines implicated in MS. Here, we explore frexalimab's long-term effects through W120 of the open-label-extension (OLE) using plasma proteomics.

Methods: 129 pwRMS were randomised (4:4:1:1) to frexalimab-1200mg/intravenous (IV) every-4-weeks (q4w), frexalimab-300mg/subcutaneous (SC)-q2w, or matching placebo. After W12, placebo recipients switched to frexalimab and entered the OLE. All SC participants switched to 1800mg-q4w for pharmacokinetic comparability with 1200mg/IV-q4w. Plasma proteomic analysis from frexalimab-1800mg/SC ($n=26$) and 1200mg/IV ($n=30$) arms was performed using NULISaseq with 249-plex-Inflammation panel, and analysed by a paired t-test and Benjamini-Hochberg correction.

Results: At W120, frexalimab treatment yielded 79 differentially abundant soluble proteins (76 downregulated, 3 upregulated) vs baseline, primarily involved in cytokine-receptor interactions, immune-cell communication, and chemotaxis. Frexalimab significantly downregulated adaptive and innate immune cell receptors/co-receptors, cytokines, chemokines, antigen-presentation and adhesion-molecules (CXCL13, CD27, BCMA, GZMA, CSF1R) associated with MS pathophysiology, including progressive disease.

Conclusion: Long-term frexalimab treatment exerts comprehensive immunomodulatory effects by downregulating inflammatory mediators associated with disease progression. These findings support understanding of frexalimab's mechanism of action, which targets immune networks involving adaptive and innate immunity, and holds the potential to mitigate acute and chronic neuroinflammation in MS.

Disclosure: Biljana Djukic, Roopali Gandhi, Galina Boldina, Nils Ternes, Bailin Zhang, Mikhail Levit, Pavithra Krishnaswami, Svend Geertsens, Stephane Saubadu, Philippe Truffinet: Employees of Sanofi (may hold shares and/or stock options in the company). Vinh Truong: an employee of Ividata Life Sciences (contracted by Sanofi), and may hold shares and/or stock options in the company. Heinz Wiendl: Honoraria for acting as a member of Scientific Advisory Boards for Biogen, Genzyme, Merck Serono, Novartis, Roche Pharma AG and Sanofi and UCB; speaker honoraria and travel support from Alexion, Biogen, Biologix, Cognomed, F. Hoffmann-La Roche Ltd, Gemeinnützige Hertie-Stiftung, Merck, Novartis, Roche Pharma AG, Genzyme, Teva and WebMD Global; paid consultant for Actelion, Argenx, Biogen, Bristol Myers Squibb, EMD Serono, Idorsia, IGES, Immunic, Immunovant, Janssen, Johnson & Johnson, Novartis,

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EPO-0781 | Baseline data of Austrian RMS patients treated earlier vs. later with Ofatumumab: The KRONOS study population

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Background and aims: The non-interventional KRONOS study investigates effects of early versus late treatment with Ofatumumab in RMS patients in real-world Austrian clinical practice. Current evidence suggests patients benefit from early highly effective therapy. KRONOS aims to support this evidence for Ofatumumab.

Methods: KRONOS (NCT05776888) is an ongoing 24-month, non-interventional study collecting retrospective and prospective data. Eligible RMS patients received Ofatumumab for ≥ 3 and ≤ 12 months at baseline. The primary endpoint is the proportion of patients with NEDA-3 in the second year after starting Ofatumumab, comparing early (Cohort1) versus late use

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FIGURE 1 KRONOS study centers.

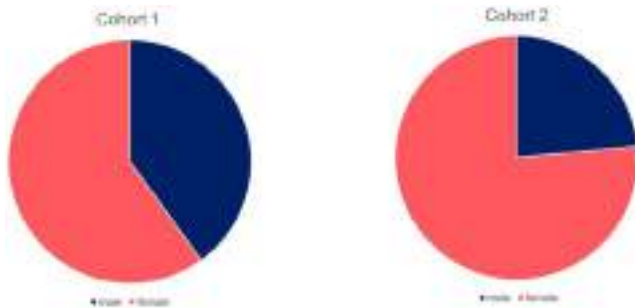


FIGURE 2 KRONOS sex distribution.

		Cohort 1 EDSS < 3 years				Cohort 2 EDSS ≥ 3 years				Total	
		n	%	n	%	n	%	n	%	n	%
Relapse-free	Yes	55	100%	4	7.7%	54	92.3%	59	100%	109	92.3%
Disability-free	Yes	48	89.1%	48	85.7%	96	85.9%	48	85.7%	96	85.9%
Disability-free	No	6	10.9%	5	9.3%	11	19.1%	6	10.9%	11	19.1%
Disability-free	Total	61	100%	9	100%	65	100%	54	100%	119	100%

TABLE 1 KRONOS EDSS change.

(Cohort 2). Secondary endpoints include demographics, individual NEDA-3 components, and safety.

Results: A total of 106 patients were enrolled at 15 centers. Cohort 1 includes treatment-naïve patients or those on disease-modifying therapy (DMT) <3 years; Cohort 2 includes patients with ≥3 years of prior DMT. Mean age was 34.3 years in Cohort 1 and 41.0 years in Cohort 2; overall, 37.5 years. Gender distribution was 40% male/60% female in Cohort 1 and 23.5%/76.5% in Cohort 2. Since disease onset, 79.5% in Cohort 1 and 40.5% in Cohort 2 had <2 relapses. Since starting Ofatumumab, 94.5% in Cohort 1 and 92.2% in Cohort 2 had no relapses. Most patients had EDSS 0.0–1.0. Overall, 13.2% showed a change in EDSS, mainly reductions (64.3% –1.0 point; 7.1% –2.0 points).

Conclusion: KRONOS patients were predominantly female with low baseline disability. Most remained relapse-free after starting Ofatumumab, and a minority showed improved EDSS. Longitudinal data will clarify longer-term outcomes and effects of early versus late Ofatumumab use.

Disclosure: This study is fully funded by Novartis Pharma GmbH, Austria.

Background and aims: Real-world data directly comparing efficacy outcomes among RRMS patients treated with high-efficacy disease modifying treatments remain limited. In this retrospective single centre study, we compared the efficacy of natalizumab (NTZ) and ocrelizumab (OCR).

Methods: We analysed relapse-free survival (RFS), MRI activity-free survival (MFS), progression-free survival (PFS – 6-month confirmed increase EDSS score) and evidence of disease activity (NEDA-3), among RRMS patients treated with OCR ($n = 677$) and NTZ ($n = 368$) at Imperial College Healthcare Trust between 2008 and 2024. After propensity score matching (1:1), outcomes were compared by using Log-rank tests. Cox model regressions were performed across pre-specified subgroups.

Results: After matching ($n = 276$ per group), 394 (71.3%) patients were females, at treatment start median age was 38 years (IQR 30–47), disease duration 4 years (IQR 1–9), and median EDSS score 2.5 (IQR 2.0–5.0). At 4 years follow up, RFS (NTZ: 89% vs OCR: 85%, $p = 0.064$) and MFS (NTZ: 86% vs OCR: 82%, $p = 0.120$) were similar between the groups, while higher proportions of NTZ-treated patients were free of progression (89% vs 85%, $p = 0.050$) and maintained NEDA-3 (74% vs 62%, $p = 0.002$) status. In subgroup analyses, NTZ was associated with a lower risk of losing NEDA 3 than OCR in patients ≤45 years (HR 0.54, 95% CI 0.37–0.78), with EDSS <4 (HR 0.60, 95% CI 0.42–0.87), and with disease duration ≤5 years (HR 0.52, 95% CI 0.35–0.76).

Conclusion: In our cohort, NTZ was superior to OCR in achieving sustained disease remission, driven by more effective prevention of disability progression

Disclosure: Nothing to disclose.

EPO-0783 | FCGR3A V158F affects ocrelizumab binding to natural killer cells and B cell repopulation during extended dosing

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Background and aims: Ocrelizumab is highly effective in multiple sclerosis (MS). We tested whether FCGR3A (CD16) V158F variant (rs396991) modifies B cell kinetics and outcomes during ocrelizumab and whether ocrelizumab binding to natural killer (NK) cells differs by genotype.

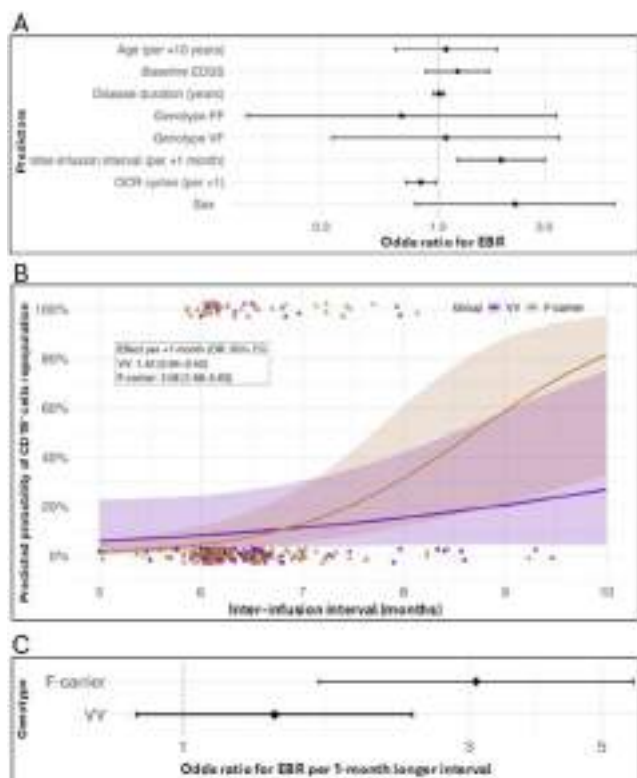


FIGURE 1 FCGR3A polymorphism effects on B cell kinetics and clinical outcomes under ocrelizumab treatment in MS subjects.

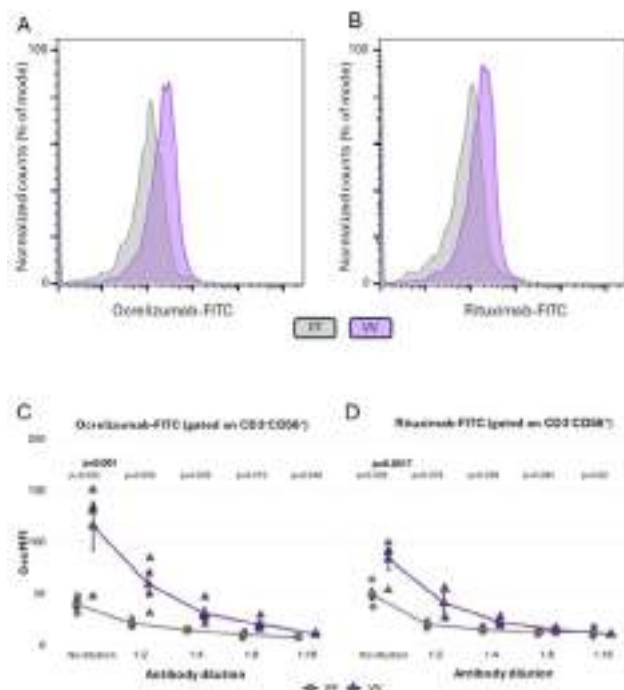


FIGURE 2 FCGR3A polymorphism effects on ocrelizumab and rituximab binding to NK cells.

Methods: We enrolled 101 people with MS receiving ocrelizumab (at least two cycles). B cell repopulation (BR) was defined as CD19 positive B cells at least 10 cells/ul. Genotype was modelled dominantly (F carriers vs VV) and as a genotype by interval interaction, adjusted for age, sex, baseline disability, disease

duration, and prior therapy class. Flow cytometry binding assays tested whether rs396991 affects ocrelizumab binding to NK cells using fluorescein labelled ocrelizumab and rituximab in eight donors (four FF, four VV).

Results: While the polymorphism did not affect B cell kinetics under standard dosing, interval extension increased early B cell repopulation in F carriers (odds ratio 2.17; p equals 0.04), and no effects were observed on disease activity or progression outcomes; per month, the odds were 3.08 in F carriers (p equals 0.00026) and 1.42 in VV (p equals 0.195). Ocrelizumab binding to NK cells was lower in FF than VV (p less than 0.001), with the same pattern for rituximab (positive control). No genotype effect was seen in monocytes or B cells, supporting a CD16 specific effect.

Conclusion: FCGR3A V158F identifies patients with earlier B cell repopulation when ocrelizumab intervals are extended, consistent with genotype dependent ocrelizumab binding on NK cells.

Disclosure: Nothing to disclose.

EPO-0784 | Switching natalizumab to cladribine: A monocentric observational trial in multiple sclerosis

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Background and aims: To test the feasibility of transitioning patients from Natalizumab to Cladribine tablets, an open-label, single-arm, one year, phase IV, research study has been performed to obtain data on safety and efficacy in Multiple Sclerosis (MS).

Methods: patients with Relapsing Remitting (RR) or Secondary Progressive MS were included. All patients were discontinued because of JCV high positivity at both STRATIFY JCV™ and IMMUNOWELL™ Anti-JCV assay. Concomitant administration of corticosteroid therapy was administered orally at the dosage of prednisone 50mg/day, tapered over 30 days or Betametasone 4 mg/day intramuscular for two weeks. Cladribine tablets were administered with the first dose given between days 3 to day 7 after last Natalizumab infusion.

Results: Sixty-five patients mean age of 42.0 years (min-max: 23.0–64.0), 80.0% female were included and all completed the study at 1 year. Most patients had RRMS (66.2%). The mean time from diagnosis was 15.0 years (SD 7.22). At baseline, none of the patients reported relapse in the previous 12 months with the mean EDSS of 3.0. All patient tested for JCV Index resulted positive above 1.5 cut-off. Three patients (4.6%) experienced a relapse at days 110, 180 and 240 after the switch. The median EDSS score was 3.0 at both 6 and 12 months. During Natalizumab treatment in the previous year, infections occurred in 30.8% of patients and 10.8% experienced an infection during Cladribine treatment.

Conclusion: Cladribine maintains disease stability and is well tolerated in patients discontinuing Natalizumab. The study provides real-world evidence supporting Cladribine as a successor therapy.

Disclosure: Nothing to disclose.

P. Vermersch¹; S. Krieger²; H. Wiendl³; C. Granziera⁴; Y. Mao-Draayer⁵; G. Cutter⁶; O. Kalbus⁷; I. Staikov⁸; M. Dufek⁹; X. Montalban¹⁰; S. Saubadu¹¹; X. Luo¹²; S. Patel¹²; B. Djukic¹³; P. Truffinet¹¹; E. Wallstroem¹³; G. Giovannoni¹⁴; on behalf of Frexalimab Phase 2 Trial Group

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Background and aims: Frexalimab is a second-generation anti-CD40L monoclonal antibody that inhibits the costimulatory CD40/CD40L pathway and may recalibrate immune networks involving adaptive and innate immunity. During the double-blind-period of the phase-2 trial (NCT04879628) in participants with relapsing multiple sclerosis (pwRMS), frexalimab-1200mg/intravenous (IV) reduced new gadolinium-enhancing (Gd+) T1-lesions by 89% vs placebo and was well-tolerated at Week (W) 12. Here, we report W144 (3-year) results of the open-label-extension (OLE).

Methods: 129 participants were randomised (4:4:1:1) to frexalimab-1200 mg/IV every-4-weeks (q4w) or frexalimab-300 mg/subcutaneous (SC)-q2w or matching placebo. After W12,

placebo recipients switched to frexalimab and 125 participants entered the OLE. All SC participants switched to 1800mg-q4w for pharmacokinetic comparability with 1200mg/IV-q4w. By W112, all SC participants switched to 1800mg-q4w, with W144 imaging data available for 55/57 participants. Key endpoints included efficacy (Gd+ T1-lesions, new/enlarging T2-lesions, annualised relapse rate [ARR]) and safety.

Results: 100 participants (78%) remained on treatment at the W144 cut-off. The total number of Gd+ T1-lesions (mean [SD]) remained low in all treatment groups (IV: 0.1 [0.3], placebo»IV: 0.3 [1.0]; SC: 0.0 [0.0], placebo»SC: 0.1 [0.3]). New/enlarging T2-lesions monthly count also remained low through W144. Further, ARR (baseline–W144) was low in the frexalimab-1200mg/IV arm; 86% of participants were relapse-free. Frexalimab was well-tolerated through W144, with stable lymphocyte counts, immunoglobulin levels and no new safety signals.

Conclusion: Frexalimab continues to exert sustained reduction in disease activity with favourable safety in pwRMS over 3 years, supporting its further development in phase-3 trials for RMS (FREXALT) and non-relapsing secondary progressive MS (FREVIVA) as a potential high-efficacy, non-lymphocyte-depleting therapy.

Disclosure: P.V: Janssen, Biogen, Sanofi, Novartis (NVS), Teva, Merck, Roche, Imcyse, AB Science, BMS-Celgene, Ad Scientiam. C.G: Actelion, NVS, Sanofi, GeNeuro, Hoffmann La Roche, Siemens, Biogen, Teva, Merck, Janssen. Y.M-D: Acorda, Bayer, Biogen, BMS/Celgene, Chugai, EMD Serono, Genentech-Roche, Horizon/Amgen, Janssen, NVS, Sanofi, Teva), NIAID Autoimmune Center of Excellence, NIH NINDS, PCORI. G.C: Applied Therapeutics, AI therapeutics, AMO Pharma, AstraZeneca, Avexis, Biolinerx, Brainstorm Cell Therapeutics, BMS/Celgene, CSL Behring, Galmed, Green Valley Pharma, Horizon, Immunic, Karuna Therapeutics, Mapi, Merck, Mitsubishi Tanabe Pharma Holdings, Opko Biologics, Prothena Biosciences, NVS, Regeneron, Sanofi, Reata, Teva, NHLBI, UT Southwestern, UPenn, Visioneering Technologies, Alexion, Antisense Therapeutics, Biogen, Clinical Trial Solutions LLC, Entelexo Biotherapeutics, Genentech, GW Pharma, Immunic, Immunosis, Klein-Buendel, Merck/Serono, Perception Neurosciences, Protalix Biotherapeutics, Regeneron, Roche, SAB Biotherapeutics, University of Alabama, Pythagoras. O.K: Sanofi, Roche, Genentech, Merck, NVS, GeNeuro, BMS/Celgene, Mapi, VielaBio, Teva. I.S: Sanofi, Ewopharma-Biogen, Shire, Gedeon-Richter, Teva, Boehringer Ingelheim, Pfizer, Bayer, F. Roche, Mylan, Polpharma, Penumbra, Adapt, Merck, Gerot Lannach, Medochemie, NVS, Viatris, Nobel Pharma. M.D: Sanofi. X.M: Abbvie, Actelion, Alexion, AstraZeneca, Autolus, Bial PD, Biogen, Bristol-Myers Squibb/Celgene, EMD Serono, Genzyme, Hoffmann-La Roche, Immunic Therapeutics, Indivi, Janssen Pharmaceuticals, Juvisé Pharmaceutical, Lilly, MedDay, Medscape, Merck, Merz Therapeutics, Mylan-Viatris, Nervgen, Neuraxpharm, NVS, PeerVoice, Rewind Therapeutics, Samsung-Biosys, Sandoz, Sanofi-Genzyme, Teva Pharmaceutical, TG Therapeutics, Zenas Biopharma, Excemed, ECTRIMS, MSIF, and NMSS or any of their affiliates. S.K: Consulting: Biogen, Cycle, EMD Serono, Genentech, MedRX, NVS and TG Therapeutics, and non-promotional speaking with Biogen, EMD Serono, Genentech, and TG. Grant and research support from Biogen, BMS, NVS and Sanofi. H.W: Biogen, Genzyme, Merck Serono, NVS, Roche Pharma AG, Sanofi, UCB, Alexion, Biologix, Cognomed, F. Hoffmann-La Roche

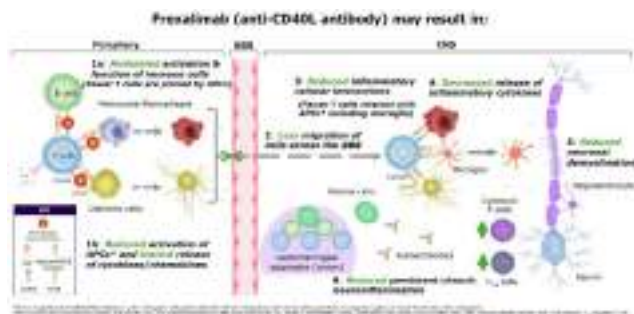


FIGURE 1 Proposed mechanism of action of frexalimab (anti-CD40L antibody) in multiple sclerosis.

Ltd, Gemeinnützige Hertie-Stiftung, Genzyme, Teva, WebMD Global, Actelion, Argenx, Bristol Myers Squibb, EMD Serono, Idorsia, IGES, Immunic, Immunovant, Janssen, Johnson & Johnson, Sanofi, the Swiss Multiple Sclerosis Society, German Ministry for Education and Research (BMBF), Deutsche Forschungsgemeinschaft (DFG), Else Kroner Fresenius Foundation, Fresenius Foundation, the European Union, Hertie Foundation, NRW Ministry of Education and Research, Interdisciplinary Center for Clinical Studies (IZKF) Muenster, GlaxoSmithKline. S.S, X.L, B.S, B.D, P.T, and E.W: Sanofi Employee. G.G: AbbVie, Actelion, Atara Bio, Biogen, Canbex, Celgene, EMD Serono, Japanese Tobacco, Sanofi, Genentech, GSK, GW Pharma, Merck, NVS, Roche, Synthon BV, Teva.

EPO-0786 | Efficacy and safety of ofatumumab in clinical practice: A multicenter study

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Background and aims: The main goal was to analyze the efficacy and safety in MS patients treated with ofatumumab.
Methods: Retrospective, multicentre cohort study including patients treated with ofatumumab from the EMCAM cohort (Multiple Sclerosis Study Group of Madrid). Demographic and clinical characteristics were collected, and effectiveness and safety outcomes were evaluated during the first two years of treatment.
Results: A total of 473 patients were included. The clinical and demographic characteristics are shown in Table 1. 20% of patients have been undergoing treatment for more than a year, and 13% for more than two years. 19.4% of patients switched to ofatumumab due to relapse, 31% due to radiological activity and 13.5% for both reasons. 36.1% switched for other reasons such as safety concerns or adverse events. Two pregnancies have been reported, one full-term and one that ended in miscarriage. 4.8% Patients have discontinued treatment. After one year of treatment with ofatumumab, the ARR decreased significantly to 0.02 and gadolinium-enhancing lesions decreased to 0.04.

Clinical and demographic characteristics of the study	N= 473
Demographic characteristics	
Mean age at inclusion (range)	40.0 (18-72)
Sex (percentage)	100% M
Clinical features	
Baseline EDSS	2.2 (0-8)
Previous relapse	14%
Baseline annualized relapse rate (ARR)	0.28
Baseline gadolinium-enhancing lesions	0.36
Patients with previous pregnancy	4.8%
Patients with relapse prior to treatment	19.4%
Patients previously treated with high or very high efficacy modifying therapies	11.8%

TABLE 1 Clinical and demographic characteristics.

EDSS remained stable at one year 2.2 (0–8). Adverse events were reported in 46% of patients after the first dose, and all were mild. One patient experienced a pulmonary thromboembolism shortly after treatment initiation; this event was deemed unrelated to therapy and did not require discontinuation. One patient suffered an ischemic stroke secondary to vertebral artery dissection three months after starting ofatumumab and has remained on treatment to date.
Conclusion: Ofatumumab was well-tolerated, with no moderate or severe adverse events. two years Significant reductions in ARR and Gd+ lesions were observed at 1 and 2 years supporting its effectiveness and safety in relapsing multiple sclerosis.
Disclosure: Nothing to disclose.

EPO-0787 | Effectiveness and safety of ponesimod in relapsing multiple sclerosis: Results from an Italian multicenter real-world observational study

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Background and aims: In 2021, the phase III OPTIMUM trial demonstrated the efficacy of Ponesimod (PON) - a sphingosine-1-phosphate receptor modulator - over teriflunomide in treating Relapsing Multiple Sclerosis (RMS). However, real-world evidence on its effectiveness and safety is still limited.

Methods: We conducted a retrospective, multicenter observational study across 12 Italian MS centers, including adult patients with RMS treated with PON. Effectiveness was evaluated in patients with at least 12 months of follow-up (FU), assessing relapse rate, MRI activity, and disability progression. Safety and tolerability were investigated by reviewing patients' clinical records, focusing on adverse events (AEs), lymphocyte counts and treatment discontinuation.

Results: We included 106 patients treated with PON. Twenty-two patients did not reach the minimum 12-month FU period: 16 for a shorter FU, 6 for treatment discontinuation (4 for AEs and 2 for breakthrough disease activity). Eighty-four (79.2%) patients completed ≥ 12 months follow-up, and 40 (37.7%) reached 24 months. During the first 12-months of FU, disease activity/PIRA occurred in 5 patients (5.8%), while 81 (94.2%) achieved NEDA-3 status. Lymphopenia developed within the first 3-months in most (90%) patients: 23% grade 1, 42% grade 2, 25% grade 3, 0% grade 4. No severe opportunistic infections were observed. Over the entire FU period, treatment discontinuation occurred in 10 patients (9.4%), most commonly due to AEs ($n=7$), with no unexpected safety signals.

Conclusion: In this Italian real-world study, PON showed high effectiveness in controlling clinical and radiological disease activity, with a favourable safety and tolerability profile.

Disclosure: A.d.A. received speaker's honoraria and/or compensation for consulting service and/or travel grants from Bristol Myers Squibb, Janssen, Juvisè, and Novartis. F.P. reports participation in conferences, advisory boards, speaking fees, and research grants with Alexion, Almirall, Amgen, Biogen, Bristol Myers Squibb, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Reload, FISM, the Italian Ministry of Universities and Research, and the Italian Ministry of Health. A.A. reports no disclosures. C.C. received sponsorship for conferences and speaking fees from Alexion, Amgen, Biogen, Novartis, and Roche. G.A.M. served on advisory boards for Biogen Idec, Genzyme, Merck Serono, Novartis, and Roche; received honoraria from Almirall, Amgen, Alexion, Biogen Idec, Merck Serono, Neuraxpharm, Novartis, Sanofi-Genzyme, Roche, Juvisè, and BMS; and was principal investigator in clinical trials for Merck Serono, Novartis, Roche, Sanofi-Genzyme, and BMS. C.G. received advisory board fees, speaking honoraria, and travel grants from Biogen, Merck Serono, Sanofi-Genzyme, Roche, Teva, Novartis, Bristol Myers Squibb, Juvisè, Almirall, and Horizon. P.A. received honoraria, advisory board fees, and/or travel support from Alexion, Almirall, Amgen, Biogen, BMS-Celgene, Janssen, Juvisè, Lundbeck, Merck, Neuraxpharm, Novartis, Roche, Sanofi-Genzyme, and Viatrix. C.G. served on scientific advisory boards and received travel support from Biogen, Roche, Novartis, Almirall,

Sanofi-Genzyme, Merck Serono, Janssen, Alexion, Juvisè, and Neuraxpharm. A.C. received research grants from ECTRIMS-MAGNIMS and Almirall; travel support from Novartis, Janssen, Roche, and Merck; and speaking honoraria from Merck, BMS, Biogen, Novartis, Roche, and Almirall. V.B.M. received research grants from the Italian Multiple Sclerosis Federation and Roche, and honoraria from Almirall, Biogen, BMS Celgene, Janssen, Merck, Novartis, Roche, Sanofi-Genzyme, and Viatrix. E.C. and J.F. served on scientific advisory boards and as speakers or consultants for Alexion, Amgen, Biogen, Bristol, Janssen, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, and Teva. E.S. received compensation from Almirall, Biogen, Sanofi, Roche, Alexion, Amgen, Novartis, and Teva for travel and advisory board activities. A.G. received honoraria from Biogen, Merck Serono, Mylan, Novartis, Roche, Sanofi-Genzyme, and Teva for consulting, speaking, and/or travel support. V.T. received honoraria, research funding, and travel grants from Alexion, Viatrix, Amgen, Merck, Sanofi, Roche, Novartis, Janssen, Bristol Myers Squibb, Horizon, and Biogen. R.D. received honoraria from Merck Serono, Roche, and Novartis, and travel funding from Almirall, Biogen, Novartis, Sanofi-Genzyme, Roche, and Merck Serono. A.B. received honoraria for consulting and/or speaking from Biogen, Roche, Merck, Celgene, and Genzyme. M.R., A.Z., P.D.F., M.B., G.S., R.M., and F.M. declare no conflicts of interest.

EPO-0788 | Long-term efficacy and safety of ponesimod in females and males: Post-hoc analysis of the ponesimod long-term extension trial

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Background and aims: Potential sex-related differences in response to disease-modifying therapies in relapsing-remitting multiple sclerosis (RRMS) are not well characterized. We assessed long-term efficacy and safety of ponesimod in females versus males in the OPTIMUM trial and its long-term extension (LTE).

Methods: After 108 weeks of double-blind treatment with ponesimod 20 mg (P) or teriflunomide 14 mg (T) in the core OPTIMUM study, participants were offered ponesimod 20 mg in the OLE for up to 5 years. Outcomes were analyzed by sex subgroups.

Results: 735 females and 398 males were included. Baseline characteristics were comparable. In the core study, mean annualized relapse rate (ARR) was 0.21 (95% CI 0.18–0.26) vs 0.31 (95% CI 0.26–0.37) in females and 0.23 (95% CI 0.18–0.29) vs 0.31 (95% CI 0.25–0.38) in males for P vs T, respectively. ARR remained low in the extension period (0.11 females; 0.12 males) and the 24-week Confirmed Disability Accumulation at Week 360 was similar for female and male patients (22.0%, 21.2%) in patients continuously treated with Ponesimod. Safety patterns across the combined (core + OLE) period were broadly similar between sexes and consistent with previously reported OPTIMUM findings. Sex-related differences were observed, with urinary tract infection more common in females (14% vs <5% males), while alanine aminotransferase elevation occurred

more often in males (32% vs 22%). Headache, fatigue and nausea were more frequent in females.

Conclusion: Ponesimod demonstrated sustained long-term efficacy and safety with comparable outcomes in both sexes. These results support consistent use of ponesimod across both sub-populations.

Disclosure: K.DK has nothing to disclose K.H. has received speaker honoraria and research support from Bayer, Biogen, Merck, Novartis, Sanofi-Genzyme, Roche and Teva, has received support for congress participation from Bayer, Biogen, Merck, Roche, Sanofi Genzyme and Teva, and has served on scientific advisory boards for Bayer, Biogen, Sanofi, Teva, Roche, Novartis and Merck M.H, S.G, A.A and A.S are employees of Juvise Pharmaceuticals.

EPO-0789 | **Efficacy and safety of ponesimod in patients aged 50years or older in OPTIMUM and its long-term extension**

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Background and aims: OPTIMUM was a 2-year phase 3 trial comparing ponesimod with teriflunomide in relapsing multiple sclerosis. In the 5years extension period, all participants received ponesimod 20 mg. This post-hoc analysis evaluated the efficacy and safety of ponesimod in patients aged 50years or older.

Methods: This post-hoc analysis included participants aged 50years or older at core study entry enrolled in OPTIMUM. Clinical efficacy outcomes include Annualized relapse rate (ARR) and disability accumulation. Imaging efficacy was assessed using cumulative combined unique active lesions (CUAL), defined as T1 gadolinium-enhancing lesions plus new or enlarging T2 lesions. Safety assessments included adverse events (AE).

Results: 68 patients were aged 50years or older (34 ponesimod and 34 teriflunomide in the core study). During the core period, ARR was numerically lower with ponesimod (0.11 vs 0.23) compared with teriflunomide [rate ratio 0.47 (95% CI 0.16 to 1.35)] (Table 1). 96.4% of ponesimod patients remained CUAL free

TABLE 1 Annualized relapse rate (ARR) estimates by randomization group and study period in patients aged over 50years; CL, confidence level; RR, rate ratio.

Period	Statistic	Randomization Group	
		Ponesimod 20 mg	Teriflunomide 14 mg
Core	Mean ARR estimate	0.108	0.229
	95% CI	(0.047, 0.252)	(0.122, 0.429)
	Treatment Effect (RR)	0.573	
	95% CI	(0.166, 1.864)	
	Number of subjects	34	34
	Number of relapses	7	16
Combined (Core + Extension) Ponesimod 20mg for all patients	Mean ARR estimate	0.094	0.130
	95% CI	(0.048, 0.185)	(0.073, 0.256)
	Treatment Effect (RR)	0.689	
	95% CI	(0.273, 1.728)	
	Number of subjects	34	34
	Number of relapses	15	22
	Total time (years)	597	582

TABLE 2 Adverse events by randomization group during combined period (core + extension) in patients aged over 50years; AE: adverse events.

Subjects with at least one:	Randomization group	
	Ponesimod 20 mg n = 33	Teriflunomide 14 mg n = 34
AE	31 (93.9%)	33 (97.1%)
Severe AE	3 (9.1%)	6 (23.5%)
Drug related AE	19 (57.6%)	29 (85.3%)
AE leading to discontinuation	6 (18.2%)	6 (17.6%)
Serious AE	7 (21.2%)	8 (23.5%)
Fatal AE	0 (0.0%)	1 (2.9%)

at the end of OPTIMUM compared to 86.2% of teriflunomide patients. During the combined period, ARR remained lower in patients receiving continuous ponesimod compared with those switching from teriflunomide (0.09 versus 0.14). At extension week 240 visit, none of the patients treated continuously with ponesimod presented CUALs. 9.1% of patients treated continuously with ponesimod during the combined period experienced severe AE and no fatal AE were reported. (Table 2).

Conclusion: In this post-hoc analysis of patients aged 50years or older, ponesimod demonstrated sustained clinical and imaging efficacy with a favorable safety profile. These findings require confirmation in larger real-world cohorts.

Disclosure: P.V. received honorarium, contributions to meetings from Biogen, Sanofi-Genzyme, Novartis, Merck, Roche, AB Science, Janssen, Ad Scientiam and Juvise Pharmaceuticals. Research supports from Novartis, Sanofi-Genzyme and Merck. M.H, S.G, A.A and A.S are employees of Juvise Pharmaceuticals.

EPO-0790 | **Long-term treatment effects of frexalimab on NFL, CXCL13, and brain volume loss in relapsing multiple sclerosis**

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Background and aims: Frexalimab, a second-generation anti-CD40L monoclonal antibody, inhibits the costimulatory CD40/CD40L pathway, and may recalibrate immune networks involving adaptive and innate immunity. During the double-blind-period of the phase-2 trial (NCT04879628) in participants with relapsing multiple sclerosis (pwRMS), frexalimab-1200mg/intravenous (IV) reduced new gadolinium-enhancing T1-lesions by 89% vs placebo at Week (W) 12, with 24% and 21% reductions in plasma neurofilament light chain (NfL) and chemokine (C-X-C motif) ligand-13 (CXCL13) levels, respectively. Here, we report W144 (3-year) results of NfL, CXCL13, and brain volume loss (BVL) during the open-label-extension (OLE).

Methods: 129 pwRMS were randomised (4:4:1:1) to frexalimab-1200 mg/IV every-4-weeks (q4w), frexalimab-300 mg/subcutaneous (SC)-q2w, or matching placebo. After W12, placebo

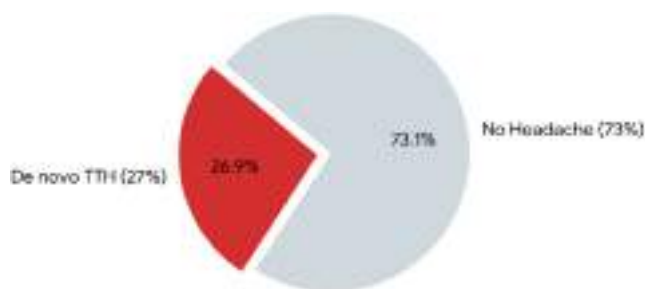


FIGURE 1 Incidence of de novo tension-type headache ($n=260$ participants).

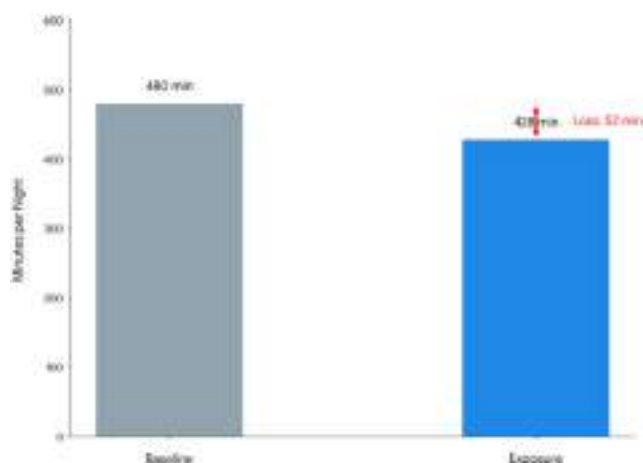


FIGURE 2 Average reduction in sleep duration (study sample: $n=260$).

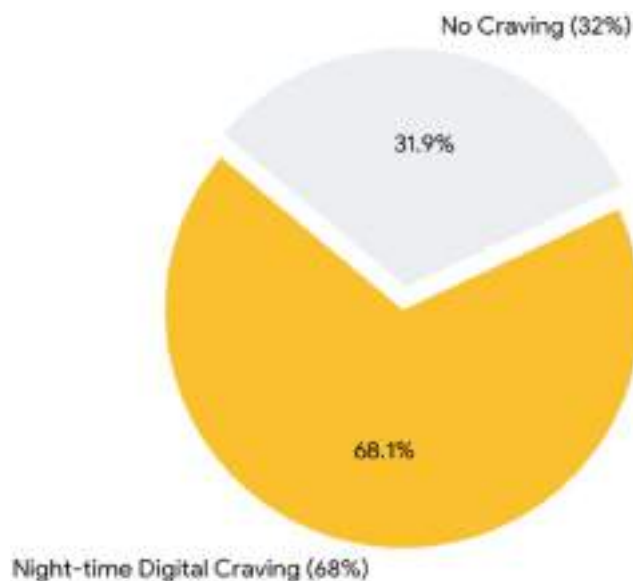


FIGURE 3 The prevalence of night time digital cravings (68 percent) as a behavioral marker identified in sleep architecture disorder participants.

insomnia. Participants participated in two hours of evening video-based short-form for 60 days. Data were collected through headache diaries and sleep records. Statistical analysis has been carried out with SPSS 26.0.

Results: 27 percent ($n=70$) developed de novo tension headache (TTH) after 60 days ($p<0.01$). The mean total amount of sleep has decreased by 52 minutes a night. The sleep latency

ranged from 15 to 60 minutes ($p<0.001$). A specific behavioural marker, night-time digital cravings (compulsive use of a smartphone when waking up in the night) was identified in 68 percent of sleep-deprivation patients. A comparative analysis showed that the effect of digital stimulation on sleep latency was greater than that of caffeine and nicotine.

Conclusion: Results Regular consumption of short-form video content is a self-reinforcing trigger of TTH and severe sleep architecture disorder. These findings support the inclusion of digital silence protocols (at least 60 minutes before bedtime) in the clinical guidelines for the management of headache and insomnia. The night time digital cravings serve as a key indicator of the behavioural addiction of this population.

Disclosure: Nothing to disclose.

EPO-0793 | Psychiatric and affective: Behavioural profile in patients with isolated REM sleep behaviour disorder

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Background and aims: Isolated REM sleep behaviour disorder (iRBD) is considered a prodromal stage of overt synucleinopathies. Increasing interest has focused on identifying biomarkers of iRBD and predictors of phenoconversion. Psychiatric comorbidities in this population remain poorly investigated. This study aimed to describe the prevalence of psychiatric comorbidities in isolated iRBD and to evaluate differences in clinical features.

Methods: Eighty-nine patients with polysomnography-confirmed isolated RBD were enrolled. Clinical history was collected; patients underwent neuropsychological testing, cardiovascular reflex tests and olfactory function assessment. Affective-behavioural features were assessed using Beck Depression Inventory-II (BDI-II), State-Trait Anxiety Inventory (STAI-S, STAI-T), Apathy Evaluation Scale (AES) and Neuropsychiatric Inventory (NPI).

Results: Among 89 patients (66 males, 23 females), 29 (32.6%) had a psychiatric diagnosis, mainly depressive and anxiety disorder; in 31% psychiatric diagnosis preceded iRBD. In the whole cohort, depressive symptoms were present in 39.3% and anxiety symptoms in 14.6%; 18.3% of patients without psychiatric diagnosis reported at least one psychiatric symptom. Patients with psychiatric comorbidity showed higher affective symptom burden for depression, anxiety and apathy (all except AES, $p=0.061$). NPI data showed significant differences in anxiety and irritability domains ($p<0.05$). No differences were observed in hyposmia, neurogenic orthostatic hypotension or mild cognitive impairment. Seventeen patients converted to a synucleinopathy, 31.1% of patients with psychiatric diagnosis vs 13.3% without ($p<0.05$).

Conclusion: Psychiatric comorbidities are common in iRBD, and psychiatric symptoms may occur even in the absence of a formal diagnosis. The presence of a psychiatric diagnosis seems associated with phenoconversion, but larger studies are needed to further characterize this clinical phenotype.

Disclosure: Nothing to disclose.

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Background and aims: REM sleep is preferentially expressed under thermoneutral conditions, likely due to reduced thermoregulatory demands. Recent evidence suggests that skin temperature (T_{skin}) and hypothalamic circuits critically modulate REM sleep. Here, we investigated how T_{skin} and melanin-concentrating hormone (MCH) neurons regulate REM sleep and cataplexy in a mouse model of narcolepsy, considering sex, thermal dynamics, and afferent thermoregulatory inputs.

Methods: Using hypocretin-knockout mice, we selectively manipulated T_{skin} during the active phase while monitoring sleep–wake states and cataplexy. To assess causality, we optogenetically inhibited or activated MCH neurons in MCH-Cre; Hcrt-KO mice. Fiber photometry was used to assess MCH neuronal activity during thermal manipulation. Monosynaptic rabies tracing combined with cFos mapping was employed to identify thermoregulatory afferent inputs to MCH neurons.

Results: Thermoneutral skin warming robustly increased REM sleep and reduced cataplexy, whereas skin cooling produced opposite effects; these dissociating effects were observed in both sexes. Non-state-specific MCH inhibition reduced REM sleep and increased cataplexy, while state-specific inhibition during cataplexy prolonged episode duration. Thermoneutral warming overrode the pro-cataplectic effect of MCH inhibition, suggesting parallel temperature-dependent mechanisms partially independent of MCH signaling. Conversely, optogenetic activation of MCH neurons increased REM sleep and decreased cataplexy. Monosynaptic rabies tracing combined with cFos mapping identified thermoregulatory inputs to MCH neurons, with the median preoptic nucleus (MnPO) showing the strongest warm-activated connectivity.

Conclusion: These findings identify skin temperature as a key physiological variable that dissociates REM sleep and cataplexy and highlight MnPO–MCH circuits as a critical interface between thermoregulation and behavioral state control.

Disclosure: Nothing to disclose.

EPO-0795 | The burden of insomnia and excessive daytime sleepiness: Switzerland's pilot study preliminary results

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Background and aims: Excessive daytime sleepiness (EDS) and insomnia (IN) are common sleep–wake complaints with substantial personal and societal burden, yet they are often underrecognized in primary care. This pilot study evaluated the feasibility of 12-month recruitment and follow-up and estimated the prevalence and correlates of EDS/IN with health-related quality of life (HRQoL), health status, and economic burden using standardized definitions and validated tools.

Methods: Adult patients were recruited from five general practices and five community pharmacies in Switzerland. A two-step approach was used: consecutive identification of self-reported sleep complaints, followed by screening with validated questionnaires. Participants exceeding clinical cutoffs (ESS >10 and/or ISI >7) were enrolled. Baseline measures included PSQI, HRQoL (SF-12, EQ-5D-5L), and a validated cost-of-illness questionnaire. Follow-up lasted 12 months.

Results: Of 821 pre-screened patients, 757 had complete data. Overall, 38% reported EDS/IN (IN 32%, EDS 19%, both 13%), and 82% had symptoms >3 months. Self-medication was reported by 22% and prescribed therapy by 28% (benzodiazepines, Z-drugs, antidepressants). Among 92 screened for follow-up, 15% did not meet questionnaire cutoffs despite subjective complaints. HRQoL was lowest in those with both EDS and IN, with mental domains more affected than physical. Health-related costs were reported by 74% (median CHF 5340). After 12 months, symptoms largely persisted, with only modest improvements in PSQI and ISI.

Conclusion: This study demonstrates feasibility and highlights the chronicity, clinical impact, and economic burden of EDS/IN in primary care, underscoring the need for improved guideline-based management.

Disclosure: Funded by the European Academy of Neurology.

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Background and aims: Narcolepsy type 1 (NT1) is caused by loss of orexin producing neurons. We report a pooled analysis of data from two randomized, double-blind, phase 3 studies (The First Light: NCT06470828; The Radiant Light: NCT06505031) that evaluated the effect of oreporexton (TAK-861) on NT1 symptoms and their severity.

Methods: Participants were aged 16–70 years, had an established diagnosis of NT1, and were randomized to twice daily oral oreporexton 1 mg (The First Light only), 2 mg, or placebo, ≥ 3 hours apart for 12 weeks. The primary endpoint was change from baseline to week 12 in mean sleep latency on the Maintenance of Wakefulness Test (MWT). Key secondary endpoints included changes from baseline in Epworth Sleepiness Scale (ESS) and Narcolepsy Severity Scale for Clinical Trials scores to week 12, weekly cataplexy rate at week 12, and Patient Global Impression of Change (PGI-C) in overall narcolepsy symptoms at week 12.

Results: Overall, 273 participants were randomized to oreporexton 1 mg/1 mg ($n = 61$), 2 mg/2 mg ($n = 136$), or placebo ($n = 76$). At week 12, the least square mean differences from placebo (95% CI) for change from baseline in MWT mean sleep latencies were 15.90 (12.65, 19.15) minutes (1 mg/1 mg oreporexton) and 19.16

(16.65, 21.68) minutes (2 mg/2 mg; both unadjusted $P < 0.0001$). Results for the other endpoints are shown in Table 1.

Conclusion: In this pooled analysis, oreporexton treatment improved measures of wakefulness, sleepiness, and cataplexy frequency and reduced the severity of narcolepsy symptoms over 12 weeks vs placebo.

Disclosure: This research was funded by Takeda Development Center Americas, Inc, who also provided funding to Envision Catalyst, an Envision Medical Communications agency, a part of Envision Pharma Group, to provide writing assistance under the direction of the authors. Markus Schmidt participated in an advisory board for Takeda. Yves Dauvilliers received funds for seminars, board engagements, and travel to conferences from Avadel, Bioprojet, Idorsia, Jazz Pharmaceuticals, Centessa, Alkermes, Pharmanovia, and Takeda. Rafael Del Rio Villegas received consultancy fees from Alkermes, Bioprojet, and Takeda, and travel funds from Bioprojet, Jazz Pharmaceuticals, and Takeda. Gert J. Lammers has received consulting fees, grants, and travel funds from Alkermes, Bioprojet, Daiichi Sankyo, Eisai, Jazz Pharmaceuticals, and Takeda. Emmanuel Mignot received consulting fees from Alkermes, Jazz Pharmaceuticals, and Takeda; research grant or trials to Stanford from Apple, Avadel, Eisai, Jazz Pharmaceuticals, and Takeda; travel funding from Harmony Biosciences, Paladin Labs, and Takeda; and stock options from Centessa. Giuseppe Plazzi received consultancy fees from Bioprojet, Jazz Pharmaceuticals, Orexia, and Takeda. Alice Cai, Ye-Ting Du, Mark Etherton, and Tina Olsson are employees of Takeda Development Center Americas, Inc, and stockholders in Takeda Pharmaceuticals Company Limited.

EPO-0797 | A hospital-based, retrospective analysis of the characteristics of periodic limb movements in sleep in children

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Background and aims: Periodic limb movements in sleep (PLMS) plays a growing role in screening patients for sleep disorders, as it is commonly associated with other sleep-related diagnoses, deteriorates nighttime sleep quality and sleep architecture. While well-studied in adults, pediatric data remains scarce. We investigated PLMS characteristics in children in our hospital-based retrospective study.

Methods: We reviewed medical records and polysomnography (PSG) of 286 patients (<18 years). 109 (38.1%) children had a PLMS index ≥ 5 /hour. Among these, 22 (20.2%) had PLMS only, and were diagnosed with Periodic Leg Movement Disorder (PLMD) due to associated symptoms. Additionally, 48 (44.0%) had pediatric obstructive sleep apnea (pOSA), and 19 (17.4%) had NREM parasomnias (NP). Overlapping diagnoses were excluded. Controls had normal PSG and no clinical sleep disorders.

Results: Demographics showed 52.8% of controls were boys, compared to 40.9% in PLMD ($p = 0.272$), 72.3% in pOSA and PLMS ($p = 0.030$), and 63.2% in NP and PLMS ($p = 0.093$). Mean age was significantly lower in pOSA+PLMS group (6.6 ± 4.3 years, $p = 0.010$) versus others (11.1 ± 4.7 in controls; 7.3 ± 3.6 in PLMD; 9.6 ± 3.0 in NP+PLMS groups). Regression

TABLE 1 Secondary endpoint data at week 12.*

Endpoint	Oreporexton 1 mg/1 mg	Oreporexton 2 mg/2 mg	Placebo
ESS Total Score; least square mean difference (95% CI) for change from baseline vs placebo	-8.27 (-10.82, -5.73)	-6.71 (-10.82, -2.60)	—
P value	<0.0001	<0.0001	—
Weekly Cataplexy Rate; incidence rate ratio (95% CI) vs placebo	0.28 (0.10, 0.41)	0.32 (0.20, 0.48)	—
P value	<0.0001	<0.0001	—
Narcolepsy Severity Scale for Clinical Trials total score; placebo adjusted least square mean (95% CI) change from baseline	-14.75 (-17.51, -11.99)	-17.40 (-19.57, -15.23)	—
P value	<0.0001	<0.0001	—
Patient Global Impression of Change; number and proportion of participants rating narcolepsy symptoms as much or very much improved	42 (72.7%)	123 (92.9%)	13 (18.8%)

*In this analysis all P values are unadjusted.

TABLE 1 PSG parameters.

Table 1. PSG parameters	Normal PSG	PLMD	pOSA+PLMS	NP+PLMS	p values
PLMS index	9.2±6.2	15.8±12.5	16.1±12.2	14.8±10.1	<0.001
AHI	0.03±0.2	0.2±0.1	13.9±11.6	0.03±0.2	
TST	464.0±40.9	467.7±25.5	464.2±44.9	466.0±28.7	
TST	407.5±74.5	427.6±65.3	436.1±59.2	434.1±56.5	
SL	21.8±24.9	18.0±19.8	16.9±18.8	11.8±12.1	
REMI	164.9±26.7	112.3±49.5	162.8±92.8	119.1±55.0	
SE	83.4±13.6	85.3±11.4	86.3±18.3	87.2±5.4	
W, %	12.7±13.5	11.2±10.2	19.8±18.0	10.2±4.5	
NI, %	4.8±3.0	5.0±2.1	3.9±2.9	5.2±3.4	
NI, %	43.3±13.3	40.2±6.8	42.3±9.7	42.7±6.5	
NI, %	24.8±7.2	28.4±8.5	28.2±18.0	23.4±4.4	
R, %	14.1±6.7	14.8±8.3	14.8±6.8	16.6±5.1	

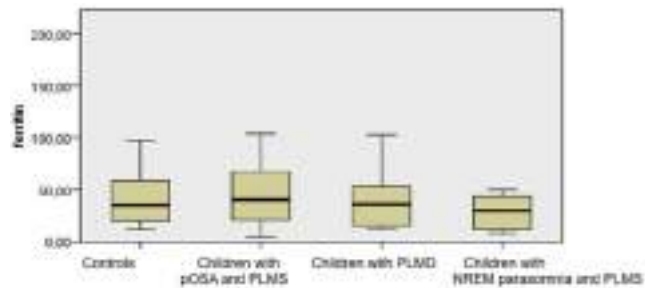


FIGURE 1 Ferritin levels across groups.

analysis adjusted for age and sex. Aside from PLMS and apnea-hypopnea indexes, PSG parameters (latency, efficiency, stages) showed no significant differences between groups. Mean ferritin levels were below 50 µg/L in all groups except pOSA, with the lowest levels in NP+PLMS (26.8 ± 16.1 µg/L, $p=0.059$).

Conclusion: PLMS is quite common in pediatric sleep center populations (~33%), frequently co-existing with pOSA and NREM parasomnias. Low ferritin levels in groups except pOSA emphasize the importance of iron deficiency. Furthermore, a 20% hospital-based PLMD prevalence is striking, suggesting this group is underestimated and warrants larger prospective studies.

Disclosure: Nothing to disclose.

EPO-0798 | Effect of the oral orexin receptor 2 agonist oreporexton (TAK-861) on functional impacts of narcolepsy type 1: Pooled data from two phase 3 studies

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TABLE 1 Least square mean change (95% CI) from baseline to week 12 versus placebo for the 6 FINI domains.*

Domain	Oreporexton 1 mg/1 mg	Oreporexton 2 mg/2 mg
Tiredness	-33.3 (-45.7, -25.3)	-43.7 (-60.4, -26.0)
Cognitive Functioning	-30.4 (-37.7, -23.3)	-34.1 (-50.8, -28.5)
Cataplexy	-18.5 (-24.6, -12.4)	-25.8 (-35.3, -16.3)
Social Activities	-20.4 (-26.2, -14.7)	-33.4 (-49.5, -27.4)
Everyday Activities	-34.5 (-41.1, -27.8)	-43.0 (-60.3, -25.6)
Everyday Responsibilities	-30.4 (-36.1, -24.8)	-36.9 (-53.7, -20.1)

Background and aims: Narcolepsy type 1 (NT1), a central hypersomnolence disorder with deficiency of hypothalamic orexin signaling, affects functioning and quality of life. We conducted a pooled analysis of data from two randomized, double-blind, oreporexton (TAK-861) phase 3 studies (The First Light: NCT06470828; The Radiant Light: NCT06505031) to investigate its effect on functional impacts using the Functional Impacts of Narcolepsy Instrument (FINI).

Methods: Eligible participants were aged 16–70 years and had a confirmed NT1 diagnosis. Participants were randomized to twice daily oral oreporexton 1 mg (The First Light only), 2 mg, or placebo, ≥3 hours apart for 12 weeks. Change from baseline to week 12 in FINI domain scores was a secondary endpoint in both trials. The FINI comprises 6 independent domains scored from 0 (best functioning) to 100 (worst functioning): Tiredness, Cognitive Functioning, Cataplexy, Social Activities, Everyday Activities, and Everyday Responsibilities.

Results: Improvements with oreporexton were observed across all FINI domains. Mean (SD) change from baseline at week 12 with oreporexton (overall) was -44.6 (23.7) for the Tiredness domain, -33.6 (25.0) for Cognitive Functioning, -24.5 (22.6) for Cataplexy, -33.8 (27.1) for Social Activities, -45.9 (23.0) for Everyday Activities, and -40.0 (28.5) for Everyday Responsibilities; corresponding changes with placebo were -5.1 (22.2), -2.4 (24.4), -5.5 (18.5), -2.6 (23.8), -6.6 (21.5), and -7.7 (28.5). Comparisons versus placebo showed improvements with oreporexton across all domains and doses (Table 1).

Conclusion: In this pooled analysis of data from two phase 3 trials, oreporexton showed improvements in narcolepsy-specific functioning versus placebo.

Disclosure: This research was funded by Takeda Development Center Americas, Inc, who also provided funding to Envision Catalyst, an Envision Medical Communications agency, a part of Envision Pharma Group, to provide writing assistance under the direction of the authors. Ramin Khatami received consulting fees, travel support, or board engagement from Bioprojet, Idorsia, Jazz Pharmaceuticals, and Takeda. Emmanuel Mignot received consulting fees from Alkermes, Jazz Pharmaceuticals, and Takeda; research grant or trials to Stanford from Apple, Avadel, Eisai, Jazz Pharmaceuticals, and Takeda; travel funding from Harmony Biosciences, Paladin Labs, and Takeda; and stock options from Centessa. Fabio Pizza participated in an advisory board for Takeda and received support for congress participation and funding from Bioprojet. Giuseppe Plazzi received consultancy fees from Bioprojet, Jazz Pharmaceuticals, Orexia, and Takeda. Stephen Crawford, Mark Etherton, Tina Olsson,

Heather Romero, Yulia Savva, and Baiyun Yao are employees of Takeda Development Center Americas, Inc, and stockholders in Takeda Pharmaceuticals Company Limited. Yves Dauvilliers received funds for seminars, board engagements, and travel to conferences from Avadel, Bioprojet, Idorsia, Jazz Pharmaceuticals, Centessa, Alkermes, Pharmanovia, and Takeda.

EPO-0799 | Differential effects of digital self-monitoring and multimodal training on sleep and psychological outcomes in adults with sleep difficulties

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Background and aims: Sleep difficulties and insomnia are widespread conditions that impair emotional well-being, daily functioning, and overall quality of life. Although digital tools are increasingly used to address sleep difficulties, the specific contribution of individual intervention components remains unclear.

Methods: We conducted a 4-week randomized study in 63 adults with sleep complaints, comparing a digital sleep diary (DSD) to a multimodal app combining self-monitoring, psychological training, and a cognitive multisensory task. Sleep and psychological outcomes were assessed pre- and post-intervention. A subsample completed resting-state fMRI to explore neural mechanisms.

Results: Both groups showed improvements in PSQI, indicating better subjective sleep quality. However, only the multimodal group showed significant ISI reductions and broader gains in depression, anxiety, stress, and attention scores. Neuroimaging revealed shared increases in nucleus accumbens (NAc) connectivity with default mode regions across groups. Unique to the multimodal group were reductions in NAc-insula and NAc-anterior cingulate connectivity, key salience network regions involved in both emotion regulation and attentional processing. Improvements in insomnia severity correlated with psychological improvements only in the multimodal group.

Conclusion: While self-monitoring improved sleep quality, only the multimodal intervention significantly reduced insomnia

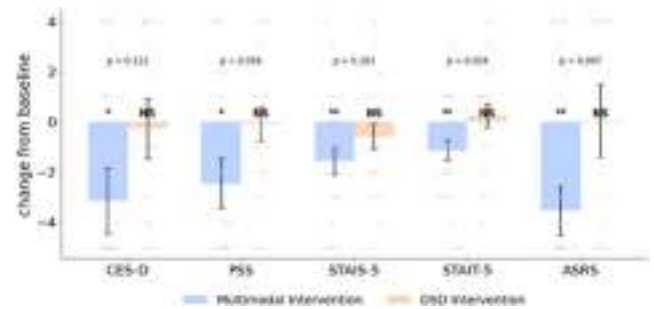


FIGURE 2 Significant improvements in affective symptoms following the Multimodal intervention.

severity and emotional symptoms. These findings highlight the value of incorporating targeted psychological and multisensory tools within digital sleep platforms and suggest that such multimodal interventions may offer a scalable and effective solution for addressing both sleep disturbance and its associated emotional symptoms in real-world settings.

Disclosure: The authors of this abstract are employees or consultants of Remepy Health Ltd, the company who developed the app and the sponsor of the study.

EPO-0800 | A multimodal neurophysiological signature of REM sleep behavior disorder: A pilot study

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Background and aims: REM Sleep Behavior Disorder (RBD) is a strong prodrome of alpha-synucleinopathies. Although brainstem dysfunction explains the loss of REM atonia, the interaction between sleep architecture, cortical activity, and motor excitability remains poorly understood. We aimed to map these multimodal interactions to clarify how cortical source signals from wake EEG relate to sleep macrostructure and daytime motor control.

Methods: Seven patients with RBD (6 males, 72.86 ± 4.01 years) underwent clinical evaluation (MMSE, MoCA, UPDRS-I, UPDRS-III), 5 minutes of resting-state High-Density EEG, full-night Polysomnography (PSG), and Transcranial Magnetic Stimulation (TMS). Cortical current density was reconstructed via eLORETA. To mitigate statistical bias from the limited sample size, we applied a Domain-Specific PCA to extract the main variance factors: EEG-Source Activity, PSG indices, TMS inhibitory metrics (Silent period, contralateral and ipsilateral), and Clinical Outcomes. Interactions were mapped using Graph Theory and Hierarchical Clustering.

Results: Source analysis revealed significant cortical hypoactivity within the Salience and the Default Mode networks

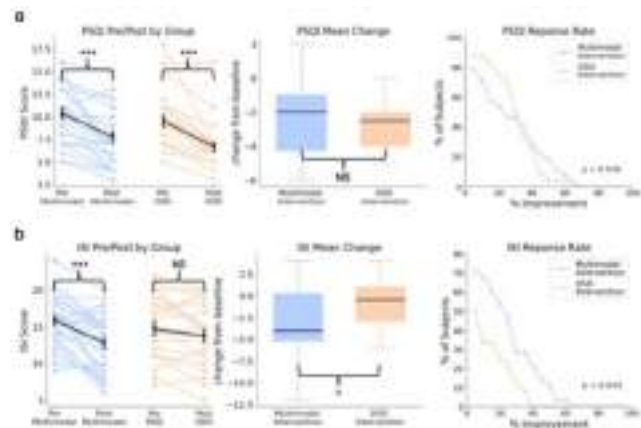


FIGURE 1 Effects of the digital interventions on sleep outcomes across groups.

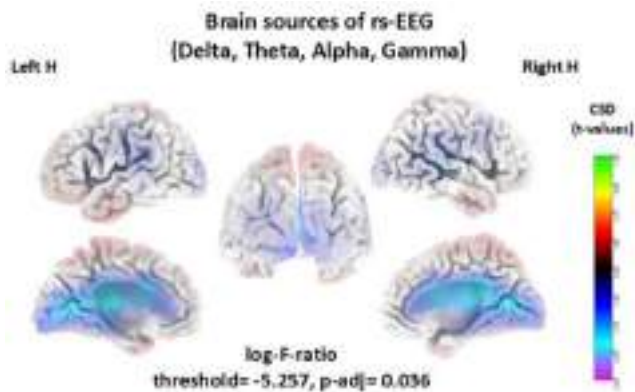


FIGURE 1 eLORETA statistical maps of source activity differences in RBD. Three-dimensional maps showing the log-F ratio distribution for reconstructed sources averaged across frequency bands (Delta, Theta, Alpha, Gamma).

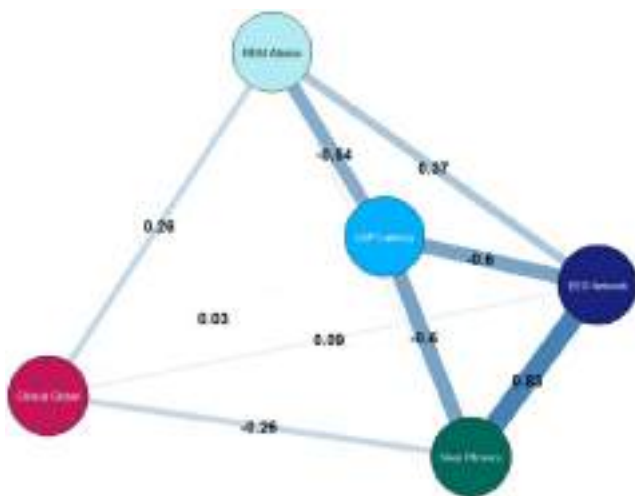


FIGURE 2 Integration of multimodal biomarkers via Graph Theory. Correlation network representing the interactions between the main variance factors extracted through Domain-Specific PCA. The nodes include the EEG-Source Activity (EEG Network), PSG indice.

(Figure 1). The PCA identified an Insular-Source Factor as a central hub. This cortical node showed a strong coupling with Sleep Efficiency ($\rho=0.83$) and a secondary association with inhibition Motor Latencies ($\rho=-0.60$) (Figure 2). In contrast, the network revealed a striking dissociation of both REM atonia and clinical motor symptoms (UPDRS-III), which exhibited the lowest centrality.

Conclusion: RBD may involve a systemic failure of cortical integration between the Salience and Default mode networks, a neurophysiological substrate for both sleep instability and slowed motor inhibitory processing. This multimodal approach provides a preliminary but promising window into the cortical network reorganization underlying RBD pathophysiology.

Disclosure: This research was supported by the following institutional grants: Italian Ministry of University and Research (MUR), BANDO PRIN 2022, project title “Caratteristiche neurofisiologiche multimodali e rischio di fenocconversione nell’iRBD” (grant number: MUR 20228XKKCM_001) to Claudio Liguori, Giuseppe Lanza, and Aurora Palmigiano; Italian Ministry of Health (MinSal), BANDO RF 2021, project title “Translational correlates of multimodal electrophysiological

markers of disease process in REM Sleep Behavior Disorder (RBD) and its progression into neurodegenerative disorders: diagnostic hints and therapeutic implications” (grant number: GR-2021-12372863) to Giuseppe Lanza, Michela Figorlli, and Francesca Ginatempo.

EPO-0801 | Abstract withdrawn

Neuroepidemiology

EPO-0802 | Leveraging a unified electronic health records system through community outreach centres to deliver tele-neurology services in Kenya: A pilot study

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Background and aims: Access to neurologists in Sub-Saharan Africa is significantly below global recommendations; Kenya has ~20 neurologists for 55 million people. While our 2021 study demonstrated tele-neurology consultations (TNCs) were feasible, critical limitations included the inability to use private insurance or access onsite investigations and/or prescriptions. We aimed to address these gaps by leveraging our tertiary hospital’s (TH) network of 55 community outreach centres (COCs)—the country’s largest, with half spread across the capital—all interlinked by a unified electronic health record (EHR) implemented in 2023.

Methods: Prospective patients booked in the TH neurology clinic were offered TNC at their nearest COC. The new integrated workflow permitted insurance processing and direct ordering of investigations/prescriptions via EHR after the TNC. We undertook a post-TNC survey after consenting participants and present our interim results.

Results: We surveyed 30 participants [median (IQR) age 45 (37.0–51.0) years, 73.3% (22/30) female]; the majority of conditions for TNC were epilepsy and migraine [53.3% (16/30), equal weighting]. 90% (27/30) reported “high satisfaction” including for accessing investigations/prescriptions at COCs. Median (IQR) savings in travel, time and overall cost were 17 (11.5–216.0) km, 3 (2.0–48.0) hours and €16.5 (IQR 7–70) per encounter respectively, and total carbon emissions saved were 2.36 tCO_{2e}.



FIGURE 1 Visual Abstract. Summary of the pilot study demonstrating the feasibility, economic impact, and high acceptability of tele-neurology delivered via Community Outreach Centres (COCs) in Kenya.

Conclusion: Our enhanced workflow, utilizing the TH's vast COC network and EHR, proved highly acceptable and successfully overcame previous insurance and investigation barriers. Interim data confirm significant reductions in travel burden, costs, and carbon emissions, supporting the integration of this model into routine specialist care.

Disclosure: Nothing to disclose.

EPO-0803 | The International Hospital Federation Geneva Sustainability Centre Toolkit to improve the sustainability of neurology services

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Background and aims: Climate change is the biggest threat to human health in the 21st Century. As a systemic issue that impacts all aspects of the healthcare sector, a common understanding of key sustainability terms and concepts is essential. Many hospitals and doctors, including neurologists, are interested in reducing their department or hospital's carbon footprint.

Methods: Presentation of the GSC Toolbox to improve the sustainability of healthcare services and practical case studies on how this can be used in clinical services.

Results: Many hospitals and health organisations around the world have now implemented the GSC Toolbox and other GSC tools. After a brief overview of all GSC tools to improve sustainability (sustainability accelerator tool, accreditation and certification, action planning, capacity strengthening), several international case studies of hospitals that have successfully implemented the GSC Toolbox will be presented.

Conclusion: Participants will learn practical tools to reduce their department or hospital's carbon footprint through case studies presented by a practising neurologist and public health physician.

Disclosure: CG is a member of the Steering Committee of the IHF Geneva Sustainability Centre.

EPO-0804 | Effectiveness of mobile stroke (MSUs) in acute ischemic stroke: An updated systematic review and meta-analysis of the five "T-Letter"

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FIGURE 1 Graphical abstract.

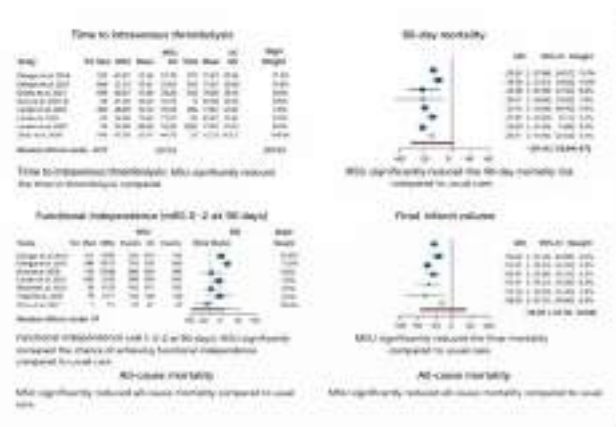


FIGURE 2 Outcome analysis.

Background and aims: Mobile stroke units may accelerate acute ischemic stroke management, but updated evidence comparing their effectiveness and safety with standard emergency medical services (EMS) remains limited, necessitating a contemporary, comprehensive meta-analysis.

Methods: This PRISMA-compliant systematic review (PROSPERO CRD420251273498) searched 5 databases for comparative studies of MSUs versus standard EMS. Random-effects models pooled treatment time, functional, mortality, and safety

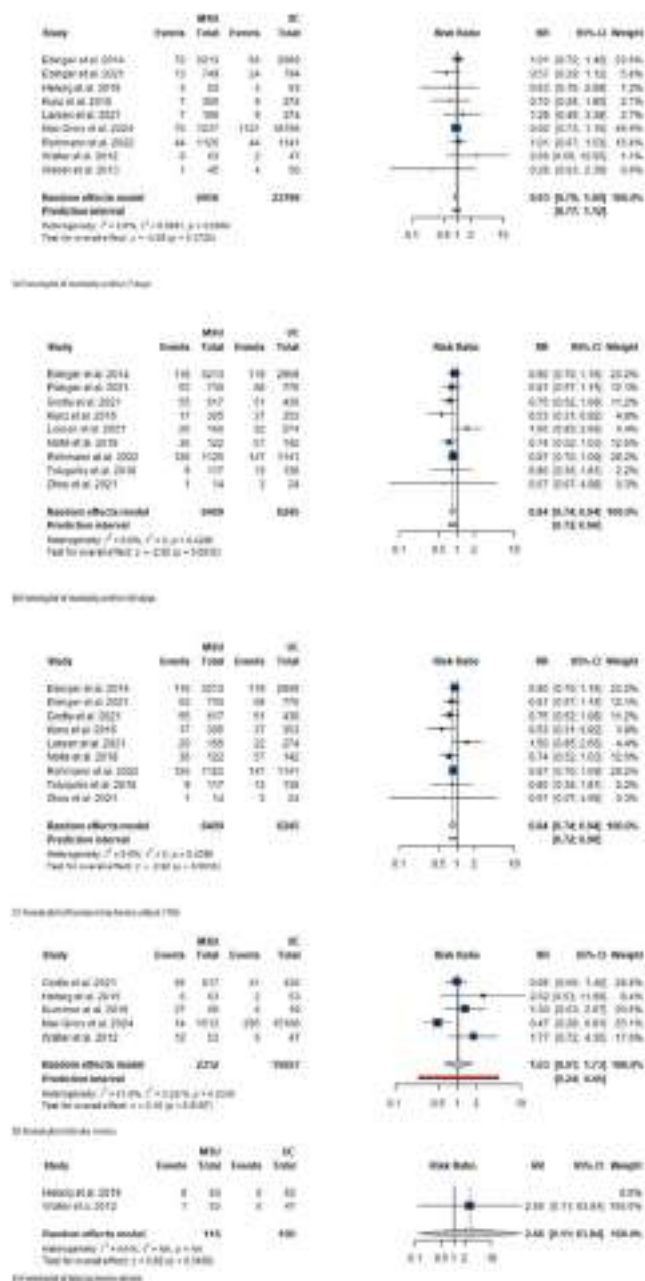


FIGURE 3 Comparison between MSU and ESU.

outcomes, with risk of bias assessed using RoB-2, ROBINS-I, and NOS tools.

Results: Across thirty three eligible studies identified, pooled analyses showed substantial reductions in treatment delays, with alarm-to-IVT/IAC time shortened by 29.1 minutes (95% CI –33.7 to –24.6; $I^2 = 97.6\%$) and time to thrombolysis reduced by 36.0 minutes (95% CI –47.4 to –24.7; $I^2 = 89.9\%$). Functional outcomes favored MSUs care across multiple thresholds: the likelihood of excellent outcome (mRS 0–1) increased by 18% (RR 1.18), good outcome (mRS 0–2) by 13% (RR 1.13), and mRS 0–3 by 10% (RR 1.10). MSUs management increased IV tPA use by 17% (RR 1.17) and markedly improved golden-hour thrombolysis (RR 4.64). While 7-day mortality did not differ (RR 0.93), 90-day mortality was reduced by 16% (RR 0.84, 95% CI 0.74–0.94). Safety outcomes, including intracranial hemorrhage (RR 1.01), seizures (RR 0.99), stroke mimics (RR 1.03), and TIA (RR 0.84),

showed no significant differences, supporting the numerical efficacy and safety profile of MSU-based stroke care.

Conclusion: MSUs care significantly reduced treatment times, increased thrombolysis rates, improved functional outcomes, and lowered 90-day mortality without increasing adverse events, demonstrating superior effectiveness and comparable safety versus standard EMS.

Disclosure: Nothing to disclose.

EPO-0805 | Dementia burden in Europe, 1990–2023: Evidence from the global burden of disease study

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Background and aims: Dementia is a leading cause of disability and mortality in Europe, yet no recent harmonised assessment has described its burden across the European Union (EU-27) and the WHO European Region.

Methods: We used data from the Global Burden of Disease Study 2023 (GBD 2023) to estimate prevalence, mortality, and disability-adjusted life years (DALYs) for Alzheimer’s disease and other dementias from 1990 to 2023. Estimates were produced for the EU-27 and the WHO European Region, by age and sex. Non-fatal outcomes were modelled using DisMod-MR 2.1, and dementia-attributable mortality was estimated using an excess-mortality framework. We also quantified DALYs attributable to six modifiable risk factors.

Results: In 2023, 7.76 million people were living with dementia in the EU-27 and 12.28 million in the WHO European Region, representing ~90% increases since 1990 despite modest declines in age-standardised prevalence. Prevalence was nearly twice as high in women as in men. Dementia rose from the eighth to the third leading cause of death in the EU-27. Dementia accounted for 5.49 million DALYs in the EU-27. An estimated 41% of DALYs in the EU-27 were attributable to modifiable risk factors, particularly ambient particulate matter pollution, high fasting plasma glucose, and high body-mass index, which are disproportionately concentrated in socioeconomically disadvantaged populations.

Conclusion: Despite modest declines in age-standardised rates, the absolute burden of dementia in Europe continues to rise, driven by population ageing. The substantial contribution of modifiable risk factors highlights major opportunities for prevention. Robust, country-specific estimates are essential to guide integrated strategies combining prevention and care planning.

Disclosure: Nothing to disclose.

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Background and aims: Large registries with real-world clinical data are essential for understanding long-term health trajectories. Large language models (LLM) reliably extract binary-coded diagnoses from clinical text (Dennstadt et al, 2024). Little is known about their ability to capture the temporal structure of diagnoses, including repeated instances. Classification of diagnostic trajectories is essential for prognostic modeling, with particular relevance for preventive medicine. **Methods:** This interim analysis is part of the ongoing “Sleep and Longevity” project within the Bern Sleep-Wake Registry (Figure 1). Six ICD-11 comorbidities with 25 subcategories were selected. They were manually scored in 1000 randomly selected

TABLE 1 LLM performance evaluated descriptively against the ground truth for exact matches.

Parameter	Stroke	Diabetes	Cystitis	Emphysema	Hyperemesis	Varicella	IGT	Mean overall
The presence of the diagnosis binary variable (750/1000)								
n	1000	1000	1000	1000	1000	1000	1000	1000
Accuracy, %	87.00%	86.80%	86.40%	86.90%	87.60%	88.80%	86.80%	87.40%
Precision, %	85.00%	86.90%	79.40%	73.20%	86.20%	75.84%	87.80%	80.60%
Recall, %	89.00%	88.24%	79.40%	81.20%	100.00%	74.10%	80.82%	85.82%
F1, %	76.50%	71.43%	73.40%	77.00%	93.32%	76.87%	71.20%	78.90%
Specificity, %	89.14%	96.30%	98.00%	87.92%	87.67%	96.95%	96.17%	92.17%
Exact matches, %	87.00%	86.80%	86.40%	86.90%	87.60%	88.80%	86.80%	87.40%
Cohen's kappa	0.90	0.89	0.85	0.88	0.90	0.82	0.89	0.88
The number of the instances of the diagnosis continuous variable (T-5)								
n	17	17	87	86	13	128	87	87
Exact matches, %	30.60%	17.60%	48.16%	40.60%	15.38%	17.89%	29.57%	29.57%
Mean difference to GT, s	-0.41	0.71	-0.36	0.16	0.65	0.32	0.30	0.30
Cohen's kappa	0.24	0.80	0.12	0.02	0.02	0.32	0.30	0.30
The number of the instances of the diagnosis with dates continuous variable (T-5)								
n	17	17	87	86	13	128	87	87
Exact matches, %	87.79%	70.59%	48.16%	85.69%	84.62%	85.86%	88.86%	80.86%
Mean difference to GT, s	-0.21	0.88	-0.26	0.08	0.18	0.21	-0.88	-0.88
Cohen's kappa	0.82	0.41	0.22	0.39	0.02	0.83	0.39	0.39
Exact date/number variable								
n	70	94	34	28	11	72	37	37
Records with identified dates, %	80.00%	55.36%	35.29%	0.00%	72.73%	77.70%	53.50%	53.50%
Exact matches, %	70.00%	55.36%	32.35%	0.00%	54.55%	65.29%	46.29%	46.29%
Mean difference to GT, days	-0.19	0.80	-0.87	0.00	-0.87	-0.37	-0.87	-0.87
Cohen's kappa for all records	0.67	0.32	0.11	0.00	0.38	0.83	0.39	0.39
Cohen's kappa for all records with identified dates	0.84	0.80	0.30	0.00	0.68	0.78	0.78	0.78

plain-text diagnostic records by two independent medical doctors. Disagreements were resolved by a third medical doctor to define the ground truth. For each diagnosis, scorers recorded its presence, type, and date, allowing up to five instances per diagnosis. LLM-based extraction was performed using the data-element-extractor Python package (v0.2.0) with GPT-OSS-20B. Only basic prompting was applied. LLM performance was evaluated descriptively against the ground truth for exact matches. **Results:** Interrater agreement between scorers was high (Cohen's kappa: median 0.96, IQR [0.79–0.98]). The LLM reliably identified diagnoses but struggled to differentiate repeated instances and their dates, occasionally returning no date (Table 1). For instances with an identified date, agreement with the ground truth was moderate to high. **Conclusion:** This is the first study to extract diagnostic trajectories from clinical text in patients with neurologic disorders using LLM. We will improve the extraction through optimized prompting, constrained outputs, and computational strategies (e.g. use of a larger model). **Disclosure:** Nothing to disclose.

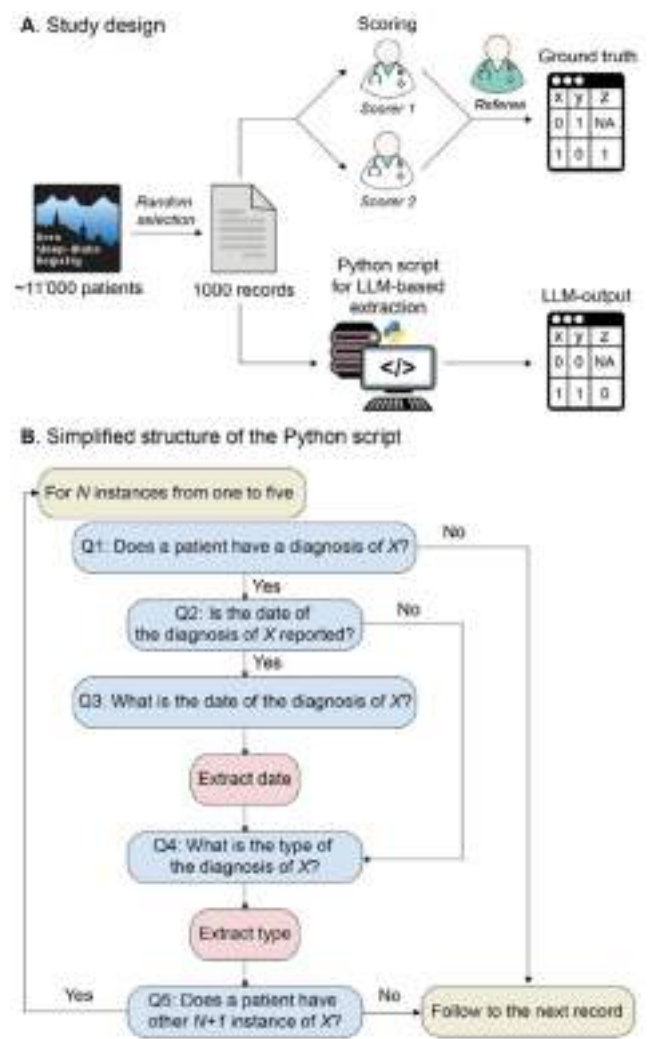


FIGURE 1 (A) Study design. (B) Simplified structure of Python script.

EPO-0807 | Risk and causes of hospitalization in persons with Parkinson's disease or atypical parkinsonism: The ParkLink Bologna cohort

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Background and aims: Parkinson's disease is among the leading neurological causes of global burden in the elderly. Risk of hospitalization in people with Parkinson's disease (PPD) is an essential aspect to consider for improving healthcare strategies.

TABLE 1 Incidence Rate Ratios (IRR) with 95% Confidence Intervals (95% CI) for the significant hospital clinical features, comparing the Parkinson's disease and atypical Parkinsonism cohorts with their respective control cohorts.

	Parkinson's disease IRR (95% CI)	Atypical parkinsonism IRR (95% CI)
All hospital admissions	1.60 (1.45–1.76)	1.61 (1.28–2.02)
Infections	2.35 (1.50–3.66)	2.51 (1.67–3.96)
Dematitis	2.28 (1.51–3.43)	2.38 (1.32–4.32)
Metabolic imbalances	1.86 (1.13–3.06)	6.25 (2.46–15.9)
Unlaid cerebrovascular disease	1.88 (1.36–2.62)	2.08 (0.99–4.01)
Myocarditis	1.79 (1.18–2.68)	0.64 (0.29–2.06)
Thromboembolism	1.27 (0.54–3.06)	3.96 (1.24–12.2)
Disorders of thyroid gland	0.38 (0.12–2.09)	6.42 (1.51–26.91)
Psychiatric events	0.38 (0.04–2.22)	1.19 (1.19–42.76)

We aimed to investigate the risk and causes of hospitalization admission in 2-year observation in PPD or people with atypical parkinsonism (PAPS) compared to a matched general population cohort.

Methods: Prospective cohort of PPD or APS compared to a general population sample, matched for sex, age, residence, and comorbidity index within the ParkLink Bologna record linkage system, an ongoing project started in February 2015 in the Local Health Care Trust of Bologna, Northern Italy. PPD/PAPS were diagnosed and recruited by neurologists during routine outpatient clinical practice. Clinical data were linked to administrative health data. Hospitalization at 2-year after recruitment was reported as Incidence Rate Ratio (IRR) between PPD/PAPS and controls.

Results: 816 PPD, 148 PAPS and 9447 controls were included. A higher risk of hospitalization was observed among PPD (IRR 1.60, 95% CI 1.45–1.76) and PAPS (IRR 1.61, 95% CI 1.28–2.02). The acute leading causes of hospitalisation were infections and metabolic imbalances for both groups, while severe trauma specifically affected PPD and thromboembolism specifically affected PAPS (Table).

Conclusion: Linking administrative data with reliable clinical data allow exploring relevant outcomes of the clinical course of PPD. Over a 2-year period, PPD and PAPS had an approximately 60% higher risk of hospitalization, respectively, mainly due to a doubled risk of infections and metabolic imbalances, compared to a matched general population cohort.

Disclosure: The study is funded by IRCCS ISNB (Bando Ricerca Corrente 2025).

EPO-0808 | 5q-associated spinal muscular atrophy in Austria: Epidemiology and co-morbidities

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Background and aims: Spinal muscular atrophy (SMA) is a genetic motor neuron disease caused by homozygous deletions or other pathogenic variants in the SMN1 gene, resulting in the degeneration of lower motor neurons associated with progressive weakness and muscle wasting. The development and approval of disease-modifying therapies (DMT) in recent years has fundamentally transformed the management, as well as the clinical and epidemiological landscape of patients with SMA.

Methods: In this study, hospitalisation and prescription databases together with the capture-recapture method are utilised to estimate the prevalence of SMA in Austria and to identify co-morbidities associated with SMA. Patients were matched to healthy controls (1:10) by sex, age and district of residence.

Results: We estimated a total population of 236 patients (95% CI 231–241) with SMA in Austria and a corrected prevalence of 2.5/100,000 persons in the year 2023. Patients with SMA showed significantly higher use of several medication classes, particularly therapies related to respiratory and infectious conditions. Healthy controls, by contrast, exhibited higher prescribing rates of cardiovascular medications, including beta-blockers and ACE inhibitors, as well as lipid-lowering agents and muscle relaxants.

Conclusion: Our findings provide a robust nationwide estimate of SMA prevalence in Austria and highlight a distinct pattern of co-morbidities in the era of disease-modifying therapies, supporting informed healthcare planning and long-term management of patients with SMA.

Disclosure: This study was financially supported by Biogen, Novartis and Roche.

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Background and aims: Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disorder characterised by the progressive loss of motor neurons, ultimately leading to death within a few years of disease onset. Although recent studies suggest that the global burden of the disease has increased over past decades, the trajectory of epidemiological metrics in Austria remain unknown.

Methods: Hospital discharge records and riluzole prescription databases were used to identify ALS cases between 2016 and 2023. The capture–recapture method was applied to estimate the incidence and prevalence of ALS in Austria. The relationship between disease prevalence and the ratio of deaths to prevalent cases (death-to-case ratio) in a given year was analysed using Pearson's correlation.

Results: A total of 3344 patients were identified during the study period, of whom 44.3% were female. While the corrected annual incidence remained stable (5.2/100,000 person-years in 2016 and 4.8/100,000 person-years in 2023), the prevalence increased steadily over the same period, rising from 8.6 to 14.5/100,000 individuals. Median survival was 605 days (95% CI 569–638). The death-to-case ratio decreased during the study period, from 0.32 in 2016 to 0.22 in 2023, which correlated negatively with the corresponding prevalence numbers ($r = -0.85$, $p = 0.0019$).

Conclusion: In this study, we report on the epidemiology of ALS in Austria between 2016 and 2023. Notably, while the annual incidence remained stable over the study period, our findings indicate an increase of prevalence, most likely due to reduced mortality rates. Our results underscore the need for national healthcare policies to address the challenges posed by the growing disease burden.

Disclosure: This study was not financially supported.

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Background and aims: Hungary has one of Europe's highest obesity rates (72.5% of adults). Excess weight is a major risk factor for stroke, contributing to substantial health burden (~€1 billion annual stroke care costs). We aimed to examine BMI

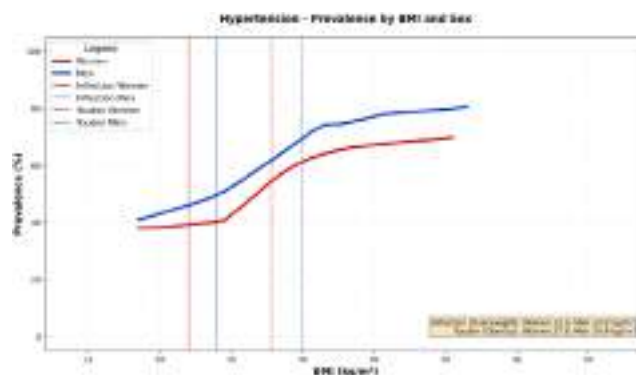


FIGURE 1 Sex-specific prevalence of hypertension across BMI.

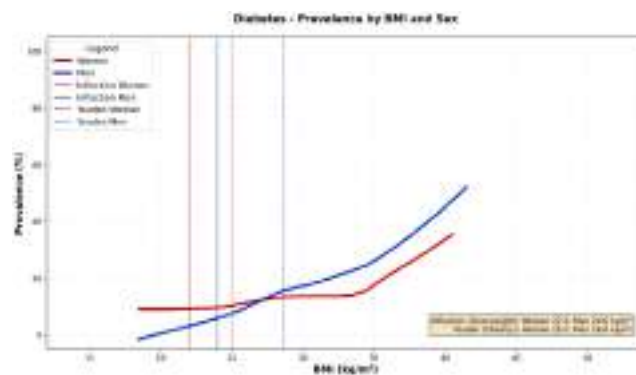


FIGURE 2 BMI-related prevalence of diabetes by sex.

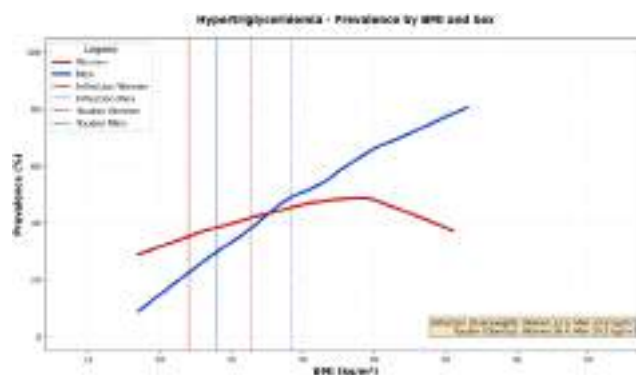


FIGURE 3 Prevalence of hypertriglyceridemia by BMI and sex.

associations with cardiometabolic risk factors in Hungarian adults and identify optimal thresholds for stroke prevention.

Methods: Cross-sectional study of 868 Hungarian adults assessing BMI associations with hypertension, type 2 diabetes, dyslipidemia, and atherosclerosis. BMI was calculated from measured height and weight; cardiometabolic conditions determined via clinical examination and laboratory-based point-of-care tests. Sex-stratified logistic regression identified BMI cut-offs for elevated cardiometabolic risk, compared to WHO standards.

Results: Higher BMI significantly associated with hypertension, diabetes, hypertriglyceridemia, and atherosclerosis ($p < 0.05$), but not hypercholesterolemia. Hungarian adults exhibited lower BMI thresholds than international standards. Women showed elevated risk at BMI ~21.8–22.3 kg/m² (normal range) and further elevation at ~25–27.8 kg/m². Men demonstrated risk inflection points at BMI ~23.8–24.3 and ~28.8–29.9 kg/m².

Conclusion: BMI thresholds for cardiometabolic risk are lower in Hungarians than WHO definitions. Hungary-specific cut-offs improve identification of high-risk individuals and enable timely intervention to prevent stroke, substantially reducing incidence and burden.

Disclosure: Nothing to disclose.

EPO-0811 | A nationwide analysis: Memantine in the treatment of dementia in Hungary

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Background and aims: Dementia is a leading cause of dependency and disability, placing a substantial burden on patients, caregivers and healthcare. The aim of this study was to provide a comprehensive overview of the use of anti-dementia drugs (ADD) – especially memantine – and their association with survival outcomes.

Methods: Using the NEUROHUN database, we analyzed health data covering the entire population of Hungary between 2010 and 2020. Descriptive statistics were calculated to assess disease incidence and prevalence, followed by Kaplan–Meier survival analyses and Cox proportional hazard regression to evaluate survival outcomes.

Results: We identified 398,584 patients with a diagnosis of dementia between 2010 and 2020. Of the total cohort, only 13.3 % of the patients were treated with at least one of the ADDs examined (memantine, donepezil or rivastigmine). Among the medication-fillers, 24.04 % patients were treated with memantine only. Kaplan–Meier analysis demonstrated that the untreated group had the shortest median survival (3.105 years, 95% CI: 3.083–3.129), whereas among patients on medication, those treated with memantine monotherapy had the shortest median survival (3.704 years, 95% CI: 3.606–3.784). In the Multivariable Cox proportional hazards model, patients receiving a combination of memantine and donepezil had the lowest mortality risk (HR: 0.581, 95% CI: [0.566–0.597]) compared to the untreated group.

Conclusion: These findings suggest a high proportion of delayed diagnoses, as memantine is typically prescribed only in advanced dementia stages. Timely diagnosis—particularly in the era of emerging disease-modifying therapies—along with prevention programs and evidence-based healthcare planning is therefore essential.

Disclosure: Nothing to disclose.

Neuroimmunology 4

EPO-0812 | Efficacy of eculizumab in the severe acute attacks of aquaporin-4 IgG-positive neuromyelitis optica spectrum disorder

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Background and aims: To compare the therapeutic efficacy of intravenous methylprednisolone (IVMP) versus IVMP combined with eculizumab in patients with neuromyelitis optica spectrum disorder (NMOSD) experiencing severe acute attacks.

Methods: The study performed a retrospective analysis of 25 severe acute attacks occurring in 17 patients with NMOSD during the acute phase. Treatment outcomes were evaluated using the Expanded Disability Status Scale (EDSS) and changes in visual acuity.

Results: Thirteen of the 17 patients received adjunctive eculizumab during the acute phase. The IVMP plus eculizumab group exhibited substantial clinical improvement at discharge and follow-up, with a mean Δ EDSS (defined as discharge EDSS minus attack EDSS) of -1.5, in contrast to a mean Δ EDSS of -0.1 in the IVMP monotherapy group.

Conclusion: The combination of IVMP and eculizumab provides superior clinical benefit over IVMP for patients with NMOSD presenting with severe acute attacks.

Disclosure: Nothing to disclose.

EPO-0813 | Impact of blood-brain barrier integrity on the clinical interpretation of serum neurofilament light as a biomarker of neuroaxonal damage

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Background and aims: Neurofilament light chain (NfL) is an established biomarker of neuroaxonal damage. Despite its relevance in disease monitoring and treatment follow-up, the influence of BBB integrity on serum NfL levels, however, remains insufficiently investigated. This study aimed to assess the impact of BBB integrity on serum NfL concentrations and its clinical relevance.

Methods: NfL concentrations were analyzed in paired cerebrospinal fluid (CSF) and serum samples. BBB integrity was assessed using the CSF/serum albumin quotient (QAlb). 109 patients were included in correlation and regression analyses, 79 patients were classified by BBB integrity (intact BBB: $n=35$; disrupted BBB: $n=44$). NfL was measured using HD-X (single molecule array assay) and Lumipulse G600 II (chemiluminescent enzyme immunoassay).

Results: Patients with a disrupted BBB showed higher NfL concentrations compared to patients with an intact BBB: HD-X CSF 453 [276–1268] pg/mL vs. 1252 [569–4622] pg/mL ($p < 0.001$); HD-X serum 9.98 [6.26–28.58] pg/mL vs. 25.46 [11.51–53.56] pg/mL ($p = 0.005$). Lumipulse CSF 334 [213–1191] pg/mL vs. 1060 [544–4260] pg/mL ($p = 0.001$); Lumipulse serum 12.33 [9.24–40.79] pg/mL vs. 35.60 [15.26–64.96] pg/mL ($p = 0.006$). CSF/serum correlations were stronger in patients with an intact BBB (HD-X: $r_s = 0.84$; Lumipulse: $r_s = 0.87$; both $p < 0.001$) than with a disrupted BBB (HD-X: $r_s = 0.73$; Lumipulse: $r_s = 0.71$; both $p < 0.001$). In the overall cohort ($N = 109$), strong CSF/serum correlations were observed (HD-X: $r_s = 0.80$, $p < 0.001$; Lumipulse: $r_s = 0.83$, $p < 0.001$). Linear regression showed no significant influence of QAlb on this relationship (interaction $p > 0.9$).

Conclusion: BBB integrity influences absolute serum NfL concentrations but not the CSF/serum NfL relationship, supporting serum NfL as a valid biomarker of neuroaxonal damage, even in BBB dysfunction.

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EPO-0814 | Diagnostic performance and pitfalls of a commercial assay for CASPR2 autoantibodies

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Background and aims: Widespread use of commercial cell-based assays (cCBA) has enhanced detection of contactin-associated protein-like 2 antibodies (CASPR2-abs) but has also raised concerns about false positive results, particularly when testing is limited to serum or antibody titres are low. However, their diagnostic performance has not been systematically validated.

Methods: We analysed serum and CSF samples from patients referred to our Neuroimmunology laboratory (Barcelona) for suspected autoimmune neurological disorders who tested positive for CASPR2-abs by cCBA (Euroimmun). Samples were re-tested using in-house rat brain immunohistochemistry and CBA.

Results: Of 105 patients with CASPR2-abs detected by cCBA, 74 (71%) were confirmed by in-house techniques (true-positive) and 31 (29%) were not (false-positive). Compared to true-positive, false-positive results occurred more frequent in females (4% vs. 52%, $p < 0.0001$) and younger patients (median age 68 vs. 58 years, $p = 0.02$). Among patients with false positive results, 23 (74%) presented with symptoms initially compatible with CASPR2 autoimmunity (although most had a final alternative diagnosis). However, only 1/20 (5%) had clinico-radiological evidence of limbic encephalitis, compared with 19/64 (30%) patients with confirmed CASPR2-abs ($p = 0.03$). False positive results occurred exclusively in serum (31 samples available). All paired CSF (21 available) from these patients were negative by both CBAs and in-house testing. In contrast, all the available CSF (37/37) from patients with true-positive serum results were also positive by cCBA.

Conclusion: CASPR2-abs detected exclusively in serum by cCBA, in the absence of CSF positivity, should be interpreted with caution, particularly in younger patients and females. CSF antibody testing is essential to minimize false-positive diagnoses.

Disclosure: Nothing to disclose.

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Background and aims: Neurosarcoidosis (NS) is a diagnosis of exclusion, with significant clinical and radiological overlap with infectious, neoplastic, and neuroinflammatory conditions. We report outcomes from the UK NS multidisciplinary team (MDT) meeting, focusing on cases where the diagnosis of NS was not confirmed.

Methods: Twenty-eight cases of suspected NS were reviewed since the MDT launch (May 2025). Clinical presentation, imaging features, diagnostic consensus, recommended investigations, onward referrals, and treatment decisions were reviewed.

Results: The primary reason for referral was diagnostic uncertainty (19/28), and, in 6/28 (21%), the MDT found insufficient evidence to confirm a diagnosis of NS. Differential diagnoses included vasculopathy and tuberculosis (6/28 each), IgG4-related disease (4/28), neoplastic and demyelinating conditions (3/28 each) and Progressive multifocal leukoencephalopathy and fungal infection (2/28 each). Of those, MDT suggested unlikely to be NS (4/6) had radiological evidence of recurrent infarcts. The most frequent clinical manifestations were brainstem symptoms (7/28), seizures, and headache (5/28 each). Frequently recommended investigations included cardiac assessment (MRI/PET), additional CSF analysis, vascular imaging, ophthalmological evaluation and full-thickness skin biopsy. Suggested management changes included discontinuation of immunosuppressive treatment (5/28), initiation of tuberculosis therapy (2/28), escalation to infliximab (7/28), cyclophosphamide (3/28), tocilizumab or adalimumab.

Conclusion: National MDT review frequently identified opportunities for additional work-up with the aim of increasing diagnostic confidence and treatment outcomes, including the rationalisation of immunosuppression. These early data highlight the importance of the national MDT in supporting neurologists managing neurosarcoidosis and other complex neuroinflammatory conditions.

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Background and aims: Chimeric antigen receptor (CAR) T-cell therapy has profoundly changed the therapeutic landscape of relapsed/refractory B-cell lymphoma. Cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS) represent relevant adverse events. Rare manifestations, including severe leukoencephalopathy, and non-ICANS complications such as myelopathy and peripheral neuropathy, have been reported.

Methods: We report the clinical course, neuroimaging findings, and treatment of a patient who developed acute leukoencephalo-myelo-radiculopathy after anti-CD19 CAR T-cell therapy.

Results: A 28-year-old woman with stage IVB diffuse large B-cell lymphoma resulted refractory to first-line chemotherapy. She underwent cyclophosphamide/fludarabine lymphodepletion and axicabtagene ciloleucel infusion. On day 1, she developed grade 2 CRS, treated with tocilizumab and corticosteroids. On day 4, she developed grade 2 ICANS, progressing to grade 4 on day 5. High-dose intravenous methylprednisolone was initiated. Brain magnetic resonance imaging (MRI) revealed diffuse leukoencephalopathy extending to the upper cervical spinal cord. On day 7, the patient was awake, but severe tetraparesis was evident. Spinal MRI demonstrated longitudinally extensive acute myelitis and thickening of the cauda equina nerve root with faint contrast enhancement. Lumbar puncture was not performed due to severe thrombocytopenia. Polymerase chain reaction testing for neurotropic viruses on peripheral blood was negative. High-dose intravenous methylprednisolone was continued, and intravenous immunoglobulin was administered, with mild neurological improvement. By day 23, clinical and radiological evidence of abdominal mass progression emerged. The patient died on day 45.

Conclusion: CAR T-cell therapy may rarely cause severe neurotoxicity affecting both the central and peripheral nervous systems. Systematic reporting is needed to improve risk stratification and management.

Disclosure: Nothing to disclose.

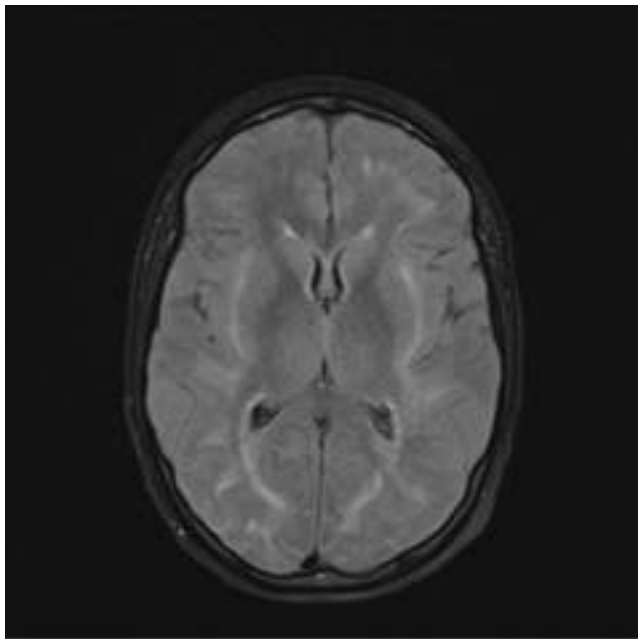


FIGURE 1 Leukoencephalopathy changes on magnetic resonance imaging: T2-weighted fluid attenuation inversion recovery axial image of the brain demonstrate symmetrical T2 hyperintensity of the deep white matter, most pronounced in the centrum semiovale.



FIGURE 3 Conus medullaris involvement in acute inflammatory myelopathy. Sagittal T2-weighted lumbar spine magnetic resonance imaging demonstrates hyperintense signal changes and mild enlargement of the conus medullaris, with associated thickening of the cauda equi.



FIGURE 2 Acute inflammatory myelopathy: Sagittal T2-weighted fat-suppressed MRI spinal magnetic resonance imaging demonstrates a longitudinally extensive intramedullary hyperintense signal involving the cervical and thoracic spinal cord, associated with mild cord.

EPO-0817 | From childhood to adulthood: Myelin autoantibody profile in acute demyelinating encephalomyelitis (ADEM)

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Background and aims: Acute disseminated encephalomyelitis (ADEM) is a rare CNS demyelinating disease, in some cases associated with antibodies to the myelin oligodendrocyte glycoprotein (MOG-IgG). Recently, antibodies to the proteolipid protein-1 (PLP1-IgG) has recently been identified in a subset of ADEM patients, though its frequency remains unknown. We aim to define the ADEM serological and clinical/paraclinical features in children versus adults.

Methods: We retrospectively screened patients with suspected ADEM referred to our laboratory and included those meeting the International Pediatric Multiple Sclerosis Study Group criteria. We collected clinical and radiological information from the clinical charts. In patients with available samples, we performed serological screening including MOG-IgG and PLP1-IgG.

Results: Ninety-eight ADEM patients were included in the study. The median age at onset was 7 years (0.03–76.27), and 71% were pediatric (≤ 16 years). Adults presented more frequently myelitis ($p = 0.002$), PNS involvement ($p < 0.001$), and sensory/sphincter symptoms ($p < 0.001$). Among 78 of 98 patients tested for anti-myelin autoantibodies, 28/78 were MOG-IgG+ (mostly children; $p = 0.054$); 10/78 were PLP1-IgG+ (mostly adults; $p < 0.001$); 34/78 were double-negative; and 6/78 were double-positive. PLP1-IgG+ patients showed more frequently PNS involvement ($p < 0.001$) and higher mRS at follow-up ($p = 0.002$). Over a median follow-up of 1.35 years, 30/98 patients relapsed without correlation with auto-antibody profile. Good recovery (mRS < 3) was associated with younger age ($p < 0.001$), myelitis ($p = 0.006$), MOG-IgG+ ($p = 0.030$) and PLP1-IgG- ($p < 0.001$).

Conclusion: Adult-onset ADEM exhibits distinct clinical/para-clinical and serological profile compared to pediatric cases. Further studies are needed to better define the relevance of PLP1-IgG in this subgroup.

Disclosure: Nothing to disclose.



FIGURE 1 Map showing thalamic nuclei significantly decreased in MOGAD compared to healthy controls.

TABLE 1 Clinical and demographic variables of patients with MOGAD, multiple sclerosis and healthy controls.

	MOG (n=25)	MS (n=37)	HC (n=38)	MOGAD 2.0 n=25	MOGAD 1.0 n=25	MOGAD 0.5 n=25
Age at onset (years)	10.5 (0.3–76.3)	35.5 (18.5–62.5)	35.5 (18.5–62.5)	10.5	10.5	10.5
Sex (M/F)	15/10	20/17	20/18	15/10	15/10	15/10
Age at last follow-up (years)	18.5 (0.3–76.3)	45.5 (18.5–72.5)	45.5 (18.5–72.5)	18.5	18.5	18.5
Duration of disease (years)	5.5 (0.3–25.5)	10.5 (0.3–35.5)	10.5 (0.3–35.5)	5.5	5.5	5.5
Relapse rate (%)	60	65	0	60	60	60
Relapse-free survival (%)	40	35	100	40	40	40
Relapse-free survival (years)	1.35 (0.3–5.5)	1.35 (0.3–5.5)	1.35 (0.3–5.5)	1.35	1.35	1.35
Relapse-free survival (months)	16.2 (0.3–66.6)	16.2 (0.3–66.6)	16.2 (0.3–66.6)	16.2	16.2	16.2
Relapse-free survival (days)	194.4 (0.3–800.0)	194.4 (0.3–800.0)	194.4 (0.3–800.0)	194.4	194.4	194.4
Relapse-free survival (weeks)	24.3 (0.3–100.0)	24.3 (0.3–100.0)	24.3 (0.3–100.0)	24.3	24.3	24.3
Relapse-free survival (months)	2.9 (0.3–12.5)	2.9 (0.3–12.5)	2.9 (0.3–12.5)	2.9	2.9	2.9
Relapse-free survival (years)	0.24 (0.3–1.0)	0.24 (0.3–1.0)	0.24 (0.3–1.0)	0.24	0.24	0.24
Relapse-free survival (months)	2.9 (0.3–12.5)	2.9 (0.3–12.5)	2.9 (0.3–12.5)	2.9	2.9	2.9
Relapse-free survival (days)	34.8 (0.3–150.0)	34.8 (0.3–150.0)	34.8 (0.3–150.0)	34.8	34.8	34.8
Relapse-free survival (weeks)	4.3 (0.3–18.2)	4.3 (0.3–18.2)	4.3 (0.3–18.2)	4.3	4.3	4.3
Relapse-free survival (months)	5.2 (0.3–21.9)	5.2 (0.3–21.9)	5.2 (0.3–21.9)	5.2	5.2	5.2
Relapse-free survival (years)	0.43 (0.3–1.8)	0.43 (0.3–1.8)	0.43 (0.3–1.8)	0.43	0.43	0.43
Relapse-free survival (months)	5.2 (0.3–21.9)	5.2 (0.3–21.9)	5.2 (0.3–21.9)	5.2	5.2	5.2
Relapse-free survival (days)	62.4 (0.3–262.8)	62.4 (0.3–262.8)	62.4 (0.3–262.8)	62.4	62.4	62.4
Relapse-free survival (weeks)	7.8 (0.3–32.9)	7.8 (0.3–32.9)	7.8 (0.3–32.9)	7.8	7.8	7.8
Relapse-free survival (months)	9.4 (0.3–39.9)	9.4 (0.3–39.9)	9.4 (0.3–39.9)	9.4	9.4	9.4
Relapse-free survival (years)	0.78 (0.3–3.3)	0.78 (0.3–3.3)	0.78 (0.3–3.3)	0.78	0.78	0.78
Relapse-free survival (months)	9.4 (0.3–39.9)	9.4 (0.3–39.9)	9.4 (0.3–39.9)	9.4	9.4	9.4
Relapse-free survival (days)	112.8 (0.3–478.8)	112.8 (0.3–478.8)	112.8 (0.3–478.8)	112.8	112.8	112.8
Relapse-free survival (weeks)	14.1 (0.3–59.9)	14.1 (0.3–59.9)	14.1 (0.3–59.9)	14.1	14.1	14.1
Relapse-free survival (months)	17.0 (0.3–71.9)	17.0 (0.3–71.9)	17.0 (0.3–71.9)	17.0	17.0	17.0
Relapse-free survival (years)	1.42 (0.3–6.0)	1.42 (0.3–6.0)	1.42 (0.3–6.0)	1.42	1.42	1.42
Relapse-free survival (months)	17.0 (0.3–71.9)	17.0 (0.3–71.9)	17.0 (0.3–71.9)	17.0	17.0	17.0
Relapse-free survival (days)	204.0 (0.3–862.8)	204.0 (0.3–862.8)	204.0 (0.3–862.8)	204.0	204.0	204.0
Relapse-free survival (weeks)	25.5 (0.3–107.4)	25.5 (0.3–107.4)	25.5 (0.3–107.4)	25.5	25.5	25.5
Relapse-free survival (months)	30.6 (0.3–129.0)	30.6 (0.3–129.0)	30.6 (0.3–129.0)	30.6	30.6	30.6
Relapse-free survival (years)	2.55 (0.3–10.8)	2.55 (0.3–10.8)	2.55 (0.3–10.8)	2.55	2.55	2.55
Relapse-free survival (months)	30.6 (0.3–129.0)	30.6 (0.3–129.0)	30.6 (0.3–129.0)	30.6	30.6	30.6
Relapse-free survival (days)	367.2 (0.3–1548.0)	367.2 (0.3–1548.0)	367.2 (0.3–1548.0)	367.2	367.2	367.2
Relapse-free survival (weeks)	45.9 (0.3–193.5)	45.9 (0.3–193.5)	45.9 (0.3–193.5)	45.9	45.9	45.9
Relapse-free survival (months)	55.1 (0.3–231.8)	55.1 (0.3–231.8)	55.1 (0.3–231.8)	55.1	55.1	55.1
Relapse-free survival (years)	4.59 (0.3–19.3)	4.59 (0.3–19.3)	4.59 (0.3–19.3)	4.59	4.59	4.59
Relapse-free survival (months)	55.1 (0.3–231.8)	55.1 (0.3–231.8)	55.1 (0.3–231.8)	55.1	55.1	55.1
Relapse-free survival (days)	661.2 (0.3–2781.6)	661.2 (0.3–2781.6)	661.2 (0.3–2781.6)	661.2	661.2	661.2
Relapse-free survival (weeks)	82.6 (0.3–347.7)	82.6 (0.3–347.7)	82.6 (0.3–347.7)	82.6	82.6	82.6
Relapse-free survival (months)	99.1 (0.3–417.2)	99.1 (0.3–417.2)	99.1 (0.3–417.2)	99.1	99.1	99.1
Relapse-free survival (years)	8.26 (0.3–34.8)	8.26 (0.3–34.8)	8.26 (0.3–34.8)	8.26	8.26	8.26
Relapse-free survival (months)	99.1 (0.3–417.2)	99.1 (0.3–417.2)	99.1 (0.3–417.2)	99.1	99.1	99.1
Relapse-free survival (days)	1189.2 (0.3–4806.0)	1189.2 (0.3–4806.0)	1189.2 (0.3–4806.0)	1189.2	1189.2	1189.2
Relapse-free survival (weeks)	148.6 (0.3–600.8)	148.6 (0.3–600.8)	148.6 (0.3–600.8)	148.6	148.6	148.6
Relapse-free survival (months)	178.3 (0.3–720.9)	178.3 (0.3–720.9)	178.3 (0.3–720.9)	178.3	178.3	178.3
Relapse-free survival (years)	14.86 (0.3–60.1)	14.86 (0.3–60.1)	14.86 (0.3–60.1)	14.86	14.86	14.86
Relapse-free survival (months)	178.3 (0.3–720.9)	178.3 (0.3–720.9)	178.3 (0.3–720.9)	178.3	178.3	178.3
Relapse-free survival (days)	2139.6 (0.3–8649.6)	2139.6 (0.3–8649.6)	2139.6 (0.3–8649.6)	2139.6	2139.6	2139.6
Relapse-free survival (weeks)	267.4 (0.3–1067.4)	267.4 (0.3–1067.4)	267.4 (0.3–1067.4)	267.4	267.4	267.4
Relapse-free survival (months)	320.9 (0.3–1283.7)	320.9 (0.3–1283.7)	320.9 (0.3–1283.7)	320.9	320.9	320.9
Relapse-free survival (years)	26.74 (0.3–106.7)	26.74 (0.3–106.7)	26.74 (0.3–106.7)	26.74	26.74	26.74
Relapse-free survival (months)	320.9 (0.3–1283.7)	320.9 (0.3–1283.7)	320.9 (0.3–1283.7)	320.9	320.9	320.9
Relapse-free survival (days)	3850.8 (0.3–15429.6)	3850.8 (0.3–15429.6)	3850.8 (0.3–15429.6)	3850.8	3850.8	3850.8
Relapse-free survival (weeks)	481.3 (0.3–1929.6)	481.3 (0.3–1929.6)	481.3 (0.3–1929.6)	481.3	481.3	481.3
Relapse-free survival (months)	577.6 (0.3–2311.7)	577.6 (0.3–2311.7)	577.6 (0.3–2311.7)	577.6	577.6	577.6
Relapse-free survival (years)	48.13 (0.3–193.0)	48.13 (0.3–193.0)	48.13 (0.3–193.0)	48.13	48.13	48.13
Relapse-free survival (months)	577.6 (0.3–2311.7)	577.6 (0.3–2311.7)	577.6 (0.3–2311.7)	577.6	577.6	577.6
Relapse-free survival (days)	6931.2 (0.3–27741.6)	6931.2 (0.3–27741.6)	6931.2 (0.3–27741.6)	6931.2	6931.2	6931.2
Relapse-free survival (weeks)	866.4 (0.3–3467.7)	866.4 (0.3–3467.7)	866.4 (0.3–3467.7)	866.4	866.4	866.4
Relapse-free survival (months)	1039.7 (0.3–4161.2)	1039.7 (0.3–4161.2)	1039.7 (0.3–4161.2)	1039.7	1039.7	1039.7
Relapse-free survival (years)	86.64 (0.3–346.8)	86.64 (0.3–346.8)	86.64 (0.3–346.8)	86.64	86.64	86.64
Relapse-free survival (months)	1039.7 (0.3–4161.2)	1039.7 (0.3–4161.2)	1039.7 (0.3–4161.2)	1039.7	1039.7	1039.7
Relapse-free survival (days)	12476.4 (0.3–50006.4)	12476.4 (0.3–50006.4)	12476.4 (0.3–50006.4)	12476.4	12476.4	12476.4
Relapse-free survival (weeks)	1559.5 (0.3–6251.7)	1559.5 (0.3–6251.7)	1559.5 (0.3–6251.7)	1559.5	1559.5	1559.5
Relapse-free survival (months)	1871.4 (0.3–7502.0)	1871.4 (0.3–7502.0)	1871.4 (0.3–7502.0)	1871.4	1871.4	1871.4
Relapse-free survival (years)	155.95 (0.3–625.2)	155.95 (0.3–625.2)	155.95 (0.3–625.2)	155.95	155.95	155.95
Relapse-free survival (months)	1871.4 (0.3–7502.0)	1871.4 (0.3–7502.0)	1871.4 (0.3–7502.0)	1871.4	1871.4	1871.4
Relapse-free survival (days)	22456.8 (0.3–90024.0)	22456.8 (0.3–90024.0)	22456.8 (0.3–90024.0)	22456.8	22456.8	22456.8
Relapse-free survival (weeks)	2807.1 (0.3–11253.0)	2807.1 (0.3–11253.0)	2807.1 (0.3–11253.0)	2807.1	2807.1	2807.1
Relapse-free survival (months)	3368.4 (0.3–138019.7)	3368.4 (0.3–138019.7)	3368.4 (0.3–138019.7)	3368.4	3368.4	3368.4
Relapse-free survival (years)	280.71 (0.3–1125.3)	280.71 (0.3–1125.3)	280.71 (0.3–1125.3)	280.71	280.71	280.71
Relapse-free survival (months)	3368.4 (0.3–138019.7)	3368.4 (0.3–138019.7)	3368.4 (0.3–138019.7)	3368.4	3368.4	3368.4
Relapse-free survival (days)	40420.8 (0.3–162048.0)	40420.8 (0.3–162048.0)	40420.8 (0.3–162048.0)	40420.8	40420.8	40420.8
Relapse-free survival (weeks)	5052.6 (0.3–19756.0)	5052.6 (0.3–19756.0)	5052.6 (0.3–19756.0)	5052.6	5052.6	5052.6
Relapse-free survival (months)	6063.2 (0.3–23707.2)	6063.2 (0.3–23707.2)	6063.2 (0.3–23707.2)	6063.2	6063.2	6063.2
Relapse-free survival (years)	505.26 (0.3–1975.6)	505.26 (0.3–1975.6)	505.26 (0.3–1975.6)	505.26	505.26	505.26
Relapse-free survival (months)	6063.2 (0.3–23707.2)	6063.2 (0.3–23707.2)	6063.2 (0.3–23707.2)	6063.2	6063.2	6063.2
Relapse-free survival (days)	72758.4 (0.3–284486.4)	72758.4 (0.3–284486.4)	72758.4 (0.3–284486.4)	72758.4	72758.4	72758.4
Relapse-free survival (weeks)	9094.8 (0.3–35560.8)	9094.8 (0.3–35560.8)	9094.8 (0.3–35560.8)	9094.8	9094.8	9094.8
Relapse-free survival (months)	10913.8 (0.3–42673.0)	10913.8 (0.3–42673.0)	10913.8 (0.3–42673.0)	10913.8	10913.8	10913.8
Relapse-free survival (years)	909.48 (0.3–3556.1)	909.48 (0.3–3556.1)	909.48 (0.3–3556.1)	909.48	909.48	909.48
Relapse-free survival (months)	10913.8 (0.3–42673.0)	10913.8 (0.3–42673.0)	10913.8 (0.3–42673.0)	10913.8	10913.8	10913.8
Relapse-free survival (days)	130965.6 (0.3–512073.6)	130965.6 (0.3–512073.6)	130965.6 (0.3–512073.6)	130965.6	130965.6	130965.6
Relapse-free survival (weeks)	16370.7 (0.3–64009.2)	16370.7 (0.3–64009.2)	16370.7 (0.3–64009.2)	16370.7	16370.7	16370.7
Relapse-free survival (months)	19644.8 (0.3–76811.0)	19644.8 (0.3–76811.0)	19644.8 (0.3–76811.0)	19644.8	19644.8	19644.8
Relapse-free survival (years)	1637.07 (0.3–6400.9)	1637.07 (0.3–6400.9)	1637.07 (0.3–6400.9)	1637.07	1637.07	1637.07
Relapse-free survival (months)	19644.8 (0.3–76811.0)	19644.8 (0.3–76811.0)	19644.8 (0.3–76811.0)	19644.8	19644.8	19644.8
Relapse-free survival (days)	235737.6 (0.3–920131.2)	235737.6 (0.3–920131.2)	235737.6 (0.3–920131.2)	235737.6	235737.6	235737.6
Relapse-free survival (weeks)	29467.2 (0.3–115016.4)	29467.2 (0.3–115016.4)	29467.2 (0.3–115016.4)	29467.2	29467.2	29467.2
Relapse-free survival (months)	35360.6 (0.3–138019.7)	35360.6 (0.3–138019.7)	35360.6 (0.3–138019.7)	35360.6	35360.6	35360.6
Relapse-free survival (years)	2946.72 (0.3–11501.6)	2946.72 (0.3–11501.6)	2946.72 (0.3–11501.6)	2946.72	2946.72	2946.72
Relapse-free survival (months)	35360.6 (0.3–138019.7)	35360.6 (0.3–138019.7)	35360.6 (0.3–138019.7)	35360.6	35360.6	35360.6
Relapse-free survival (days)	424327.2 (0.3–1656230.4)	424327.2 (0.3–1656230.4)	424327.2 (0.3–1656230.4)	424327.2	424327.2	424327.2
Relapse-free survival (weeks)	53040.9 (0.3–207028.8)	53040.9 (0.3–207028.8)	53040.9 (0.3–207028.8)	53040.9	53040.9	53040.9
Relapse-free survival (months)	63649.1 (0.3–248434.6)	63649.1 (0.3–248434.6)	63649.1 (0.3–248434.6)	63649.1	63649.1	63649.1
Relapse-free survival (years)	5304.09 (0.3–20702.9)	5304.09 (0.3–20702.9)	5304.09 (0.3–20702.9)	5304.09	5304.09	5304.09
Relapse-free survival (months)	63649.1 (0.3–248434.6)	63649.1 (0.3–248434.6)	63649.1 (0.3–248434.6)	63649.1	63649.1	63649.1
Relapse-free survival (days)	763789.6 (0.3–3001171.2)	763789.6 (0.3–3001171.2)	763789.6 (0.3–3001171.2)	763789.6	763789.6	763789.6
Relapse-free survival (weeks)	95473.7 (0.3–375146.4)	95473.7 (0.3–375146.4)	95473.7 (0.3–375146.4)	95473.7	95473.7	95473.7
Relapse-free survival (months)	114568.4 (0.3–450175.7)	114568.4 (0.3–450175.7)	114568.4 (0.3–450175.7)	114568.4	114568.4	114568.4
Relapse-free survival (years)	9547.37 (0.3–37514.6)	9547.37 (0.3–37514.6)	9547.37 (0.3–37514.6)	9547.37	9547.37	9547.37
Relapse-free survival (months)	114568.4 (0.3–450175.7)	114568.4 (0.3–450175.7)	114568.4 (0.3–450175.7)	114568.4	114568.4	114568.4
Relapse-free survival (days)	1374820.8 (0.3–5402107.2)	1374820.8 (0.3–5402107.2)	1374820.8 (0.3–5402107.2)	1374820.8	1374820.8	1374820.8
Relapse-free survival (weeks)	171852.6 (0.3–675263.4)	171852.6 (0.3–675263.4)	171852.6 (0.3–675263.4)	171852.6	171852.6	171852.6
Relapse-free survival (months)	206223.2 (0.3–810316.1)	206223.2 (0.3–810316.1)	206223.2 (0.3–810316.1)	206223.2	206223.2	206223.2
Relapse-free survival (years)	17185.26 (0.3–67526.3)	17185.26 (0.3–67526.3)	17185.26 (0.3–67526.3)	17185.26	17185.26	17185.26
Relapse-free survival (months)	206223.2 (0.3–810316.1)	206223.2 (0.3–810316.1)	206223.2 (0.3–810316.1)	206223.2	206223.2	206223.2
Relapse-free survival (days)	2478672.0 (0.3–9723801.6)	2478672.0 (0.3–9723801.6)	2478672.0 (0.3–9723801.6)	2478672.0	2478672.0	2478672.0
Relapse-free survival (weeks)	309834.0 (0.3–1215475.2)	309834.0 (0.3–1215475.2)	309834.0 (0.3–1215475.2)	309834.0	309834.0	309834.0
Relapse-free survival (months)	371800.8 (0.3–1458570.2)	371800.8 (0.3–1458570.2)	371800.8 (0.3–1458570.2)	371800.8	371800.8	371800.8
Relapse-free survival (years)	30983.4 (0.3–121547.5)	30983.4 (0.3–121547.5)	30983.4 (0.3			

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Background and aims: As multiple analytical platforms for serum NfL (sNfL), an established biomarker of neuroaxonal damage, are now available, assay comparability and harmonization are critical for clinical and research use. This study compared sNfL measurements across three assays—NF-light Advantage V2/Plus (Quanterix), Lumipulse G NfL blood (FujiRebio), and Elecsys NfL (Roche Diagnostics) and suggested conversion formulas to support standardization.

Methods: Serum samples from healthy donors ($n = 303$) and patients with multiple sclerosis (MS; $n = 181$) were analyzed. Inter-assay agreement was assessed using Passing–Bablok regression and correlation analyses. Assays were further evaluated for their ability to detect MS disease activity. In addition to original data, this poster will integrate and compare published conversion formulas and provide data on NfL levels across neurological diseases.

Results: All assays demonstrated strong correlations (Pearson $r > 0.98$; Spearman $\rho > 0.90$; $p < 0.001$). Fourteen of 484 samples (2.9%) showed stable, platform-specific deviations (> 4 SD). After excluding outliers, conversion equations to the NF-light Advantage scale were derived: $NfL = 6.31 \times [Elecsys] + 2.33$ ng/L; $NfL = 0.94 \times [Lumipulse] + 1.30$ ng/L. All platforms detected significant differences between MS relapse and remission ($p < 0.001$). Age-adjusted Z scores values yielded the largest effect sizes.

Conclusion: The three platforms show high concordance and comparable clinical performance. However, rare but marked assay-specific deviations underscore the need for continued harmonization. Age-adjusted Z scores may further enhance interpretability. The consistency and generalizability of published conversion formulas across broader NfL ranges warrant further investigation.

Disclosure: Assay kits were provided by Roche Diagnostics and FujiRebio; all analyses were conducted independently.

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Background and aims: Cognitive impairment is increasingly recognized as a relevant clinical manifestation of neuromyelitis optica spectrum disorders (NMOSD), but its underlying mechanisms and early predictors remain poorly understood. Alterations in cortical excitability, measurable through transcranial magnetic stimulation (TMS), may reflect subclinical cortical dysfunction and represent a potential biomarker of future cognitive decline.

Methods: Forty-eight patients with newly diagnosed NMOSD and twenty-five matched healthy controls underwent baseline neurophysiological assessment using TMS, including measures of resting motor threshold, short-interval intracortical inhibition (SICI), and intracortical facilitation (ICF). Comprehensive neuropsychological testing was performed at baseline and at 5 years. Cognitive impairment was defined as performance ≥ 1.5 SD below controls on ≥ 2 cognitive domains. Multivariate regression analyses were used to identify baseline predictors of cognitive decline, adjusting for age, education, disease duration, and relapse rate.

Results: At diagnosis, NMOSD patients showed significantly reduced SICI and increased ICF compared with controls ($p < 0.01$), indicating early cortical disinhibition. After 5 years, 37.5% of patients developed cognitive impairment, primarily affecting attention, executive function, and processing speed. Baseline SICI reduction independently predicted cognitive decline ($\beta = -0.42$, $p = 0.004$), even after controlling for demographic and clinical covariates. No association was found between relapse frequency or EDSS progression and cognitive outcomes.

Conclusion: Early alterations in cortical excitability, reflecting GABAergic dysfunction, are strong predictors of long-term cognitive deterioration in NMOSD. TMS-derived measures may serve as valuable biomarkers to identify patients at higher risk for cognitive decline and guide early neuroprotective interventions.

Disclosure: Nothing to disclose.

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Background and aims: International evaluation of rapid iDOT aquaporin-4 assay

Methods: Sera from centres in China, France, Italy, Korea, Spain, UK were sent coded to the coordinating centre in Italy from four patient groups (1. AQP4 antibody positive NMOSD patients 2. MOG-IgG clear positive MOGAD patients 3. Clinically definite pwMS 4. Sera difficult on any assay). iDOT kits from China were sent to Italy. All samples were recoded and shipped with kits to 8 testing centres (Austria, Brazil, India, Sri Lanka, France, China, Korea, UK). iDOTs were performed at all centres; ICBA in Korea and UK; fCBA in China. Test sensitivity was based on positive results in group 1; specificity was based on negative results in groups 2–4.

Results: Unblinding revealed 381 sera were tested: Group 1: 126, Group 2: 106, Group 3: 93, Group 4: 56. Kits were employed close to/after expiry date in seven centres. The median sensitivity was 84.9% (range 39.7–92.9%); median specificity was 99.2% (range 98.8–99.6%). 72% of group 1 samples were positive on all iDOTs across 7 centres with only 7 (6%) samples positive in fewer than 4/7 centres. The China team performed the assay on iDOT kits 2 months old with a sensitivity of 100% and specificity of 98.8%. The fCBA was 91.2% sensitive. ICBA were 83.7% and 100% sensitive. All three were 100% specific.

Conclusion: The iDOT kit performed well despite being near the end of their shelf-life. A relatively new kit performed in-house had equal sensitivity to the best ICBA and highlights great promise for this system.

Disclosure: Nothing to disclose.

EPO-0822 | Efficacy and safety analysis of efgartigimod in patients with very-late-onset myasthenia gravis

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Background and aims: Myasthenia gravis (MG) is an autoimmune disorder caused by pathogenic IgG autoantibodies disrupting the neuromuscular junction. Very-late-onset MG (VLOMG, onset ≥ 65 years) poses greater management challenges, with scarce data on efgartigimod (a novel FcRn antagonist) in this population.

Methods: Retrospective analysis of 62 AChR antibody-positive VLOMG patients treated with efgartigimod (10 mg/kg IV weekly for 4 weeks, repeated after 4-week intervals) at Affiliated Hospital of Qingdao University (Nov 2023–Sep 2025), assessing MSE rate, corticosteroid reduction, MG-ADL/MG-QoL15r scores, muscle function, and adverse events (AEs).

Results: A total of 62 eligible VLOMG patients were enrolled, of these, 15 completed 3 or more treatment cycles, 10 completed 2 cycles, and 37 completed 1 cycle. At the study endpoint, 41.9% (26/62) of patients had achieved MSE. MG-ADL/MG-QoL15r scores and corticosteroid doses significantly decreased across subgroups (1, 2, ≥ 3 cycles, all $P < 0.05$). MG-ADL/MG-QoL15r

TABLE 1 Patient characteristics at efgartigimod initiation.

	MG patients (n = 62)
Age, years, median (IQR)	72.4 (69.9, 76.0)
Sex, n (%)	
Male	33 (53.2)
Female	29 (46.8)
Duration of MG, months, median (IQR)	12.0 (2.9, 42.5)
Complications, n (%)	
Thymoma	5 (8.1)
Thymectomy	1 (1.6)
Hypertension	30 (48.4)
Diabetes	19 (30.6)
Cardiovascular and cerebrovascular diseases	13 (21.0)
Malignant tumor	2 (3.2)
Other complications	23 (37.1)
Scores (mean $\pm$ SD)	
QMG	12.4 $\pm$ 5.6
ADL	4.9 $\pm$ 2.3
QoL 15r	18.2 $\pm$ 11.9
MGFA, n (%)	
II	32 (51.6)
III	17 (27.4)
IV	13 (21.0)
Key muscle groups, median (IQR)	
Respiratory muscles	0.0 (0.0, 6.0)
Bulbar muscles	1.0 (0.0, 7.0)
Limb muscles and axial muscles	7.5 (5.0, 10.3)
Extracranial Muscles	2.0 (1.0, 4.3)
Anti-AChR antibody level, nmol/L, n (%)	
<10	15 (24.2)
10–20	23 (37.1)
>20	26 (41.9)

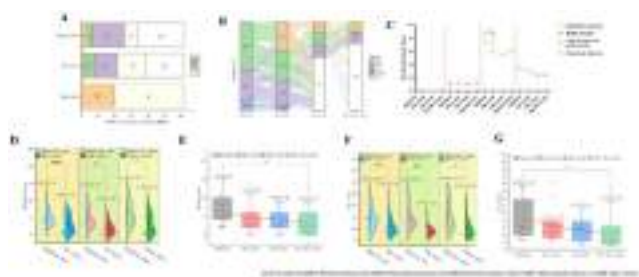


FIGURE 1 (A) Dynamics of MSE criteria-meeting patients ($n=62$). (B) MG-ADL score changes ($n=62$). (C) Key muscle group changes (≥ 3 cyc, $n=15$). (D) MG-ADL scores by treatment cycle. (E) MG-ADL scores (≥ 3 cyc). (F) QoL scores by treatment cycle. (G) QoL scores (≥ 3 cyc).

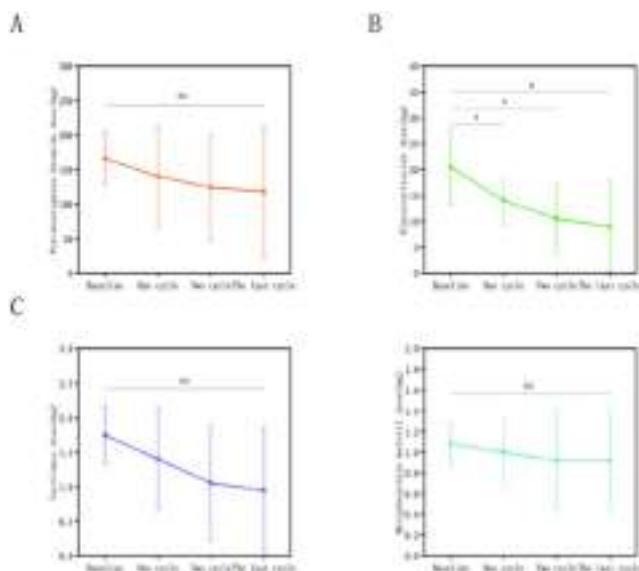


FIGURE 2 Conventional immunosuppressant usage changes in MG patients with ≥ 3 efgartigimod cycles.

scores with progressive improvement in ≥ 3 -cycle patients. No serious AEs/deaths occurred; only 1 allergic rash.

Conclusion: Efgartigimod is safe and effective for VLOMG, ameliorating symptoms, improving quality of life, and facilitating corticosteroid reduction.

Disclosure: Nothing to disclose.

EPO-0823 | Steroids with or without intravenous immunoglobulin for immune checkpoint inhibitor-related neuropathies: A French multicenter cohort study

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Background and aims: Current guidelines for immune-related neurotoxicities recommend discontinuation of immune checkpoint inhibitors (ICIs) and steroid therapy in severe cases. However, the management of ICI-related neuropathies remains less standardised, with intravenous immunoglobulin (IVIg) frequently combined with steroids despite limited evidence.

Methods: We conducted a retrospective analysis of patients with ICI-related neuropathies from six French centers. Clinical, biological and electrophysiological data were collected at baseline, 3 months, and 6 months. The outcome was defined by changes in the modified Rankin Scale (mRS) and the Inflammatory Neuropathy Cause and Treatment (INCAT) disability scores between baseline and follow-up visits.

Results: Forty-eight patients with ICI-related neuropathies were identified including 24 demyelinating neuropathies, 18 axonal neuropathies, and 6 multifocal radiculopathies. Most presented with subacute onset and predominant sensory symptoms. Immunomodulatory therapy was initiated for severe cases, primarily steroids alone ($n=25$) or steroids combined with IVIg ($n=14$). 90% of patients achieved a favourable and rapid outcome: 11 experienced complete recovery, 32 partial or subtotal improvement. No significant advantage was observed in adding IVIg to steroids. In the analysis of other variables, a higher baseline disability score (INCAT) was the only factor significantly associated with poorer outcomes. ICIs were reintroduced in ten patients, with good tolerance observed.

Conclusion: Our study does not support the routine use of IVIg in addition to steroids as first-line treatment for ICI-related neuropathies, even in demyelinating cases. Reintroduction of ICIs after resolution of neurological impairment appears safe and feasible.

Disclosure: AFC is a consultant for BMS. No disclosures for the other authors.

EPO-0824 | **Imaging-informed cerebral hemodynamic insights into Moyamoya disease and stroke risk: A systematic review**

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Background and aims: Moyamoya disease (MMD) is a progressive cerebrovascular disorder characterized by intracranial arterial stenosis, leading to impaired cerebral perfusion and increased risk of ischemic and hemorrhagic stroke. Although advanced imaging is routinely used for diagnosis and follow-up, the hemodynamic mechanisms underlying disease progression, stroke risk, and post-revascularization outcomes remain incompletely understood. This systematic review synthesizes imaging-informed hemodynamic evidence to support pathophysiological understanding and clinical decision-making in MMD.

Methods: This systematic review followed PRISMA guidelines. A comprehensive literature search of PubMed, Scopus,

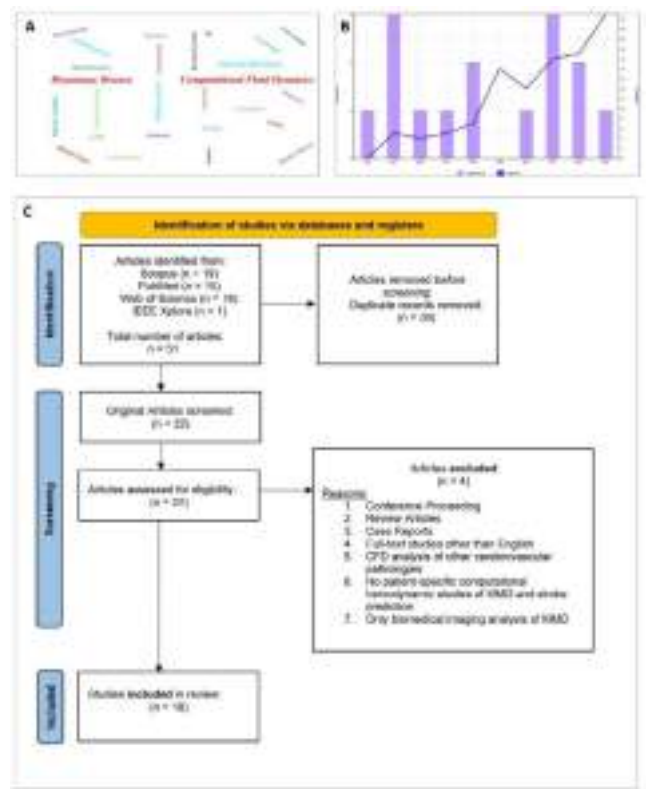


FIGURE 1 Overview of the systematic review methodology for computational bio-fluid mechanics studies in Moyamoya disease: (A) keyword cloud illustrating the literature search strategy, (B) temporal trend of publications and citations.

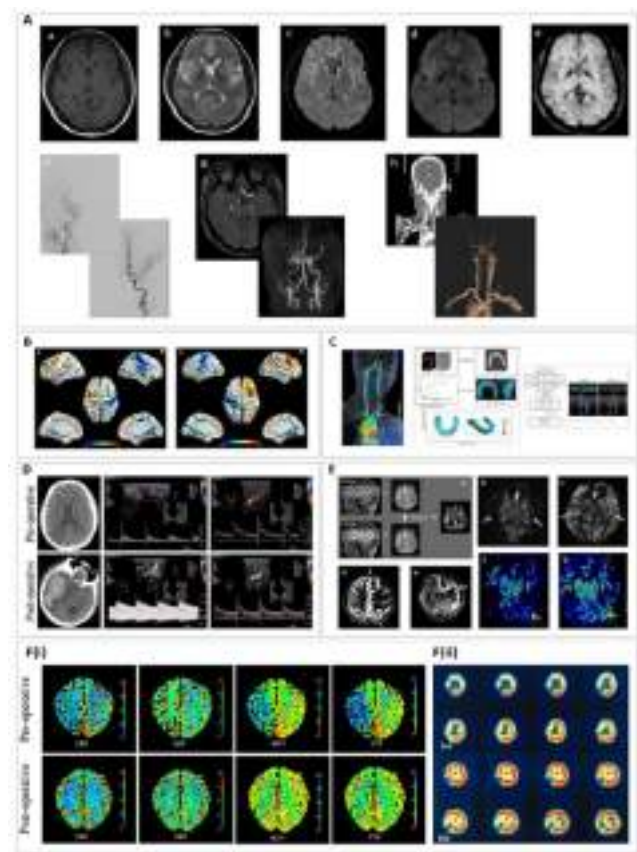


FIGURE 2 Multimodal clinical imaging approaches for evaluating cerebrovascular structure, perfusion, and hemodynamics in Moyamoya disease. Shown are representative MRI sequences and angiographic techniques (DSA, TOF-MRA, CTA), functional MRI, 4D flow MRI.

Web of Science, and IEEE Xplore was conducted up to March 2024 using the keywords “Moyamoya disease” AND “computational fluid dynamics.” Original computational studies investigating cerebral hemodynamics, cerebrovascular biomechanics, patient-specific blood-flow modeling, stroke prediction, or relevant idealized models were included. Eighteen studies were selected for qualitative synthesis.

Results: Eighteen imaging-informed computational studies were analyzed. Neuroimaging modalities, including digital subtraction angiography, CT perfusion, ASL-MRI, and phase-contrast MRI, were frequently integrated with computational fluid dynamics to quantify flow redistribution, perfusion deficits, and vascular remodeling. Consistent findings revealed abnormal flow patterns, elevated wall shear stress, pressure drops, and impaired distal perfusion in stenotic internal carotid and middle cerebral arteries. Pressure- and shear-based metrics emerged as potential biomarkers for disease progression, surgical outcome assessment, and stroke risk stratification.

Conclusion: Patient-specific modeling integrated with clinical imaging shows promise for disease assessment, surgical planning, and postoperative evaluation. Future work should emphasize physiologically realistic fluid–structure interaction frameworks, standardized boundary condition calibration, and integration with advanced imaging and physics-guided AI to enable robust, non-invasive, personalized clinical decision-making.

Disclosure: Nothing to disclose.

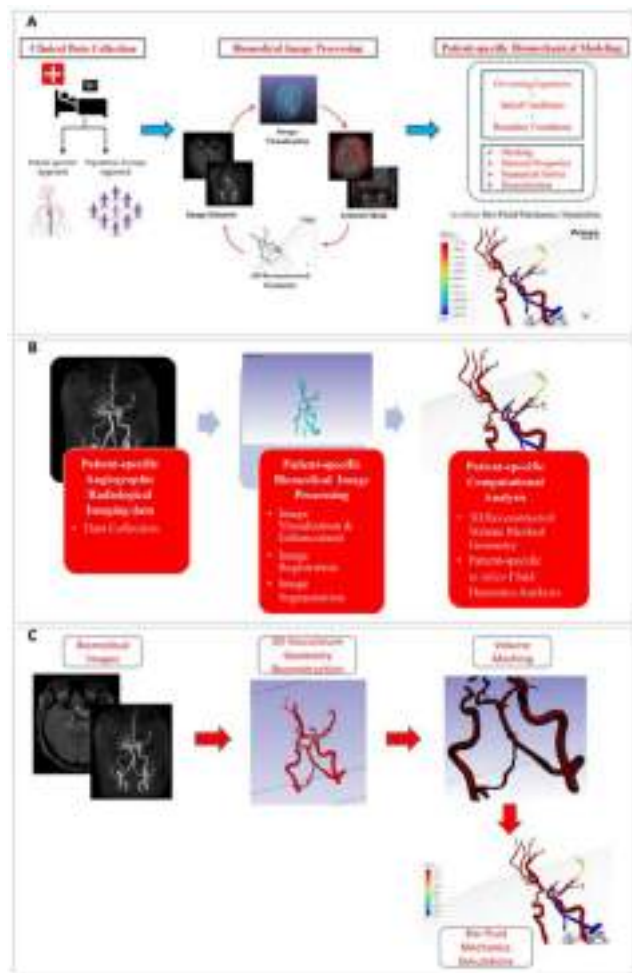


FIGURE 3 Schematic overview of the in silico biomechanical analysis pipeline for Moyamoya disease and cerebral stroke risk assessment, illustrating biomedical image processing, patient-specific vascular reconstruction, and computational fluid dynamics–ba.

EPO-0825 | Impact of endovascular treatment in patients aged ≥ 85 years with large vessel occlusion ischemic stroke

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Background and aims: To evaluate functional outcomes and mortality in patients aged ≥ 85 years with large vessel occlusion (LVO) ischemic stroke managed with endovascular therapy (EVT).

Methods: We conducted a prospective, multicenter observational study including patients aged ≥ 85 years with LVO treated in seven Stroke Centres in Madrid over one year (from February 2024). Patients treated with EVT were compared with those managed without EVT. Inverse probability of treatment weighting (IPTW) based on propensity scores was applied to adjust for baseline differences between groups.

Results: A total of 277 patients were included (71% women), with a mean age of 89.2 ± 3.11 years and a median baseline mRS of 1 (IQR 0–4). EVT was performed in 188 patients (67.8%). Patients treated with EVT had less pre-stroke cognitive impairment and higher NIHSS scores. After IPTW adjustment, there were no significant differences in functional independence at 3 months (mRS 0–2) between EVT and non-EVT patients (31.1% vs 24.7%; $p = 0.31$; OR = 1.41, 95% CI 0.72–2.72). However, EVT was associated with a higher proportion of favorable outcomes when considering the mRS 0–3 (50.0% vs 31.9%; $p = 0.035$; OR = 1.92, 95% CI 1.05–3.52) and with lower 3-month mortality (29.3% vs 50.9%; $p = 0.038$; OR = 0.51, 95% CI 0.27–0.96).

Conclusion: In our cohort, EVT did not significantly increase the likelihood of full independence or mild disability (mRS 0–2) at 3 months. However, EVT was associated with reduced mortality and a lower degree of disability among dependent patients.

Disclosure: Nothing to disclose.

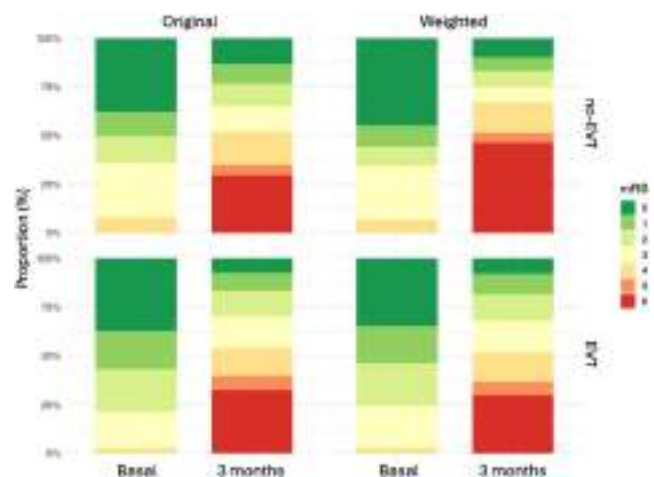


FIGURE 1 Grotta bar charts comparing baseline and 3-month modified Rankin Scale (mRS) distributions in the basal and weighted cohorts.

EPO-0826 | Two circulations, different outcomes?
Clinical and paraclinical results of mechanical thrombectomy in anterior and posterior circulation strokes

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Background and aims: Mechanical thrombectomy (MT) is the standard of care for acute ischemic stroke due to large vessel occlusion (LVO). The outcomes of thrombectomy for anterior circulation infarction (ACI) and posterior circulation infarction (POCI) may differ due to inherent clinical and paraclinical particularities of the respective vascular territories. Objective: To compare technical and clinical outcomes of MT in patients with POCI versus ACI.

Methods: Retrospective analysis of consecutive patients who underwent MT for POCI between July/2023-July/2025, and for ACI between January-July/2025. Demographic, clinical, and procedural variables were systematically collected.

Results: A total of 148 patients were included (49 POCI, 99 ACI), 78 (52.7%) female. No significant differences were found in age ($p=0.321$) or interhospital transfer rates ($p=0.666$). POCI patients more frequently presented with Glasgow Coma Scale (GCS) score ≤ 8 at admission than ACI patients (17/49 vs. 4/99, $p<0.001$). Compared with ACI, POCI was associated with lower neurological improvement at 24 hours (38.8% vs. 69.7%, $p<0.001$), longer median onset-to-puncture time (627 vs. 416 minutes, $p=0.001$), and higher two-month mortality (16/49 vs. 9/99, $p<0.001$). Puncture-to-recanalization time (31.9 ± 24.6 vs. 36.4 ± 22.1 minutes, $p=0.324$) and successful reperfusion rates (TICI ≥ 2 C; 63.3% vs. 59.6%, $p=0.720$) were similar between groups. However, procedure-related complications occurred more frequently in POCI (8/49 vs. 2/99, $p=0.001$).

Conclusion: Despite similar technical performance regarding time to recanalization and rate of successful reperfusion, POCI patients exhibited distinct clinical trajectories, which may be associated with lower admission GCS and delayed symptom-to-puncture times.

Disclosure: Nothing to disclose.

EPO-0827 | Early evolocumab reduces early neurological deterioration in acute non-cardioembolic stroke: A real-world retrospective cohort study

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Background and aims: Early neurological deterioration (END) complicates 15–20% of acute ischemic strokes despite standard care. The acute neuroprotective potential of PCSK9 inhibition, mediated by rapid lipid-lowering and anti-inflammatory mechanisms, remains unexplored.

Methods: This retrospective cohort study at Central Veterans Hospital, Republic of Korea (2022–2025) enrolled consecutive patients with acute non-cardioembolic ischemic stroke (large artery atherosclerosis or small vessel occlusion) presenting

Characteristic	Evolocumab (n=96)	Control (n=120)	p-value
Age (mean $\pm$ SD)	72.1 $\pm$ 10.5	72.3 $\pm$ 10.2	0.98
Sex (male, %)	58.3	57.5	0.92
NIHSS (mean $\pm$ SD)	5.2 $\pm$ 3.1	5.4 $\pm$ 3.3	0.63
LDL-C (mg/dL)	149 $\pm$ 32	145 $\pm$ 35	0.85
hs-CRP (mg/L)	5.2 $\pm$ 2.1	5.0 $\pm$ 2.3	0.89
Time to puncture (min)	627	416	0.001
Time to recanalization (min)	31.9 $\pm$ 24.6	36.4 $\pm$ 22.1	0.324
TICI ≥ 2 C (%)	63.3	59.6	0.720
Successful reperfusion (%)	63.3	59.6	0.720
Neurological improvement at 24h (%)	38.8	69.7	<0.001
Two-month mortality (%)	32.7	9.1	<0.001

Outcome	Evolocumab (n=96)	Control (n=120)	p-value
END (≥ 2 points) (%)	8.3	18.3	0.04
Adjusted OR	0.41	0.38	0.03
Functional independence at 90 days (%)	71	63	0.31
No symptomatic intracranial hemorrhage (%)	100	100	1.00
No serious adverse events (%)	100	100	1.00

FIGURE 1 Baseline characteristics, primary outcome.

within 48 hours. The evolocumab group ($n=96$, 100% Korean Asian, mean age 72 ± 10 years, 58% male) received subcutaneous evolocumab 140mg within 24 hours plus moderate-to-high-intensity statins (atorvastatin 40–80mg or rosuvastatin 20–40mg). Historical controls ($n=120$) received high-intensity statins alone, matched 1:1.25 for age (± 5 years), sex, and baseline NIHSS (± 2 points). The primary endpoint was END (NIHSS increase ≥ 2 points) within 7 days, assessed daily by certified neurologists blinded to treatment allocation.

Results: Baseline characteristics were balanced (baseline NIHSS 5.2 ± 3.1 vs 5.4 ± 3.3 , $p=0.63$). Evolocumab achieved superior LDL-C reduction versus controls ($149 \pm 32 \rightarrow 61 \pm 18$ mg/dL [59% reduction] vs $145 \pm 35 \rightarrow 89 \pm 24$ mg/dL [39% reduction]; between-group difference -32 mg/dL, 95% CI -40 to -24 , $p<0.001$) and greater hs-CRP reduction ($5.2 \pm 2.1 \rightarrow 2.8 \pm 1.5$ mg/L [46% reduction] vs $5.0 \pm 2.3 \rightarrow 4.1 \pm 1.9$ mg/L [18% reduction], $p<0.001$). END occurred in 8/96 (8.3%) versus 22/120 (18.3%) patients (unadjusted OR 0.41, 95% CI 0.17–0.95, $p=0.04$; adjusted OR 0.38, 95% CI 0.15–0.91, $p=0.03$), representing 55% relative risk reduction and a number-needed-to-treat of 10. Functional independence at 90 days was 71% versus 63% ($p=0.31$). No symptomatic intracranial hemorrhage or serious adverse events occurred.

Conclusion: Early evolocumab demonstrated potent dual lipid-lowering and anti-inflammatory effects, independently associated with reduced END.

Disclosure: Nothing to disclose.

EPO-0828 | Predictors and clinical impact of incomplete recanalization from endovascular treatment in acute ischemic stroke: A single-centre cohort study

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Background and aims: Poor recanalization at the end of endovascular treatment (EVT) despite successful catheter access

Disclosure: Nothing to disclose.

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Results: Clinically relevant thrombophilia testing was positive in 57/112 (50.9%). Management changed in 27/112 (24.1%) and was more frequent in thrombophilia-positive than thrombophilia-negative patients (46% vs 2%, $p < 0.001$). Thrombophilia-positive patients were younger (42.4 (4.9) vs 45.9 (6.1) years, $p = 0.001$)

Figure 1 consists of two bar charts. The top chart, titled 'Feedback Change Rate by Positive Item', shows the relationship between the number of positive feedback items (1 to 5) and the change rate. The y-axis is 'Feedback Change Rate (%)' ranging from 0 to 30. The x-axis is 'Number of Positive Items'. The change rate increases from approximately 18% for 1 item to 28% for 5 items. The bottom chart, titled 'Frequency Rate of (Nonnegative Item) (N=121)', shows the relationship between the number of nonnegative items (1 to 5) and the frequency rate. The y-axis is 'Frequency Rate (%)' ranging from 0 to 30. The x-axis is 'Number of Nonnegative Items'. The frequency rate increases from approximately 15% for 1 item to 25% for 5 items.

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	WHSS	TG/HDL	HDL	LDL	TG	Thrombophilic_pos_count	APS_ang_pos	SI_high
WHSS	1.00	0.80	-0.01	0.63	0.77	0.07	0.05	0.79
TG/HDL	0.80	1.00	-0.01	0.76	0.90	0.06	0.05	0.73
HDL	-0.01	-0.01	1.00	-0.01	-0.06	-0.12	-0.27	-0.01
LDL	0.63	0.76	-0.01	1.00	0.79	0.04	0.09	0.73
TG	0.77	0.90	-0.06	0.79	1.00	0.06	0.05	0.68
Thrombophilic_pos_count	0.07	0.06	-0.12	0.04	0.06	1.00	0.05	0.66
APS_ang_pos	0.05	0.05	-0.27	0.09	0.05	0.05	1.00	0.39
SI_high	0.79	0.73	-0.01	0.73	0.68	0.66	0.39	1.00

and had higher National Institutes of Health Stroke Scale (NIHSS) scores (7.8 (7.9) vs 1.8 (3.7), $p < 0.001$), more frequent SII-high status (40% vs 7%, $p < 0.001$), and higher TG/HDL-C

(11.7 (11.0) vs 4.2 (4.9), $p < 0.001$). Very high TG/HDL-C risk level was more common in the thrombophilia-positive group (53% vs 15%, $p < 0.001$). TG/HDL-C correlated strongly with NIHSS (Spearman correlation coefficient 0.85, $p < 0.001$).

Conclusion: In young cryptogenic ischemic stroke, clinically relevant thrombophilia findings were frequent and were associated with higher NIHSS and adverse atheroinflammatory profiles (SII and TG/HDL-C). The management impact of thrombophilia testing was higher than prior reports, suggesting a more favorable benefit-harm balance when testing is focused on selected young cryptogenic patients.

Disclosure: Nothing to disclose.

EPO-0830 | **Efficacy and safety of bridging intravenous thrombolysis prior to endovascular treatment in patients over 80years old with acute ischemic stroke**

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Background and aims: Endovascular treatment (EVT) is an effective treatment for patients with acute ischemic stroke (AIS). However, it is unclear if treatment with intravenous thrombolysis (IVT) prior to EVT confers any benefit in octogenarians and older. This study aimed to address if bridging tPA has improved functional outcomes or complications in patients 80years

Methods: This multicentre retrospective cohort study included patients 80years old and above who underwent endovascular therapy for large vessel occlusion acute ischaemic stroke in 10 compressive stroke centres across China and Singapore between 2018 and 2024. Clinical and procedural factors of patients in Singapore and China were compared using multivariate binary logistic regression. The primary outcome measured was achieving successful recanalization. Secondary outcomes included 3-month functional independence defined as modified rankin

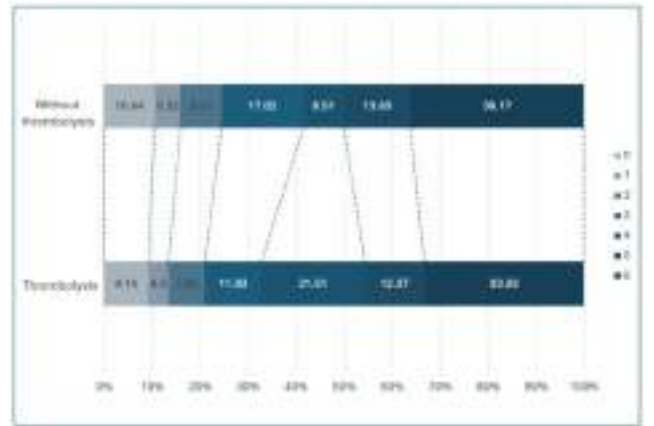


FIGURE 1 Comparison of modified rankin scale scores between patients with bridging intravenous thrombolysis prior to endovascular therapy and those without.

TABLE 1 Baseline characteristics of patients.

		Without Thrombolysis N=186	Thrombolysis N=94	P value
Age, Median (IQR)		84.0 (82.0,86.0)	84.9 (82.0,85.0)	0.251
Male sex, N (%)		79 (42.47)	42 (44.68)	0.725
Hypertension, N (%)		150 (81.08)	77 (81.91)	0.866
DM, N (%)		40 (21.51)	19 (20.21)	0.802
CAD, N (%)		47 (25.27)	25 (26.60)	0.810
Smoker history, N (%)		14 (7.53)	8 (8.51)	0.773
AF, N (%)		124 (66.67)	60 (63.83)	0.637
Prior stroke, N (%)		39 (20.97)	10 (10.64)	0.032
Dyslipidemia, N (%)		53 (28.57)	33 (35.11)	0.346
Pre-EVT NIHSS, Median (IQR)		18.0 (13.8,22.0)	16.5 (12.8, 21.0)	0.303
ASPECTS, Median (IQR)		9.0 (8.0, 10.0)	9.0 (8.0, 10.0)	0.897
CPT, min, Median (IQR)		315.0 (190.0, 648.5)	190.0 (143.3, 356.0)	0.008
Vessel	ICA	28 (13.98)	25 (26.60)	0.100
	M1	95 (51.08)	38 (40.43)	
	M2	22 (11.83)	10 (10.64)	
	ICA+M1	31 (16.67)	17 (18.09)	
	Basilar	12 (6.45)	4 (4.26)	

Abbreviations: Atrial Fibrillation - AF; Coronary Artery Disease - CAD; Diabetes Mellitus - DM; modified Thrombolysis in Cerebral Infarction - mTICI

TABLE 2 Comparing recanalization, safety and outcomes between elderly patients who underwent bridging thrombolysis and those who did not.

		Without Thrombolysis	Thrombolysis	P value
Successful recanalization		163 (87.63)	84(89.36)	0.672
Complete recanalization		121 (65.05)	57(60.64)	0.468
Parenchymal haematoma		23 (12.37)	13(13.83)	0.730
Any haemorrhage		49 (26.34)	36(38.30)	0.048
3-month functional independence (mRS 0-2)		39 (20.97)	23(24.47)	0.505
3-month independent ambulation (mRS 0-3)		61 (32.88)	39 (41.49)	0.512
3-month mortality		63(33.33)	34(36.17)	0.637
3-month mRS	0	17(9.14)	30(10.64)	0.298
	1	8(4.30)	5(5.32)	
	2	14(7.53)	8(8.51)	
	3	22(11.83)	30(17.02)	
	4	40(21.51)	8(8.51)	
	5	23(12.37)	13(13.83)	
	6	62(33.33)	34(36.17)	

scale (mRS) 0–2, 3-month independent ambulation as defined as mRS 0–3, and 3-month mortality rates. Data on complications such as parenchymal haematoma and any haemorrhage was also collected.

Results: Bridging IVT was not associated with increased rates of successful recanalisation (89.36% vs 87.63%; $p=0.672$), improvement in 3-month functional independence, improvement in 3-month independent ambulation or 3-month mortality rates. Instead, patients who underwent bridging IVT had higher rates of haemorrhage compared to patients who did not undergo bridging IVT even after adjusting for confounding factors (OR=1.921; 95% CI 1.026–3.596; $p=0.041$).

Conclusion: In patients over 80years old with AIS, bridging IVT is not associated with improved functional independence or reduced mortality at three months. However, it is associated with increased risk of hemorrhage.

Disclosure: Nothing to disclose.

EPO-0831 | Anticoagulation and stroke risk in dialysis patients with atrial fibrillation: A single-centre study in Malta

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Background and aims: Anticoagulation is central to stroke prevention in atrial fibrillation (AF), but its use in end-stage renal failure (ESRF) patients on dialysis is challenging. Warfarin has traditionally been used, though it is associated with labile INR, drug interactions, vascular calcification, and increased bleeding risk. Direct oral anticoagulants (DOACs), particularly apixaban, are increasingly prescribed off-label due to predictable pharmacokinetics and no need for routine monitoring, but evidence of their safety and efficacy in ESRF is limited and conflicting.

Methods: This single-centre retrospective study included all ESRF patients on chronic dialysis in Malta with documented AF. Data collected included demographics, comorbidities,

anticoagulation type (warfarin, DOAC, or none), and major bleeding events. Patients were followed for one year to assess cerebrovascular outcomes, including ischaemic stroke or transient ischemic attack (TIA).

Results: Of 358 ESRF patients, 43 (12%) had AF: 7 (16.3%) received apixaban, 30 (69.8%) warfarin, and 5 (11.6%) no anticoagulation. Preliminary findings show 3 patients (60%) without anticoagulation experienced a cerebrovascular event, while no events occurred in warfarin or apixaban groups. Labile INR was noted in 11 warfarin patients (36.7%), and 4 experienced major bleeding; no major bleeding occurred with apixaban.

Conclusion: These results suggest warfarin and apixaban may be similarly effective for stroke prevention in ESRF patients with AF, but warfarin carries a higher bleeding risk. Although limited by small sample size, this audit included the entire ESRF population in Malta, providing valuable insight into real-world anticoagulation practices in this high-risk group.

Disclosure: Nothing to disclose.

EPO-0832 | Diagnostic value of microbiological analysis of cerebral thrombi in infective endocarditis-related stroke: A tertiary-center descriptive study

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Background and aims: Infective endocarditis (IE) is an uncommon but severe cause of cardioembolic ischemic stroke. Since 2023, a positive microbiological analysis of the retrieved thrombus has been recognized as a pathological criterion for definite IE, enabling diagnosis to be established based on a single microbiological isolation.

Methods: Patients with ischemic stroke secondary to IE were included and divided into two groups according to the clinical sequence of diagnosis. Clinical, radiological, microbiological and prognostic characteristics were compared between groups (Table 1). The primary objective was to assess the diagnostic value of microbiological analysis of thrombi retrieved during mechanical thrombectomy.

Results: Seventeen patients were included (8 presenting with stroke as the first manifestation and 9 with a previous diagnosis of IE). The median age was 67years and 82.4% were male. Thrombus microbiological analysis was performed in 13 cases. In patients presenting with stroke as the first manifestation, thrombus cultures were positive in 100% of cases compared with 33.3% in patients with prior infective endocarditis ($p=0.021$), with 100% concordance with blood culture isolates (Table 2). Patients with stroke as the initial manifestation showed a higher proportion of favorable functional outcome at 3 months (mRS 0–2: 87.5% versus 44.4%), showing a trend toward statistical significance ($p=0.05$).

Conclusion: Microbiological analysis of cerebral thrombi may provide relevant diagnostic value in IE, particularly when stroke

TABLE 1 Characteristics of patients with ischemic stroke associated with infective endocarditis, stratified by study group: Group A (stroke as first clinical manifestation) and Group B (infective endocarditis diagnosed before stroke).

Baseline characteristics			
Variable	Group A	Group B	P
Age	63.5 (IQR 58.2–65.8)	69.0 (IQR 67.8–74.0)	0.194
Male sex	8 (100%)	6 (66.7%)	0.206
Hypertension	3 (37.5%)	7 (77.8%)	0.153
Diabetes mellitus	1 (12.5%)	4 (44.4%)	0.294
Dyslipidemia	3 (37.5%)	5 (55.6%)	0.335
Smoker	1 (12.5%)	2 (22.2%)	0.620
Atrial fibrillation	0 (0.0%)	4 (44.4%)	0.082
Coronary artery disease	0 (0.0%)	2 (22.2%)	0.471
Chronic kidney disease	0 (0.0%)	1 (11.1%)	1.000
Prosthetic heart valve	3 (37.5%)	4 (44.4%)	1.000
Implanted intracardiac device	1 (12.5%)	0 (0.0%)	1.000
Baseline mRS 0–2	8 (100%)	6 (100%)	1.000
Stroke characteristics			
Variable	Group A	Group B	P
NIHSS	13 (IQR 9–18)	11 (IQR 7–20)	0.87
Fever on admission	6 (75%)	4 (44.4%)	0.350
Wake up or unknown onset stroke	2 (25%)	6 (66.7%)	0.170
Thrombolysis	3 (37.5%)	1 (11.1%)	0.29
Pain and neuroimaging			
Variable	Group A	Group B	P
ASPECTS	8.5 (IQR 7–10)	9 (IQR 7–10)	0.79
Duration to reevaluation time (min)	13 (IQR 10–28)	14 (IQR 12–62)	0.9
Number of passes TICI 2b–3	2 (IQR 1–4.5)	1 (IQR 1–3)	0.48
TICI 2b–3	8 (100%)	8 (88.9%)	1.00
Complications			
Variable	Group A	Group B	P
Valvular abscess	0 (0.0%)	1 (11.1%)	1.00
Mycotic aneurysm	0 (0.0%)	1 (11.1%)	1.00
Acute heart failure	3 (37.5%)	5 (55.6%)	0.44
Valve surgery	2 (25.0%)	4 (44.4%)	0.63
Symptomatic intracranial hemorrhage	1 (12.5%)	0 (0.0%)	1.00
Mortality	1 (12.5%)	2 (22.2%)	1.00
Prognostic data			
Variable	Group A	Group B	P
Baseline NIHSS	11 (IQR 9–16)	11 (IQR 7–20)	0.87
NIHSS at discharge	3 (IQR 0–6)	3 (IQR 0–6)	1.00
mRS 0–2 at discharge	5 (62.5%)	3 (33.3%)	0.347
mRS 0–2 at 3 months	7 (87.5%)	4 (44.4%)	0.05

TABLE 2 Comparison of thrombus and blood culture microorganisms in endocarditis-related stroke by group (A: stroke first, B: endocarditis first). In Group A, thrombus culture showed 100% concordance with blood cultures.

	Thrombus culture	Blood culture
#01 – Group A	<i>Streptococcus equinus</i>	<i>Streptococcus equinus</i>
#02 – Group A	<i>Staphylococcus epidermidis</i>	<i>Staphylococcus epidermidis</i>
#03 – Group A	<i>Enterococcus faecalis</i>	<i>Enterococcus faecalis</i>
#04 – Group A	<i>Staphylococcus epidermidis</i>	<i>Staphylococcus epidermidis</i>
#05 – Group A	<i>Staphylococcus epidermidis</i>	<i>Staphylococcus epidermidis</i>
#06 – Group A	<i>Streptococcus faecalis</i>	<i>Streptococcus faecalis</i>
#07 – Group A	<i>Streptococcus gordonii</i>	<i>Streptococcus gordonii</i>
#08 – Group B	<i>Staphylococcus aureus</i> methicillin sensible	<i>Staphylococcus aureus</i> methicillin sensible
#09 – Group B	Negative	<i>Aerococcus urinae</i>
#10 – Group B	<i>Candida parapsilosis</i>	<i>Candida parapsilosis</i>
#11 – Group B	Negative	<i>Enterococcus faecalis</i>
#12 – Group B	Negative	<i>Streptococcus galloyticus</i>
#13 – Group B	Negative	<i>Staphylococcus aureus</i> methicillin sensible

is the first clinical manifestation. Early diagnosis of IE supported by thrombus analysis may enable earlier targeted treatment and be associated with improved long term functional outcomes.

Disclosure: Nothing to disclose.

EPO-0833 | Development of the GLaSSES score for predicting neurological deterioration after acute ischaemic stroke

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Background and aims: Patients with acute ischaemic stroke (AIS) may experience neurological deterioration (ND) within the first seven days, defined as an increase in the National Institutes of Health Stroke Scale (NIHSS) score. ND incidence ranges from 5% to 40% and is linked to increased morbidity and mortality. Despite ongoing research, no practical predictive tool is currently available.

Methods: We conducted a retrospective observational study of patients aged >18 years hospitalised for AIS (excluding reperfusion therapies). ND occurrence, causes, and associated mortality were recorded. A multivariable logistic regression model with forward stepwise selection identified independent predictors. Model performance was evaluated using receiver operating characteristic (ROC) curve analysis.

Results: Of 444 patients (mean age 71.3 years), ND occurred in 20.3%, mainly due to ischaemic core progression (46%). Five independent risk factors were identified: hyperglycaemia (OR 15.9, 95% CI 8.1–33.2), leukocytosis (OR 10.1, 95% CI 4.8–23.1), baseline NIHSS severity (OR 3.3, 95% CI 1.3–8.9), symptomatic intracranial stenosis (OR 3.8, 95% CI 2.5–5.5), and extracranial stenosis (OR 6.0, 95% CI 1.6–8.9). The GLaSSES score stratified patients into four risk categories: ND incidence 1.9% (very low),

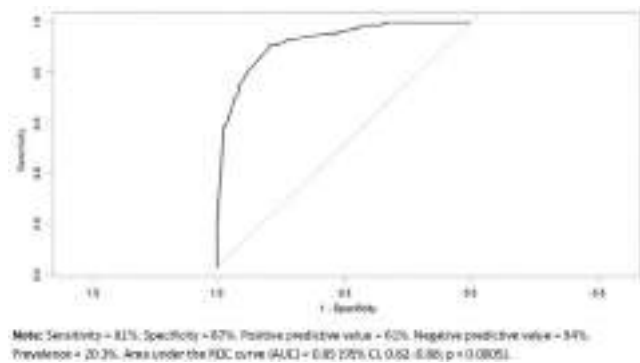


FIGURE 1 Receiver operating characteristic (ROC) curve of the predictive model.

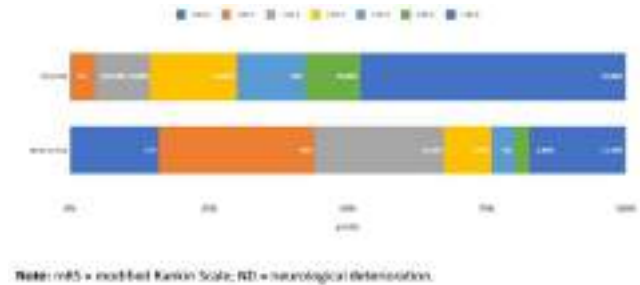


FIGURE 2 Distribution of the modified Rankin Scale score at 7 days in patients with neurological deterioration.

17.7% (low), 47.5% (moderate), 85.7% (high). The model showed good discrimination (AUC 0.85, 95% CI 0.82–0.88). Mortality was 47.8% in ND patients.

Conclusion: The GLaSSES score is a promising, simple tool for predicting ND risk in AIS patients. External validation in larger, prospective cohorts is warranted to confirm its clinical utility and improve outcomes.

Disclosure: Nothing to disclose.

EPO-0834 | Mechanical thrombectomy in patients with low ASPECTS: Real-world outcomes compared with SELECT-2 and LASTE

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Background and aims: Randomised trials (SELECT-2, LASTE) support mechanical thrombectomy (MT) in patients with large ischaemic core infarction defined by low ASPECTS (≤ 5). Real-world safety and functional outcomes remain less well described.

Methods: We conducted a retrospective observational study including consecutive patients with acute ischemic stroke and low ASPECTS treated with MT at a tertiary hospital.

Results: Sixty-six patients were included (mean age 67 years; 56.1% women). Median baseline NIHSS was 20 and median ASPECTS was 4. Intravenous fibrinolysis was given in 22.7%. Successful reperfusion (mTICI 2b-3) was achieved in 83.3%. sICH occurred in 10.6%. Median 90-day mRS was 4 and 18.8% achieved mRS 0–2. Baseline characteristics and functional outcomes were broadly comparable to SELECT-2 and LASTE, with similar reperfusion rates and functional status (both, median 90-day mRS of 4).

Conclusion: In routine practice, MT in patients with ASPECTS ≤ 5 achieved high reperfusion rates with functional outcomes consistent with trial populations, supporting external validity and ongoing implementation in carefully selected patients.

Disclosure: Nothing to disclose.

EPO-0835 | Cerebral hemodynamic signatures linking mild cognitive impairment and late-life depression: A multimodal transcranial doppler and MRI Study

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Background and aims: Late-life depression (LLD) and mild cognitive impairment (MCI) frequently coexist in older adults and share common neurovascular mechanisms. Cerebral small vessel dysfunction has been implicated in both cognitive and affective decline, yet clinically accessible markers linking these conditions remain limited. We investigated whether cerebral hemodynamic parameters assessed by transcranial Doppler (TCD), combined with MRI markers, identify shared neurovascular signatures between MCI and LLD.

Methods: In this cross-sectional study, 96 older adults with depressive symptoms underwent standardized cognitive assessment (MMSE-2, MoCA), affective evaluation (GDS-15), brain MRI with Fazekas scoring of white matter lesions (WMLs), and TCD examination of the middle cerebral artery. Hemodynamic parameters included mean blood flow velocity (MBFV), end-diastolic velocity (EDV), pulsatility index (PI), and resistive index (RI). Logistic regression and receiver operating characteristic (ROC) analyses identified independent predictors of MCI.

Results: Compared with cognitively unimpaired participants, individuals with MCI showed significantly lower MBFV and EDV and higher PI and RI (all $p < 0.05$). In multivariable analysis, altered MBFV, EDV, PI, and RI remained independently associated with MCI. RI demonstrated the highest discriminative accuracy (AUC=0.919), followed by MBFV (AUC=0.879). PI correlated with depressive symptom severity, while RI was associated with greater WML burden.



FIGURE 1 Transcranial Doppler ultrasound recording of the middle right cerebral artery (MCA). The spectrum shows peak systolic velocity (PSV), end-diastolic velocity (EDV), and mean blood flow velocity (MBFV) used for the calculation of the pulsatility index (PI) and resistivity index (RI).

TABLE 1 Predictors for MCI.

Univariate Logistic Regression	Odds Ratio	95% CI	p Value
Age (years)	0.88	0.79–0.97	0.01
GDS-15	1.66	1.04–2.66	0.03
MRI Fazekas visual scale	2.17	1.07–4.41	0.03
MBFV (cm/s)	0.64	0.49–0.84	<0.01
PSV (cm/s)	1.32	1.03–1.69	0.02
EDV (cm/s)	0.78	0.66–0.91	<0.01
PI (%)	0.79	0.63–1.00	0.04
RI (%)	5.4	2.97–9.88	0.02
Multivariate Logistic Regression	Odds Ratio	95% CI	p Value
MBFV (cm/s)	0.64	0.43–0.94	0.02
EDV (cm/s)	0.70	0.53–0.91	0.03
PI (%)	3.2	0.74–13.9	<0.01
RI (%)	1.9	0.22–2.66	<0.01

Abbreviations: MRI, magnetic resonance imaging; GDS-15, 15-item Geriatric Depression Scale; MBFV, mean blood flow velocity; PI, pulsatility index; RI, resistivity index; PSV, peak systolic blood flow velocity; EDV, end-diastolic blood flow velocity.

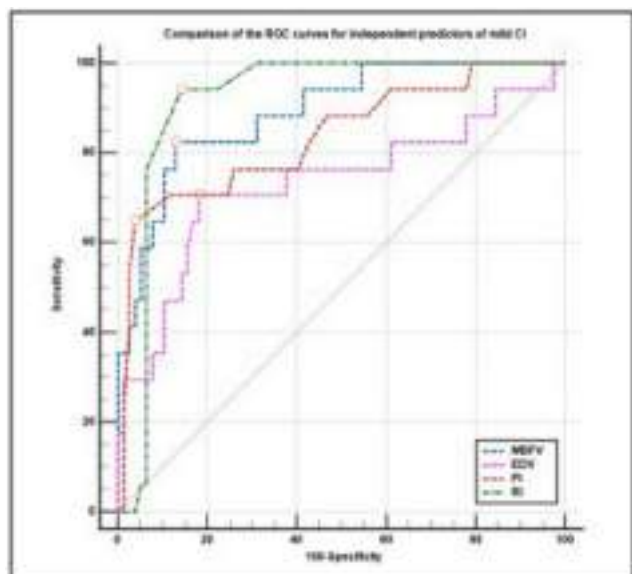


FIGURE 2 A comparative ROC curve of all four predictors.

Conclusion: Altered cerebral hemodynamics represent a shared neurovascular substrate linking MCI and late-life depression. Integrating TCD-derived indices with MRI markers offers a clinically feasible multimodal approach for early identification of vascular cognitive and affective vulnerability in older adults.
Disclosure: Nothing to disclose.

Neuro-Ophthalmology/Neuro-Otology 2

EPO-0836 | The vertigo challenge: Diagnostic variability across acute referral pathways

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Background and aims: Acute vertigo, a common emergency presentation, places considerable strain on acute care resources. Stroke-focused pathways drive extensive investigations, despite a low likelihood of a central cause. We evaluated diagnostic agreement between the emergency, stroke referral services, and specialist neuro-otology in acute vertigo patients to identify gaps and inform targeted strategies.

Methods: This single-centre retrospective cohort included new referrals with acute vertigo between August 2023 and July 2025. Of approximately 300 adult patients, 151 were analyzed. Data included referral source (ED, Acute Medicine Unit (AMU), Stroke Unit, TIA clinic, Neurology SDEC), vascular risk factors, imaging, and initial versus specialist diagnosis based on clinical history and imaging as a gold standard.

Results: Referrals originated from the ED/AMU (42.4%) and neuro-SDEC/stroke/TIA services (57.6%). Overall diagnostic agreement was 32% higher for neurology-led services (39.8%) than emergency services (23.5%). Overall, agreement was highest for stroke/TIA (75%) and lowest for chronic cardiovascular causes (6.7%). Additionally, 27.8% had no referral diagnosis, and most (65.56%) underwent neuroimaging.

Conclusion: Neurology-led teams showed higher diagnostic accuracy than emergency services, which have limited neurology expertise, and this may explain the variability. Structured triage systems are essential to reduce misclassification, avoid unnecessary imaging, and support frontline teams.

Disclosure: Nothing to disclose.

EPO-0837 | Tolosa-Hunt syndrome versus its mimics: Comparative analysis of clinical and radiological characteristics for accurate diagnosis

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Background and aims: Tolosa-Hunt syndrome (THS) is a rare cause of painful ophthalmoplegia, diagnosed by its steroid response [1]. However, its presentation is mimicked by serious pathologies, often leading to initial misdiagnosis. This study analyzes the key differences between true THS and its clinical mimics to improve diagnosis accuracy.

Methods: A systematic review was performed following PRISMA guidelines. Out of 22 studies, 15 were included. PubMed and BVS were searched using the keywords “Tolosa-Hunt syndrome,” “painful ophthalmoplegia,” and “cavernous sinus syndrome.” Data was extracted for comparative analysis of clinical and diagnostic variables.

Results: 222 patients, 125 females and 97 males, were included, with a mean age of 35.87 years. True THS accounted for 168 patients (75%). The other 58 cases were diagnosed with conditions including Rosai-Dorfman Disease and neoplastic conditions involving cavernous sinus and orbit. True THS has slight predominance in females (86, 52.4%) than in male (78, 47.6%). True THS was involved unilaterally (>90%), more frequent on the left side in majority of patients. MRI abnormalities identified in 90% of cases with true THS predominant to isolated cavernous sinus

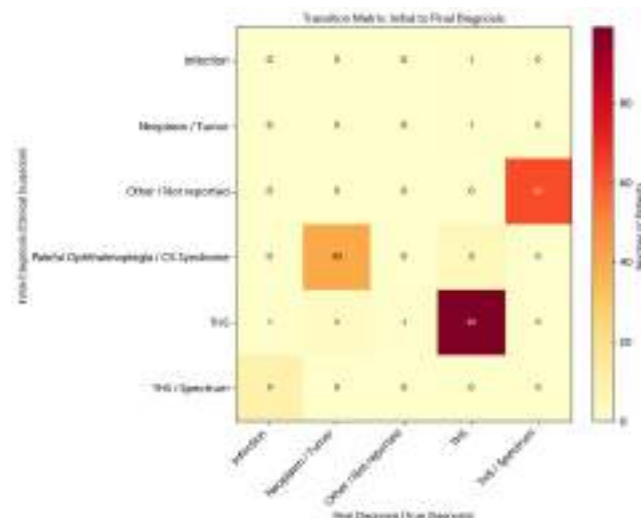


FIGURE 1 Initial diagnosis versus final diagnosis in clinical suspicion of Tolosa-Hunt Syndrome.

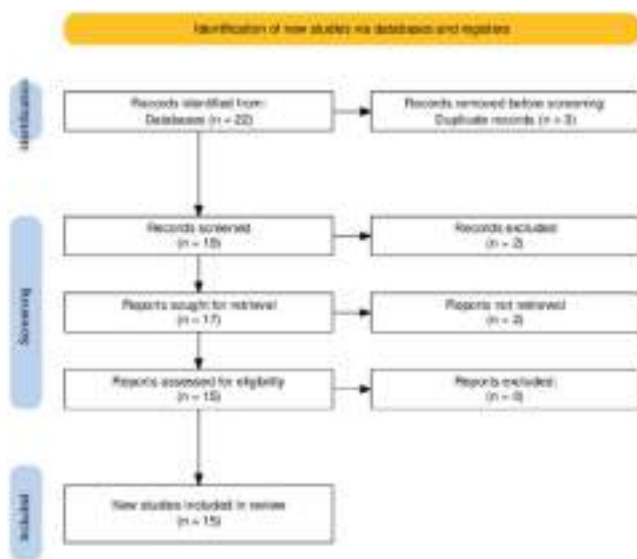


FIGURE 2 Prisma-P flowchart of the process of inclusion of studies in the systematic review.

inflammation; >95% of false TSH showed mass-like infiltrative lesions extending beyond the cavernous sinus. 85–90% of false cases demonstrated partial improvement after corticosteroid therapy, true cases showed >95% response and pain relief.

Conclusion: Most cases labeled as THS are true THS; but some represent neoplastic disorders. Persistent or bilateral symptoms, mass-like MRI lesions beyond the cavernous sinus, and incomplete steroid response suggest false-positive THS, supporting early reassessment and targeted testing.

Disclosure: Nothing to disclose.

EPO-0838 | Brain MRI abnormalities along the visual pathways in subacute leber hereditary optic neuropathy

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Background and aims: Brain MRI abnormalities along the visual pathways are historically considered an uncommon feature of LHON at difference with inflammatory optic neuritis. We aimed at describing the prevalence of such MRI abnormalities in subacute LHON patients, correlating with clinical and genetic characteristics.

Methods: We retrospectively analysed clinical, genetic and ophthalmological data from 40 genetically-confirmed LHON patients. MRI data performed within 6 months from onset were collected. Best corrected visual acuity (BCVA), retinal nerve fiber layer (RNFL) thickness and visual fields parameters were compared between patients with and without MRI abnormalities at time of MRI execution and at follow-up (≥ 24 months). Linear mixed models were applied to account for inter-eye correlation, with clinical and genetic predictors as covariates.

Results: MRI abnormalities along the visual pathways were found in 20/40 (50%) patients. Optic nerve hyperintensity was

present in 45%, chiasm and optic tract involvement in 20%, and contrast enhancement of the optic nerve in 22%. At time of MRI execution, optic nerve hyperintensity resulted significantly associated with worse BCVA and visual field parameters, while optic nerve contrast enhancement was selectively associated with thinner temporal RNFL. At follow-up (25 patients), long-term BCVA was significantly influenced by mutation type, with rare mutations showing better outcomes, while MRI abnormalities were not prognostic. As exploratory analysis, high-dose steroid therapy at onset showed mild influence on functional parameters at follow-up.

Conclusion: MRI abnormalities are frequent in LHON and likely reflect early axonal damage and possibly an ongoing inflammatory component, but their independent prognostic value is still under evaluation.

Disclosure: Nothing to disclose.

EPO-0839 | Saccadic velocity dynamics abnormalities and levodopa-induced dyskinesia in Parkinson's disease: An exploratory study

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Background and aims: Oculomotor abnormalities were described in Parkinson's disease (PD) as an expression of disease-related basal ganglia dysfunction, showing coherent relations with the disease's clinical features. Nevertheless, the possible relation between saccadic performances and levodopa-induced dyskinesia (LID) has never been addressed to date. This exploratory study aimed to investigate possible associations between saccadic eye movements features and LID in PD.

Methods: Horizontal and vertical visually-guided saccades (VGS) were analysed through Eyelink 1000 Plus in healthy controls (HCs), PD without LID (PD-LID-) and PD with LID (PD-LID+) in their OFF motor state. The following VGS metrics were assessed: latency, amplitude, duration, mean and peak velocity, gain. VGS dynamics were also assessed by: peak velocity/amplitude ratio, reflecting main sequence linear relation, and Q-ratio (peak/mean velocity ratio), used to quantify and detect abnormalities of VGS velocity profile (i.e. higher Q-ratio).

Results: Thirty-two HCs, $N=16$ PD-LID- and $N=26$ PD-LID+ were enrolled. After adjusting by possible confounders, lower horizontal and vertical VGS amplitude, mean, peak velocity and gain were found in PD-LID- as compared to HCs while no significant differences on VGS dynamics were found. On the other hand, reduced amplitude and gain as well as increased peak velocity/amplitude and Q-ratios were shown in PD-LID+ as compared to HCs. Significantly higher peak velocities, peak velocity/amplitude and Q-ratios were demonstrated in PD-LID+ as compared to PD-LID-.

Conclusion: This exploratory study firstly highlights the association between saccadic velocity dynamics abnormalities as assessed in the OFF motor state and LID in PD. Clinical applications should be further investigated.

Disclosure: Nothing to disclose.

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Background and aims: This study investigated vestibular function in Alzheimer's disease (AD) and amnesic mild cognitive impairment (aMCI), to examine the prevalence of vestibular impairment and its association with domain-specific cognitive decline.

Methods: The study included 91 participants: 30 AD, 30 aMCI, and 31 controls. All participants underwent neuropsychological testing and vestibular assessments using video Head Impulse Test (vHIT) for semicircular canals (SCCs) and vestibular-evoked myogenic potentials (VEMPs) for otolith function. Overall vestibular scores were defined as abnormal results on ≥ 2 of 10 subtests. Cognitive domains assessed included memory, executive function, attention, visuospatial, and language (Table 1).

Results: (1) Abnormal vHIT results were significantly higher in AD and aMCI (50% each) versus controls (12.9%; $p=0.002$)

TABLE 1 The descriptive statistics of the demographics and neuropsychological tests.

Demographics	HCA (n = 31)	aMCI (n = 30)	AD (n = 30)	
Age	67.77 $\pm$ 7.08	71.39 $\pm$ 5.55	71.43 $\pm$ 7.57	[F(2,88) = 2.846, $p=0.062$]
Sex (F/M)	20/11	18/12	15/15	[$\chi^2(2) = 3.378$, $p=0.302$]
Education	12.42 $\pm$ 3.24	9.27 $\pm$ 3.53	9.03 $\pm$ 3.44	[H(2) = 9.909, $p=0.007$]
yGDS	6.48 $\pm$ 4.99	9.10 $\pm$ 3.29	6.56 $\pm$ 3.87	[H(2) = 3.132, $p=0.076$]
BAI	4.13 $\pm$ 4.07	4.30 $\pm$ 4.79	2.46 $\pm$ 3.08	[H(2) = 3.319, $p=0.070$]
Disease duration (mo)	-	25.97 $\pm$ 22.47	23.00 $\pm$ 13.75	[U = 411.000, $z=-0.389$, $p=0.550$, $r=-0.08$]

HCA, healthy controls; aMCI, amnesic mild cognitive impairment; AD, Alzheimer's disease; F, female; M, male; yGDS, Yesavage Geriatric Depression Scale; BAI, Beck Anxiety Inventory; mo, month.

TABLE 2 The proportions of subjects with abnormal responses in vestibular tests.

All vestibular tests	HCA (n = 31)	aMCI (n = 30)	AD (n = 30)	
Overall vestibular impairment score	14 (45.16%)	20 (66.67%)	19 (63.33%)	$\chi^2(2) = 3.796$, $p=0.183$
Video Head Impulse Test				
Overall vHIT	4 (12.9%)	15 (50%)	15 (50%)	$\chi^2(2) = 11.819$, $p=0.002$
Adjusted residual	3.47	1.75	1.78	
p	0.009	0.062	0.062	significant at $p < 0.009$
Lateral vHIT	0 (0%)	6 (20%)	4 (13.3%)	$\chi^2(2) = 6.086$, $p=0.049$
Adjusted residual	2.8	1.9	0.3	
p	0.098	0.089	0.517	significant at $p < 0.009$
Anterior vHIT	2 (6.3%)	7 (23.3%)	6 (20%)	$\chi^2(2) = 3.598$, $p=0.169$
Posterior vHIT	6 (19.3%)	9 (30%)	12 (40%)	$\chi^2(2) = 5.762$, $p=0.056$
Vestibular-evoked myogenic potentials				
VEMPs total	17 (54.8%)	23 (76.7%)	19 (63.3%)	$\chi^2(2) = 3.206$, $p=0.199$
cVEMP total	13 (41.9%)	16 (53.3%)	17 (56.7%)	$\chi^2(2) = 0.423$, $p=0.808$
cVEMP AC	14 (45.2%)	19 (63.3%)	10 (33.3%)	$\chi^2(2) = 3.223$, $p=0.074$
cVEMP BC	23 (74.2%)	25 (83.3%)	22 (73.3%)	$\chi^2(2) = 1.042$, $p=0.599$
sVEMP total	11 (35.5%)	17 (56.7%)	11 (36.7%)	$\chi^2(2) = 2.807$, $p=0.245$
sVEMP AC	13 (41.9%)	20 (66.7%)	20 (66.7%)	$\chi^2(2) = 3.596$, $p=0.169$
sVEMP BC	28 (90.3%)	22 (73.3%)	21 (70%)	$\chi^2(2) = 0.569$, $p=0.752$

HCA, healthy controls; aMCI, amnesic mild cognitive impairment; AD, Alzheimer's disease; vHIT, video Head Impulse Test; VEMPs, vestibular-evoked myogenic potentials; cVEMP, cervical VEMP; AC, air-conducted; BC, bone-conducted; sVEMP, ocular VEMP; VEMPs total indicates overall cVEMP total and sVEMP total responses. cVEMP total indicates overall cVEMP AC and BC responses. sVEMP total indicates overall sVEMP AC and BC responses.

while VEMPs and overall vestibular scores showed no group differences; (2) Specific vestibular impairments were associated with distinct cognitive domains; (2a) in AD with vestibular impairment (ADwVi), memory linked to posterior SCC impairment and visuospatial to overall vestibular scores ($p=0.029$). In aMCI with vestibular impairment (aMCIwVi), visuospatial associated with abnormal ocular VEMP (oVEMP; $p=0.046$); (2b) oVEMP showed contrasting associations: language in aMCIwVi ($p=0.009$) versus, Mini-Mental State Examination (MMSE) and executive function in AD without vestibular impairment ($p=0.033$ and $p=0.023$, respectively); and (3) the overall vestibular scores, though not differing between groups ($p=0.063$), correlated with memory, visuospatial, language performance, and age (Table 2).

Conclusion: These findings indicate subclinical vestibular impairment, particularly involving SCCs, is prominent in AD and aMCI. Specific vestibular-cognitive associations suggest vestibular assessment could serve as a valuable supplementary tool for identifying and monitoring cognitive decline in at-risk populations.

Disclosure: Nothing to disclose.

EPO-0841 | Can perfusion-weighted imaging improve diagnostic accuracy in transient vascular vertigo?

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Background and aims: Transient vestibular syndrome (TVS) is a frequent manifestation of transient ischemic attacks (TIA) in the vertebrobasilar territory. However, the underlying etiology often remains unidentified in more than half of these patients, even with detailed neuro-otological examinations and standard MRI, including diffusion-weighted imaging (DWI). The aim of this study is to evaluate the clinical utility of perfusion-weighted imaging (PWI) in diagnosing acute transient vascular vertigo (TVV).

Methods: We retrospectively reviewed patients with recurrent transient vertigo/dizziness between October 2023 and November 2025. Inclusion criteria were: no alternative vestibular diagnosis, negative findings on initial brain MRI/DWI, and evaluation within 7 days of onset. Comprehensive neuro-otological assessments (VOG, vHIT, cVEMP, SVV/SVH), lipid profiles, and PWI were analyzed.

Results: Thirty-one patients were included (mean age 67 ± 13 years; 14 males). While all patients were DWI-negative, PWI revealed perfusion deficits in 18 patients (58%). Posterior circulation vascular stenosis or hypoplasia was identified in 24 patients (77%). Regarding neuro-otological findings, none showed spontaneous nystagmus or skew deviation, though apogeotropic nystagmus was observed in 12 patients. Abnormal cVEMP and vHIT were found in 13% and 22.5% of patients, respectively. All patients possessed at least three vascular risk factors, most commonly diabetes and dyslipidemia. Notably, 7 patients (22.6%) progressed to cerebral infarction within one week.

Conclusion: PWI is a valuable diagnostic tool for identifying transient vascular vertigo, especially when DWI is negative. The

presence of posterior circulation stenosis and multiple vascular risk factors should raise clinical suspicion of TVV to prevent subsequent stroke.

Disclosure: Nothing to disclose.

EPO-0842 | Interpretation of HINTS findings in idiopathic sudden sensorineural hearing loss with vertigo

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Background and aims: The head impulse-nystagmus-test of skew (HINTS) examination is widely used to distinguish peripheral from central causes of acute vestibular syndrome, but its diagnostic performance in idiopathic sudden sensorineural hearing loss (SSNHL) with vertigo remains uncertain. This study aimed to assess the accuracy and clinical utility of the HINTS examination in patients with idiopathic SSNHL accompanied by vertigo.

Methods: We prospectively evaluated 121 patients with idiopathic SSNHL and vertigo at two university hospitals. The patients underwent detailed neuro-otological examinations, including nystagmus assessment, caloric testing, head impulse tests (HITs), and, as well as brain MRI to exclude central lesions. HINTS findings were classified as “peripheral” or “central” according to established criteria, and false-positive and true-negative rates were calculated.

Results: Spontaneous nystagmus (SN) was present in 59 patients (49%), with no cases of direction-changing nystagmus and only one case of skew deviation. Abnormal caloric responses were observed in 34/71 (48%), whereas abnormal horizontal HIT findings occurred in 35/119 (29%). Concordance between the two tests was noted in 80% of patients, while 17% showed abnormal caloric responses despite normal horizontal HIT. Among 59 patients with SN, 36 (61%) demonstrated “central” HINTS patterns despite normal brain MRI findings, indicating a high false-positive rate. Misclassification was significantly more frequent in patients with ipsilesional SN than in those with contralesional SN (89% vs 31%, $p < 0.001$).

Conclusion: In idiopathic SSNHL with vertigo, the HINTS examination frequently yields central patterns despite peripheral pathology, reflecting limited specificity. Thus, careful interpretation with complementary audiovestibular testing is required for accurate diagnosis.

Disclosure: Nothing to disclose.

EPO-0843 | When LHON mimics demyelination: Area postrema syndrome in Biallelic DNAJC30 variant

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Background and aims: Biallelic pathogenic variants in a nuclear-encoded gene DNAJC30 cause autosomal recessive form of Leber hereditary optic neuropathy (arLHON), commonly considered to be mitochondrially inherited. Although most affected individuals present with isolated optic neuropathy, further research expanded phenotypic spectrum of diseases linked to DNAJC30. We report a patient with LHON and area postrema syndrome (APS).

Methods: 13-year-old patient was referred due to blurred vision with central scotoma of the left eye, recurrent vertigo, and vomiting for 2 months. He presented horizontal gaze-evoked nystagmus and convergence insufficiency of the left eye. Ophthalmological evaluation showed bilateral papilledema with telangiectatic vessels and significant vision impairment of the left eye. Laboratory tests did not detect any specific antibodies. MRI identified brain stem T2 high-signal lesions of suspected inflammatory etiology. Thus, intravenous steroids were commenced, followed by immunoglobulin therapy, without significant vision improvement. Follow-up MRI showed T2 high-signal lesions in AP and optic nerves enhancement, indicative for NMOSD. Despite further immunosuppressive treatment visual function did not improve. On the contrary, progressive vision impairment was observed. Hence, whole exome sequencing was performed, identifying homozygous pathogenic DNAJC30 variant c.152A>G, p.(Tyr51Cys).

Results: The patient was diagnosed with arLHON with symptoms of APS and provided with idebenone, resulting in significant vision improvement.

Conclusion: DNAJC30-associated disease may mimic neuromyelitis optica spectrum disorder (NMOSD). We highlight the need of early genetic diagnosis in patients with optic neuropathy and APS who do not respond to immunotherapy, as targeted treatment with idebenone may lead to significant clinical and radiological improvement.

Disclosure: Nothing to disclose.

EPO-0844 | When atypical is common: Rethinking optic neuritis in multiple sclerosis

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Background and aims: Optic neuritis (ON) is a classic manifestation of multiple sclerosis (MS). If atypical features are present (AON), these generally suggest a distinct underlying etiology from MS. We aimed to ascertain the prevalence of AON in MS and to further investigate it as a subgroup.

Methods: A retrospective analysis of clinical/paraclinical data from MS patients who presented with acute optic neuritis in our department was conducted. All patients underwent

neuro-ophthalmic observation, retinography, optical coherence tomography, and perimetry at baseline, and 1- and 6-month follow-ups. These were further separated into typical (TON) and AON groups.

Results: We included 62 patients (47 females [75.8%]; mean age 38.9 ± 11.9). AON was present in 62.9% patients. If not considering lack of pain as an atypical feature, AON was still present in 40.3%. Visual acuity was $\leq 20/400$ at baseline, $\leq 20/200$ at 1-month and $\leq 20/100$ at 6-months, in 21%, 4.8% and 4.8% patients, respectively. Ten (16.1%) patients were ≥ 50 years old at the time of optic neuritis. There was no pain in 35.5% patients. There was severe optic disc edema in 3.2%, and peripapillary hemorrhages in 1 (1.6%) patient. Four (6.5%) patients had bilateral involvement. In 3.2% patients, chiasmal involvement was documented. EDSS score, disease duration and number of relapses in the last two years did not significantly differ between groups.

Conclusion: Approximately 40% of MS patients show ≥ 1 atypical feature at the time of their optic neuritis. While AON still requires the active exclusion of an alternative diagnosis, atypical optic neuritis is not uncommon in MS.

Disclosure: Nothing to disclose.

EPO-0845 | **Pupillodynamics: A potential indicator of autonomic dysfunction in Parkinson disease**

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Background and aims: The pupillary light reflex is an important metric of autonomic function. In Parkinson's disease (PD) autonomic function is often impaired, even in early stages of the disease. Therefore, we aimed to identify disease specific alterations by dynamic pupillometry.

Methods: Dynamic pupillometric parameters were assessed in 116 patients with PD and 188 healthy controls (HC) using

TABLE 1 Group differences in pupillometric parameters between Parkinson's disease and healthy controls adjusted for age and sex. *F*, *F* statistic from the general linear model; η^2 , partial eta squared (effect size); SE, standard error.

Parameter	Healthy controls Mean $\pm$ SE	Parkinson disease Mean $\pm$ SE	<i>F</i> (1, df)	<i>p</i>	η^2
Baseline pupil size after dark adaptation (mm)	3.689 $\pm$ 0.047	3.648 $\pm$ 0.053	4.254	.040	0.015
Pupil size after light exposure (mm)	2.329 $\pm$ 0.028	2.384 $\pm$ 0.033	28.133	<.001	0.093
Minimal pupil size at 10 s (mm)	2.887 $\pm$ 0.032	2.870 $\pm$ 0.043	28.178	<.001	0.093
Minimal pupil size at 15 s (mm)	2.681 $\pm$ 0.021	2.833 $\pm$ 0.039	66.965	<.001	0.137
Changing ratio dilation 5–8 s	0.375 $\pm$ 0.006	0.417 $\pm$ 0.013	10.218	.001	0.035
Changing ratio dilation 10–12 s	0.327 $\pm$ 0.007	0.339 $\pm$ 0.008	0.031	.860	0.000
Changing ratio constriction 2–5 s	0.334 $\pm$ 0.005	0.327 $\pm$ 0.006	0.896	.347	0.003
Changing ratio constriction 8–10 s	0.262 $\pm$ 0.004	0.232 $\pm$ 0.005	16.885	<.001	0.056
Changing ratio constriction 13–15 s	0.277 $\pm$ 0.004	0.231 $\pm$ 0.006	36.004	<.001	0.120
Peak velocity dilation (mm/s)	1.549 $\pm$ 0.042	1.279 $\pm$ 0.063	5.948	.018	0.021
Peak velocity constriction (mm/s)	−4.219 $\pm$ 0.080	−4.139 $\pm$ 0.075	0.642	.422	0.002
Peak acceleration dilation (mm/s ²)	11.193 $\pm$ 0.435	10.556 $\pm$ 0.538	0.828	.364	0.003
Peak acceleration constriction (mm/s ²)	−39.936 $\pm$ 3.490	−38.521 $\pm$ 0.822	0.396	.532	0.001
Latency dilation (ms)	552.4 $\pm$ 20.8	537.0 $\pm$ 28.1	1.706	.196	0.006
Latency constriction (ms)	385.3 $\pm$ 2.9	376.1 $\pm$ 3.7	2.248	.135	0.008

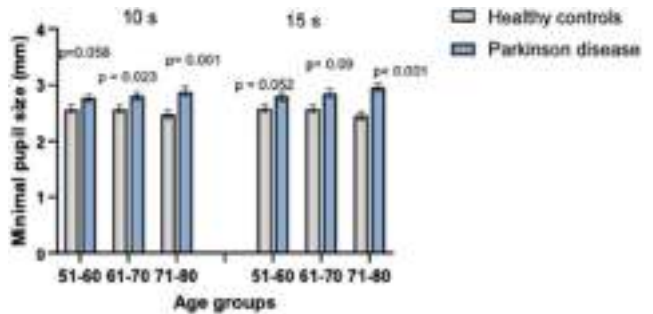


FIGURE 1 Age-stratified comparison of minimal pupil size at 10 and 15 s in HC and PD. Bars represent estimated marginal means $\pm$ SE. Group sizes: 51–60 years: HC *n* = 27, PD *n* = 29; 61–70 years: HC *n* = 37, PD *n* = 37; 71–80 years: HC *n* = 29, PD *n* = 31.

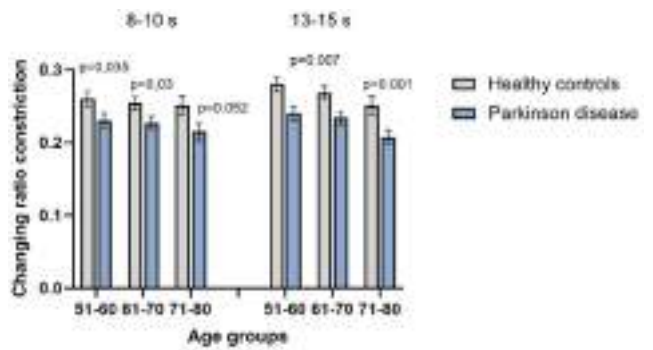


FIGURE 2 Age-stratified comparison of constriction dynamics (8–10 s and 13–15 s) in HC and PD. Bars represent estimated marginal means $\pm$ SEM. Group sizes: 51–60 years: HC *n* = 27, PD *n* = 29; 61–70 years: HC *n* = 37, PD *n* = 37; 71–80 years: HC *n* = 29, PD *n* = 31.

BulbiCamTM. Group differences were analyzed with univariate general linear models for each pupillometric parameter, including diagnosis as the between-subject factor and age and sex as covariates. Additional age-stratified analyses were performed within the 51–60, 61–70, and 71–80 years subgroups.

Results: Patients with PD showed larger baseline pupil size after dark adaptation and after light exposure, as well as significantly larger minimal pupil size at 10 and 15 s, indicating reduced and less sustained pupillary constriction (medium to large effect sizes, η^2 up to 0.137). In PD pupillary dynamics were selectively altered, with markedly reduced late phase constriction (8–10 and 13–15 s). Peak velocity of dilatation was modestly reduced in PD, whereas peak acceleration and pupillary latencies did not differ significantly between groups (Table). Age-stratified analyses demonstrated that group differences in tonic pupil size and minimal pupil diameter were preserved or enhanced in older age groups (Figure 1), while impaired late phase pupillary constriction were consistently present across all age strata (Fig. 2).

Conclusion: PD is associated with selective alterations of pupillary control, predominantly affecting pupil size regulation and late-phase pupillary constriction. These findings support disease-specific autonomic pupillary dysfunction in PD.

Disclosure: M. Strupp is Joint Chief Editor of the Journal of Neurology, Editor in Chief of Frontiers of Neuro-otology and Section Editor of F1000. He has received speaker's honoraria

from Abbott, Auris Medical, Biogen, Eisai, Grünenthal, GSK, Henning Pharma, Interacoustics, J&J, MSD, NeuroUpdate, Otometrics, Pierre-Fabre, TEVA, UCB, and Viartis. He received support for clinical studies from Decibel, U.S.A, Cure within Reach, U.S.A. and Heel, Germany. He distributes “M-glasses” and “Positional vertigo App”. He acts as a consultant for Abbott, AurisMedical, Bulbitech, Heel, Sensorion, Vifor and Vertify. He is a scientific founder, investor and share-holder of IntraBio.

EPO-0846 | Translating RNFL thickness into papilledema grades in idiopathic intracranial hypertension

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Background and aims: The Frisén scale is widely used to grade papilledema, but it remains subjective with notable inter-observer variability. Quantitative OCT provides objective and reproducible assessment of papilledema with micrometer-level precision. This study aimed to define retinal nerve fiber layer (RNFL) thickness thresholds that correspond to modified Frisén papilledema grades.

Methods: A total of 49 patients with newly diagnosed idiopathic intracranial hypertension (98 eyes) were followed for up to two years. 64 healthy controls were included. RNFL thickness was compared across papilledema grades, and ROC was applied to identify cut-off values.

Results: RNFL thickness correlated strongly with papilledema levels according Frisén grading and moderately with intracranial pressure. RNFL thickness increased progressively with higher papilledema grades and differed significantly between all grade groups ($p < 0.001$). Threshold values of approximately 105, 140, 232, and 400 μm distinguished Grade 0 from I, I from II, II from III, and III from IV, respectively, with AUCs ranging from 0.77 to 0.95.

Conclusion: The results demonstrate that RNFL measurements can be translated into clinically meaningful papilledema severity levels. Defining RNFL-based cut-offs offers a more objective, sensitive, precise and reproducible approach to grading papilledema, supporting improved monitoring and management in IIH.

Disclosure: Nothing to disclose.

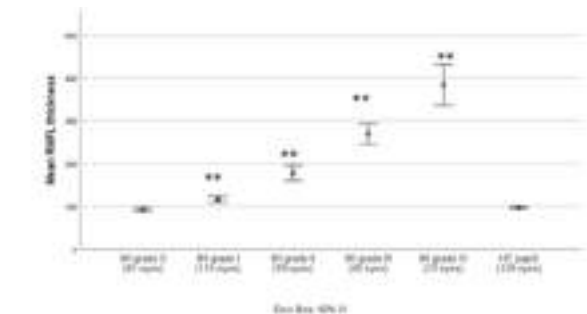


Figure 1. Mean retinal nerve fiber layer (RNFL) thickness in eyes with idiopathic intracranial hypertension (IIH) across different papilledema grades (0-IV) and in healthy controls (HC). RNFL thickness increased progressively with papilledema severity, with significant differences observed between grade groups. Statistical comparisons were performed using one-way ANOVA with post hoc Bonferroni correction. Error bars indicate 95% confidence intervals. ** $p < 0.01$ versus HC.

Spinal Cord and Root Disorders

EPO-0847 | Spinal cord compression due to plexiform neurofibromas in NF1

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Background and aims: Neurofibromatosis type 1 (NF1) is an autosomal dominant neurocutaneous disorder caused by mutations in the NF1 gene. It is characterised by Schwann cell proliferation, leading to cutaneous neurofibromas and plexiform neurofibromas, which may cause neurological complications due to mass effect.

Methods: We present the case of a 26-year-old male with a known diagnosis of NF1 who presented with a six-month history of intermittent, sudden-onset jerking sensations affecting the chest and upper limbs, associated with pins and needles and tingling. Clinical examination and neuroimaging findings were reviewed.

Results: Neurological examination revealed multiple cutaneous neurofibromas, particularly over both wrists, with bilateral thenar eminence wasting. Muscle tone was normal, with preserved power except for weakness of thumb abduction. Upper limb reflexes were brisk. In the lower limbs, there was pyramidal-pattern weakness with an absent left ankle reflex. MRI of the brain and spine demonstrated extensive cervical plexiform neurofibromas causing compression of the cervical spinal cord at the C2–3 level, with no radiological evidence of malignant transformation.

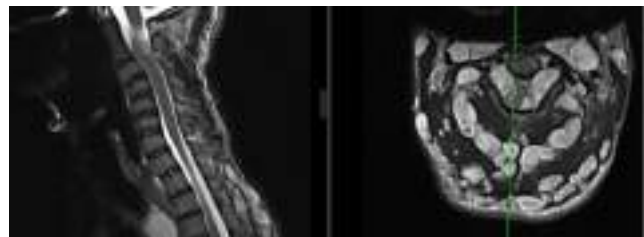


FIGURE 1 Sagittal T2 and axial T2 showing distortion and compression of cervical cord.

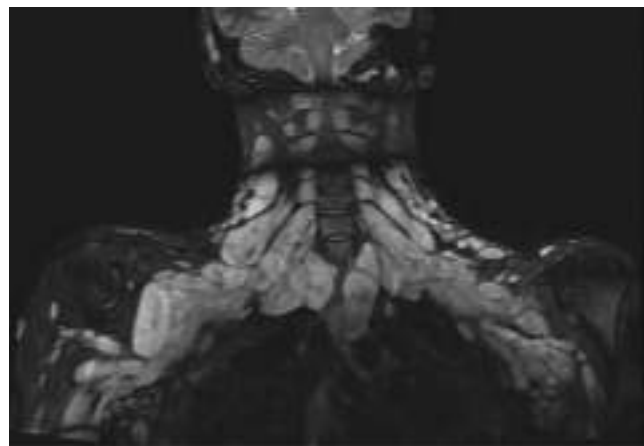


FIGURE 2 Brachial plexus STIR view showing huge large plexiform neurofibromas.

Conclusion: NF1 is an important cause of nerve root hypertrophy and spinal cord compression. Plexiform neurofibromas can result in significant cervical cord compromise, highlighting the need for early recognition and surveillance imaging in symptomatic patients.

Disclosure: Nothing to disclose.

EPO-0848 | A systematic review of the natural history of degenerative thoracic myelopathy

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Background and aims: Degenerative thoracic myelopathy (DTM), a recently proposed term, encompasses a spectrum of degenerative spine conditions that cause thoracic spinal cord compression and progressive neurological dysfunction. The natural history of DTM is poorly characterised, hindering prompt diagnosis and management. This systematic review aimed to assess the initial symptoms, progression, and prognostic factors. Studying DTM as a unified condition provides a larger body of evidence to draw conclusions from, analogous to Degenerative Cervical Myelopathy.

Methods: A systematic literature search was performed in Embase and MEDLINE, identifying papers assessing DTM. Relevant outcomes included initial symptoms, symptom progression, duration of disease and prognostic factors. This review followed PRISMA guidelines.

Results: Sixteen studies met the inclusion criteria. Sensory disturbances were more common than motor symptoms in the early stages of DTM, with all 8 studies reporting initial symptoms finding that numbness, tingling or pain were most common. Gait disturbances and motor symptoms such as weakness were commonly reported later in disease progression. The prevalence of symptoms increased over time. Symptom progression was highly variable, with a mean duration from symptom onset to surgery of 25.5 months. Risk factors for progression include the underlying aetiology, age and the presence of T2 hyperintensity on spinal MRI.

Conclusion: Despite a paucity of literature on the natural history of DTM, this review identifies several patterns which provide actionable guidance for clinicians. Recognising early symptoms may reduce diagnostic delay, prognostic features can inform risk-stratified surveillance and understanding progression may inform treatment thresholds. Importantly, diagnostic delay is associated with poorer post-operative outcomes in DTM.

Disclosure: Nothing to disclose.

EPO-0849 | Abstract withdrawn

EPO-0850 | Clinical characteristics of toxocara canis myelitis in Lebanon: Largest single center cohort study

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Background and aims: Neurotoxocariasis is a rare manifestation of human toxocariasis presenting as myelitis and literature is limited to small case series. This study describes the clinical characteristics, diagnostic findings, treatment results, and prognostic factors of Toxocara-associated myelitis.

Methods: This is a retrospective cohort study conducted at a tertiary center, including 44 patients diagnosed with Toxocara myelitis between 1998 and 2025. Diagnosis was based on clinical presentation, magnetic resonance imaging (MRI), serology, cerebrospinal fluid (CSF), eosinophilia, and treatment response. Clinical improvement was defined as $\geq 50\%$ or $< 50\%$ from baseline. Predictors of outcome were obtained by bivariate and multivariate analyses.

Results: Majority of patients were males (84%) with median age 41 years. All patients were ambulatory at baseline with sensory symptoms, however autonomic dysfunction and motor weakness were present in 59% and 57% of cases, respectively. Peripheral eosinophilia was in 46%, CSF pleocytosis in 21%, CSF eosinophilia in 5% and CSF oligoclonal bands in 44%. 95% had positive serology in blood and/or CSF. All patients were treated with Albendazole, 82 % also received corticosteroids. Of the 41 reevaluated patients: 71% improved, and 51% attained $\geq 50\%$ recovery. Early Albendazole initiation was the only predictor of a favorable outcome (median 10 vs. 32 weeks from symptom onset; $p=0.012$), confirmed controlling for age, sex, and treatment with corticosteroids. Importantly, demographic characteristics, clinical presentation, laboratory data, and corticosteroid use were not associated with recovery.

Conclusion: Toxocara myelitis is considered a potentially reversible subacute myelitis. The most important factor for neurological recovery is the early diagnosis and administration of albendazole.

Disclosure: Nothing to disclose.

EPO-0851 | Paraneoplastic sensory ganglionopathy as the initial manifestation of a small pancreatic neuroendocrine tumor

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Background and aims: Paraneoplastic neurological syndromes (PNS) are rare immune-mediated disorders, affecting fewer than 1% of patients with cancer, and may precede the diagnosis of an underlying malignancy. Sensory ganglionopathy is a severe and disabling manifestation, classically associated with small-cell lung carcinoma but also described in other tumors, including neuroendocrine neoplasms. The frequent absence of detectable onconeural antibodies may delay diagnosis, emphasizing the importance of systematic malignancy screening in unexplained sensory neuronopathies.

Methods: We report a 56-year-old woman presenting with subacute progressive sensory ataxia, marked proprioceptive loss, generalized areflexia, postural instability, and functional dependence. Motor weakness was predominantly secondary to sensory deafferentation. Cranial nerve examination were normal.

Results: Brain and spinal MRI showed no structural abnormalities. Cerebrospinal fluid analysis revealed mild protein elevation

without pleocytosis, oligoclonal bands, or infectious markers. Extensive metabolic, infectious, toxic, and autoimmune investigations, including neuronal surface antibodies, were negative. Neurophysiological studies were consistent with moderate sensory ganglionopathy. Given the unexplained progressive course, systemic malignancy screening was pursued. Abdominal imaging identified a small lesion in the pancreatic head. Functional imaging with PET-DOTANOC confirmed a somatostatin receptor-positive pancreatic neuroendocrine tumor. The patient underwent pancreaticoduodenectomy following multidisciplinary discussion. While awaiting surgery, recurrent hypoglycaemic episodes occurred, consistent with hormonally active disease, requiring glucagon therapy. Histopathological examination confirmed a well-differentiated, low-grade pancreatic neuroendocrine tumor (WHO grade 1). Despite tumor resection and immunomodulatory therapy, neurological deficits persisted, consistent with irreversible dorsal root ganglion damage.

Conclusion: Sensory ganglionopathy may be the initial manifestation of pancreatic neuroendocrine tumors, supporting early comprehensive oncological screening in unexplained subacute sensory neuronopathy.

Disclosure: Nothing to disclose.

EPO-0852 | Spontaneous spinal epidural hematoma secondary to a dural arteriovenous fistula as a cause of acute myelopathy

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Background and aims: Spinal dural arteriovenous fistulas are vascular malformations that typically cause slowly progressive myelopathy. However, a rare but severe complication in this setting is spontaneous spinal epidural hematoma, which may present as acute spinal cord compression.

Methods: We report the case of an 81-year-old man who presented to the emergency department with acute lumbar pain followed by sudden bilateral lower-limb weakness. Neurological examination revealed paraparesis with muscle strength graded as follows: hip flexion/extension 3-/2, knee flexion/extension 1/1, ankle dorsiflexion/plantarflexion 3+/0. Patellar and Achilles reflexes were absent, with bilateral indifferent plantar responses. Sensory examination showed bilateral hypoesthesia to pain and touch below the knees. Acute myelopathy was suspected, and urgent spinal MRI demonstrated an extensive extradural collection compatible with spinal epidural hematoma from T7 to L3, with maximal compression and myelopathy associated at T11–T12. Tortuous perimedullary vessels of uncertain significance were also observed. Emergency D11–D12 laminoplasty with hematoma evacuation was performed by the Neurosurgery ward.

Results: Given the suspicion of an underlying vascular malformation, the case was discussed with the Neurointerventional Radiology service, and spinal angiography was performed 48 hours later, identifying a dural arteriovenous fistula at the left D6 neural foramen. Neurological outcome was favorable, with

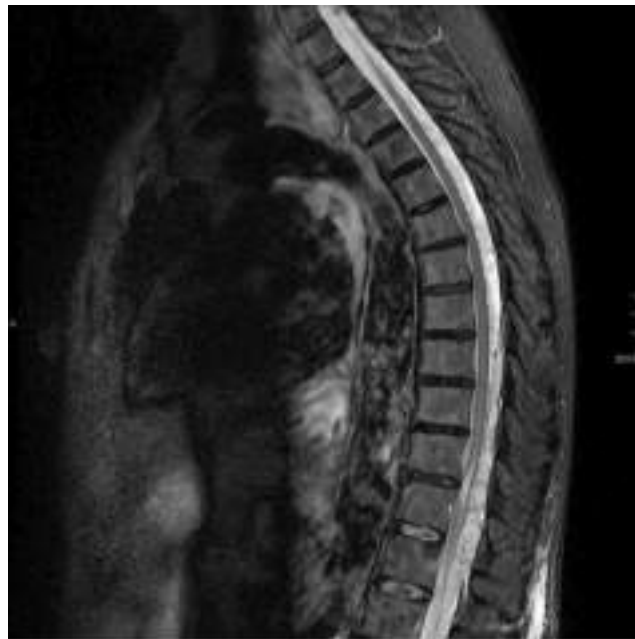


FIGURE 1 Sagittal STIR spinal MRI demonstrating an extensive hyperintense extradural collection consistent with an acute spinal epidural hematoma extending from T7 to L3, with maximal spinal cord compression and associated myelopathy at T11–T12.

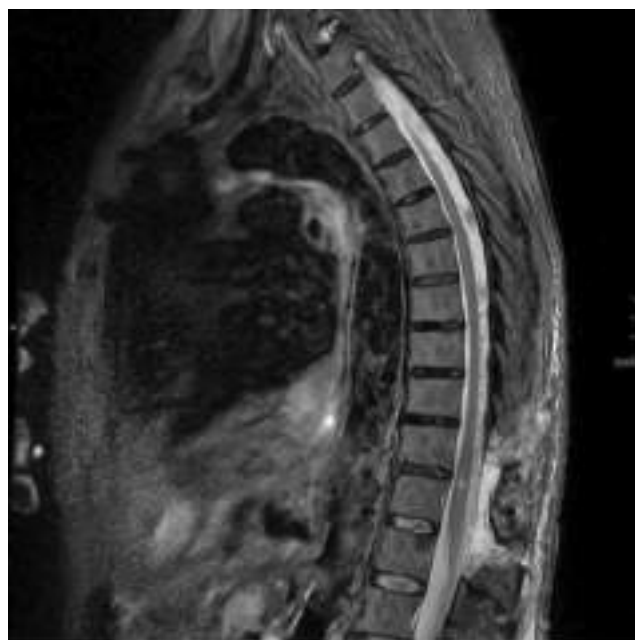


FIGURE 2 Sagittal STIR spinal MRI showing postoperative changes at the level of the previous hematoma, with persistent but reduced extent of myelopathy. Engorged perimedullary vessels surrounding the spinal cord are observed, raising suspicion of an underlying vas.

muscle strength improving to 5/5 in the right lower limb and 2/5 in the left lower limb within 24 hours. Assisted ambulation was achieved after two weeks.

Conclusion: Dural arteriovenous fistula should be considered in cases of acute myelopathy secondary to spontaneous spinal epidural hematoma. Spinal angiography is essential for diagnosis and enables definitive treatment.

Disclosure: Nothing to disclose.

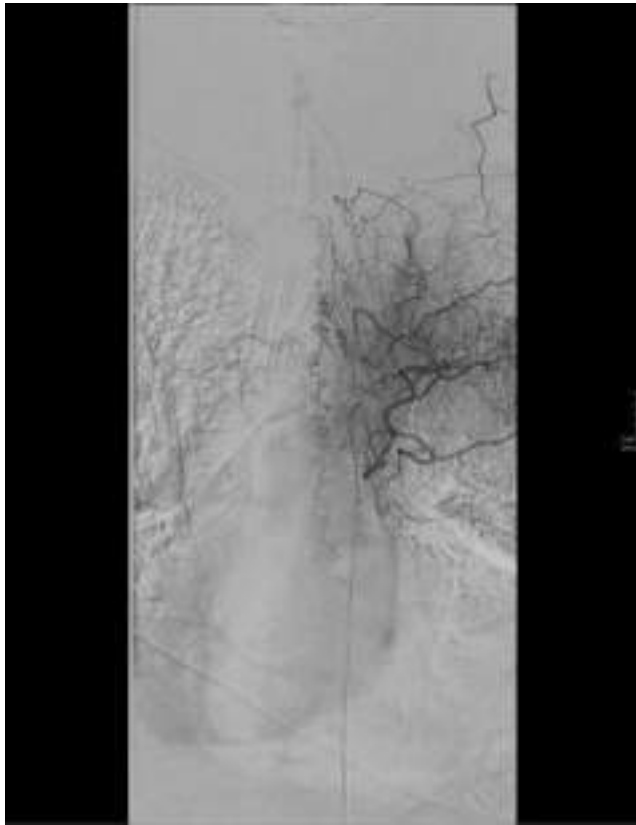


FIGURE 3 Spinal angiography demonstrating an arteriovenous shunt between radiculomedullary arteries and perimedullary veins, consistent with a dural arteriovenous fistula at the left D6 neural foramen.

EPO-0853 | Isolated seronegative longitudinally extensive transverse myelitis in children: A Tunisian case series

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LR18SP04 and Department of Pediatric Neurology, National Institute Mongi Ben Hmida of Neurology, Tunis, Tunisia

Background and aims: Pediatric longitudinally extensive transverse myelitis (LETM) classification has evolved with antibody discovery. These cases are heterogeneous with variable clinical features and outcomes.

Methods: We retrospectively reviewed eight Tunisian children diagnosed with isolated seronegative inflammatory LETM from 2020 to 2025. Demographics, clinical presentation, neuroimaging, cerebrospinal fluid (CSF) findings, treatments, and outcomes were collected. Functional status and follow-up MRI findings were analyzed. All patients were tested for serum and CSF myelin oligodendrocyte glycoprotein antibodies and serum anti-aquaporin 4.

Results: Eight patients (five females, three males) had a median age of 13.5 years (range 5–17). A history of a preceding infection was noted in three patients. All presented with paraparesis. Six had sensory symptoms. Four had urinary dysfunction. CSF pleocytosis was present in six patients. CSF oligoclonal bands were positive in three. MRI showed cervical cord involvement in seven and conus medullaris involvement in two. Two patients had tumefied lesions. Two showed multifocal contrast

enhancement. All received intravenous methylprednisolone. Six continued oral corticosteroids for 3–8 months. One received intravenous immunoglobulin. Five patients improved clinically. Two patients relapsed. One patient worsened. At a median follow-up of two years, five had improved motor function. Follow-up MRI showed spinal cord atrophy in two, stable myelitis in two, and lesion enlargement in one.

Conclusion: Seronegative pediatric LETM presents with variable clinical and radiological features. Despite early corticosteroid therapy, many patients have incomplete recovery. Careful long-term follow-up is required to better understand this entity. Further studies are needed to identify prognostic factors and optimize treatment strategies.

Disclosure: Nothing to disclose.

EPO-0854 | Spinal dural arteriovenous fistula as a diagnostic pitfall: A case report and literature

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Background and aims: Spinal dural arteriovenous fistulas (sDAVFs) are rare but potentially reversible vascular causes of progressive myelopathy resulting from venous hypertensive congestion of the spinal cord. Because of their insidious onset and nonspecific clinical and radiological features, they are frequently misclassified in acute and pre-hospital settings, leading to delayed neurology referral and preventable neurological disability.

Methods: We report a 61-year-old man with several months of progressive gait impairment, lower limb numbness, and urinary retention. Examination showed spastic paraparesis (Aminoff–Logue G4 U3). Spinal MRI demonstrated longitudinal T2 hyperintensity from T7 to L1 with dilated perimedullary flow voids. Cerebrospinal fluid showed elevated protein, initially suggesting inflammatory or degenerative etiologies. However, progressive symptoms with sphincter dysfunction and characteristic imaging raised suspicion for a vascular cause. Digital subtraction angiography confirmed a right-sided dural arteriovenous fistula at L2. Endovascular embolization resulted in clinical improvement with recovery of motor function and resolution of urinary symptoms (Aminoff–Logue G3 U1).

Results: A brief literature review indicates that median diagnostic delays range from 12 to 44 months, primarily due to

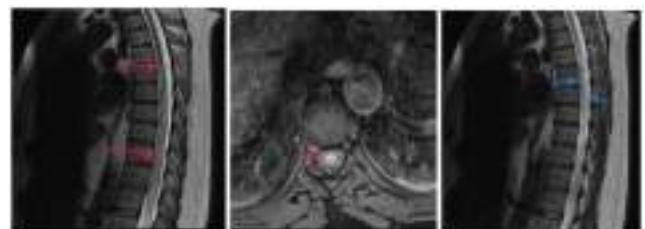


FIGURE 1 MRI shows medullary edema (red arrow) from T7 to L1 with the presence of perimedullary vessels (blue arrow).

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Background and aims: Spinal dural arteriovenous fistulas (sDAVF) are the most common intradural spinal vascular malformations, yet they remain frequently underdiagnosed. Incidious clinical onset and misinterpretation of initial MRI can lead to delayed recognition and progressive venous hypertensive myelopathy. Using statistical techniques, this research analyzes the clinical features, diagnostic delay, and short-term outcomes following endovascular therapy to investigate disability predictors.

Methods: A retrospective observational study including 9 patients diagnosed with sDAVF from 2022–2025. All patients underwent complete spinal MRI and selective spinal angiography. Functional status was assessed using the Aminoff–Logue (AL) gait and sphincter scores and the modified Rankin Scale (mRS). Associations between diagnostic delay and disability scores were explored with Pearson or Spearman correlation, as appropriate. A simple linear regression model evaluated diagnostic delay as a predictor of AL scores (β -coefficient, 95% CI, p -value). Statistical significance was set at $p < 0.05$, outcomes before and after treatment compared using paired t -tests.

Results: Mean age was 54.5 years; 7/9 were male. Median diagnostic delay was 7.11 months. Common comorbidities included degenerative spine disease (56.4%), hypertension (35.6%), and type 2 diabetes (23.4%). Post-treatment analysis demonstrated significant improvement in AL gait and sphincter scores (Wilcoxon $p < 0.05$). Linear regression showed a positive β -trend between longer diagnostic delay and worse baseline AL scores, although not statistically significant ($p > 0.05$), likely due to the small sample size.



FIGURE 2 Medullary angiography reveals aright lateral dural arteriovenous fistula at the L2 level (yellow arrow), with hypertrophic perimedullary vessels (orange arrow).



FIGURE 3 Post-embolization arteriography demonstrates reduced hypertrophic plexuses.

misinterpretation of MRI, attribution of symptoms to nonvascular spinal disorders, and low awareness during stroke-mimic triage and early neurological assessment. Prolonged delays are consistently associated with poorer neurological outcomes.

Conclusion: This case emphasizes the importance of including sDAVF in stroke-mimic algorithms and neurology referral pathways for patients with unexplained progressive myelopathy, to enable timely angiographic confirmation and definitive treatment.

Disclosure: Nothing to disclose.

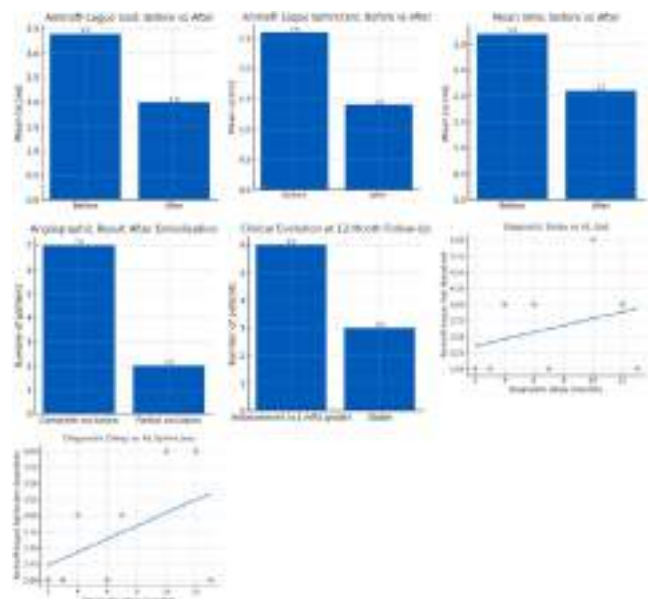


FIGURE 1 Statistical results of the study.

Conclusion: Diagnostic delays in sDAVF remain common and correlate with greater neurological disability. Endovascular treatment achieved marked functional improvement. Incorporating structured MRI protocols and multidisciplinary evaluation is essential to reduce delays and improve outcomes. **Disclosure:** Nothing to disclose.

EPO-0856 | Peripheral arteriovenous fistula and non-traumatic myelopathy: Diagnostic challenges in a case of acute tetraparesis

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Background and aims: Acute tetraparesis is a neurological emergency requiring prompt diagnostic evaluation. While traumatic or intrinsic spinal cord vascular causes are more frequent, rarer conditions such as hemodynamic changes induced by peripheral arteriovenous fistulas (AVFs) should be considered in selected patients.

Methods: Retrospective review of a patient's clinical file.

Results: A 57-year-old man presented with severe back pain followed by sudden tetraparesis evolving over 1 hour. He had no history of trauma. Past medical history included chronic kidney disease and prior hemodialysis via a right elbow AVF. Neurological examination revealed tetraparesis with lower limb plegia and MRC 3/5 upper limbs weakness, sensory level at C4, and absent reflexes. Cervical MRI revealed a posterior epidural collection from C3-D10, suggestive of acute hemorrhage, and spinal cord hyperintensity from C5–C7 with diffusion restriction, compatible with ischemic injury. Emergent C5–C7 laminectomy and drainage of the epidural collection were performed, however follow-up MRI revealed progression of spinal cord hyperintensity from C4–D1. Dural AVF was suspected and the patient underwent diagnostic angiography, excluding this hypothesis, but revealing an AVF of the elbow with a broad thrill and exuberant development of the right brachial territory and cervical collaterals, associated with subclavian-steal syndrome. The elbow AVF was considered the likely cause of cervical vascular ectasia and consequent spinal ischemia. The patient underwent elbow AVF ligation and was discharged after 40 days, referred for rehabilitation.

Conclusion: This rare case highlights the importance of considering peripheral vascular causes as a potential etiology of non-traumatic myelopathies, especially in patients with a history of hemodialysis.

Disclosure: Nothing to disclose.

Pain 2

EPO-0857 | From peripheral hemodynamics to central connectivity: A multi-modal framework towards precision medicine in trigeminal neuralgia

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Background and aims: Trigeminal neuralgia (TN) management is evolving towards precision medicine, necessitating biomarkers that delineate pathological neurovascular conflict

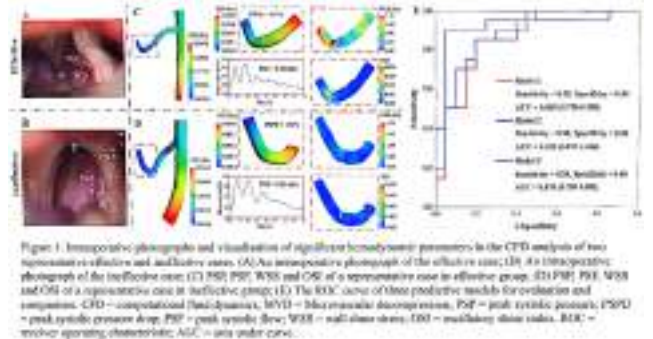


FIGURE 1 Effective vs. ineffective surgical outcomes in CFD analysis. (A, B) Intraoperative views. (C, D) Key hemodynamic parameters (PSP, PSF, WSS, OSI). (E) ROC curve comparison of predictive models.

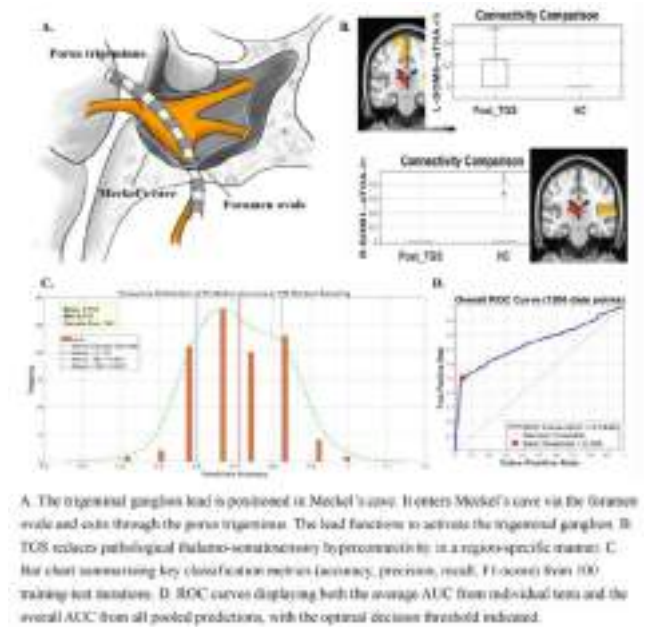


FIGURE 2 (A) Trigeminal ganglion lead placement. (B) TGS reduces thalamo-somatosensory hyperconnectivity. (C) Classification performance metrics. (D) ROC curves with AUC and optimal threshold.

(NVC) and its central consequences. We propose a multi-modal framework integrating peripheral hemodynamics and central connectivity to guide treatment.

Methods: In Study 1 (CFD-MVD), In 56 classical TN patients, computational fluid dynamics (CFD) quantified hemodynamic parameters (e.g. peak systolic flow-PSF, max wall shear stress) at neurovascular conflict (NVC) sites to predict microvascular decompression (MVD) efficacy. In Study 2 (TGS-fMRI), In 29 TN patients, longitudinal resting-state fMRI assessed thalamo-somatosensory functional connectivity (FC) changes before and after trigeminal ganglion stimulation (TGS). Machine learning evaluated FC as a treatment-response biomarker.

Results: Study 1 revealed that “effective” NVC for MVD was characterised by significantly lower PSF (0.202 ± 0.136 vs. 0.306 ± 0.142 mL/s, $p=0.007$) and higher Max WSS (3.231 vs. 2.197 Pa, $p=0.024$). A prediction model incorporating these parameters achieved an AUC of 0.920 for MVD efficacy. Study 2 demonstrated that TN patients exhibited pathological hyperconnectivity within the thalamo-somatosensory circuit compared to healthy controls. Successful TGS significantly normalised this aberrant FC in a region-specific manner. The SVM classifier reliably distinguished pre- from post-TGS states (AUC = 0.74), validating these FC patterns as stable neuromodulation biomarkers.

Conclusion: This framework links peripheral CFD biomarkers (guiding MVD selection) with central fMRI biomarkers (assessing TGS response), enabling personalised TN therapy based on distinct pathophysiological profiles.

Disclosure: Nothing to disclose.

EPO-0858 | Clinical features distinguishing fibromyalgia from small fiber neuropathy

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Background and aims: Fibromyalgia syndrome (FMS) has been described associated with small fiber pathology and it manifests with a distinct clinical picture compared with pure small fiber neuropathy (SFN). However, some severe fibromyalgic patients may present with overlap phenotypes. We compared sensory and autonomic symptoms and signs across FMS, FMS associated with SFN and pure SFN.

Methods: We retrospectively analyzed 115 patients with FMS and 67 with biopsy-confirmed SFN without FMS. Within FMS, 91 patients were classified as FMS+SFN, when associated with SFN symptoms and signs, otherwise as FMS ($n=24$). We collected pain descriptors and distribution, sensory signs (allodynia, hyperalgesia, hypoesthesia to pinprick/touch), autonomic symptoms and comorbidities.

Results: Our population (mean age 46.1 ± 14.3 years) was mainly composed of women (FMS+SFN 90.1%; FMS 79, 2%; SFN 68,7%). Compared with FMS, FMS+SFN more often reported burning pain (91.2% vs 58.3%), hyperalgesia (23.1% vs 0%) and allodynia (47.3% vs 0%) and less frequently was associated

with dysmetabolic comorbidities (15.4% vs 37.5%). Compared with SFN, FMS+SFN more frequently reported excruciating pain (91.2% vs 67.2%), painful cold (19.8% vs 7.5%), pins and needles (19.8% vs 7.5%), tingling (54.9% vs 32.8%), pinprick hyperalgesia (23.1% vs 3.0%), mechanical allodynia (47.3% vs 29.9%), itch (65.9% vs 38.8%), hypoesthesia to pinprick (82.4% vs 54.0%) and widespread pain distribution (95.6% vs 40.2 %). Overall, FMS+SFN presented more frequently autonomic symptoms (mostly urinary) and autoimmune comorbidities.

Conclusion: Patients with FMS/SFN phenotype overlap show a more severe sensory/autonomic burden than FMS and should prompt evaluation for the search of potentially treatable causes.

Disclosure: Nothing to disclose.

EPO-0859 | Sympathetic skin response in fibromyalgia patients with small fibers pathology

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Background and aims: Fibromyalgia (FM) is a chronic disease characterised by small fibre pathology and autonomic dysfunction. Sympathetic Skin Response (SSR) is easily obtained in clinical practice as a marker of autonomic dysfunction. The present study aims to assess SSR abnormalities in small fibre pathology associated with the disease.

Methods: This was a retrospective, observational, monocentric, cross-sectional study including 393 patients with FM, who underwent skin biopsy from the thigh and ankle, SSR obtained with a standard electromyographic active electrode attached to the right palm and sole, and clinical evaluation.

Results: SSR at the hand and foot was similarly absent in the three FM groups: those with normal skin biopsy, proximal denervation, and proximal and distal denervation. Latency at the foot was significantly prolonged in patients with proximal denervation ($z=2.33$, $p=0.019$). Amplitude was reduced in patients with distal denervation compared with the normal skin biopsy group ($z=2.30$, $p=0.020$). SSR had low predictive performance with respect to skin biopsy results ($r^2=0.04$). Latency of SSR at the foot and intraepidermal nerve fibre density (IENFD) correlated with the score for associated symptoms. Amplitude of SSR at the foot also negatively correlated with anxiety, depression, and fatigue scores.

Conclusion: SSR abnormalities did not correspond with small fibre impairment as shown by skin biopsy in FM patients, but instead appear to reflect different aspects of the complex clinical picture, including psychopathological features and fatigue, as distinct biomarkers of the symptom constellation.

Disclosure: Nothing to disclose.

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Background and aims: Differential diagnosis of somatoform pain disorder (SPD) from other chronic pain (CP) conditions presents significant diagnostic challenges due to the overlap in symptoms. It is known that CP leads not only to anxiety, depression and sleep disturbances, but also to a specific neurochemical imbalance in the brain. In addition, sleep disorders and CP negatively affect brain homeostasis by reducing glymphatic clearance. To identify differences in neurochemical imbalance and glymphatic clearance using MRI spectroscopy (MRS) and MRI tractography in patients with SPD and CP.

Methods: 27 patients were diagnosed with SPD according to DSM-5 criteria (1st group), and 32 patients with chronic secondary musculoskeletal pain according to ICD-11 criteria (2nd group). We used MRS and MRI with DTI-ALPS index analysis, questionnaires and scales to assess pain characteristics, anxiety levels, depression and quality of life (table).

Results: No differences were found in anxiety, depression, VAS, or quality of life. 1st group had the worst scores in the mental health component and the distress subscale. The 2nd group showed a decrease in the DTI-ALPS index and NAA with an increase of the glial metabolites, which was expressed in NAA/

TABLE 1 Comparative assessment of patients with somatoform pain disorder, chronic neuropathic pain and a control group.

Parameters	Control group n = 27	1 st group n = 27	2 nd group n = 32
Mood (Q28)			
Age, years	48 (44 – 56)	46 (44 – 51)	48 (42 – 54)
Men / Women	12/15	10/17	15/17
Disease duration (months)	–	23 (18 – 32)	24 (8 – 36)
Visual Analogue Scale	–	7 (6 – 8)	8 (4 – 7)
4DSQ total	38 (27 – 46)	62 (53 – 71)	84 (58 – 78) ^Δ
Distress	23 (19 – 38)	28 (22 – 35)	23 (19 – 28) ^Δ
Depression	3 (2 – 5)	6 (3 – 9)	6 (2 – 8)
Anxiety	7 (5 – 9)	14 (6 – 18)	33 (7 – 16) ^Δ
Somatisation	32 (20 – 38)	24 (18 – 30)	23 (17 – 28) ^Δ
Sympt Sleep Questionnaire	28 (17 – 24)	16 (11 – 20)	34 (20 – 30)
MFI-36	88 (66.5 – 16.5)	87.1 (61.8 – 41.8)	44.8 (48.6 – 66.1) ^Δ
GAD-7	7 (5 – 10)	14 (9 – 18)	35 (19 – 39)
SP-36	14 (8 – 8)	42 (38 – 46)	45 (35 – 47) ^Δ
Physical Component Summary	71 (58 – 85)	37 (30 – 32)	40 (33 – 47) ^Δ
Mental Component Summary	73 (56 – 85)	32 (25 – 41)	46 (24 – 49) ^Δ
DTI-ALPS index (left)	1.57 (1.41 – 1.88)	1.55 (1.47 – 1.82)	1.38 (1.33 – 1.48) ^Δ
DTI-ALPS index (right)	1.62 (1.43 – 1.87)	1.59 (1.45 – 1.88)	1.42 (1.31 – 1.54) ^Δ
MRS (thalamus / amygdala / prefrontal cortex)			
NAA/Cr	1.86 (1.55 – 2.33) ^Δ	1.79 (1.49 – 1.98) ^Δ	1.34 (0.81 – 1.57) ^Δ
NAA/MI	1.73 (1.47 – 2.0)	1.68 (1.34 – 1.87) ^Δ	1.11 (1.13 – 1.62) ^Δ
NAA/Cho	1.74 (1.48 – 1.78)	1.65 (1.35 – 1.74)	1.43 (1.18 – 1.83) ^Δ
NAA/MI	2.0 (1.62 – 2.25) ^Δ	1.85 (1.66 – 1.92) ^Δ	1.45 (0.95 – 1.68) ^Δ
NAA/Cho	1.85 (1.58 – 1.94) ^Δ	1.71 (1.62 – 1.93) ^Δ	1.64 (1.58 – 1.77) ^Δ
NAA/Cho	1.74 (1.61 – 1.87)	1.69 (1.45 – 1.88)	1.32 (1.28 – 1.84)
NAA/Cho	2.07 (1.64 – 2.23) ^Δ	1.88 (1.49 – 1.97) ^Δ	1.35 (0.74 – 1.63) ^Δ
NAA/Cho	1.87 (1.61 – 1.95) ^Δ	1.75 (1.55 – 1.88) ^Δ	1.48 (0.92 – 1.66) ^Δ
NAA/Cho	1.72 (1.58 – 1.83)	1.61 (1.48 – 1.77)	1.35 (1.32 – 1.72)

4DSQ – Four-Dimensional Symptom Questionnaire
MFI-36 – Multidimensional Fatigue Inventory
GAD-7 – Generalized Anxiety Disorder 7
SP-36 – 36-Item Short-Form Survey
DTI-ALPS – Diffusion tensor image analysis along the perivascular space
NAA – N-acetylcholine, Cr – Creatine, MI – Myo-Inositol, Cho – Choline
^Δ – Statistically significant difference (p < 0.05) between the 1st group and 2nd group (Mann-Whitney)
^Δ – Statistically significant difference (p < 0.05) between controls, 1st group and 2nd group (Kruskal-Wallis test)

Cr, NAA/MI and NAA/Cho ratio reduced in the thalamus, amygdala, prefrontal region compared to the 1st group and the controls (Table).

Conclusion: Despite the fact that both types of pain disorders are characterized by disturbances in the functional relationship between the affective and cognitive systems, the present study shows that SPD does not lead to significantly metabolic changes and glymphatic dysfunction characteristic of CP, which can be used to differentiate these conditions.

Disclosure: Nothing to disclose.

EPO-0861 | **Immune activity in skull bone marrow and the periphery in chronic pain: A TSPO PET/MRI and LPS-stimulated cytokines study**

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Background and aims: Chronic pain is increasingly recognized as a neuroinflammatory condition involving interactions between peripheral immune processes and the central nervous system. The skull bone marrow (SBM) is a critical interface in this peripheral-central immune axis, yet cytokine signatures and SBM activation patterns in distinct chronic pain conditions remain unclear.

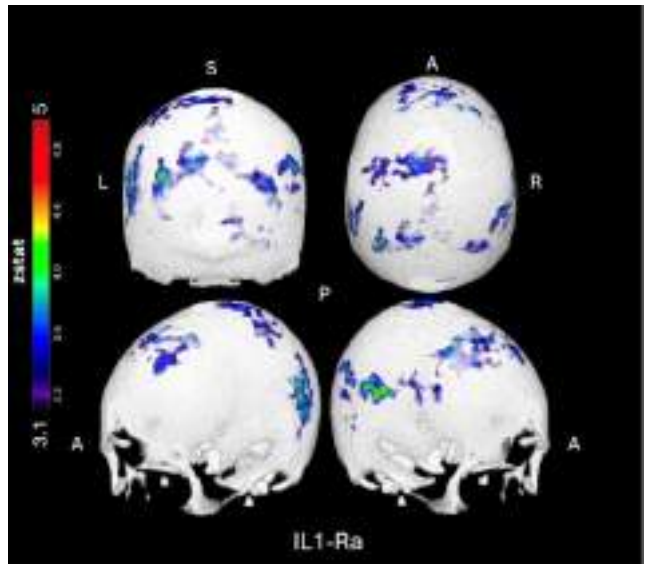


FIGURE 1 3D mapping of voxels in patients with chronic pain showed a voxel-wise positive correlation between skull bone marrow [11C]PBR28 TSPO signal and IL-1Ra fold change (LPS-stimulated/base-line), overlaid onto a 3D skull model in standard MNI152 space.

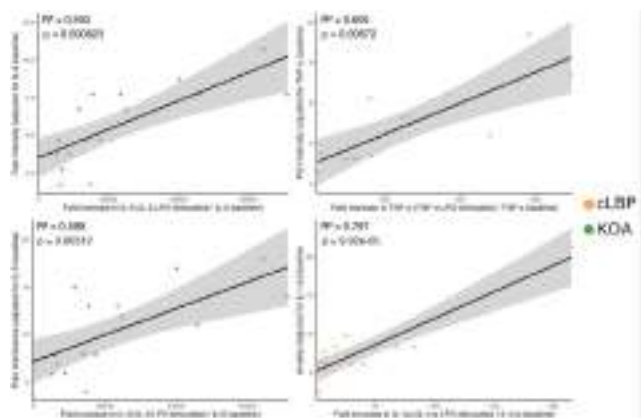


FIGURE 2 Scatter plots of the partial linear regression and 95% confidence interval showing associations between PROMIS-29 scores and cytokine fold change (LPS-stimulated/baseline) in cLBP and KOA patients. All analyses were adjusted for baseline cytokine levels.

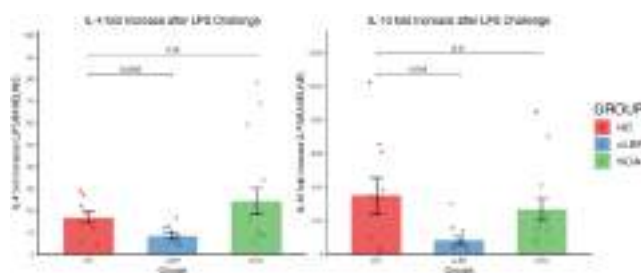


FIGURE 3 Bar plots showing group differences in IL-10 and IL-4 fold change (LPS-stimulated/baseline) between healthy controls, KOA, and cLBP. Bars represent group means $\pm$ SEM. Group comparisons were assessed using *t*-tests

Methods: We assessed lipopolysaccharide (LPS)-induced cytokine reactivity in peripheral blood cells from patients with chronic lower back pain (cLBP; $n = 13$) and knee osteoarthritis (KOA; $n = 17$), alongside healthy controls ($n = 10$). Cytokine fold changes were analyzed in relation to pain intensity, pain interference, depression, and anxiety using partial linear regression adjusting for baseline cytokine levels. SBM immune cell density was quantified via [^{11}C] PBR28 PET/MRI, indexing 18 kDa translocator protein (TSPO) expression.

Results: Compared to controls, only cLBP patients exhibited significantly reduced LPS-induced IL-4 and IL-10 reactivity ($p < 0.05$). In cLBP, pain intensity showed associations with IL-1 β , TNF- α , GM-CSF, and IFN- γ reactivity ($p < 0.05$), with a trend for IL-6. In contrast, KOA demonstrated broader clinical associations, with both pain intensity and pain interference correlating with IL-1 β , IL-6, MCP-1, and MIP-1 α ($p < 0.05$). Notably, depressive and anxiety symptoms correlated with IL-1 receptor antagonist (IL-1RA) reactivity exclusively in cLBP ($p < 0.001$). Voxelwise analyses revealed significant associations between SBM TSPO signal and IL-1RA, IL-6, IL-8, and GM-CSF reactivity ($p < 0.01$).

Conclusion: These findings reveal condition-specific peripheral immune signatures in chronic pain and provide in vivo evidence linking SBM activation with peripheral cytokine reactivity, supporting a functional skull-immune-brain axis in human pain pathophysiology.

Disclosure: Nothing to disclose.

EPO-0862 | Characteristics of migraine patients who successfully discontinued anti-CGRP monoclonal antibodies: A single-center retrospective study

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Background and aims: Randomized controlled trials have established anti-CGRP monoclonal antibodies (mAbs) as effective for migraine prevention. While some patients discontinue treatment following substantial improvement—a process termed “graduation”—the clinical characteristics associated with success remain poorly understood. This study investigated factors associated with successful graduation from anti-CGRP mAbs in a real-world setting.

Methods: We retrospectively evaluated 257 migraine patients (ICHD-3 criteria) treated with anti-CGRP mAbs at our institution. Clinical characteristics, including medication overuse headache (MOH), baseline monthly migraine days (MMDs), and MIDAS/HIT-6 scores, were compared between “graduates” and “non-graduates.” Multivariable logistic regression analysis was performed to identify independent factors associated with graduation.

Results: The anti-CGRP mAbs administered included galcanezumab ($n = 96$), erenumab ($n = 66$), and fremanezumab ($n = 95$). Thirty patients (11.7%) successfully graduated. Univariate analyses showed graduates had a significantly lower prevalence of MOH (20% vs. 66%, $p = 0.015$) and fewer baseline MMDs (median 11 vs. 16, $p = 0.042$). In multivariable logistic regression, absence of MOH (adjusted OR: 0.22; 95% CI: 0.05–0.89; $p = 0.034$) and lower baseline MMDs (adjusted OR: 0.93; 95% CI: 0.87–0.99; $p = 0.048$) were independently associated with successful graduation. No significant interaction was observed between MOH and MMDs.

Conclusion: Successful discontinuation of anti-CGRP mAbs was independently associated with fewer baseline MMDs and absence of MOH. Early initiation of preventive therapy—before high frequency and medication overuse develop—may enhance the likelihood of successful treatment withdrawal, supporting a treat-to-target strategy aimed at eventual treatment graduation.

Disclosure: Noboru Imai reports being an advisor for Sawai and received speaker fees from Daiichi Sankyo, Eli Lilly, Otsuka, and Amgen; however, these companies had no relation to the study.

TABLE 1 Logistic regression analysis of factors associated with “graduation.”

Variable	Univariate OR (95% CI)	<i>p</i>	Multivariate adjusted OR (95% CI)	<i>p</i>
Sex (female)	1.0	>0.2	N/A	>0.2
Age at onset (per year)	n.s.	>0.2	n.s.	>0.2
Disease duration (per year)	1.0	>0.1	N/A	>0.1
MOH (yes)	0.11 (0.04–0.47)	0.020	0.22 (0.05–0.89)	0.034
Baseline migraine days (per day)	0.93 (0.84–0.99)	0.042	0.93 (0.87–0.99)	0.048
MOH $\times$ MMD interaction	—	—	N/A	>0.1

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Background and aims: Pain is a common and disabling symptom in multiple sclerosis (MS), but the clinical and functional impact of neuropathic pain (NP) versus musculoskeletal pain (MSP) is not well defined in large real-world cohorts.

Methods: In this cross-sectional study, 1,503 people with MS attending a tertiary MS center were assessed. Pain subtypes were classified using PainDETECT and the Nordic Musculoskeletal Questionnaire into no pain, MSP, or NP. Disability was measured with the Expanded Disability Status Scale (EDSS). Health-related quality of life (HRQoL) was evaluated with EQ-5D-3L and EQ-VAS.

Results: Pain was reported by 68.2% of participants; 50.5% had MSP and 17.7% had NP. The NP group had higher disability (median EDSS 2.0) than MSP or no-pain groups ($p < 0.001$). HRQoL was poorest in NP, with significantly worse EQ-5D domain scores and EQ-VAS compared with MSP and no pain (all $p < 0.001$). MSP most frequently involved the lower back (33.4%), neck (31.3%), knees (31.0%), and ankles/feet (29.4%).

Conclusion: In MS, NP is associated with greater disability and markedly worse HRQoL than MSP or no pain, supporting routine screening for pain subtypes and early, targeted management to reduce functional and psychosocial burden.

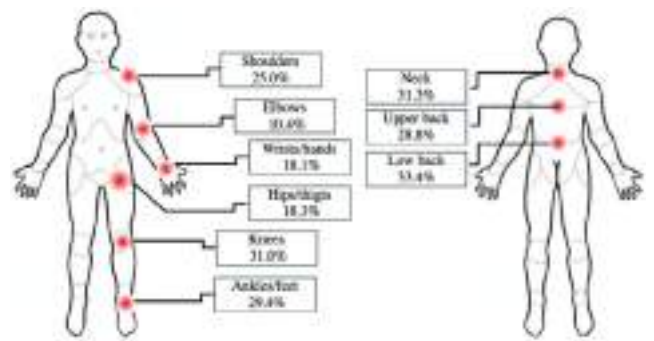


FIGURE 1 Anatomical distribution of musculoskeletal pain in people with multiple sclerosis (pwMS).

TABLE 1 Comparison of demographic and clinical features of the participants ($n = 1503$) Data are presented as mean $\pm$ SD or median (IQR) unless otherwise indicated. Significant p -values (< 0.05) are shown in bold. EDSS, Expanded Disability Status Scale.

	Participants without pain ($n = 478$)	Participants with MSP ($n = 759$)	Participants with NP ($n = 265$)	p
Age (years)	38.61 $\pm$ 11.55	37.01 $\pm$ 11.52	38.26 $\pm$ 10.90	0.163*
EDSS	1.0 (0-2.0)	1.0 (0-2.0)	2.0 (1.0-3.0)	$< 0.001^{**}$
Disease duration (years)	6.0 (1.0-12.0)	5.0 (1.0-12.0)	5.0 (2.0-11.0)	0.756**
Gender (Female/Male)	311/167 (65.1%/34.9%)	549/210 (72.3%/27.7%)	204/62 (76.7%/23.3%)	0.002
MS type (RR/Progressive)	415/61 (87.2%/12.8%)	667/84 (88.0%/11.2%)	233/31 (88.3%/11.7%)	0.689

TABLE 2 Comparison of HRQoL between groups.

	Participants without pain ($n = 302$)	Participants with MSP ($n = 481$)	Participants with NP ($n = 199$)	p	η^2	Post-hoc
EQ-5D - mobility	1.0 (1.0-1.0)	1.0 (1.0-1.0)	2.0 (1.0-2.0)	< 0.001	0.037	MSP>No pain ($p = 0.007$) NP>No pain ($p = 0.001$) NP>MSP ($p = 0.001$)
EQ-5D - self-care	1.0 (1.0-1.0)	1.0 (1.0-1.0)	1.0 (1.0-1.0)	< 0.001	0.019	NP>No pain ($p = 0.001$) NP>MSP ($p = 0.001$)
EQ-5D - usual activity	1.0 (1.0-1.0)	1.0 (1.0-1.0)	2.0 (1.0-2.0)	< 0.001	0.048	MSP>No pain ($p = 0.001$) NP>No pain ($p = 0.001$) NP>MSP ($p = 0.001$)
EQ-5D - pain/discomfort	1.0 (1.0-1.0)	2.0 (1.0-2.0)	2.0 (2.0-2.0)	< 0.001	0.135	MSP>No pain ($p = 0.001$) NP>No pain ($p = 0.001$) NP>MSP ($p = 0.001$)
EQ-5D - anxiety/depression	1.0 (1.0-1.0)	2.0 (1.0-2.0)	2.0 (1.0-2.0)	< 0.001	0.060	NP>No pain ($p = 0.001$)
EQ-VAS	56.0 (30.0-85.0)	56.0 (70.0-85.0)	75.0 (50.0-85.0)	< 0.001	0.041	MSP>No pain ($p = 0.001$) NP>No pain ($p = 0.001$) NP>MSP ($p = 0.001$)

Disclosure: Nothing to disclose.

EPO-0864 | Microstructural changes at the trigeminal root entry zone and their role in classical trigeminal neuralgia: A systematic review

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Background and aims: Classical trigeminal neuralgia is linked to neurovascular compression at the root entry zone. This review aimed to summarise evidence on structural and physiological mechanisms contributing to pain generation.

Methods: A systematic search of PubMed, Scopus, and Web of Science was conducted (2000–2024). Eligible studies included human clinical, neuroimaging, neurophysiological, or histopathological investigations examining the REZ in classical TN. Data were extracted on REZ morphology, diffusion tensor imaging (DTI) findings, electrophysiological abnormalities, and correlations with pain severity or treatment outcomes. Screening and qualitative synthesis followed PRISMA guidelines.

Results: A total of 47 studies met inclusion criteria. Neurovascular compression was consistently associated with focal demyelination at the REZ, with 78% of imaging studies reporting reduced fractional anisotropy in the affected nerve. Electrophysiological data demonstrated abnormal trigeminal reflex latencies consistent with impaired axonal conduction. Across clinical studies, greater structural or electrophysiological disruption was associated with higher pain intensity, increased attack frequency, and reduced responsiveness to sodium-channel blockers. Evidence also showed that patients with severe REZ distortion were more likely to benefit from microvascular decompression compared with medical therapy alone.

Conclusion: Across diverse methodologies, current evidence supports a shared mechanism in TN centred on REZ demyelination, abnormal impulse generation, and ectopic cross-talk between trigeminal fibres. These mechanistic insights reinforce



FIGURE 1 Educational infographic illustrating the role of MRI in identifying neurovascular compression in trigeminal neuralgia. Included to contextualise the diagnostic relevance of imaging referenced in this review.

the value of integrating neuroimaging and neurophysiology into routine evaluation to improve treatment selection. A unified mechanistic framework may also guide future biomarker development for personalised TN management.

Disclosure: Nothing to disclose.

EPO-0865 | An artificial intelligence-based decision-support tool to predict outcomes in low back pain

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Background and aims: The study aimed to create a predictive tool (an artificial intelligence-based decision-support tool), which merges Clinical Intelligence and Radiomics to generate customized therapy plans for LBP patients.

Methods: The tool uses deep learning models to perform automated segmentation which enables the extraction of geometrical parameters including disc height and width, vertebrae height and width, canal diameter, disc height index, signal intensity, and disc volume. A total of 402 patient's mid-sagittal T2-weighted MRIs have been utilized for the segmentation model while 4260-disc slice with radiological geometric parameters for spondylosis grading. The creation of a labeled dataset utilized

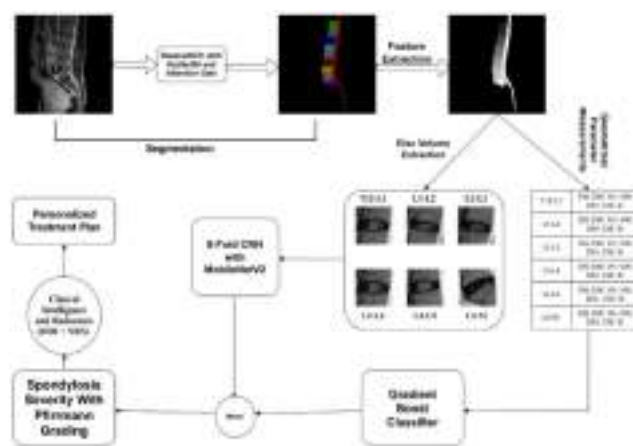


FIGURE 1 The workflow of our study.

expert-verified Pfirrmann and spondylosis severity gradings (11 grades, shown in fig. 2) to address the clinical issues stemming from manual grading variability and subjectivity. The machine learning algorithms use this combined dataset to predict results and combined with clinical scores (Oswestry Disability Index and Numerical Rating Scale) to recommend personalized treatment plans.

Results: The DeepLabV3+ segmentation model with ResNet50 encoder reached 95.5% accuracy, which increased to 98.7% after 8-fold cross-validation and simultaneously improved precision (96.95%), recall (97.1%), Dice coefficient (96.9%), and IoU (94.8%). The CNN with MobileNetV2 achieved 97.84% accuracy and 96.76% IoU for spondylosis severity prediction after cross-validation. The Gradient Boost classifier demonstrated the best results with geometrical data by achieving 91.65% accuracy and 84.59% IoU.

Conclusion: The tool introduces an innovative method to customize LBP treatment through the combination of AI technology with radiological data and clinical expertise.

Disclosure: The study was sponsored by DHR-MRU grant. Nothing to disclose Part of work is published (doi:10.1007/s10278-025-01517-3).

EPO-0866 | A role for glycine transporter 2-inhibitors in modulating levels of dopamine in the nucleus Accumbens

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Background and aims: Impaired glycinergic neurotransmission in the spinal cord which disinhibits nociceptive signaling to higher cortical centers is implicated in states of pathological pain. Glycine transporter 2 (GlyT2)-inhibition, which augments glycine receptor (GlyR) signaling has emerged as a novel

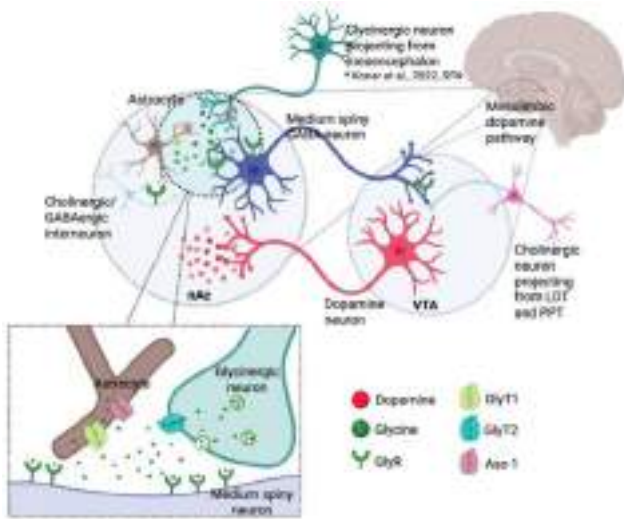


FIGURE 1 Overview of hypothesized glycinergic signaling in the nAc. Enhanced glycinergic tone in the nAc, produced by e.g. GlyT1- or GlyT2-inhibition disinhibits VTA-nAc dopaminergic projections, thereby slightly elevating accumbal dopamine output.

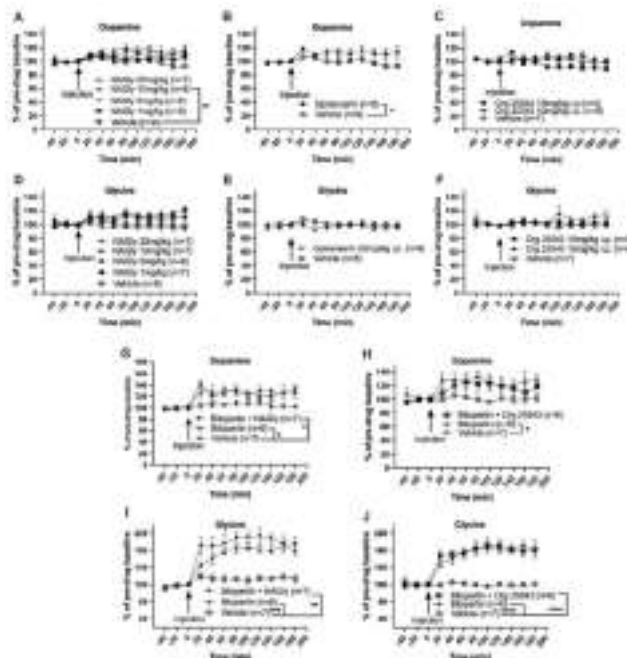


FIGURE 2 Systemic treatment with partial but not full GlyT2-inhibitors slightly elevate extracellular dopamine but not glycine levels in vivo in the nAc. Combined GlyT1- and GlyT2-inhibition did not produce any statistically significant additive effects.

pharmacological treatment concept for pain, devoid of adverse effects and effective in achieving allodynia in a phase III-trial. In rats, GlyRs also regulate dopamine (DA) levels in the nucleus Accumbens (nAc), a pivotal region for motivational processes. Recently, the presence of GlyT2-expressing glycinergic axons in the mouse nAc has been established.

Methods: This study in rats examined if GlyT2-inhibition alters accumbal DA and glycine content as measured by in vivo microdialysis. The presence of GlyT2-expressing axons and GlyRs in the nAc was examined by immunohistochemistry (IHC).

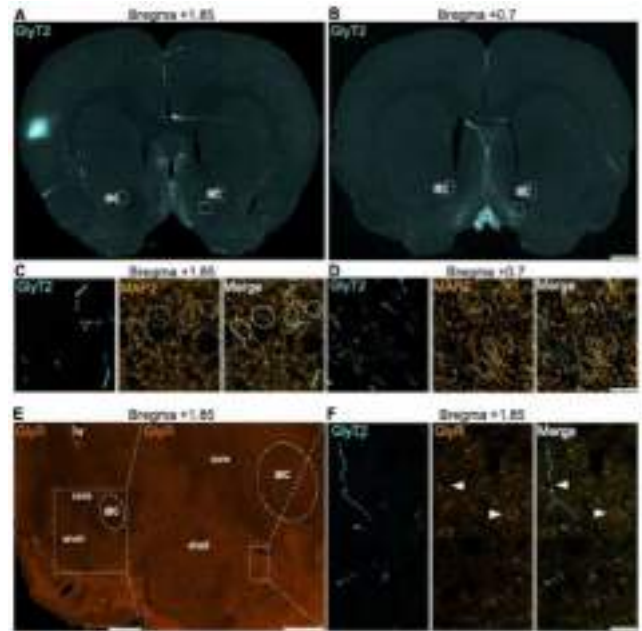


FIGURE 3 Immunoreactivity for GlyT2 and GlyR was confirmed in the nAc at the A/P level of probe insertion for microdialysis (bregma +1.85 mm) and more caudally (+0.7), although with a higher signal for GlyT2 in e.g. septal regions.

Results: The partial GlyT2-inhibitors NAGly (10mg/kg i.p.) and Opiranserin (20 mg/kg i.p.), but not the full GlyT2-inhibitor Org 25543, slightly elevated accumbal DA levels, while no effects on extrasynaptic glycine levels were observed. The reversible GlyT1-inhibitor Bitopertin (10mg/kg p.o.) produced a pronounced glycine and DA elevation but combined GlyT1- and GlyT2 inhibition did not yield any statistically significant additive effects. The IHC study confirmed the presence of GlyT2 and GlyRs in the nAc.

Conclusion: To conclude, this study indicates that GlyT2-expressing glycinergic neurons are involved in regulating nAc DA levels, albeit to a lesser extent than extrasynaptic GlyRs situated nearby GlyT1s. The extraspinal effects mediated by systemic treatment with GlyT2-inhibitors, i.e. involvement of the mesolimbic DA system, should be further explored.

Disclosure: Nothing to disclose.

EPO-0867 | Central mechanisms and therapeutic interventions in trigeminal neuralgia: A systematic neuroimaging and neuromodulation study

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Background and aims: Microvascular decompression (MVD) targets peripheral neurovascular conflict in trigeminal neuralgia (TN), yet its variable efficacy highlights the critical role of central nervous system adaptations. Vascular compression pathophysiology, central sensitization neuroimaging correlates, and their treatment response predictive value remain unclear. This study combined multimodal neuroimaging with MVD, rTMS and TGS interventions to elucidate TN central mechanisms and explore personalized therapeutic strategies.

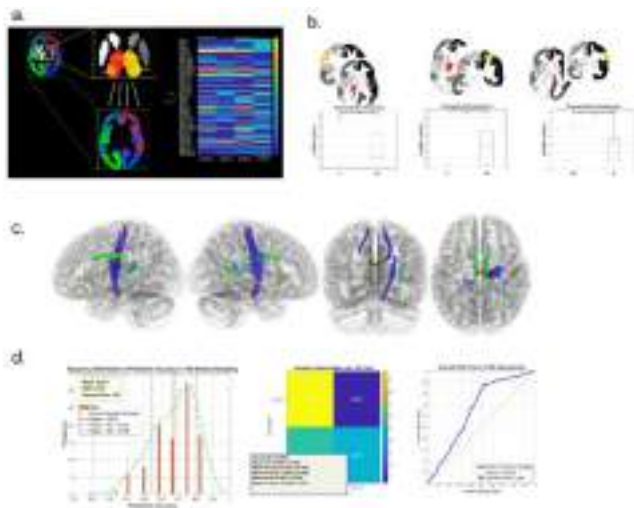


FIGURE 1 (a) rs-fMRI workflow: acquisition→preprocessing→ROI extraction→FC matrix construction; (b) NE vs E: enhanced thalamo-somatosensory FC ($p < 0.05$); (c) Subcortical somatomotor FA ↓ with longer disease duration ($FDR \leq 0.05$); (d) SVM MVD prediction performance.

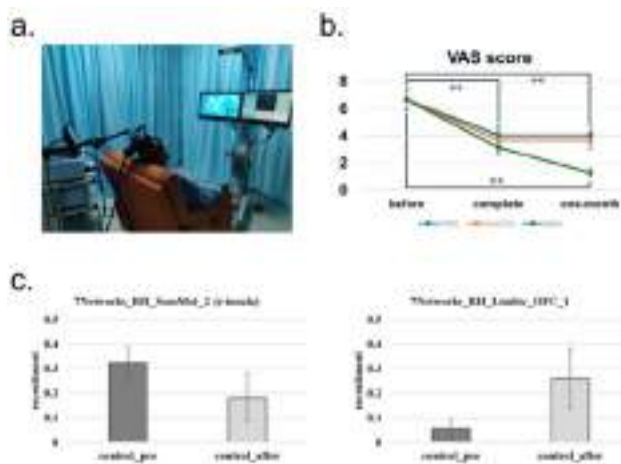


FIGURE 2 rTMS neural mechanisms. (a) Study design: randomization to active M1-rTMS, sham, active DLPFC-rTMS; (b) Post-intervention VAS pain reduction in all groups; (c) Divergent activation: M1-rTMS ↓ insula recruitment, sham/DLPFC ↑ prefrontal activity.

Methods: Three complementary studies: ① 60 TN patients (30 MVD responders/non-responders) and 30 HCs underwent pre-operative rs-fMRI and DTI, with thalamo-S1 FC analyzed (pipeline: Fig. 1a) and an SVM prediction model constructed; ② 34 early TN patients were randomized to active M1-rTMS, sham stimulation, active DLPFC-rTMS (Fig. 2a), with clinical scales and rs-fMRI assessed pre-/post-intervention for network reorganization; ③ 29 TN patients received longitudinal rs-fMRI before/after TGS (30 HCs as controls, electrode placement: Fig. 3a), with FC dynamics analyzed and feature stability verified via SVM.

Results: ① MVD non-responders showed enhanced thalamo-somatosensory FC (Fig. 1b), milder vascular compression, and reduced subcortical FA with structural-functional decoupling (Fig. 1c); ② SVM predicted MVD outcomes with 67.33% accuracy (Fig. 1d); ③ All rTMS groups achieved pain relief (Fig. 2b), with active M1-rTMS suppressing insula activation and sham/

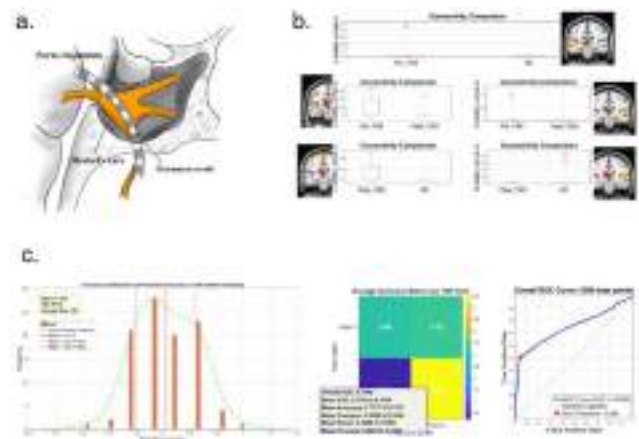


FIGURE 3 TGS study: (a) Electrode placement; (b) Pre-/post-TGS connectivity normalization; (c) SVM validation of treatment response (71.17% accuracy).

DLPFC groups enhancing prefrontal activity (Fig. 2c, targeted vs placebo-mediated); ④ TGS normalized aberrant thalamo-somatosensory hyperconnectivity (Fig. 3b, with regional heterogeneity), and SVM distinguished pre-/post-TGS states with 71.17% accuracy (Fig. 3c).

Conclusion: This study confirms TN as a spectrum disorder from peripheral compression to central sensitization, with thalamo-somatosensory FC as a core biomarker. It enables pre-operative MVD patient stratification, guides neuromodulation target optimization, and advances TN management from empirical surgery to circuit biomarker-informed personalized therapy.

Disclosure: Nothing to disclose.

Peripheral Nerve Disorders 2

EPO-0868 | Atherogenic lipid indices and electrophysiological severity in Carpal Tunnel Syndrome

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Background and aims: Carpal tunnel syndrome (CTS) is the most common entrapment neuropathy, yet the role of metabolic and vascular factors in disease severity is not fully understood. Composite lipid indices may better reflect microvascular and metabolic alterations than conventional lipid fractions. This study compared atherogenic lipid indices between CTS patients and healthy controls and evaluated their association with electrophysiological severity.

Methods: In this cross-sectional case-control study, 124 CTS patients and 113 age- and sex-matched controls were enrolled. Electrophysiological grading followed the Padua classification. Fasting total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), and remnant cholesterol (RC) were measured. Composite indices included the TG/HDL, non-HDL/HDL, and atherogenic index of plasma (AIP). Between-group comparisons

TABLE 1 Demographic and baseline biochemical characteristics of patients with carpal tunnel syndrome and healthy controls.

	CTS (n = 124)	Controls (n = 113)	p value
Age, years (mean ± SD)	46.3 ± 8.6	48.0 ± 12.8	0.098
Female sex, n (%)	92 (74.2)	81 (71.7)	0.664
Fasting glucose, mg/dL	96 (20.3)	93 (13)	0.001
HbA1c, %	5.7 (0.10)	5.5 (0.30)	<0.001
HDL-cholesterol, mg/dL	53 (2)	45 (14)	<0.001
LDL-cholesterol, mg/dL	116.6 (16.9)	100 (31.9)	<0.001
Total cholesterol, mg/dL	201 (17)	175 (40)	<0.001
Triglycerides, mg/dL	117 (11.5)	139 (10.2)	0.293
Remnant cholesterol, mg/dL (RC)	30 (9)	26 (20.5)	0.029
Atherogenic index of plasma (AIP)	0.35 (0.06)	0.44 (0.44)	0.100
TG/HDL ratio	2.21 (0.35)	2.76 (2.81)	0.100
Non-HDL/HDL ratio	2.78 (0.45)	2.72 (1.12)	0.125

Values are presented as mean ± SD or median (IQR). Between-group comparisons were performed using t-test or Mann-Whitney U test. CTS: carpal tunnel syndrome; RC: remnant cholesterol; AIP: atherogenic index of plasma.

TABLE 2 Biochemical parameters according to carpal tunnel syndrome severity.

	Mild CTS (n = 37)	Moderate CTS (n = 47)	Severe CTS (n = 40)	p value
Fasting glucose, mg/dL	98.5 (22.5)	96 (20)	99 (16)	0.202
HbA1c, %	5.65 (0.10)	5.70 (0.10)	5.60 (0.30)	<0.001
HDL-cholesterol, mg/dL	53 (1)	52 (3)	54 (2)	<0.001
LDL-cholesterol, mg/dL	107 (11.5)	116.6 (12.4)	120.9 (6.45)	<0.001
Total cholesterol, mg/dL	190 (6)	201 (10)	204 (7)	0.001
Triglycerides, mg/dL	117 (1)	115 (13)	121 (24)	0.010
Remnant cholesterol, mg/dL (RC)	30 (0.4)	32.4 (10)	29.1 (4.67)	0.023
Atherogenic index of plasma (AIP)	0.34 (0.01)	0.35 (0.10)	0.35 (0.06)	0.004
TG/HDL ratio	2.24 (0.33)	2.87 (0.71)	2.78 (0.15)	0.028
Non-HDL/HDL ratio	2.58 (0.37)	3.10 (0.78)	2.80 (0.46)	<0.001

Values are presented as median (IQR). Comparisons across CTS severity groups were performed using the Kruskal-Wallis test with Bonferroni-corrected post hoc analyses. CTS: carpal tunnel syndrome; AIP: atherogenic index of plasma; RC: remnant cholesterol.

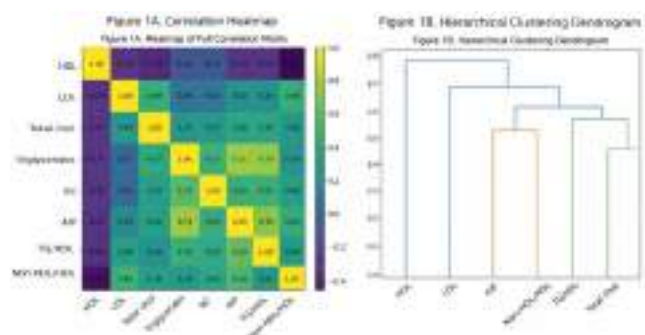


FIGURE 1 Correlation heatmap and hierarchical clustering of lipid parameters in CTS.

and correlations with severity were analyzed using non-parametric tests and Spearman correlation.

Results: Compared with controls, CTS patients had higher fasting glucose, HbA1c, total cholesterol, LDL-C, HDL-C, and remnant cholesterol (all $p < 0.05$). Composite indices (TG/HDL, non-HDL/HDL, AIP) were similar between groups. Electrophysiological severity showed significant associations with multiple lipid measures, with total cholesterol, LDL-C, TG, TG/HDL, non-HDL/HDL, AIP, and remnant cholesterol increasing progressively from mild to moderate and severe CTS (all $p < 0.05$). No significant sex-related differences were observed.

Conclusion: Composite atherogenic indices and RC were associated with electrophysiological severity rather than CTS presence, supporting a severity-focused metabolic framework. Atherogenic burden may contribute to progressive median nerve dysfunction beyond mechanical compression. Routine lipid-derived indices may aid risk stratification and enhance understanding of CTS pathophysiology.

Disclosure: Nothing to disclose.

EPO-0869 | Autonomic dysfunction in hereditary transthyretin amyloidosis: Real-world experience of stabilization under vutrisiran therapy

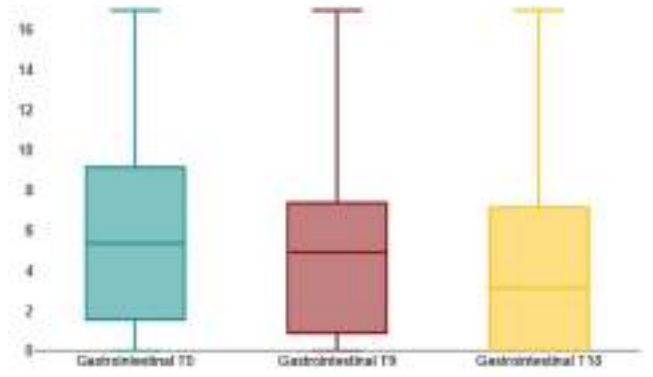
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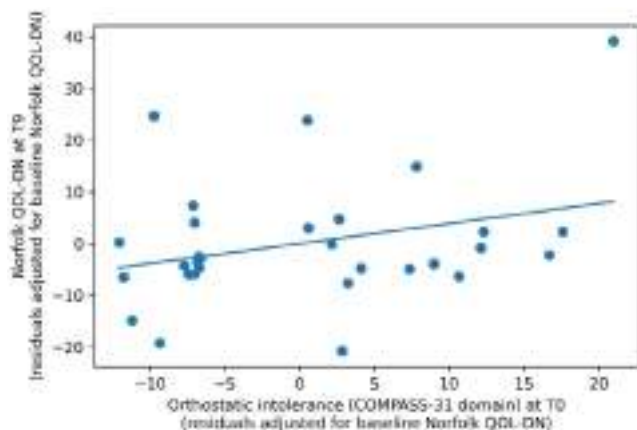
Background and aims: Hereditary transthyretin amyloidosis (ATTRv) is a systemic disease caused by the deposition of misfolded transthyretin (TTR), frequently affecting both peripheral nerves and the heart. Amyloid deposition prematurely involves autonomic ganglia, leading to early and often progressive autonomic dysfunction. Vutrisiran (Amvuttra), an RNA interference therapeutic targeting TTR, is approved for the treatment of hATTR and has demonstrated benefits on both neurological and cardiac outcomes.

Methods: We retrospectively analysed 57 patients with ATTRv treated with vutrisiran and followed within the Emilia-Romagna regional network. Demographic and clinical data were collected from 9 months before treatment initiation through 18 months of follow-up. Autonomic symptoms were assessed using the Composite Autonomic Symptom Score-31 (COMPASS-31), and disability was evaluated with the Norfolk Quality of Life-Diabetic Neuropathy (Norfolk QOL-DN) questionnaire.

Results: Repeated measures analyses of COMPASS-31 domains demonstrated overall longitudinal stability across follow-up, with particular regard to the gastrointestinal domain, which exhibited a directional trend toward improvement. Norfolk QOL-DN scores



Longitudinal distribution of COMPASS-31 gastrointestinal domain scores at baseline (T0), 9 months (T9), and 18 months (T18). Box plots show median, interquartile range and overall distribution of scores over time.



The figure shows the association between baseline orthostatic intolerance (COMPASS-31 domain) at T0 and Norfolk Quality of Life–Diabetic Neuropathy (Norfolk QOL-DN) at T9, with both variables adjusted for baseline Norfolk QOL-DN.

displayed a descriptive reduction from baseline to 18 months. Baseline COMPASS-31 domains were not independently associated with Norfolk QOL-DN at 9 months after adjustment for baseline disability; however, orthostatic intolerance showed a positive directional trend, suggesting that higher autonomic symptom burden at baseline may contribute to worse outcome.

Conclusion: Our real-world findings highlight the role of autonomic dysfunction in determining clinical disability in ATTRv and suggest that treatment with vutrisiran may be effective in stabilizing autonomic symptom progression. These results support the relevance of targeting dysautonomia as a key component of disease management in ATTRv.

Disclosure: Alnylam Pharmaceuticals.

EPO-0870 | Sensory neuronopathy in chronic alcohol users: A retrospective bicentric cohort study

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Background and aims: Chronic alcohol misuse typically causes length-dependent axonal polyneuropathy. Purely sensory ataxic presentations have been reported, but data are limited. We aimed to characterise sensory neuronopathy (SNN) associated with chronic alcohol misuse.

Methods: In a retrospective two-centre cohort (2008–2024), we analysed adults meeting clinical and electrophysiological criteria for SNN, reporting chronic alcohol misuse (≥ 100 g ethanol/week for ≥ 3 months), without any alternative aetiology. Severity was assessed using the modified Rankin Scale and a neurophysiological sensory score based on ten sensory nerves. Patients with and without a favourable outcome (mRS improvement ≥ 1) were compared.

Results: Twenty-five patients were included (median age 50 years; 64% female). They presented pure sensory non-length-dependent ataxic neuropathy (100%), of acute/subacute onset (88%), highly disabling (mRS ≥ 3 : 76%), painful (68%), asymmetrical (44%), with central nervous system involvement (32%). Precipitating events included unintentional weight loss (64%) and alcohol intake escalation (32%). Reported weekly consumption did not correlate with baseline severity. Markers of malnutrition (11/23, 48%) and vitamin deficiencies (18/23, 78%) were frequently observed, mostly involving B1, B9, and B12 pathways. Outcome was favourable in 16/21 (71%) and was more frequent among patients receiving B1 supplementation ($p=0.047$).

Conclusion: Our findings are consistent with an association between chronic alcohol misuse and a distinct SNN phenotype marked by rapid onset, pain, and favourable outcomes despite high baseline disability. Findings support a predominantly nutritional contribution, without ruling out direct alcohol neurotoxicity. Rapid-onset SNN should prompt assessment of alcohol exposure and nutritional status, with early vitamin supplementation when misuse is identified.

Disclosure: Nothing to disclose.

EPO-0871 | The electrophysiology of disease remission in chronic inflammatory demyelinating polyneuropathy

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Background and aims: Whether serial electrophysiology may be useful in chronic inflammatory demyelinating polyneuropathy (CIDP), is unknown.

Methods: We retrospectively studied electrophysiological findings during active disease and after remission off-treatment, in subjects with CIDP, at 2 UK neuropathy centres.

Results: We included 24 consecutive subjects. Eighteen subjects (75%) had typical CIDP, 3 (12.5%) had motor CIDP and 3 (12.5%) had distal CIDP. Mean age was 61.3 years. Mean interval between electrophysiological study during active disease and post-remission, was 63.25 months (SD: 52.0). Mean distal motor latency improved for median ($p<0.001$), ulnar ($p<0.001$), fibular ($p=0.01$) and tibial ($p=0.002$) nerves. Mean motor conduction velocity improved for median ($p<0.001$), ulnar ($p<0.001$) and fibular ($p=0.012$) nerves. Mean minimum F-wave latency improved for median ($p=0.003$), ulnar ($p=0.002$), and tibial ($p=0.027$) nerves. The proportion of nerves showing conduction block was reduced post-remission compared to during active disease (10/67 vs. 26/66; $p=0.002$). Demyelinating criteria were fulfilled by all subjects during active disease vs. 14/24 (58.3%) after remission ($p=0.006$). Electrophysiological quasi-normalization correlated with shorter pre-treatment disease duration ($p=0.002$). Amplitude of functional improvement correlated with fibular nerve CMAP amplitude at the first study ($p=0.024$).

Conclusion: Electrophysiological quasi-normalisation may occur in a third of subjects with CIDP in remission off-treatment and may be more likely with prompt diagnosis/treatment. Amelioration of most commonly studied parameters may be

observed in all tested nerves. Pre-treatment fibular nerve axonal loss may predict clinical improvement amplitude.

Disclosure: YAR has received consultancy honoraria from Sanofi, Argenx, Janssen, LFB, Polyneuron, Grifols, Takeda, Dianthus, Vitaccess; has received educational sponsorships from LFB and CSL Behring; has obtained research grants from LFB. CO has received speaker/consultancy honoraria from Takeda, Grifols and Terumo BCT. JF, RI and DA have no disclosures.

EPO-0872 | Comparative efficacy of treatments for chronic inflammatory demyelinating polyneuropathy: A network meta-analysis

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Background and aims: Chronic inflammatory demyelinating polyneuropathy is an immune-mediated neuropathy with multiple therapeutic options but limited head-to-head randomized comparisons. This study aimed to systematically compare and rank the efficacy and safety of available treatments for chronic inflammatory demyelinating polyneuropathy using a network meta-analysis of randomized controlled trials.

Methods: A systematic literature search was conducted across PubMed, EMBASE, Scopus, CENTRAL, and ClinicalTrials.gov from inception to January 14, 2025. Randomized controlled trials evaluating pharmacological treatments for chronic inflammatory demyelinating polyneuropathy were included. Outcomes assessed were disability measured by the Inflammatory Neuropathy Cause and Treatment score, functional independence measured by the Inflammatory Rasch-built Overall Disability Scale, muscle strength measured by the Medical Research Council sum score, grip strength, and safety outcomes. A frequentist network meta-analysis with random-effects modeling was performed, with Bayesian sensitivity analyses conducted to assess ranking consistency.

Results: Seventeen randomized controlled trials involving 1,079 participants were included. Subcutaneous immunoglobulin at 0.4g per kilogram demonstrated the greatest improvement in disability. Intravenous immunoglobulin at 1.0g per kilogram showed the strongest improvement in muscle strength. Efgartigimod significantly improved functional independence and ranked consistently across analyses. Plasma exchange showed the largest improvement in grip strength but was supported by a single trial. Corticosteroids ranked lowest across efficacy outcomes. Treatment rankings were consistent between frequentist and Bayesian analyses.

Conclusion: Intravenous immunoglobulin, subcutaneous immunoglobulin, and efgartigimod are the most effective therapies for improving disability, function, and strength in chronic inflammatory demyelinating polyneuropathy. These findings support individualized treatment selection based on patient-specific clinical goals.

Disclosure: Nothing to disclose.

EPO-0873 | Burden of Guillain-Barré syndrome in the United States in 2025

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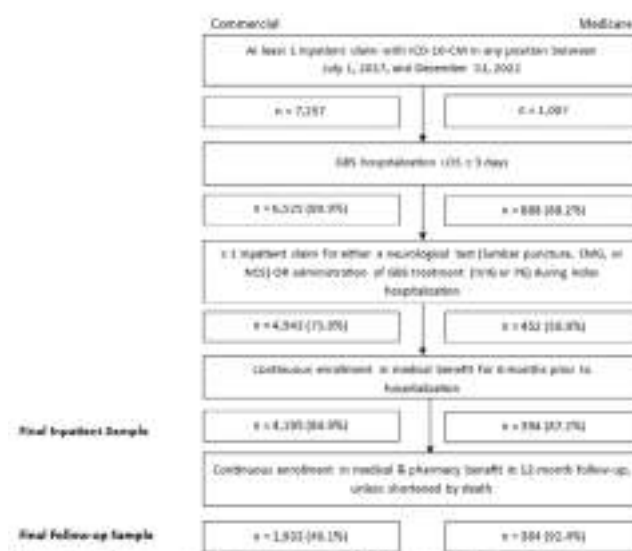
¹Oxford University Hospitals NHS Foundation Trust, Oxford, UK; ²Annexon Biosciences, Brisbane, USA; ³Annexon Biosciences, Brisbane, USA, and HealthEcon Consulting, Inc, Ancaster, Canada; ⁴Curta Inc, Seattle, USA; ⁵Magnolia Market Access, Bridgewater, USA; ⁶University of Minnesota, Minneapolis, USA

Background and aims: Guillain-Barre Syndrome (GBS) imposes substantial disability and economic burden, extending well beyond the initial hospitalisation. Real-world data quantifying this burden remain limited. This study characterised real-world GBS incidence, healthcare resource utilisation (HCRU), and costs in the US.

Methods: We analysed 2017–2023 data from a large US commercial claims database and Medicare Fee-for-Service (FFS) 5% sample to identify patients with GBS (Figure 1). Annual incidence rates were projected to the 2025 US population, and costs were adjusted for age and sex. Direct medical costs were estimated with paid amounts during the index hospitalisation and subsequent year, adjusted to 2024 USD. HCRU was measured during the 12 months following discharge from index hospitalisation.

Results: An estimated 8,039 GBS cases [95% CI: 7,734–8,364] occurred in the US in 2025, corresponding to an incidence of 2.39 per 100,000. During the index hospitalisation, 28.0% of commercial and 34.9% of Medicare patients required ICU care [mean (SD) ICU stay of 7.8 (14.2) and 9.1 (13.3) days, respectively] (Table 1), and age/sex-adjusted GBS-related medical costs averaged \$77,096 and \$50,429 for commercial and Medicare patients, respectively (Table 2). During the 12-month follow-up, GBS-related and all-cause medical costs averaged \$32,786 and \$67,822 for commercial and \$62,322 and \$104,919 for Medicare

Figure 1. Study Case Selection Algorithm Patient Attrition



Abbreviations: EMG, electromyography; GBS, Guillain-Barre Syndrome; ICD-10-CM, International Classification of Diseases, Tenth Revision, Clinical Modification; IVIg, intravenous immunoglobulin; ICD, length of stay; PE, plasma exchange; PE, plasmapheresis.

FIGURE 1 Study case selection algorithm patient attrition.

TABLE 1 Post-discharge GBS-related healthcare resource utilization in 12-month follow-up.

	Commercial (N=1,323)		Medicare (N=364)	
Index Hospitalization by HCBA Agency				
Mean (SD) Total LOS, days	12.4	18.2	18.3	31.4
Any ICU Stay, n (%)	541	38.6%	127	34.9%
Mean (SD) ICU LOS, days	7.8	18.2	9.1	33.3
12 Month Post-Discharge Follow-up GBS-related HCBA Metrics				
Inpatient Hospitalization, n (%)	219	38.5%	85	26.1%
Mean (SD) Visits	1.38	0.85	1.45	0.9
Emergency Department, n (%)	73	3.7%	31	8.0%
Mean (SD) Visits	1.24	0.52	1.36	0.67
Outpatient Hospital Visit, n (%)	853	42.1%	159	43.7%
Mean (SD) Visits	5.5	8.34	4.1	3.28
Outpatient Office Visit, n (%)	3,289	69.4%	390	52.2%
Mean (SD) Visits	7.67	11.68	6.24	8.88
Outpatient Rehabilitation, n (%)	297	36.6%	82	25.0%
Mean (SD) Visits	31.49	15.84	7.62	9.81
Inpatient Rehabilitation, n (%)	567	29.1%	115	32.7%
Mean (SD) Visits	1.3	0.37	1.18	0.37
Skilled Nursing Facility (SNF), n (%)	396	5.0%	73	20.1%
Mean (SD) Visits	1.00	0.34	1.45	0.87
Long-Term Care Facility (LTCF), n (%)	76	3.0%	28	8.0%
Mean (SD) Visits	1.13	0.35	1.24	0.58
Home Health, n (%)	525	22.2%	86	28.1%
Mean (SD) Visits	7.5	8.33	62.9	81.8
DME, n (%)	704	36.4%	140	49.3%
Wheelchair, n (%)	414	23.4%	85	23.4%
Walker, n (%)	328	17.0%	64	17.6%
Oxygen Support Equipment, n (%)	219	13.3%	37	10.2%

Abbreviations: DME, durable medical equipment; ICU, intensive care unit; LOS, length of stay; SD, standard deviation.
*Mean visits calculated among patients with at least one visit.

TABLE 2 Weighted medical direct cost components of resource utilization for hospitalization and 12-month follow-up, adjusted to 2024 dollars using the US medical CPI.

	Commercial (N=1,323)		Medicare (N=364)	
	Average	95% CI	Average	95% CI
Index Hospitalization	\$71,085	\$70,908 – \$83,647	\$50,439	\$44,354 – \$17,500
All Medical Costs, 12-month follow-up period				
GBS-related Medical Costs	\$31,786	\$27,894 – \$38,638	\$62,322	\$58,796 – \$74,238
All-Cause Medical Costs	\$67,822	\$60,339 – \$75,963	\$184,918	\$81,268 – \$118,462
All Medical Costs, including index hospitalization and 12-month follow-up period				
GBS-related Medical Costs	\$109,883	\$108,619 – \$119,848	\$152,793	\$99,088 – \$128,576
All-Cause Medical Costs	\$144,918	\$133,383 – \$157,678	\$155,347	\$128,593 – \$170,825
Pharmacy				
All-Cause Pharmacy Costs	\$4,487	\$3,528 – \$5,680	\$4,497	\$2,311 – \$7,512
Total Medical Costs including Index Hospitalization				
All-Cause Medical Costs	\$149,405	\$137,638 – \$162,008	\$159,847	\$129,904 – \$178,385

Abbreviations: CI, confidence interval; CPI, Consumer Price Index; DME, Durable Medical Equipment.

patients. Total GBS-related and all-cause medical costs averaged \$109,883 and \$144,918 for commercial and \$112,751 and \$155,347 in Medicare patients.

Conclusion: GBS imposes a substantial and persistent burden on patients and the US healthcare system, highlighting the need for more effective treatments.

Disclosure: This study was sponsored by Annexon. SR: Medical Advisory Board member of the Guillain-Barré syndrome and Associated Inflammatory Neuropathies (GAIN) patient charity. His department receives payment for the provision of diagnostic antibody testing, including of nodal/paranodal antibodies. He has received payment from Argenx for preparation and delivery of conference presentation on clinical trial, payment for serving on Argenx, Annexon, Dianthus and Hansa clinical trial advisory boards, and payment from CSL for delivering a conference symposium lecture. MK: Employment with and shareholder of Annexon Biosciences SV: Employee of HealthEcon Consulting and employee and shareholder of Annexon EB, LB: Employee of Magnolia Market Access BT, JD: Employee of Curta Inc JAA: Consultant fees: Akcea therapeutics, Alexion, Alnylam Pharmaceuticals, Annexon Biosciences, Argenx, CSL Behring, Grifols, Immunovant, Immupharma, Johnson & Johnson, Pfizer, and Takeda

EPO-0874 | Premature death as a major driver of total societal cost of Guillain-Barré syndrome in the United States

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Background and aims: Guillain-Barre Syndrome (GBS) is associated with substantial economic burden, including direct costs (hospitalisation and rehabilitation) and indirect costs (lost productivity and increased risk of premature death). We present the indirect cost of premature deaths among US patients hospitalised with GBS (USD 2025).

Methods: Incidence and mortality rates for patients hospitalised with GBS, derived from a claims analysis of commercially insured and Medicare Fee-For-Service 5% standard analytical files from 2017–2023, were projected to the 2025 US populations aged <65 and ≥65 years (average age: 45 and 70, respectively). Excess mortality was estimated using age-adjusted background mortality (Centers for Disease Control and Prevention Life Tables). A value of statistical life (VSL) was applied to excess deaths ≤12 months post-hospitalisation to estimate total premature mortality cost.

Results: 8,039 US GBS cases (95% CI: 7,734–8,364) were projected for 2025 (<65 years: 67%, ≥65: 33%). Excess mortality rates ≤12 months from hospitalisation were 4.98% (<65) and 22.1% (≥65) (including inpatient mortality: 1.8% vs 7.7%). Premature mortality costs (using the US Department of Health and Human Services age-agnostic 2025 central VSL estimate; \$13.2M) were \$3.446B (<65) and \$7.669B (≥65). The total annual estimate was \$11.114B for deaths ≤12 months from hospitalisation. Applying age-specific central estimates for VSL (age-specific US EQ-5D scores, Hamner 2026. 45yr: \$10.8M; 70yr: \$5.6M), resulted in premature mortality costs of \$6.086B (<65: \$2.812B, ≥65: \$3.274B). Results were robust regardless of statistical method.

Conclusion: Premature death in patients hospitalised with GBS is a major driver of GBS total costs from a US societal perspective.

Disclosure: This study was funded by Annexon Biosciences. SR: Medical Advisory Board member of the Guillain-Barré syndrome and Associated Inflammatory Neuropathies (GAIN) patient charity. His department receives payment for the provision of diagnostic antibody testing, including of nodal/paranodal antibodies. He has received payment from Argenx for preparation and delivery of conference presentation on clinical trial, payment for serving on argenx, Annexon, Dianthus and Hansa clinical trial advisory boards, and payment from CSL for delivering a conference symposium lecture. JAA: Consultant fees: Akcea therapeutics, Alexion, Alnylam Pharmaceuticals, Annexon Biosciences, argenx, CSL Behring, Grifols, Immunovant, Immupharma, Johnson & Johnson, Pfizer, and Takeda MK: Employee and shareholder of Annexon Biosciences SV: Employee of HealthEcon Consulting and employee and shareholder of Annexon Biosciences SCK, LB, BB,

EPO-0875 | De novo mutations in charcot-marie-tooth disease of Korean origin

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Background and aims: Charcot–Marie–Tooth disease (CMT), a genetically and clinically heterogeneous group of peripheral neuropathies, is characterized by progressive muscle weakness and significant limb deformities. De novo mutations are an important source of pathogenic variation in autosomal dominant CMT, yet their overall frequency, parental origin, and clinical implications beyond recurrent copy number variants remain poorly defined. The aim of this study was to systematically identify de novo mutations in autosomal dominant CMT and to investigate their genetic features, parental origin, and associated clinical phenotypes.

Methods: We examined 151 genetically diagnosed Korean parent–offspring trio families with autosomal dominant CMT, excluding cases with CMT1A duplication. Pathogenic variants were identified using Sanger sequencing, whole-exome sequencing, or targeted gene panels, and de novo status was confirmed by parental testing and paternity verification. Parental origin, CpG methylation status, mutation spectra, and clinical characteristics were analyzed.

Results: We identified 49 de novo point or small insertion/deletion mutations in 61 patients, corresponding to a de novo frequency of 40.4%. De novo mutations showed marked gene-specific differences, with high frequencies in INF2, MORC2, PMP22, and KIF5A. Most mutations were paternally derived and preferentially occurred at highly methylated CpG sites. Clinically, affected children carrying de novo mutations demonstrated significantly earlier disease onset compared with their parental founders, consistent with phenotypic anticipation.

Conclusion: De novo mutations contribute substantially to autosomal dominant CMT and exhibit distinct molecular and clinical patterns. These findings emphasize the value of trio-based analyses for genetic diagnosis and provide insights into mutational mechanisms and disease progression in inherited peripheral neuropathies.

Disclosure: Nothing to disclose.

EPO-0876 | Phenotypic severity in patients with hereditary motor and sensory neuropathy with somatic mosaic mutations

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Background and aims: Hereditary motor and sensory neuropathies (HMSN) are inherited peripheral neuropathies characterized by progressive muscle weakness and sensory dysfunction. Although somatic mosaicism in HMSN-associated genes has been reported, most cases show asymptomatic or mild phenotypes. However, some mosaic cases resemble patients with germline mutations. This study aimed to characterize somatic mosaicism and its clinical impact in a Korean HMSN family.

Methods: Whole exome sequencing (WES) followed by variant filtering was used to identify genetic causes. The degree of somatic mosaicism was quantified by analyzing approximately 100 cloned sequences per mutation site and calculating variant ratios. Clinical phenotypes were compared with those of patients carrying typical germline mutations.

Results: Two cases of somatic mosaicism were identified: p.Cys104Tyr in INF2 and p.Ser729Arg in TRPV4. The estimated mosaicism levels in blood were approximately 24% and 30%, respectively. The individual with the INF2 mutation exhibited mild peripheral neuropathy, whereas the individual with the TRPV4 mutation showed severe clinical manifestations. The INF2 case was considered gonadal mosaicism. Notably, the p.Cys104Tyr mutation in INF2 was associated with demyelinating peripheral neuropathy without focal segmental glomerulosclerosis (FSGS), expanding the known phenotype of INF2-related disease.

Conclusion: This study represents one of the first reports describing somatic mosaicism involving INF2 and TRPV4 in HMSN. The severity of clinical phenotypes in mosaic cases appears to depend on both the mutation site and the proportion of mutant alleles in affected tissues. These findings suggest that somatic mosaicism may play an important role in inter- and intra-familial phenotypic heterogeneity in HMSN.

Disclosure: Nothing to disclose.

EPO-0877 | Patient-reported outcomes and safety from the phase 2 ARDA study of Empasiprubarb in multifocal motor neuropathy

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Background and aims: Multifocal motor neuropathy (MMN) is a rare, chronic, predominantly complement-driven neuropathy, causing axonal degeneration and progressive, disabling, asymmetric limb weakness. Empasiprubart binds C2, blocking the classical and lectin complement pathways while leaving the alternative pathway intact. The phase 2 ARDA study (NCT05225675) assessed safety and efficacy of empasiprubart in adults with MMN.

Methods: Enrolled participants had probable/definite MMN (2010 European Federation of Neurological Societies/Peripheral Nerve Society guidelines) and proven intravenous immunoglobulin (IVIg) dependency and were on a stable IVIg regimen before randomisation. Participants were assigned to 1 of 2 dosing cohorts, each randomised 2:1 to empasiprubart or placebo. Efficacy endpoints included patient-reported outcomes (PROs) measured by the Chronic Acquired Polyneuropathy Patient-Reported Index (CAP-PRI), the Fatigue Severity Scale (FSS), the Patient Global Impression of Change (PGI-C), the Treatment Satisfaction Questionnaire for Medication (TSQM-14), and the Health-Related Productivity Questionnaire (HRPQ). Safety and tolerability were evaluated throughout the study.

Results: Overall, 27 participants (empasiprubart, $n=18$; placebo, $n=9$) were randomised per cohort (total $N=54$). Participants in cohort 2 were older, with longer duration of disease and time since first IVIg treatment versus cohort 1. Overall, empasiprubart was generally well tolerated. Most adverse events

TABLE 1 HRPQ results for work activities and for household chores activities.

	Cohort 1				Cohort 2			
	Baseline		Time of last assessment (DBT)		Baseline		Time of last assessment (DBT)	
	Empasiprubart (n=12)	Placebo (n=6)	Empasiprubart (n=6)	Placebo (n=3)	Empasiprubart (n=12)	Placebo (n=6)	Empasiprubart (n=6)	Placebo (n=3)
Percentage of participants who were able to perform or partially completed employment, recreation (DBT)	12.7 (0.0–25.0)	0.0 (0.0–0.0)	20.0 (0.0–25.0)	33.3 (0.0–40.0)	7.5 (0.0–20.0)	0.0 (0.0–0.0)	0.0 (0.0–0.0)	20.0 (0.0–25.0)
Percentage of participants who were able to perform or partially completed household activities (DBT)	8.3 (0.0–12.5)	0.0 (0.0–0.0)	33.3 (0.0–40.0)	33.3 (0.0–40.0)	0.0 (0.0–0.0)	0.0 (0.0–0.0)	16.7 (0.0–20.0)	0.0 (0.0–0.0)

DBT, double-blind treatment period; HRPQ, Health-Related Productivity Questionnaire.

were mild/moderate. Improvements in PROs were generally observed with empasiprubart compared with placebo in both cohorts, including CAP-PRI (Figure 1A), FSS (Figure 1B), PGI-C (improvement: 94% vs 11% in cohort1; 83% vs 44% in cohort 2), TSQM-14 (Figure 2A–C), and HRPQ (Table 1).

Conclusion: Empasiprubart improved PROs versus placebo, with patients consistently reporting less fatigue and better quality of life.

Disclosure: MP: advisory board for Alexion, Amgen, Annexon, argenx, Catalyst, CSL Behring, Dianthus, EMD Serono/Merck, Immunovant, Janssen, Johnson & Johnson, Medlink, Momenta, Octapharma, Sanofi Genzyme, Takeda, TerumoBCT, UCB; KB: advisory board and research support for argenx, CSL Behring, Johnson & Johnson, Takeda, Sanofi, Octapharma; KLG: nothing to disclose; TH: research or institutional support for Annexon, argenx, Dianthus, Immunovant, Janssen, Nuvig, Sanofi, Takeda; CH: research or institutional support, advisory board, speaker honoraria for argenx, Alexion, Avidity, Biogen, Dyne, Immunovant, Johnson & Johnson, Lupin, Remgen, Roche, UCB, Vertex; CK: consulting agreement, honoraria, advisory board for Acceleron, Alpine, Alexion, Alnylam, Amgen, Amicus, Annexon, argenx, AstraZeneca, Biogen, Corino, CSL Behring, Genentech, Ionis, Johnson & Johnson, Neuroderm, Novo Nordisk, Octapharma, Pfizer, Sanofi, Takeda, UCB, Vertex, Zai lab; SC: consulting agreement for argenx, via PPD, part of Thermo Fisher Scientific; EKP and MV: employees of argenx; LQ: research or institutional support for Alnylam, Annexon, argenx, Avilar, Biogen, CIBERER, CSL Behring, Dianthus, Fundació La Marató, Grifols, Janssen, LFB, Lundbeck, Merck, Novartis, Octapharma, Roche, Sanofi, UCB; JAA: consulting agreement for argenx, Alexion, Alnylam, Annexon, CSL Behring, Grifols, Immunovant, Immupharma, Pfizer, Takeda; SP: research or institutional support for ADOC, argenx, Berlin-Chemie Menarini, Kedrion, Mylan, Octapharma, Pfizer, Roche, Salveo, Sanofi Genzyme, Teva Actavis, Wörwag.

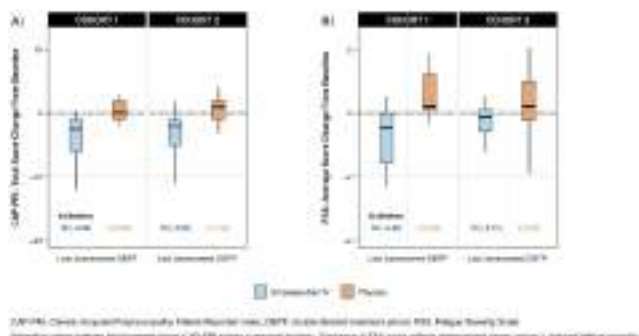


FIGURE 1 Box plots of (A) CAP-PRI* total score and (B) FSS† average score of change from baseline at last assessment during the DBTP.

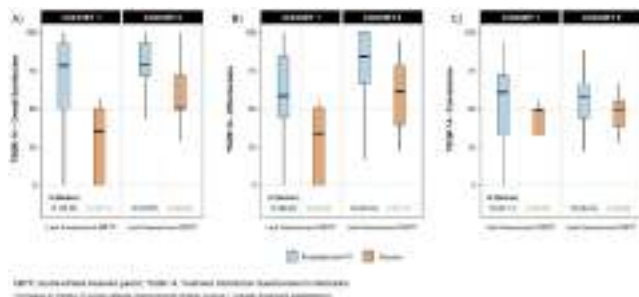


FIGURE 2 Box Plots of values of (A) TSQM-14* (overall satisfaction), (B) TSQM-14* (Effectiveness), and (C) TSQM-14* (Convenience).

EPO-0878 | Early onset radiation-induced leukoencephalopathy in patients treated for a glioblastoma by STUPP protocol and risk factors evaluation

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Background and aims: Radiation-induced leukoencephalopathy (RIL) is a known late complication of brain radiotherapy in glioblastoma (GBM) survivors, whereas early-onset RIL remains poorly described. This ancillary study of the phase III ASTER trial evaluated the incidence of early-onset RIL and explored associated risk factors, including exposure to losartan, a peroxisome proliferator-activated receptor gamma (PPAR γ) agonist.

Methods: We retrospectively analyzed ASTER trial participants treated with standard radiotherapy plus temozolomide (STUPP protocol). Brain MRI performed before radiotherapy and at six months post-treatment was centrally reviewed. Early-onset RIL was defined by new or progressive confluent T2/FLAIR white-matter hyperintensities with associated cortico-subcortical atrophy and no contrast enhancement, quantified using a simplified Scheltens scale. Cognitive function and quality of life were assessed using the Montreal Cognitive Assessment (MoCA) and EORTC QLQ-C30/BN20 questionnaires. Clinical, treatment-related, and genetic factors (PPAR γ) were analyzed.

Results: Thirty-one patients were evaluable. Early-onset RIL was identified in 29% ($n=9$), with high inter-rater diagnostic agreement. No significant associations were observed with age, MGMT promoter methylation, IDH1 mutation, or PPAR γ polymorphism. Losartan exposure was not associated with reduced RIL incidence. At six months, patients with RIL exhibited significantly lower MoCA scores and impaired physical and emotional functioning compared with patients without RIL.

Conclusion: Early-onset RIL affects nearly one-third of GBM patients treated with the STUPP protocol and is associated with early cognitive and functional decline. No specific clinical, genetic, or treatment-related risk factors, including losartan exposure, were identified. Early monitoring and larger prospective studies are warranted.

Disclosure: No disclosure

EPO-0879 | Application of the PNS-care score in real world clinical practice: Insights from a neuro-immuno-oncology perspective

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Background and aims: To describe the diagnostic criteria compliance of patients with suspicion of paraneoplastic neurologic syndromes (PNSs) who have positive antibody testing.

Methods: We performed an observational retrospective study of a prospectively collected cohort of patients under PNSs suspicion with positive paraneoplastic antibodies between January-2024 and December-2025. Patients were classified using the PNS-Care Score.

Results: We included 14 patients, 8 (57%) were male, median of age of 66 (SD: 19) years. The PNSs-Care Score was definite in 3 (21%), probable in 1 (7%) and possible in 4 (29%) with 6 (43%) patients classified eventually as non-PNSs. In the definite PNS group, 1/3 (33%) patient presented with Morvan syndrome with anti-titin, anti-LGI1 and anti-CASPR2 positive antibodies, and was diagnosed with metastatic thymoma; 1/3 (33%) patient had anti-Hu and Anti-SOX1 positive antibodies presenting with Eaton-Lambert phenotype and small-cell lung cancer (SCLC); and 1/3 (33%) patient had also anti-Hu positive antibody and SCLC presenting with sensory neuronopathy. The patient with probable PNS showed a positive anti-SOX1 antibody, but did not present with a defined phenotype nor and a tumor was not found after diagnosis workup. In patients with possible PNSs 2 (50%) had anti-NMDAR encephalitis, 1 (25%) had epileptic crisis with anti-recoverin, anti-Ma2 and anti-CV2 antibodies, and 1 (25%) had anti-Yo positivity with atypical parkinsonism.

Conclusion: These findings offer an insight of the application of the PNS-Care Score in real world clinical practice, supporting the association between “high-risk antibodies” and “high or intermediate risk neurologic phenotypes” with cancer. Further multicenter studies focusing on PNS-Care Score applications are granted.

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Background and aims: This study aimed to explore the correlation between the effectiveness of second-line chemotherapy in patients with recurrent glioblastoma (rGBM) who underwent second surgery and drug treatment tests on patient-derived explants.

Methods: Sixteen adult patients diagnosed with IDH wild-type glioblastoma underwent surgery for recurrent GBM, with 9 cases studied retrospectively and 7 prospectively. Patient-derived 3D glioblastoma organoids (GBM-EXPs) were established from resected tumors and treated with chemotherapy. NADH FLIM method was used to assess drug-sensitivity in vitro.

Results: Among patients progressing during first-line temozolomide (TMZ) therapy or within three months after discontinuation, most exhibited TMZ resistance by NADH-FLIM ($p=0.02$), with a longer PFS1 in TMZ-sensitive compared with TMZ-resistant rGBM-EXPs (15.9 vs 10.9 months; $p=0.07$), independent of MGMT promoter methylation. In the second-line setting, patients treated with regorafenib whose rGBM-EXPs were regorafenib-sensitive experienced a significantly longer PFS2 than those with resistant organoids (7.5 vs 3.6 months; $p=0.04$). In the prospective cohort, ex-vivo testing prior to second-line therapy was associated with a trend toward longer PFS2 (7.4 vs 4.6 months; $p=0.08$) and a significantly improved OS2 (not reached vs 6.3 months; $p=0.03$) compared with the retrospective cohort, with no difference in MGMT promoter methylation rates. Paired longitudinal analyses demonstrated acquisition of TMZ resistance in rGBM-EXPs compared with untreated GBM-EXPs.

Conclusion: This study shows that the sensitivity of rGBM-EXP to TMZ mirrors the timing of recurrence observed in vivo. NADH-FLIM-based drug response assessment in rGBM-EXP may play a role in predicting the efficacy of second-line therapies.

Disclosure: Nothing to disclose.

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Background and aims: In the management of patients diagnosed with high-grade glioma (HGG), practices and rehabilitation programs display significant variability in real-world settings. The HGG Rehab survey aimed to glean insights from physicians specialized in neuro-oncology and affiliated with scientific societies in Italy: AINO, SINch and SIRN.

Methods: The questionnaire was released in June 2023, and responses were recorded until February 2024. One hundred eleven responses were collated.

Results: A median value of 40% of HGG patients from Neurosurgical and Neuro-oncologic/Oncologic Divisions were evaluated for rehabilitation, and a median value of 20% were deemed eligible for a rehabilitation. The rate of requests for physiatric evaluation was progressively higher as KPS increased, especially patients with KPS 60–70 (80%). Over 50% of responders reported viable outpatient rehabilitation programs during adjuvant radio-chemotherapy, with good tolerance (>55%). Seventy percent of practitioners recommended rehabilitation evaluation for newly diagnosed GBM patients and over 50% for those with recurrent GBM. Clinical benefits were observed in a median of 80% of patients, ranging from 30% to 100%. Recognized benefits of rehabilitation encompassed facilitating home discharges (82%), psychological benefits (65%), aiding the progress of adjuvant oncologic therapies (58%), and enhancing quality of life (40%). Key rehabilitation time points for newly diagnosed HGG patients included 5 days post-surgery, after histo-molecular diagnosis/treatment plan definition, and at recurrence.

Conclusion: The HGG Rehab Survey offers invaluable insights shared by three scientific societies into rehabilitation pathways for HGG patients in Italy. It highlights the usual criteria employed for the selection of patients and time points for outcome evaluation.

Disclosure: Nothing to disclose.

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Background and aims: Glioblastoma (GBM) lethality stems from its aggressive infiltration of healthy brain tissue.



FIGURE 1 Automated segmentation and tracking of patient-derived glioblastoma (GBM) cells. Representative single frame from a 72-hour live-cell microscopy video showing patient-derived GBM cells. The cells were automatically segmented using the Cellpose software.

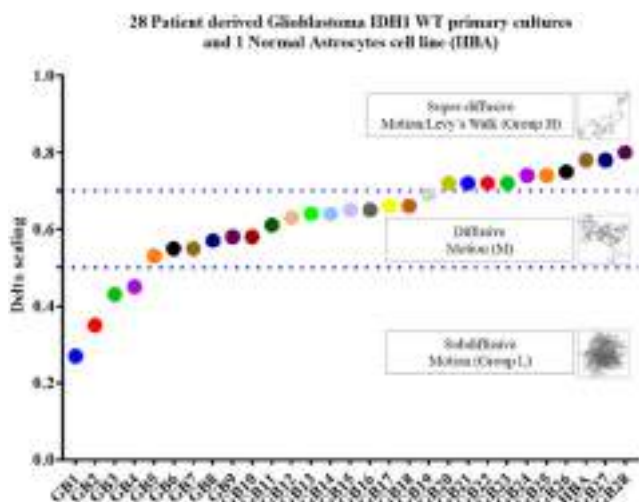


FIGURE 2 Delta Scaling (δ) Analysis of Glioblastoma Cell Lines via Diffusion Entropy Analysis This figure illustrates the distribution of the Delta scaling parameter values, derived from the Diffusion Entropy Analysis (DEA), for 28 patient-derived GB IDH1 Wild Type.

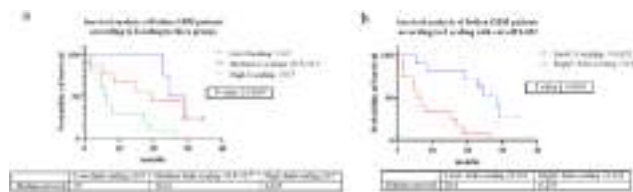


FIGURE 3 Survival stratification of GBM IDH-wildtype patients based on migration efficiency. (a) Kaplan-Meier survival curves for the initial three-tier stratification (low, medium, and high δ scaling).

Traditional metrics often fail to capture the complexity of this movement. This study aimed to characterize GBM cell motility using advanced modeling to identify new therapeutic vulnerabilities and stratification markers.

Methods: We developed Single-Cell Behavior Live Imaging (ScBLI), integrating live imaging with Diffusion Entropy Analysis (DEA) across 28 primary patient-derived cultures. By analyzing 3,842 trajectories, we classified cells into Low (L), Medium (M), and High (H) groups based on their δ scaling parameter.

Results: High δ scaling cells exhibited Lévy-like, persistent migration. Paradoxically, while these cells were slower and covered less total distance, they demonstrated the highest migratory efficiency and superior chemotactic performance. Clinical correlation revealed a clear linear progression: High δ Scaling: Shortest patient survival (poorer prognosis). Low δ Scaling: Longest patient survival. Transcriptomic analysis confirmed distinct gene expression signatures corresponding to these behavioral clusters, suggesting a biological basis for these movement strategies.

Conclusion: This study introduces migratory efficiency as a superior predictor of GBM malignancy over traditional metrics like speed or distance. We demonstrate that a highly efficient spatial exploration strategy—rather than the sheer magnitude of movement—is a critical driver of tumor invasiveness and poor clinical outcomes. These findings redefine GBM motility, establishing δ scaling as a novel biomarker for aggressive disease.

Disclosure: Nothing to disclose.

EPO-0883 | Clinical, molecular, and transcriptomic factors associated with seizures at onset in patients with glioblastoma

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Background and aims: Epileptogenesis in glioblastoma (GBM) primarily arises within the peritumoral cortex, yet the contribution of this region to tumor progression and seizure generation remains poorly understood. This study investigated the interactions between the tumor core and the peritumoral microenvironment by comparing gene expression profiles between GBM patients presenting with seizures at onset (GBM-EP+) and those with other symptoms (GBM-EP-). We also evaluated differences in clinical and molecular characteristics, as well as overall survival (OS) and progression-free survival (PFS).

Methods: We analyzed a retrospective cohort of 32 GBM patients using spatial transcriptomics. All patients had confirmed GBM, and complete clinical and molecular data. Molecular profiling, including IDH mutation and MGMT promoter methylation status, was performed by next-generation sequencing. Spatial transcriptomic analyses were conducted using the GeoMx DSP® platform.

Results: Seizures at presentation were not associated with OS or PFS, and no clinical or molecular variables were significantly linked to seizure occurrence. Spatial analyses revealed limited differences between tumor and peritumoral compartments. However, distinct transcriptional programs characterized the two groups. EP+ tumors exhibited broader gene deregulation with a mesenchymal-like invasive phenotype, whereas EP- GBMs showed a more proliferative profile marked by enhanced protein synthesis and mitochondrial activity, along with a relatively more inflamed immune microenvironment. Transcription factor analysis identified BRD4 as a key regulator of the mesenchymal program in EP- GBMs.

Conclusion: These results highlight the peritumoral microenvironment's role in shaping GBM phenotype and demonstrate the value of spatial transcriptomics in linking tumor biology with epilepsy.

Disclosure: Nothing to disclose.

EPO-0884 | A new diagnostic and therapeutic care pathway for early palliative care integration in patients with glioblastoma: A single centre experience

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Background and aims: Glioblastoma is the most aggressive primary brain tumour in adults characterised by major clinical challenge due to multidimensional symptoms, rapid progression and poor prognosis. Early integration of palliative care (PC) from diagnosis can improve quality of life, symptom control and psychosocial well-being. We developed and implemented an integrated care pathway for early PC in patients with glioblastoma.

Methods: The diagnostic and therapeutic care pathway (PDTA) was designed between February 15th 2024 and December 13th 2024 in a tertiary oncological institute involving oncologists, radiotherapists, PC specialists, nurses, and quality department. Implementation began on January 1st 2025 as standard care. Demographic and clinical data were collected from patient charts and analysed descriptively.

Results: The PDTA allows systematic identification of PC needs in patients with glioblastoma hospitalised for diagnosis or surgery. A neuro-oncology nurse specialist assesses PC needs and arranges either an in-hospital PC consultation or an outpatient PC visit with oncologist/radiotherapist within four to seven weeks after diagnosis. Between January 1st to December 31st 2025, 24 patients underwent in-hospital or outpatient PC consultations. The PDTA significantly shortened the time to the first PC consultation—from 80 days in 2024 to 37 days in 2025. Main clinical issues addressed included symptom management, advance care planning, and respite care.

Conclusion: Integrating early PC as a standard of care for glioblastoma enables systematic needs assessment, enhances quality of life, and fosters communication on treatment goals and preferred care settings. These findings encourage broader adoption of coordinated and patient-centred models to optimize outcomes.

Disclosure: Nothing to disclose.

EPO-0885 | Machine learning to predict distant recurrence spread in Glioblastoma: A radiomic signature from standard T1-CE tumor core and peritumoral margins

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Background and aims: Predicting tumor spread in Glioblastoma (GB) is essential for prognosis and guiding treatment decisions. The clinical challenge lies in distinguishing whether recurrence will remain confined to the original tumor bed (local) or manifest as distant spread. While perfusion MRI is often used for this purpose, it requires complex protocols. This study evaluated whether Radiomic features derived solely from standard contrast-enhanced T1-weighted (T1-CE) images could effectively predict this dissemination pattern.

Methods: We analyzed 109 patients with confirmed recurrence. Tumor was manually segmented by two experienced radiologists. Peritumoral margins were generated via a 5mm isotropic dilation of the core mask. Radiomic features were extracted separately from both regions using PyRadiomics. The pipeline included Z-score normalization and Mutual Information feature selection. Eight classifiers were benchmarked: Multi-Layer Perceptron, Support Vector Machine, XGBoost, K-Nearest

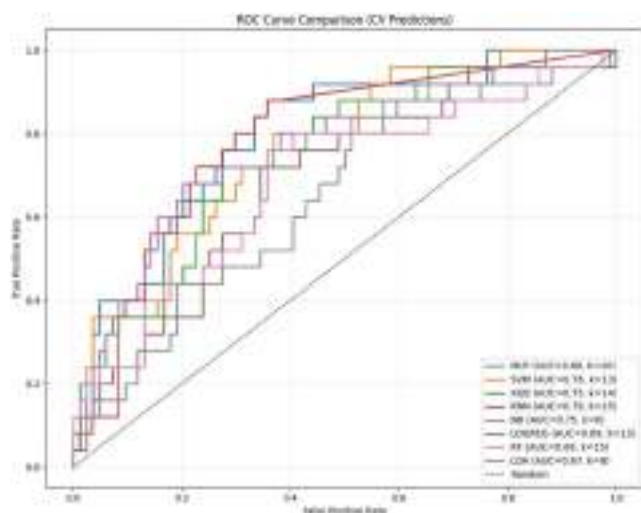


FIGURE 1 Receiver operating characteristic (ROC) curve comparison. The plot displays the performance of the eight machine learning classifiers benchmarked for distinguishing local from distant Glioblastoma recurrence.

Neighbors, Naive Bayes, Logistic Regression, Random Forest, and Linear Discriminant Analysis. Class weighting was applied where applicable, and all models were validated via Leave-One-Out Cross-Validation (LOOCV).

Results: The MLP model achieved the highest performance (AUC 0.80, accuracy 82.6%, Figure 1). Feature selection analysis revealed a robust "Radiomic signature" of 8 features consistently selected across different classifiers. Notably, 3 of these predictors were derived from the geometric dilated peritumoral margins.

Conclusion: This analysis demonstrates that T1-CE Radiomics provides a robust method for predicting GB recurrence topography. The significant contribution of marginal features confirms that infiltration signals are detectable in structural images. While these preliminary findings warrant prospective validation, this approach transforms widely available standard baseline imaging into robust non-invasive prognostic biomarkers.

Disclosure: Nothing to disclose.

EPO-0886 | Neurological immune related adverse events of checkpoint inhibitors: A prospective study

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Background and aims: Neurological immune related adverse events (nirAEs) of checkpoint inhibitors (ICIs) are increasingly recognized in clinical practice. Retrospective studies may underreport nirAEs. The aim of this study was to prospectively determine the incidence of nirAEs, including grade 1 events.

Methods: Electronic PRO-CTCAE based questionnaire was administered to immunotherapy-naïve ICIs treatment candidates at baseline and at 3, 6 and 12 months to assess neurological and

psychiatric symptoms including: insomnia, fatigue, symptoms of depression, poor concentration, memory problems, dizziness, problems with vision, swallowing, urination, numbness/weakness/clumsiness in extremities, nausea, vomiting, headache, muscle pain, joint pain. Reported symptoms were grouped into putative clinical syndromes and analyzed in relation to other clinical characteristics (age, sex, type of tumor and treatment).

Results: Seventy-nine of 89 patients responded at baseline. Fifty-nine patients responded to at least one follow-up questionnaire (age median 66 [36–79], twenty-three [39.0%] females). The most common tumor was non-small cell lung carcinoma (22; 37.3%). Nineteen (32.2%) patients reported new or worsened symptoms indicative of polyneuropathy (10), myositis/myopathy (5) and/or encephalopathy (4). 7/10 patients with polyneuropathy symptoms had prior or ongoing chemotherapy. Thus, possible nirAEs occurred in 12/59 (20.3%) patients. Age, gender, tumor type, treatment (PD1/PDL1 ICIs alone or in combination with CTLA4 ICIs) and a history of autoimmune disease were not found to be statistically significant risk factors.

Conclusion: The incidence of adverse reactions, especially those of a lower grade, may be underestimated. Dedicated questionnaires can improve the detection of these events.

Disclosure: This work was supported by the Charles University project GA UK No 171324. PRO-CTCAE questionnaire (<https://healthcaredelivery.cancer.gov/pro-ctcae/>) was used in this study.

EPO-0887 | Neurological vulnerability to ICANS in CAR T-cell therapy: Impact of comorbidity and EEG-cognitive monitoring

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Background and aims: Immune effector cell-associated neurotoxicity syndrome (ICANS) frequently complicates chimeric antigen receptor (CAR) T-cell therapy, yet neurological vulnerability factors remain poorly defined. This study aimed to identify neurological predictors of ICANS and assess combined EEG-MoCA monitoring.

Methods: We conducted a retrospective monocentric study (Hospices Civils de Lyon), including adults treated with CAR T-cells for B-cell lymphoma between 2019 and 2024. Neurological comorbidity was defined as any pre-existing central nervous system disorder, including lymphoma involvement, cerebrovascular, neuroinflammatory, traumatic, neoplastic, or neurodegenerative disorders, or prior seizure history. EEG and MoCA were obtained at baseline and day 7. ICANS risk factors were assessed using logistic regression, and longitudinal MoCA changes using linear mixed-effects models.

Results: Among 356 patients, 152 (43%) developed ICANS, including 29 (18%) with severe forms. Neurological comorbidity was independently associated with ICANS (adjusted odds ratio [aOR] 2.07, 95% confidence interval [CI] 1.22–3.55; $p=0.007$), along with older age (per 10-year increase: aOR 1.42, 95% CI 1.19–1.70; $p<0.001$) and treatment with Yescarta versus other CAR T-cell products (aOR 2.44, 95% CI 1.53–3.94; $p<0.001$). EEG abnormalities during days 4–10 affected up to 68% of patients with ICANS. A MoCA decline of at least 2 points at day 7 was

TABLE 1 Baseline characteristics according to immune effector cell-associated neurotoxicity syndrome status.

Table 1. Baseline characteristics by ICANS status			
Variable	No-ICANS, N = 201	ICANS, N = 152	p-value
Age	63 [18–82]	67 [29–88]	<0.003
Woman	37% (75/204)	38% (58/152)	0.8
CAR-T product			0.002
Yescarta	93% (188/204)	78% (117/152)	
Other CD19 CAR-T	39% (80/204)	27% (41/152)	
Other	7.3% (16/204)	2.6% (4/152)	
Lines before lenisaparesis	3 [1–9]	2 [1–11]	0.2
LDS	235 [123–1,487]	247 [338–1,678]	0.2
Comorbidities			
Cardio-vascular	32% (65/204)	41% (61/152)	0.080
Other cancer	13% (25/204)	12% (18/152)	>0.5
Autoimmune	4.0% (8/200)	6.0% (9/150)	0.5
Psychiatric	3.1% (7/200)	2.0% (3/150)	0.5
Neurological	17% (33/204)	31% (47/152)	0.003
Neurological exploration			
Abnormal EEG	13% (21/159)	14% (18/134)	0.9
Background EEG frequency (Hz)	9.00 [5.00–12.00]	9.00 [7.00–12.00]	0.029
NoCA score	27.00 [15.00–30.00]	26.00 [13.00–30.00]	0.044

Median [Min–Max]; % (n/N)
Wilcoxon rank-sum test, Fisher's exact test
Data are presented as % (n/N) for categorical variables and as median [range] for continuous variables.
P values were calculated using Pearson χ^2 test, Fisher exact test, or Wilcoxon rank-sum test, as appropriate.

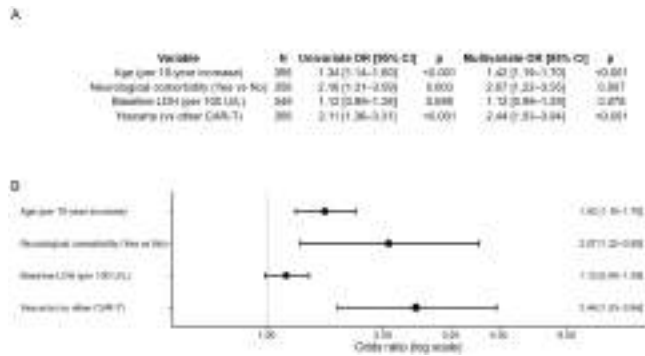


FIGURE 1 Neurological predictors of immune effector cell-associated neurotoxicity syndrome (ICANS). (A) Univariable and multivariable logistic regression analyses. (B) Forest plot of adjusted odds ratios with 95% confidence intervals.

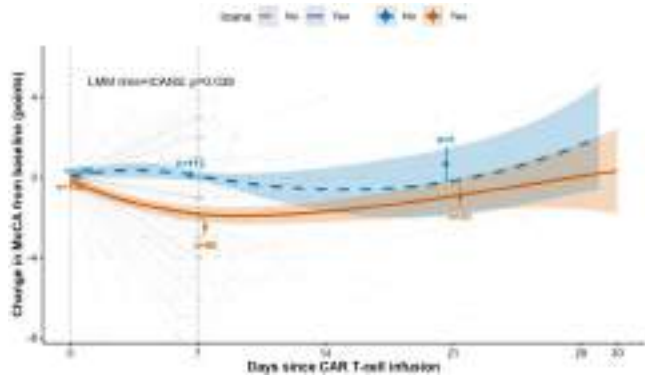


FIGURE 2 Change in Montreal Cognitive Assessment score after CAR T-cell infusion according to ICANS status. Individual trajectories, smoothed group trends with 95% confidence intervals, and group means at days 0, 7, and 21 are shown.

more frequent in ICANS (31.2% vs 12.8%; $p < 0.001$), with greater longitudinal decline (beta -0.09 points per day; $p = 0.038$).

Conclusion: Pre-existing neurological comorbidities are independently associated with an increased risk of ICANS.

Combined EEG and MoCA monitoring provides a pragmatic framework to characterize neurological vulnerability and follow ICANS-related cognitive trajectories.

Disclosure: Nothing to disclose.

EPO-0888 | Peripheral nerve toxicity of immune checkpoint inhibitors and CAR-T therapy: A multicentric cohort study

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Background and aims: Among neurological complications of cancer immunotherapy peripheral neuropathies are recognized immune-related adverse events (NP-irAE) of checkpoint inhibitors (ICI), whereas CART-related neuropathies (NP-CART) are rarely reported. We aim to describe clinical features, treatment, and outcomes of this complication.

Methods: Retrospective multicenter study of NP-irAE and NP-CART across 20 Spanish hospitals (2018–2025). Neuropathies were classified as acute (<4 weeks), subacute (4–8w) or chronic (>8w). We assessed severity with INCAT scale and outcomes at one month and last follow-up (good outcome: complete recovery or ≥ 2 -point mRS decrease).

Results: NP-irAE occurred in 13/144 (9%) neurological irAE; 3 male (23%), median age 72 years (IQR: 68–74). Median onset was 44 days (24–113) post-ICI initiation, median INCAT 0+3.5. Twelve (92%) had polyradiculopathy, and 1 (8%) sensory neuropathy. Three (23%) had CNS involvement with high-risk antibodies (2 anti-Hu, 1 anti-Ma2). Twelve (92%) received steroids and 8 (62%) IVIg. At 1 month, 11 (85%) improved; at last follow-up (median 33 months, 15–54), 7 (54%) had good outcomes. Among 600 anti-CD19 CAR-T recipients, 4 (0.6%) developed NP-CART; 3 male (75%), median age 54.5 years (45–57). One patient (25%) had prior severe ICANS. Median onset was 26 days (16–35) post-infusion. Three (75%) had polyradiculopathy with median INCAT 2+5; 1 (25%) cranial neuropathy. Two received steroids and 2 IVIg. All improved at 1 month; at last follow-up (median 40 months, 9–86), 2 (50%) had good outcomes.

Conclusion: NP-CART occurred earlier than NP-NiRAE. Although both are severe, high therapy responsiveness highlights the importance of prompt recognition and treatment.

Disclosure: Nothing to disclose.

Movement Disorders 11

EPO-0889 | Characteristics of the intestinal microbiome in Parkinson's disease patients

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Background and aims: Growing evidence implicates the gut-brain axis and gut microbiome dysbiosis in PD pathogenesis via immune mediators, the enteric nervous system and the vagus nerve, leading to microglial activation and chronic inflammation that may promote α -synuclein aggregation.

Methods: The cohort included 73 PD patients followed at a municipal outpatient clinic in Tashkent. Gastrointestinal autonomic dysfunction was quantified using the gastrointestinal domain of SCOPA-AUT, with a working severity grading (0–3 minimal; 4–7 mild; 8–12 moderate; ≥ 13 severe). Gut microbiota composition was analyzed using Osipov's gas chromatography–mass spectrometry (GC–MS) method, based on detection of microbial markers (specific fatty acids and related compounds) without culture.

Results: Signs of dysbiosis were identified in 90% of patients. Total bacterial load ranged from 16,848 to 38,784 units (mean $27,488 \pm 8,012$), exceeding reference values for healthy individuals; 44.4% showed hypercolonization ($>35,000$), potentially linked to slowed motility and constipation. The “microbiota core” (Firmicutes + Actinobacteria) varied between 28–48% (mean $39.2 \pm 6.9\%$), and was reduced in 20% of patients, indicating depletion of obligate anaerobes. Fungal flora (mainly *Candida*) exceeded the accepted threshold in 55.5%. The viral component averaged $7.0 \pm 2.3\%$ (reference $\sim 6\%$), with elevated levels in $\sim 20\%$ of patients, accompanied by higher endotoxin and a more pronounced anaerobic deficit; a positive correlation between viral load and *Clostridium* spp. was reported ($r \approx 0.58$).

Conclusion: Overall, the findings indicate a persistent dysbiotic profile in PD, with depletion of obligate flora and increased opportunistic and viral components, potentially contributing to chronic inflammation and disease progression.

Disclosure: Nothing to disclose.

EPO-0890 | Inflammatory mechanisms in Parkinson's disease and vascular Parkinsonism: IL-6, fecal calprotectin, and *Helicobacter pylori*

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Background and aims: This study aimed to evaluate local and systemic inflammatory markers (interleukin-6, fecal

calprotectin) and *Helicobacter pylori* infection in patients with Parkinson's disease (PD) and vascular parkinsonism, and to determine their clinical and laboratory associations.

Methods: A total of 52 patients were consecutively enrolled (32 with PD and 20 with vascular parkinsonism) and followed in an outpatient setting in Tashkent during 2020–2025. Clinical and demographic parameters were recorded; disease stage was assessed using the Hoehn and Yahr scale, and motor severity was evaluated with UPDRS. Fecal calprotectin was measured by ELISA, serum IL-6 by ELISA, and *H. pylori* infection by the urease breath test. Statistical analysis included parametric and non-parametric comparisons, chi-square/Fisher's exact tests, and Spearman correlations, with $p < 0.05$ considered significant.

Results: Elevated fecal calprotectin was detected in 34.4% of PD patients versus 9.3% in vascular parkinsonism ($p < 0.05$). Calprotectin levels correlated with Hoehn and Yahr stage ($r = 0.42$; $p < 0.05$) and UPDRS III ($r = 0.38$; $p < 0.05$), showing a tendency toward higher values at later disease stages. Increased IL-6 was more frequent in PD (85%; mean 10.28 ± 4.30 pg/mL) than in vascular parkinsonism (59.4%; 8.13 ± 3.13 pg/mL; $p < 0.05$). In addition, *H. pylori* prevalence was higher in PD (37.5% vs 8.3%; $p < 0.05$) and was associated with longer disease duration and greater motor impairment. Positive correlations were also observed between *H. pylori* parameters and both calprotectin and IL-6.

Conclusion: The findings support the contribution of intestinal and systemic inflammation to parkinsonism mechanisms and suggest that these markers may be useful for differential diagnosis, risk stratification, and more personalized clinical.

Disclosure: Nothing to disclose.

EPO-0891 | Rapidly progressive posterior cortical syndrome with alien limb: Diagnostic clues to sporadic Creutzfeldt–Jakob disease

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Background and aims: Corticobasal syndrome (CBS) is classically attributed to corticobasal degeneration (CBD), yet sporadic Creutzfeldt–Jakob disease (sCJD) can present with a CBS phenotype, often driven by posterior network dysfunction.

Methods: We reviewed the “CJD-CBS/alien limb” literature to extract specific discriminators—particularly patterns of cognitive involvement and structural involvement that can meaningfully shift pre-test probability early.

Results: Posterior alien limb phenomenon with non-dominant parietal dysfunction and visual-parietal signs (optic ataxia/neglect) supported a reproducible clinico-anatomical motif in CJD-CBS. While most CJD-CBS reports include striatal/basal ganglia abnormalities, a small subset shows complete structural and metabolic sparing of basal ganglia with dominant posterior cortical DWI changes and posterior hypometabolism on FDG-PET. This “striatum-sparing, posterior-cortical” phenotype is unusual for typical sCJD and can misdirect clinicians toward CBD unless specifically recognized. Contrary to the heuristic that early dementia is obligatory in CJD, several CJD-CBS cases show minimal cognitive impairment at onset, with later emergence of visuospatial deficits consistent with posterior network

primacy and helping explain early diagnostic anchoring on CBD. A parallel biomarker–imaging dissociation with markedly elevated total tau and normal p-tau plus RT-QuIC positivity supports prion disease even when amyloid markers are reduced, as low Aβ42 in CJD may not imply concomitant Alzheimer pathology.

Conclusion: The combination of posterior ALP + visual-parietal signs should trigger a topographic differential: assess posterior cortical DWI ribboning, actively interrogate basal ganglia involvement (or sparing) on MRI/FDG-PET, and integrate RT-QuIC with tau patterns—especially when cognition is initially preserved. Recognizing the “posterior, striatum-sparing CJD-CBS” subgroup may prevent systematic misclassification as CBD and accelerate appropriate prion-focused work-up.

Disclosure: Nothing to disclose.

EPO-0892 | Essential tremor five years follow-up study of MRgFUS in a real-world setting

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Background and aims: This study evaluated the long-term outcomes of magnetic resonance-guided focused ultrasound (MRgFUS) thalamotomy in patients with medication-refractory essential tremor (ET). ET is a common neurological condition. Unilateral and staged-bilateral (≥9 months apart) MRgFUS thalamotomy are CE-approved. This global, open-label FDA Mandated extension study enrolled participants after completion of the pivotal trial as the first cohort in a real-world setting. It further investigates long-term follow-up outcomes after MRgFUS thalamotomy.

Methods: Treatment effectiveness was assessed by changes in tremor severity and motor function using the Clinical Rating Scale for Tremor (CRST) and quality of life with the Quality of Life in Essential Tremor (QUEST) questionnaire. Safety was monitored through documentation of adverse events (AEs).

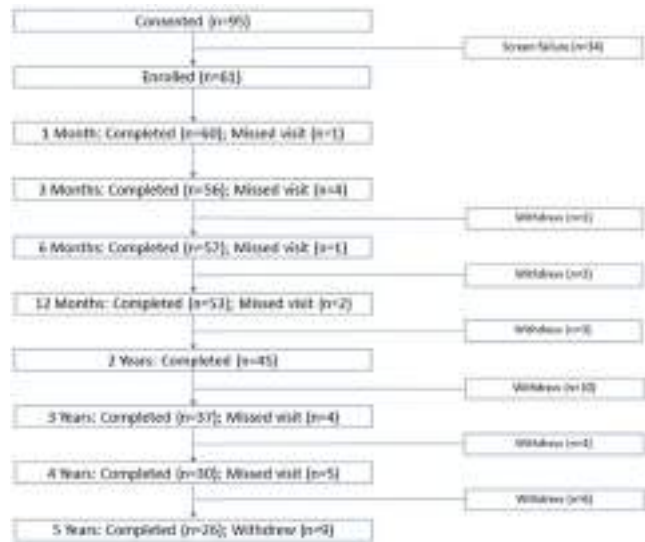


FIGURE 1 Patient disposition.



FIGURE 2 CRST scores and QUEST.

TABLE 1 Number of subjects reporting adverse events.

Adverse event	Total (n = 61)	1 Month (n = 11)	2 Months (n = 11)	3 Months (n = 11)	4 Months (n = 11)	5 Months (n = 11)	6 Months (n = 11)	7 Months (n = 11)	8 Months (n = 11)	9 Months (n = 11)
Headache	20 (33%)	14	11	10 (16%)	10	10	8	8	8	8 (13%)
Headaches/migraine	10 (16%)	14	11	10 (16%)	10	10	8	8	8	8 (13%)
Fatigue	20 (33%)	14	11	10 (16%)	10	10	8	8	8	8 (13%)
Dizziness	10 (16%)	14	11	10 (16%)	10	10	8	8	8	8 (13%)
Joint discomfort	10 (16%)	14	11	10 (16%)	10	10	8	8	8	8 (13%)
Stomach	10 (16%)	14	11	10 (16%)	10	10	8	8	8	8 (13%)
Depression	10 (16%)	14	11	10 (16%)	10	10	8	8	8	8 (13%)
Insomnia	10 (16%)	14	11	10 (16%)	10	10	8	8	8	8 (13%)
Weight	10 (16%)	14	11	10 (16%)	10	10	8	8	8	8 (13%)

Results: Among 61 subjects (mean age 69.5 ± 14.0 years; 67.2% male), 26 (42.6%) completed 5-year follow-up (Figure 1). MRgFUS thalamotomy produced sustained improvements in tremor, motor function, and disability. From baseline to 1 and 5 years, percentage improvements were: CRST A+B 62.2% and 51.9%, tremor severity 75.6% and 67.4%, and CRST-C 65.4% and 35.4%, respectively. Quality-of-life scores improved 53.6% at 1 year and 43.7% at 5 years (Figure 2). Most treatment-related AEs were mild (85%) or moderate (12%), 3% were severe; over half resolved within six months (Table 1).

Conclusion: These findings complement the original pivotal data outcome supporting long-term safety and effectiveness of MRgFUS thalamotomy in 1st side medication-refractory ET patients. Further studies should assess 2nd-side treatment requirement and long-term outcomes. Meanwhile, MRgFUS thalamotomy offers a safe, durable, and meaningful option for improving quality of life in ET.

Disclosure: All authors are Insightec employees.

EPO-0893 | Homer 3 antibodies: Emerging biomarkers in cerebellar dysfunction

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Background and aims: Homer-3 is a postsynaptic scaffolding protein expressed in Purkinje cells and plays a crucial role in calcium signaling mediated by metabotropic glutamate receptors. Cerebellitis associated with anti-Homer-3 antibodies is rare. Most reported patients present with vertigo and gait disturbance, while neurological examination almost invariably reveals ataxia and nystagmus. Cerebrospinal fluid (CSF) analysis typically shows pleocytosis and type 2 oligoclonal bands, whereas brain MRI findings are usually non-specific. Anti-Homer antibodies may be detected in serum or CSF. Various immunotherapeutic strategies have been used, including corticosteroids, intravenous immunoglobulin (IVIG), plasmapheresis, mycophenolate mofetil, and rituximab, with variable clinical responses

Methods: A 51-year-old woman presented with subacute cerebellar ataxia, choreiform movements, and inability to walk, since five months. Corticosteroid therapy worsened choreiform movements. Neurological examination revealed gaze-evoked nystagmus, dysmetria, choreiform movements in all four limbs, impaired deep sensation, and inability to stand unassisted. Deep tendon reflexes were absent. CSF analysis showed a normal cell count and type 1 oligoclonal bands. Brain MRI and EEG

were unremarkable. Extensive immunological, virological, and screening for malignancy, revealed no pathological findings. Serum testing was positive for anti-Homer-3 antibodies, while CSF was negative.

Results: Treatment with IVIG resulted in significant clinical improvement, allowing ambulation with bilateral support.

Conclusion: Anti-Homer-3 cerebellitis should be considered in the differential diagnosis of subacute-onset ataxia, as early identification and timely immunotherapy may improve outcome if initiated before irreversible neuronal damage occurs.

Disclosure: Nothing to disclose.

EPO-0894 | Phase angle as a robust indicator of clinical severity in patients with Huntington's disease

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Background and aims: Huntington's disease (HD) is a hereditary neurodegenerative disorder involving progressive motor, cognitive, and neuropsychiatric impairment, alongside systemic alterations in body composition. Bioelectrical impedance analysis (BIA)-derived parameters, specifically phase angle (PA), are potential markers of cellular integrity but remain underexplored in HD.

Methods: We enrolled 23 patients with genetically confirmed HD and 25 age- and sex-matched healthy controls (HC). Body composition and hydration were assessed via BIA. Within the HD group, we analysed clinical severity (UHDRS motor score ≤ 25 vs > 25) and genetic burden (CAG repeats < 42 vs ≥ 42). Multivariable regression models adjusted for age, sex, and height were used to control for anthropometric confounding. Partial correlations controlling for age were also performed.

Results: Compared to HC, HD patients showed significantly lower PA ($p = 0.008$) and body cell mass index (BCMI) ($p = 0.013$), with higher extracellular and lower intracellular water percentages ($p < 0.02$). Within the HD cohort, higher UHDRS scores were independently associated with lower PA ($p < 0.03$), while differences in muscle mass did not survive adjustment. Higher CAG repeats were associated with increased extracellular water volume ($p < 0.02$) but not with PA. In multivariable models, the UHDRS score was the primary predictor of PA.

Conclusion: HD is associated with reduced cellular integrity and altered fluid distribution. Phase angle appears to be a robust, severity-sensitive marker independent of body size and genetic load. BIA-derived PA may represent a clinically meaningful biomarker of disease severity in HD, warranting further longitudinal investigation.

Disclosure: Nothing to disclose.

“favorable outcome” (FO; mRS < 5). Biomarkers were compared with ANCOVA, correlations tested, and ROC analysis performed.

Results: Compared with controls, iNPH patients showed higher triglycerides, triglyceride/high-density lipoprotein (TG/HDL) and neutrophil/HDL ratios, and lower HDL. Within iNPH, patients with “high-grade” CSO-EPVS exhibited higher TG/HDL and neutrophil/HDL ratios than those with low-grade. PO patients had higher TG and TG/HDL and lower HDL than FO patients. A TG/HDL cut-off of 2.91 best predicted PO at 5 years.

Conclusion: iNPH patients exhibit a characteristic lipid profile within metabolic dysregulation and peripheral inflammation, tracked on a structural level by increased CSO-EPVS, a marker of glymphatic impairment. TG/HDL index appears to predict long-term clinical outcome, supporting its potential as prognostic marker in iNPH.

Disclosure: Nothing to disclose.

EPO-0897 | Deep stimulation in brain the Middle East: First UAE experience comparing subthalamic and pallidal targets for Parkinson's disease

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Background and aims: Deep brain stimulation (DBS) of the subthalamic nucleus (STN) and globus pallidus internus (GPi) is an established therapy for advanced Parkinson's disease (PD). However, comparative outcome data from the Middle East are limited, and it remains unclear whether target-specific differences reported in randomized trials translate to regional real-world practice. We report outcomes of STN and GPi DBS from a single tertiary center in the United Arab Emirates.

Methods: We retrospectively reviewed 50 consecutive patients with PD who underwent bilateral STN or GPi DBS between 2020 and 2025 (37 STN, 13 GPi). Demographics, clinical phenotype, levodopa equivalent daily dose (LEDD), Unified Parkinson's Disease Rating Scale part III (UPDRS-III) scores, and adverse events were collected. Outcomes were summarized by target, with exploratory comparisons acknowledging baseline heterogeneity and limited statistical power.

Results: GPi patients had earlier motor symptom onset and universal dyskinesias, while STN candidates demonstrated

more heterogeneous phenotypes. Both targets were associated with substantial LEDD reduction (STN $49.9\% \pm 32.4$; GPi $37.5\% \pm 22.0$). Variability and unequal sample sizes limited definitive target comparisons. Mean OFF-medication to DBS-ON UPDRS-III improvement exceeded 80% for both targets, likely influenced by high levodopa responsiveness, non-blinded assessments, and variable postoperative timing.

Conclusion: In this first comparative DBS analysis from the UAE, both STN and GPi DBS produced meaningful medication reduction and robust motor improvement. Methodological limitations preclude conclusions on target superiority, supporting individualized, phenotype-guided target selection and the need for larger prospective regional studies.

Disclosure: Nothing to disclose.

EPO-0898 | Spatial multi-omics identifies early synaptic pruning and context-specific dopaminergic vulnerability in Synucleinopathies

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Background and aims: The molecular events that precede overt neurodegeneration in Parkinson's disease (PD) remain poorly defined. Incidental Lewy body disease (iLBD), a prodromal state characterized by brainstem-predominant Lewy pathology in the absence of clinical symptoms, provides a unique opportunity to investigate early pathogenic mechanisms underlying PD.

Methods: We applied a multi-omic spatial profiling approach to post-mortem midbrain tissue obtained from neurologically unaffected controls, iLBD cases, and patients with PD. This integrated strategy combined spatial transcriptomics, proteomics, and α -synuclein seed amplification assays (SAA), along with proximity ligation assay (PLA), to assess molecular alterations and misfolded α -synuclein across disease stages.

Results: We identified early and spatially distinct molecular changes in the substantia nigra pars compacta (SNpc) of iLBD cases, including a marked upregulation of the microglial synaptic pruning factor C1QC. These alterations occurred independently of overt Lewy pathology or detectable misfolded α -synuclein, as determined by SAA and PLA, and were preferentially enriched at GABAergic synapses, indicating early vulnerability of inhibitory circuits. Notably, similar changes were absent in Alzheimer's disease cases with comorbid SNpc Lewy pathology, suggesting that α -synuclein aggregation alone is insufficient to drive SNpc remodeling.

Conclusion: Our findings implicate microglia-mediated synaptic dysfunction as an early, preclinical feature of PD and suggest that targeting microglial-synaptic interactions may represent a

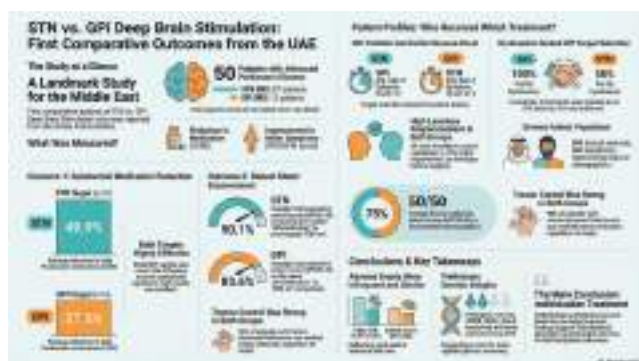


FIGURE 1 STN vs. GPi deep brain stimulation: First comparative outcomes from the UAE.

therapeutic strategy to halt disease progression prior to the onset of neurodegeneration.
Disclosure: Nothing to disclose.

EPO-0899 | **Gastrointestinal autonomic dysfunction defines an axial-functional vulnerability phenotype in drug-naïve Parkinson's disease**

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Background and aims: Autonomic dysfunction is common in Parkinson's disease (PD) from its prodromal stage, yet its heterogeneity and relationship with clinical features remain incompletely understood.
Methods: We analyzed a two-center cohort of 88 newly diagnosed, drug-naïve PD patients who were assessed using the SCOPA-AUT (The scales for autonomic dysfunction) questionnaire. Motor-related ADL and severity were evaluated using UPDRS parts II and III, with predefined subscores for bradykinesia, rigidity, tremor, and postural instability/gait difficulty (PIGD). Autonomic burdens, especially gastrointestinal (GI) dysautonomia severity, were examined using a median split to define mild and moderate to severe subgroups, and the comparisons between the groups were performed.

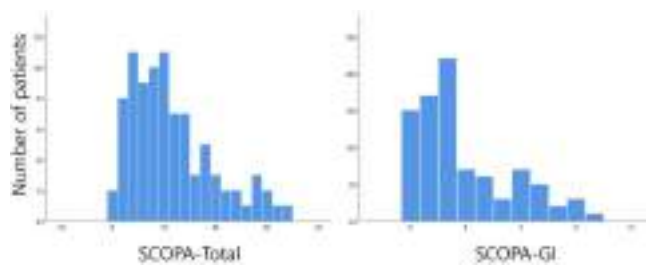


FIGURE 1 The distribution of autonomic symptoms.

TABLE 1 Correlation between autonomic symptoms and motor ADL and severity.

SCOPA-AUT Domain	r	P-value	r	P-value
Subtotal score	0.645	<0.001	0.675	<0.001
Urinary	0.391	0.001	0.325	0.001
Cardiovascular orthostatic	0.327	0.001	-0.008	0.924
Thermoregulatory	0.401	<0.001	0.099	0.561
Papillospasm	0.118	0.280	-0.019	0.918
Sexual function-III	0.187	0.340	0.031	0.881
Sexual function-IV	0.005	0.958	0.116	0.462
SCOPA-AUT score	0.588	<0.001	0.228	0.008
SCOPA-AUT Domain	Median subscores ^a			
Subtotal score	Intactness	Rigidity	Tremor	PIGD
Subtotal score	0.218 (0.000)	0.217 (0.000)	0.147 (0.071)	0.208 (0.000)
Urinary	0.224 (0.000)	0.000 (0.707)	0.004 (0.925)	0.188 (0.000)
Cardiovascular orthostatic	-0.009 (0.906)	-0.102 (0.446)	-0.025 (0.848)	0.146 (0.395)
Thermoregulatory	0.006 (0.970)	0.011 (0.932)	-0.006 (0.984)	0.006 (0.997)
Papillospasm	-0.009 (0.904)	0.013 (0.911)	0.019 (0.830)	0.119 (0.000)
Sexual function-III	0.071 (0.692)	-0.006 (0.958)	-0.002 (0.991)	0.061 (0.000)
Sexual function-IV	-0.101 (0.561)	0.114 (0.520)	-0.240 (0.178)	0.133 (0.401)
SCOPA-AUT score	0.267 (0.007)	0.000 (0.489)	0.009 (0.980)	0.188 (0.110)

^a Reporting as r/sd
^b Sexual dysfunction are included from the estimation of total SCOPA-AUT score

TABLE 2 Comparison of characteristics of drug-naïve PD patients according to GI severity (A) and clinical indicators highly associated with moderate to severe GI severity calculated by logistic regression (B).

(A)					(B)				
Characteristic	GI-Mild (n=48)	GI-Moderate to Severe (n=40)	P-value	OR (95% CI)	Characteristic	GI-Mild (n=48)	GI-Moderate to Severe (n=40)	P-value	OR (95% CI)
Age (years)	60.8 ± 8.2	60.9 ± 7.5	0.988		UPDRS II	10.8 ± 2.5	12.5 ± 2.5	<0.001	1.05 (1.02-1.08)
Sex (male/female)	28/20	25/15	0.845		UPDRS III	10.5 ± 2.5	12.0 ± 2.5	<0.001	1.05 (1.02-1.08)
UPDRS II	10.8 ± 2.5	12.5 ± 2.5	<0.001		UPDRS Total	21.3 ± 3.0	24.5 ± 3.0	<0.001	1.05 (1.02-1.08)
UPDRS III	10.5 ± 2.5	12.0 ± 2.5	<0.001		MoCA-K	28.5 ± 1.5	27.5 ± 1.5	<0.001	0.95 (0.92-0.98)
UPDRS Total	21.3 ± 3.0	24.5 ± 3.0	<0.001		PIGD	1.5 ± 0.5	2.0 ± 0.5	<0.001	1.05 (1.02-1.08)
MoCA-K	28.5 ± 1.5	27.5 ± 1.5	<0.001		Tremor	0.5 ± 0.5	0.5 ± 0.5	0.988	
PIGD	1.5 ± 0.5	2.0 ± 0.5	<0.001		Sexual function-III	0.5 ± 0.5	0.5 ± 0.5	0.988	
Tremor	0.5 ± 0.5	0.5 ± 0.5	0.988		Sexual function-IV	0.5 ± 0.5	0.5 ± 0.5	0.988	
Sexual function-III	0.5 ± 0.5	0.5 ± 0.5	0.988		Sexual function-IV	0.5 ± 0.5	0.5 ± 0.5	0.988	
Sexual function-IV	0.5 ± 0.5	0.5 ± 0.5	0.988						

Results: SCOPA-AUT scores showed non-normal, right-skewed distributions, indicating substantial heterogeneity in autonomic burden. Among autonomic domains, GI symptoms showed the strongest correlation with activities of daily living (UPDRS II; $r=0.64, p<0.001$), exceeding other autonomic domains. GI dysautonomia correlated with overall motor severity ($r=0.38, p<0.001$), bradykinesia ($r=0.31, p=0.003$), and PIGD score ($r=0.28, p=0.008$), but not with tremor severity. Compared with GI-mild patients, the GI-moderate to severe subgroup demonstrated significantly worse UPDRS II and III scores, higher PIGD burden, lower MoCA-K scores, and a higher proportion of PIGD motor subtype, without increased motor asymmetry. In multivariable analysis, functional disability (UPDRS II) and cognitive performance independently predicted GI-moderate to severe status.
Conclusion: In drug-naïve PD, GI autonomic dysfunction identifies a distinct axial-functional vulnerability phenotype characterized by greater postural-gait impairment, functional disability, and cognitive vulnerability. Early GI dysautonomia may represent a key clinical marker of PIGD type with higher motor burden at diagnosis.
Disclosure: Nothing to disclose.

EPO-0900 | **Parkinson's disease patients present with more pronounced impairment of vertical eye movements**

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Background and aims: Eye movement impairment is present in 75% of patients with Parkinson's disease (PD). Most pronounced impairment is in smooth pursuit which become hypometric with prolonged latency. We aimed to investigate differences in smooth pursuit eye movements in PD patients, depending on the movement axis.
Methods: This is a cross-sectional study with idiopathic PD patients. Velocity, phase, and number of fixations of horizontal

TABLE 1 Characteristics of PD patients.

	Mean/median	SD/IQR
Age (years)	65.06	+9.1
Age at PD onset (years)	59.48	+9.62
PD duration (years)	4	2-7
HDS-UPDRS III score	12.5	3-18.25
HDS-UPDRS IV score	0	0-0
LEDD (mg)	596	400-840

*PD, Parkinson's disease; SD, standard deviation; IQR, interquartile range; LEDD, Levodopa Equivalent Daily Dose.



FIGURE 1 Comparison of smooth pursuit eye movements with axis of movement.

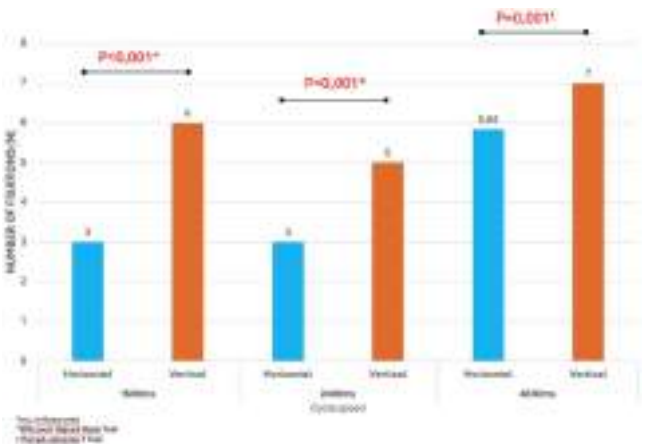


FIGURE 2 Comparison of number of fixations during smooth pursuit eye movements with axis of movement.

and vertical smooth pursuit eye movements were evaluated using eye-tracker analysis with a battery of tests. The results for pursuit gain, speed, and range of motion are expressed as mean square error (MSE), ranging from 0 to 1, with lower values indicating smaller deviation from expected values.

Results: Fifty subjects (36 men and 14 women) with early-stage idiopathic PD were evaluated. Most patients (38%) were in Hoehn and Yahr stage II. Patients' characteristics can be seen in Table 1 PD patients showed significantly more impaired gain, velocity, and range of motion (Figure 1) of smooth pursuit eye movements, as well as an increased number of eye fixations (Figure 2) in the vertical axis compared to the horizontal axis. Differences were more pronounced during slow movement tasks.

Conclusion: Vertical smooth pursuit is more prominently impaired, with significantly lower gain and prolonged latencies. This axis-specific deficit is present even in mild cases and is especially evident during slower cycles, when horizontal movement is still preserved. A possible cause is more complex interactions between the frontal and parietal eye fields and the basal ganglia, mainly the substantia nigra pars reticulata.

Disclosure: Nothing to disclose.

Sleep-Wake Disorders 2

EPO-0901 | Sleepwalking and risk of Parkinson's disease: A pooled analysis of 183,848 adults from the UK and USA

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Background and aims: Sleepwalking is a non-rapid eye movement (NREM) parasomnia that commonly occurs in Parkinson's disease (PD). Whether sleepwalking may precede the development of PD is unknown. This study investigated the association between sleepwalking and the risk of incident PD in the general population.

Methods: This prospective study used data from 183,848 community-dwelling adults without PD (ages 18–96, 58% female) pooled from the UK Biobank Study (UK) and Parkinson's Progression Markers Initiative Online Study (USA). Sleepwalking (ever vs never) was self-reported at baseline. Incident PD at follow-up was defined by doctor-diagnosis. Multivariable Firth logistic regression was used to estimate odds ratios (OR) for incident PD according to baseline sleepwalking status.

Results: Amongst 183,848 participants included at baseline, 10,316 (5.6%) had a history of sleepwalking. During a maximum follow-up of 3.6 years, 284 participants were diagnosed with PD. After adjusting for demographic characteristics, participants who sleepwalked had a 2-fold risk of developing PD during follow-up (adjusted OR: 1.99; 95% CI 1.27–3.12; $p=0.003$). The association between sleepwalking and incident PD was similar after adjusting for a wide range of possible confounders, including polygenic risk score for PD and idiopathic REM sleep behaviour disorder at baseline.

Conclusion: In this large prospective study spanning the UK and US, sleepwalking was independently associated with an increased risk of developing PD, suggesting it may represent a prodromal symptom of PD. These novel findings may help to identify individuals at risk of PD and could facilitate early prevention strategies.

Disclosure: Nothing to disclose.

EPO-0902 | Nightmares predict future cardiometabolic diseases: A pooled analysis of 113,073 participants across three continents

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Background and aims: Nightmares are cross-sectionally associated with cardiometabolic diseases (CMD), which are major contributors to neurodegeneration and premature mortality. Whether nightmares increase the risk of developing CMD is unknown. This prospective study investigated whether nightmares predict incident CMD in the general population.

Methods: Data from 113,073 CMD-free participants (ages 31–96; 64% female) from seven population-based cohort studies across three continents (UK Biobank; Midlife in Japan; Midlife in the United States; Wisconsin Sleep Cohort; The Osteoporotic Fractures in Men Study, Study of Osteoporotic Fractures, and Study of Women's Health Across the Nation) were used in this longitudinal analysis. Nightmare frequency was self-reported at baseline. Incident CMD at follow-up was defined as doctor-diagnosed myocardial infarction, stroke, hypertension, or diabetes. Multivariable logistic regression was used to examine the association between nightmare frequency and incident CMD. Participants' genetic risk for CMD was measured at baseline using polygenic risk scores.

Results: During a maximum follow-up of 17 years, 2,786 participants developed a CMD. After adjustment for potential confounders, a higher frequency of nightmares was linearly associated with a greater risk of incident CMD (p for trend < 0.001). Compared with participants who had no nightmares at baseline, those with weekly nightmares had a 1.3-fold risk of developing a CMD (adjusted odds ratio = 1.28; $p = 0.002$). Notably, there was no interaction between nightmare frequency and genetic risk ($p = 0.32$).

Conclusion: Nightmare frequency was independently associated with the development of CMD, regardless of genetic risk. Future studies are needed to determine whether treating nightmares could help reduce CMD risk in the general population.

Disclosure: Nothing to disclose.

EPO-0903 | REM sleep without atonia across dementia with Lewy body stages: Correspondence with clinical RBD and dopaminergic correlates

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Background and aims: Dementia with Lewy bodies (DLB) and its prodromal stage with mild cognitive impairment (MCI-LB), are frequently accompanied by REM sleep behavior disorder (RBD). REM sleep without atonia (RSWA), its neurophysiological correlate, is increasingly recognized as a biomarker of synucleinopathy; however, polysomnographic features across

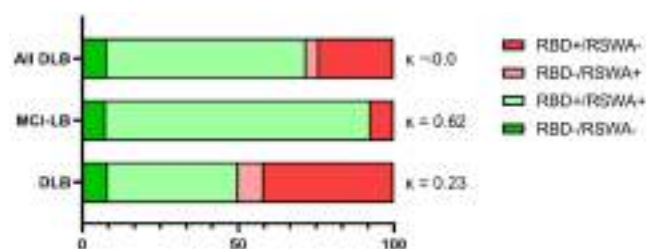


FIGURE 1 Correspondence between clinical RBD and pathological RSWA. Bar plots show the distribution of concordant (dark green and light green) and discordant (dark red and light red) classifications in patients with MCI-LB and DLB.

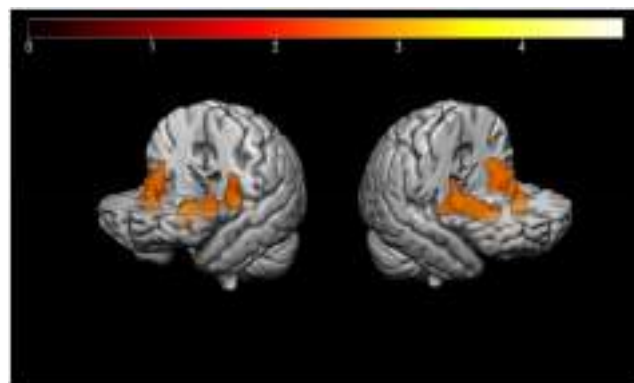


FIGURE 2 Brain rendering showing clusters obtained in SPM regression analysis for the negative correlation between DaT uptake and RSWA index.

disease stages and their relationship with dopaminergic dysfunction remains incompletely elucidated.

Methods: Thirty-three patients (17 MCI-LB, 16 DLB) underwent overnight polysomnography (PSG) and dopamine transporter (DaT)-SPECT. PSG parameters were compared using ANCOVA adjusted for age, sex, levodopa and acetylcholinesterase-inhibitor doses. Concordance between clinical RBD and pathological RSWA was explored in patients with artifact-free RSWA quantification. Associations between RSWA and regional DaT availability were assessed using voxel-wise regressions.

Results: Groups were comparable for age, sex, and motor severity, while cognitive fluctuations were more prevalent in DLB. DLB also showed longer nighttime total sleep time and greater daytime sleepiness (Epworth Sleepiness Scale). Conversely, RSWA-index values were higher in MCI-LB than in DLB. Concordance between clinical RBD and pathological RSWA was only partial (Cohen's $k = 0.23$, see Fig. 1), indicating incomplete overlap between clinical and physiological definitions. Across the whole cohort, higher RSWA was associated with reduced DaT uptake in bilateral insulae, extending to the claustrum and right caudate (see Fig. 2).

Conclusion: Sleep macrostructural alterations are equally present at the prodromal stage of DLB. Partial correspondence between clinical RBD and RSWA supports the added value of quantitative analysis, and the association between RSWA and extrastriatal dopaminergic dysfunction highlights RSWA as a translational biomarker linking sleep physiology with underlying neurodegenerative mechanisms.

Disclosure: Nothing to disclose.

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Background and aims: Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disorder characterized by motor and non-motor manifestations, including sleep disturbances and fronto-temporal spectrum cognitive impairment. The relationship between sleep disorders and cognitive alterations in ALS remains insufficiently explored: this study aimed to assess the

TABLE 1 Linear regression model predicting memory performance from T90 (%), hypoxic burden (%). The overall model was significant ($p < 0.001$), explaining 91.6% of the variance in memory scores (adjusted $R^2 = 0.885$).

Model Fit Measures				Overall Model Test			
Model	R	R ²	Adjusted R ²	F	df1	df2	p
1	0.957	0.916	0.885	29.3	3	8	<.001

Omnibus ANOVA Test					
	Sum of Squares	df	Mean Square	F	p
T90 (%)	89.5	1	89.53	17.3	.003
Hypoxic Burden (%)	58.6	1	58.65	11.4	.010
Pathological Memory*	151.5	1	151.50	29.8	<.001
Residuals	41.3	8	5.17		

Note. Type 3 sum of squares
* Fixed factor

Model Coefficients - Memory				
Predictor	Estimate	SE	t	p
Intercept ^a	18.105	0.8444	21.44	<.001
T90 (%)	-1.745	0.4189	-4.17	.003
Hypoxic Burden (%)	-0.290	0.0869	-3.37	.010
Pathological Memory ^a				
Yes - No	-8.522	1.5736	-5.42	<.001

^a Represents reference level
* Fixed factor

• Assumption Checks:

Durbin-Watson Test for Autocorrelation		
Autocorrelation	DW Statistic	p
-0.170	1.97	.922

Normality Test (Shapiro-Wilk)	
Statistic	p
0.971	.923

prevalence, characteristics, and cognitive impact of sleep disorders in a local ALS cohort.

Methods: Newly diagnosed ALS patients evaluated at our Neurology Unit in Parma (Italy) were prospectively enrolled. Participants underwent an overnight type III polysomnography and standardized questionnaires assessing disease severity, multidomain cognitive function, sleep quality, daytime sleepiness, and quality of life.

Results: Among 22 recruited ALS patients, 57.1% were affected by obstructive sleep apnea (OSA), with 23% deemed as moderate-to-severe forms. Numerous nocturnal respiratory parameters were strongly correlated ($p = 0.01$) with immediate and delayed memory impairment. Particularly, the hypoxic burden (HB) and the time spent with a peripheral blood oxygen saturation level below 90% were significantly predictive of memory compromise (Table 1). According to current guidelines, most patients with significant memory dysfunction were affected by mild OSA. Memory compromise is commonly described for patients with moderate-to-severe OSA. Our results suggest a higher prognostic relevance of the central hypoxic load than the apnoeic events count and support an increased sensitivity of ALS patients to the hypoxic damage.

Conclusion: Sleep breathing disorders may adversely affect not only survival but also cognitive performance in ALS, particularly memory. A comprehensive sleep evaluation emphasizing hypoxic overload rather than apnea frequency may improve clinical management and outcomes in this population.

Disclosure: Nothing to disclose.

EPO-0905 | iSPHYNCS: Over five years of the international Swiss Primary Hypersomnolence and Narcolepsy Cohort Study – Where we are and where we are heading

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Background and aims: With the aim to discover new biomarkers for narcolepsy type 1 (NT1) and the narcolepsy borderland (NBL), and in order to facilitate diagnosis and find more individualized treatment options, the prospective, longitudinal Swiss Primary Hypersomnolence and Narcolepsy Cohort Study (SPHYNCS) was started in 2020 and expanded internationally in 2023 (iSPHYNCS). New technologies, hypotheses- and data driven approaches have the potential to improve patient care and management.

Methods: The study has recently included new study sites in Norway and Austria and now counts 12 active study sites all over Europe. It plans to include 500 CDH patients and 60 healthy controls (HC) by the end of 2026. The multi-modal approach includes questionnaires, clinical assessments, video-polysomnography, the Multiple Sleep Latency Test (MSLT), vigilance tests, actigraphy, long-term activity monitoring with Fitbit, immunological studies, quantitative hypocretin measurements, proteomics, gut microbiomics, and genetics/epigenetics. AI-powered analyses, including unsupervised clustering, are used for data-driven patient phenotyping.

Results: 366 participants, including 13 children, have been recruited. The study population comprises 81 individuals with NT1, 233 individuals of the NBL, as well as 52 HC. So far, 6 publications have resulted from the study and have revealed notable differences among the study groups by analyzing questionnaires, neuropsychiatric profiles, Actigraphy and Fitbit, more are currently in review.

Conclusion: The study is heading towards its inclusion goal of 500 participants. In the next phase, the focus will be on the longitudinal data, “omics”- analyses and comprehensive machine learning analyses of (a) different raw data types and (b) multi-modal data integration.

Disclosure: The authors declare no conflict of interest. The study is supported by the Swiss National Science Foundation (SNF 320030_185362; SNF 32003B_215721).

EPO-0906 | Symptom severity in central disorders of hypersomnolence is associated with cerebrospinal fluid hypocretin-1 concentration

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Background and aims: Central disorders of hypersomnolence (CDH) are marked by excessive daytime sleepiness and/or increased sleep duration; narcolepsy type 1 is defined by hypocretin-1 deficiency. This study aimed to identify symptoms beyond cataplexy that signal hypocretin-1 deficiency and to examine how CDH symptom severity relates to hypocretin-1 levels.

Methods: CDH patients from the European Narcolepsy Network database were included: 257 had low hypocretin-1 (≤ 200 pg/mL; 163 ≤ 40 , 59 at 41–110, 35 at 111–200 pg/mL) and 45 had levels >200 pg/mL. Stochastic gradient boosting identified non-cataplexy predictors of hypocretin-1 deficiency, while logistic and quantile regression assessed associations between hypocretin-1 levels and 26 demographic, clinical, polysomnographic, MSLT variables, adjusted for age, sex, and BMI. Cataplexy features were analyzed only in hypocretin-1 ≤ 200 pg/mL cases.

Results: The machine-learning model predicted degrees of hypocretin-1 deficiency with moderate accuracy. Across all CDH patients, lower hypocretin-1 levels were associated with shorter REM and sleep latencies, more SOREMPs, higher Epworth scores, increased periodic limb movements, and more

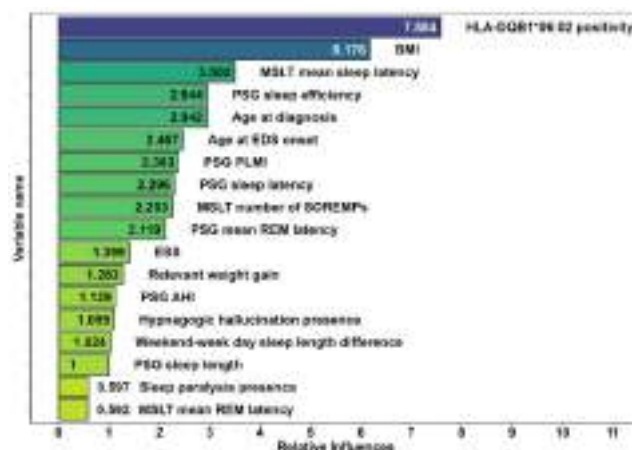


FIGURE 1 Predictors with relative influence greater than 0.5 in machine learning.

TABLE 1 Logistic and quantile regression (T=0.5) results for clinical variables using absolute hypocretin-I levels in CDH individuals.

Variable	Regression model	Coeff / exp(OR)	SE	Wald Z	P value
Gender	LRM	0.8768	0.0436	-4.5580	0.5811
BDI	QRM	0.0496	0.0247	-1.6238	0.1060
Behavior weight gain	LRM	0.5737	0.0233	-3.7670	0.0004
Age at diagnosis	QRM	0.0867	0.0266	-1.9421	0.0534
Age at onset	QRM	-0.0807	0.0389	-1.9981	0.0433
HLA-DQB1*03:02 positivity	LRM	0.5661	0.0638	-4.4880	0.0001
MSLT mean sleep latency	QRM	0.0296	0.0133	-14.031	0.0001
MSLT mean REM latency	QRM	0.0204	0.0143	-12.946	0.0001
MSLT number of SOREMPs	LRM	0.0131	0.0616	-1.9420	0.0534
Epworth Sleepiness Scale	QRM	-0.0609	0.0333	-1.8940	0.0604
Daytime sleep duration	LRM	0.0546	0.0478	-4.9780	0.0311
Sleep drunkenness	QRM	1.1711	0.0491	-1.8480	0.0661
PSG AHI	QRM	-0.0515	0.0195	-6.2716	0.0001
PSG PLMI	QRM	-0.0818	0.0366	-1.8884	0.0609
PSG mean REM latency	QRM	1.0417	1.1553	-1.8941	0.0604
PSG mean sleep latency	QRM	0.0266	0.0230	-1.6834	0.0961
PSG sleep efficiency	QRM	0.0510	0.0300	-1.6831	0.0961
PSG sleep length	QRM	0.0461	0.0310	-1.5087	0.1330
Disturbed nocturnal sleep	LRM	0.0808	0.0480	-1.7580	0.0806
Hypnagogic hallucinations presence	LRM	1.0811	0.0471	-6.1180	0.0001
Sleep paralysis presence	LRM	-0.0891	0.0461	-6.2080	0.0001
Sleep length mean	QRM	0.0537	0.0296	-1.9226	0.0534
Weekend-weekday sleep length difference	QRM	0.0338	0.0238	-1.8749	0.0638

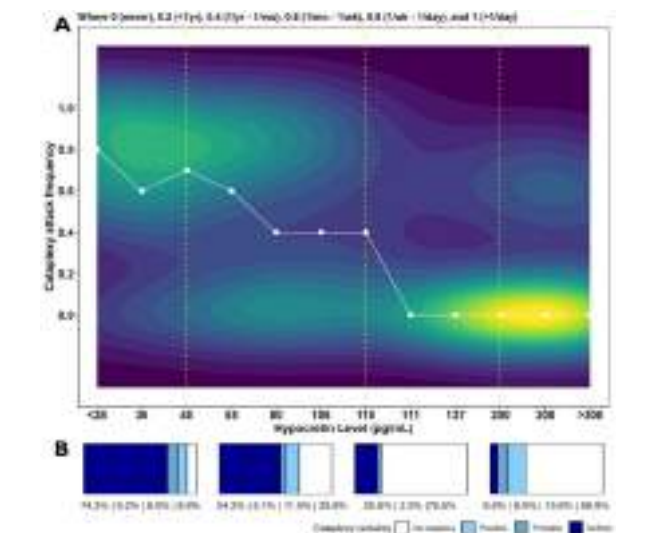


FIGURE 2 Cataplexy frequency and certainty across hypocretin-1 levels. (A) frequency coding: 0=never, 0.2=1/day; (B) hypocretin groups: ≤40pg/mL; 41–110pg/mL; 111–200pg/mL; >200pg/mL.

disturbed nocturnal sleep. In patients with cataplexy and hypocretin-1 ≤200pg/mL, lower levels were linked to earlier onset of excessive daytime sleepiness, younger age at diagnosis, greater certainty of hypnagogic hallucinations and sleep paralysis, and more frequent and complete cataplexy. In contrast, sleep drunkenness was more common at higher hypocretin-1 levels and associated with longer daytime sleep duration.

Conclusion: Lower hypocretin-1 levels were linked to greater symptom severity across CDH and within hypocretin-deficient cataplexy patients (≤200pg/mL). Even within this range, level differences explained clinical variability, highlighting a larger role for hypocretin-1 in CDH heterogeneity than previously recognized.

Disclosure: Nothing to disclose.

EPO-0907 | Neurometabolic differentiation of central disorders of hypersomnolence: A harmonized spectroscopy study

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Background and aims: Narcolepsy type 1 (NT1), narcolepsy type 2 (NT2), and idiopathic hypersomnia (IH) are central disorders of hypersomnolence (CDH); NT1 involves hypocretin deficiency, while NT2 and IH lack reliable biomarkers. We examined whether default mode network neurometabolites differ from controls, relate to symptoms and diagnostic differentiation. **Methods:** Sixty-two CDH patients (NT1 n=20, NT2 n=15, IH n=27) and 18 well-rested and sleep-restricted healthy controls underwent GABA-edited MEGA-PRESS 3T MRI of the medial prefrontal (mPFC) and posterior cingulate (PCC) cortices. Water-scaled, tissue-corrected GABA⁺, glutamate-glutamine, excitatory-inhibitory ratio, and secondary metabolites were analyzed using linear mixed models adjusted for age, sex, site, and IQ. Correlations assessed links with clinical measures, and ROC analyses evaluated diagnostic discrimination.

Results: No GABA⁺ or glutamate-glutamine differences survived FDR-correction between CDH and rested controls. Sleep-restricted controls showed higher PCC excitatory-inhibitory ratio and lower PCC myo-Inositol than CDH. Across CDH, higher mPFC total-N-acetylaspartate correlated with poorer sleep quality, and higher PCC total-Choline with longer MSLT sleep latency. Subtype-specific associations emerged: in NT2, lower mPFC glutamate-glutamine related to greater

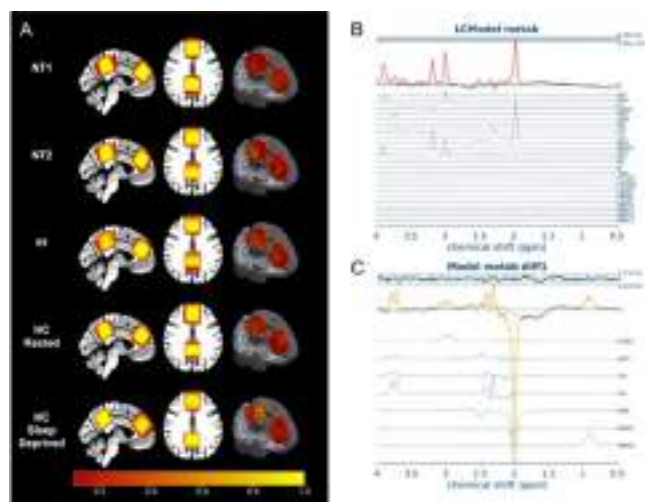


FIGURE 1 Voxel placement and representative spectra. A. Group-level voxel placement overlap maps for the medial prefrontal cortex and posterior cingulate cortex. Representative LCModel reconstruction for the standard MEGA-PRESS editing scheme.

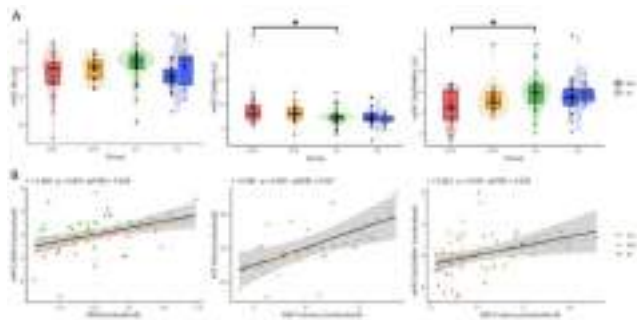


FIGURE 2 Group differences in mPFC metabolites (A) and preregistered clinical associations (B).

sleepiness; in IH, PCC glutamate-glutamine and excitatory-inhibitory ratio correlated with disease duration. ROC analyses showed good discrimination of NT1 vs NT2 (mPFC glutamate-glutamine, myo-Inositol) and NT2 vs IH (PCC total-Choline, total-N-acetylaspartate).

Conclusion: Despite no differences versus rested controls, subgroup analyses revealed distinct frontal (NT1/NT2) and posterior (IH) default mode network neurometabolic profiles. Frontal metabolites differentiated NT1-NT2 and tracked NT2 severity, while posterior markers distinguished IH-NT2 and related to disease duration, suggesting biologically grounded signals for improved CDH subtype differentiation beyond current criteria.

Disclosure: Nothing to disclose.

EPO-0908 | Phenoconversion in isolated REM sleep behavior disorder: Long-term data from the Innsbruck cohort

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Background and aims: Polysomnographically-proven isolated REM sleep behaviour disorder (iRBD) is a prodromal alpha-synucleinopathy, with over 90% phenoconverting eventually to Parkinson's disease (PD), Lewy body dementia (LBD) or multiple system atrophy (MSA). This study aimed at analyzing phenoconversion characteristics along with clinical-demographic features in the Innsbruck iRBD cohort.

Methods: We included 166 iRBD patients followed-up longitudinally; those developing an overt alpha-synucleinopathy were classified as “phenoconverters,” those remaining iRBD as “non-converters”. Survival analyses assessed phenoconversion characteristics by demographic and clinical factors.

Results: Thirty-nine (23.4%) subjects phenoconverted over a median of 62.1 (IQR 12.5–249.6) months follow-up, mostly to PD (64.1%), followed by LBD (25.7%) and MSA (10.2%).

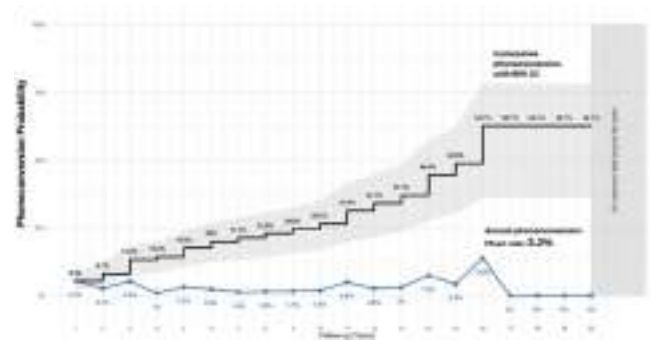


FIGURE 1 Kaplan-Meier-based cumulative and annual incremental phenoconversion plot.

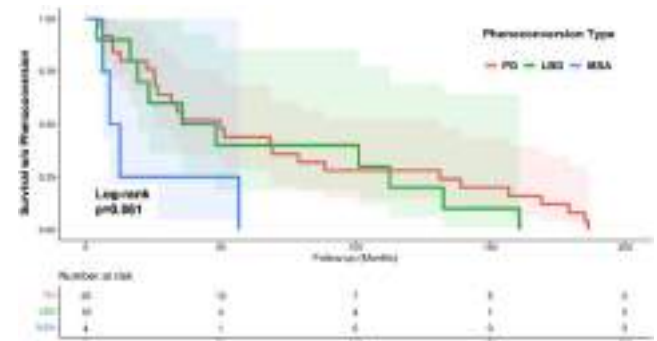


FIGURE 2 Survival curves of the phenoconverters grouped by phenoconversion type, abbreviated as Parkinson's disease: PD, Lewy body dementia: LBD, and multiple system atrophy: MSA.

Phenoconverters were older at diagnosis (68.3 vs 64.0y, $p=0.017$), had longer follow-up (80.6 vs 51.1 months, $p=0.008$), higher mortality (51% vs 13%, $p<0.001$) and more cardiovascular (44% vs 25%, $p=0.028$) and cerebrovascular (28% vs 12%, $p=0.014$) comorbidities. Cumulative phenoconversion risk (Kaplan-Meier analysis) was 13.4% at 3-years, 17.6% at 5-years, 26.5% at 10-years, and 62.7% at 20-years (annual phenoconversion rate: 3.2%, Figure1) without sex differences. We found a trend toward faster phenoconversion in MSA compared to PD and LBD (log-rank $p=0.061$, Figure2). LBD phenoconverters presented more depressive symptoms (LBD 70%, PD 20%, MSA 0%, $p=0.010$).

Conclusion: Our findings are consistent with prior reports, although with relatively more PD and MSA and fewer LBD cases compared to other cohorts. Beyond age and disease duration, our data suggest that cardio/cerebrovascular comorbidities may influence phenoconversion. Prospective studies are needed to determine whether targeted vascular risk management influences phenoconversion probability or timing.

Disclosure: Nothing to disclose.

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Background and aims: Olfactory dysfunction in isolated REM sleep behaviour disorder (iRBD) is a known risk factor of short-term phenoconversion to overt alpha-synucleinopathy. Yet data about longitudinal changes in olfactory function is scarce. This study aimed at investigating long-term olfactory function changes in iRBD.

Methods: This long-term observational study included iRBD with annual olfactory testing using the Sniffin' Stick 16-item identification task. Those developing overt-synucleinopathy were classified as "phenoconverters," those remaining iRBD as "non-converters". Longitudinal olfaction scores were used to model the predictive power of olfactory function on phenoconversion.

Results: 89 patients were included; 14 (15.7%) phenoconverted over 4.82 ± 3.87 years follow-up (age at baseline: 65.02 ± 10.19 y, M/F:80/9). The baseline olfactory score was 11.3 ± 3.1 in non-converters, 10.1 ± 3.5 in phenoconverters ($p=0.162$). The olfactory score at last follow-up was significantly higher in non-converters compared to phenoconverters (11.0 ± 3.6 vs

8.5 ± 2.8 , $p=0.019$). Longitudinal mixed-effects model (Figure1) showed lower olfactory scores in phenoconverters ($\beta=-1.664$, $p=0.047$). Higher olfactory score reductions were associated with longer follow-up time ($\beta=-0.107/\text{year}$, $p=0.017$) and older age at diagnosis ($\beta=-0.088/\text{year}$, $p=0.002$), with no sex effect ($p=0.802$). In Kaplan-Meier analysis (Figure 2), baseline olfactory scores ≤ 12 were associated with higher phenoconversion risk (HR=1.168 per 1-point decrease; $p<0.001$).

Conclusion: In our iRBD cohort, olfactory identification performance showed a gradual decline over time, underscoring its role as dynamic biomarker. Olfactory dysfunction was more severe at last follow-up in phenoconverters, confirming the previously-reported association with short-term phenoconversion risk. The strong association between lower baseline olfactory scores and increased phenoconversion risk supports the prognostic value of quantitative olfactory testing in prodromal alpha-synucleinopathies

Disclosure: Nothing to disclose.

EPO-0910 | Long-term assessment of social jetlag by Fitbit smartwatch in patients with central disorder of hypersomnolence

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Background and aims: Central disorders of hypersomnolence (CDH) are characterized by excessive daytime sleepiness and altered sleep-wake patterns. Social jetlag (SJL), reflecting misalignment between circadian rhythm and social schedules, has rarely been examined in CDH. Using smartwatch data, we characterized SJL in CDH and hypothesized greater SJL compared to healthy controls (HC).

Methods: Sleep-wake data from Fitbit smartwatches were analyzed in 37 HC, 50 patients with narcolepsy type-1 (NT1), and 116 narcolepsy borderline (NBL) patients enrolled in the Swiss Primary Hypersomnolence and Narcolepsy Cohort Study. Weekly SJL was defined as the difference between weekend and weekday mid-sleep times; monthly SJL was averaged across weeks. Linear-mixed-models assessed effects of diagnosis on

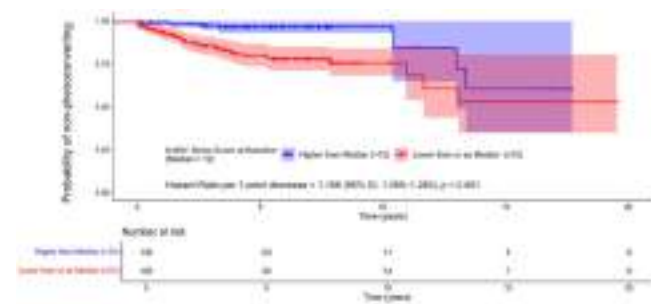


FIGURE 1 Kaplan-Meier analysis of phenoconversion-free survival in iRBD stratified by baseline Sniffin' Sticks scores. iRBD, isolated REM sleep behaviour disorder.

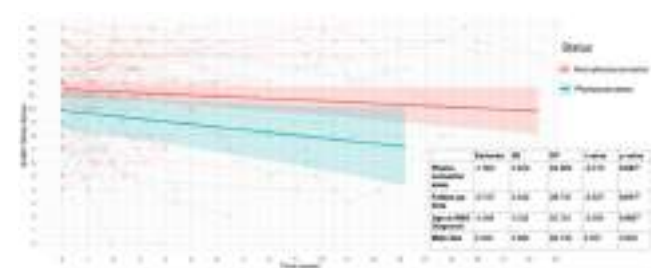


FIGURE 2 Linear mixed model aims to predict Sniffin' Sticks score over the follow-up period. DF, degrees of freedom; iRBD, isolated REM sleep behaviour disorder, SE, standard error.

monthly SJL, adjusting for age, BMI, gender, season, daylight saving time (DST), and nocturnal sleep duration (NSD).

Results: NBL patients showed significantly larger SJL than HC (19.0 min, $p=0.0015$), while NT1 showed a trend toward larger SJL (12.3 min, $p=0.074$). Differences between NBL and NT1 were not significant. Findings were replicated during summer-time DST, whereas in winter-time only NBL differed from HC. HC exhibited greater SJL in winter versus summer, a seasonal effect absent in CDH. SJL was unrelated to BMI or gender but negatively associated with NSD. CDH patients showed greater weekend variability in NSD than HC.

Conclusion: Wearables enable longitudinal assessment of SJL in CDH. Patients exhibit increased SJL, particularly in summer-time, with DST effects differing from HC. Persistent SJL in NBL may represent a disease marker, while reduced seasonal adaptability in CDH suggests limited compensation for circadian disruption or sleep debt.

Disclosure: Nothing to disclose.

EPO-0911 | Heterogeneous daily vigilance responses during supervised in-lab CBT-I in insomnia

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Background and aims: Cognitive behavioral therapy for insomnia (CBT-I) is the first-line treatment for chronic insomnia disorder (CID), with sleep restriction therapy (SRT) as a core component. Previous home-based CBT-I studies reported increased daytime sleepiness and impaired vigilance during SRT, findings that may be confounded by limited adherence and supervision. We therefore investigated changes in daytime sleepiness and vigilance during a tightly supervised, in-lab CBT-I program.

Methods: We retrospectively analyzed data from 56 CID patients who completed a standardized two-week in-house CBT-I program including SRT (5–6 h/night). Daily Stanford Sleepiness Scale (SSS), subjective sleep quality (SQ), and afternoon psychomotor vigilance tests (PVT) were collected over 10 working days. Linear mixed-effects models assessed changes in vigilance (inverse mean reaction time), SSS, and SQ, adjusting for age and sex. Subject-level bootstrap analyses were used to evaluate robustness of individual vigilance trajectories.

Results: SQ improved significantly over time ($p<0.001$), whereas vigilance and SSS showed no systematic change. Higher sleepiness was consistently associated with poorer vigilance ($p<0.001$). Individual responses were heterogeneous: 19 patients showed improving and 26 declining vigilance. Subgroups did not differ in demographics or fatigue severity, but higher depressive symptom severity (Beck Depression Inventory score ≥ 16) tended to be more frequent in patients with declining vigilance ($p=0.092$). SQ was positively associated with vigilance only in the improving subgroup ($p=0.032$).

Conclusion: Unlike prior home-based studies, supervised in-lab CBT-I did not produce consistent increases in daytime sleepiness or vigilance impairment during SRT. Improved sleep quality may mitigate adverse daytime effects despite restricted sleep opportunity.

Disclosure: Nothing to disclose.

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EPO-0912 | Redefining the accuracy of clinical diagnosis of Parkinson's disease: A systematic review and meta-analysis

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Background and aims: Accurate diagnosis of Parkinson's disease (PD) remains challenging, particularly in early stages, with major implications for patient care, biomarker research, and clinical trials. Although modern diagnostic criteria such as the Movement Disorder Society (MDS) 2015 criteria were designed to improve diagnostic precision, their real-world performance relative to earlier frameworks is incompletely defined.

Methods: We conducted a systematic review and meta-analysis of studies evaluating the accuracy of clinical PD diagnosis against neuropathological confirmation. Studies using UK Parkinson's Disease Society Brain Bank (UKPDSBB), Gelb, and MDS-PD 2015 criteria were included. Bayesian bivariate random-effects models were used to estimate pooled sensitivity and specificity for early PD (first clinical assessment) and late PD (final diagnosis before death), and to explore between-study heterogeneity and temporal trends.

Results: Twelve studies including 9,659 participants were analysed. In early PD, pooled sensitivity and specificity were 57% and 76%, respectively, with substantial heterogeneity across studies. Conversely, late PD showed markedly higher accuracy, with pooled sensitivity of 87% and specificity of 89%. Among late-stage criteria, UKPDSBB provided the highest sensitivity (91%), whereas the MDS-PD 2015 criteria achieved the highest specificity and overall diagnostic precision. Meta-regression demonstrated progressive improvement in specificity over recent decades, with a modest decline in sensitivity, suggesting increasing diagnostic stringency.

Conclusion: Clinical diagnosis of PD is strongly stage-dependent and suboptimal in early disease. Although modern criteria, particularly the MDS-PD 2015, improved specificity and overall precision, misclassification in early PD remains common, underscoring the need for objective biomarkers to enhance diagnostic reliability in clinical practice and trials.

Disclosure: Nothing to disclose.

EPO-0913 | Evolving drivers of advanced therapy selection in essential tremor: European neurologists show a measurable shift toward patient-centered priorities

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Background and aims: The introduction of new advanced therapies reshaped the landscape for patients with essential tremor (ET). This study aimed to assess whether these developments influence the factors considered by neurologists when selecting advanced therapies.

Methods: A cross-sectional survey was conducted among 270 general neurologists (GN) and movement disorder neurologists (MDN) across Europe. Prescribing drivers, perceptions of advanced therapies, and unmet educational needs were assessed. Outcomes were compared with the results of the same survey performed in 2023.

Results: Compared with 2023, improvement in quality of life became the leading driver of choice for advanced therapy selection (2023: $13.9 \pm 2.9\%$. 2025: $26.9 \pm 4\%$). Patients' willingness to undergo therapy ranked second in Italy (15%), Germany (13.3%) and UK (17.5%) (Figure 1). This factor had a greater influence on prescribing MRgFUS (22.9%) than DBS (4.1%) (Figure 2). Familiarity with MRgFUS increased across Europe (2023: $86.9 \pm 6.5\%$. 2025: $96.5 \pm 3.5\%$), becoming the most frequently cited advanced therapy for ET in Spain (23.6%) and Italy (16.7%). When asked about unmet educational needs, neurologists reported a marked increase, compared with 2023, in the need to



FIGURE 1 Neurologists' clinical priorities for advanced therapies in ET.



FIGURE 2 Patient profile besides severity for advanced therapies.



FIGURE 3 Recommendations for education.

improve patient information and education (2023: $14.2 \pm 5.1\%$. 2025: $25.2 \pm 9.1\%$) (Figure 3).

Conclusion: These findings demonstrate a shift in clinical decision-making within the Neurology community towards patient-centered priorities. Patient preferences and quality-of-life considerations are gaining importance alongside traditional efficacy and safety endpoints. Future efforts should enhance patient education, awareness and access to advanced therapies, ultimately enhancing their quality of life and treatment satisfaction.

Disclosure: All authors are Insightec employees.

EPO-0914 | Atypical late-onset PRKN-related Parkinson's disease due to compound heterozygosity: A case report

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Background and aims: Approximately 5–10% of Parkinson's disease (PD) cases have a monogenic cause. PRKN is the commonest autosomal recessive gene. The typical phenotype involves early-onset PD, limb dystonia and slow progression.

Methods: Case report

Results: A 58-year-old man (patient 1) presented with a 7-year history of resting tremor, initially affecting the right lower limb and later the left upper limb, with gait impairment. Family history revealed a brother (patient 2) with similar symptoms beginning around 50s and a deceased father with PD. Both neurological examinations showed tremor-predominant parkinsonism with more evident resting tremor in the right lower limb and bradykinesia and rigidity in the contralateral left upper limb. Cognitive functions were preserved, and no pyramidal, cerebellar, or autonomic signs were observed. They were investigated with brain MRI and laboratory panel, unremarkable. Both showed a good response to levodopa. Genetic testing started in patient 1, with LRRK2 single test that was negative. A NGS panel identified two heterozygous pathogenic variants in the PRKN gene, which confirmed to be present in the brother (patient 2), and in the descendant (son of patient 2) it was only found a single pathogenic PRKN variant, supporting a compound heterozygous configuration in trans in the affected cases.

Conclusion: We report two cases of genetic PD due to compound heterozygous PRKN variants, with an unusual phenotype:

involvement of opposite limbs, absence of dystonia despite lower-limb onset, and a later age-at-onset. The distinctive clinical presentation, positive family history, slow progression, good response to levodopa and confirmation of trans configuration allow a genotype–phenotype correlation.

Disclosure: Nothing to disclose.

EPO-0915 | Caregiver burden in Parkinson's disease and implications for a CBT/ACT-based caregiver-intervention (Care-PD)

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Background and aims: Parkinson's disease (PD) progressively increases patients' needs for medical care and everyday support, most of which is provided by relatives without medical education. These informal caregivers frequently experience substantial physical and psychological strain summarized as caregiver burden (CB). The CARE-PD study aims to quantify CB in caregivers of individuals with PD and identify patient- and caregiver-related predictors to develop a targeted psychological caregiver intervention.

Methods: PD patients and their primary informal caregivers were recruited at the Charité movement disorders day clinic between December 2024 and May 2025. CB was measured using the Zarit Burden Interview (ZBI). Patient assessments included motor severity (MDS-UPDRS-III) and depressive symptoms (BDI-II). Caregiver assessments included BDI-II, mindfulness (MAAS), psychological inflexibility/experiential avoidance (AAQ-II), and sociodemographic data. Multiple linear regression models were used to identify predictors of ZBI scores.

Results: 57 PD patients (age=58.79 years, 19 female) and 57 caregivers (age=63.71 years, 38 female) completed the study. Caregivers reported mild to moderate burden (ZBI=21.30±8.64). Higher UPDRS-III predicted higher CB ($p=0.006$), whereas BDI-II did not ($p=0.838$). Higher caregiver depressive symptoms ($p=0.002$), lower psychological flexibility (AAQ-II; $p=0.046$), and female caregiver gender ($p=0.033$) were significant predictors of CB; mindfulness was not ($p=0.549$).

Conclusion: CB in PD is driven by both patient motor impairment and caregiver psychological vulnerability, particularly depressive symptoms and psychological inflexibility, with female caregivers at increased risk. These findings can support early identification of at-risk caregivers and guide the development of targeted cognitive behavioral therapy (CBT)/Acceptance and Commitment Therapy (ACT)-based interventions to mitigate CB and enhance care delivery.

Disclosure: Nothing to disclose.

EPO-0916 | Baseline dopaminergic treatment profiles of patients with advanced Parkinson's disease prior to Levodopa-Entacapone-Carbidopa intestinal gel infusion

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Background and aims: Continuous intra-jejunal infusion of Levodopa-Entacapone-Carbidopa intestinal gel (LECIG) is an effective therapy for the treatment of advanced Parkinson's Disease (APD) associated with severe motor fluctuations with-or-without dyskinesias. The objective was to describe the characteristics of the last dopaminergic treatment in a large group of patients with APD in which current non-invasive therapy alone was no longer effective.

Methods: We conducted a retrospective review of all APD patients treated in our department before the initiation of LECIG treatment between December 2021 and November 2025. We analyzed the use of dopaminergic agents based on the most recent treatment recommendations.

Results: During the four years that were included in the study, we initiated LECIG treatment in 50 patients with APD; mean age 65.2 ± 8.65 (mean ± SD), disease duration 11.2 ± 4.6 years. The average treatment duration with Levodopa was 10.0 ± 4.01 years. Patients were on relatively high-dose levodopa regimens (mean: 857 ± 240 mg/day), administered at 5.34 ± 0.56 doses daily (5 to 7 times daily). Most patients also had treatment with dopamine agonists (82%; pramipexole $n=10$, 2.35 ± 0.87 mg; ropinirole $n=11$, 12.36 ± 5.5 mg; rotigotine patch $n=21$, 8.48 ± 1.89 mg) as well as selective monoamine oxidase B inhibitors (MAO-Bi, 60%, $n=30$), apomorphine pen (14%, $n=7$), Amantadine (28%, $n=14$) and Entacapone (70%, $n=35$).

Conclusion: The usage of dopaminergic treatments in patients with APD prior to LECIG treatment was more complex than those described in the literature. Such analyses help general neurologists identify the limits of conventional dopaminergic therapy and prompt timely referral to tertiary centers.

Disclosure: Nothing to disclose.

EPO-0917 | From scores to signals: A task-level atlas of movement disorder rating scales

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Background and aims: Clinical observation and rating scales are the currency of movement-disorder assessment, guiding diagnosis, tracking progression, and judging treatment response. Digital tools can quantify motor behaviour at scale in real-world settings, but to translate this into clinically meaningful endpoints we must first understand what existing scales actually “sample.” We therefore mapped the content of widely used movement-disorder

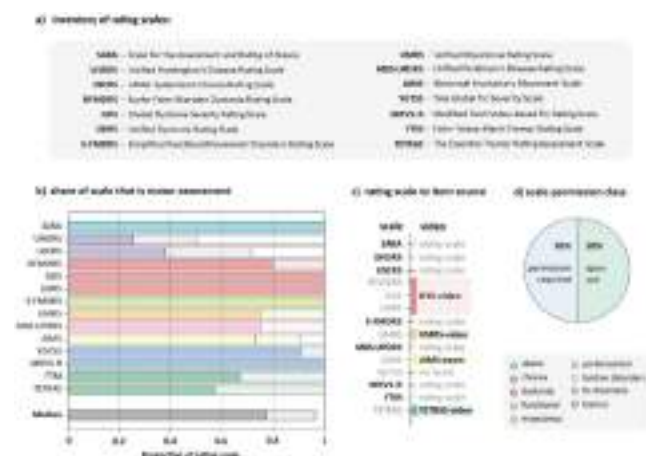


FIGURE 1 Inventory of movement disorder rating scales. (a) Candidate scales for motor phenotypes. (b) Proportion devoted to motor (dark), function (pale), or non-motor (white) assessment. (c) Source of motor-assessment items. (d) Licensing status of scales.

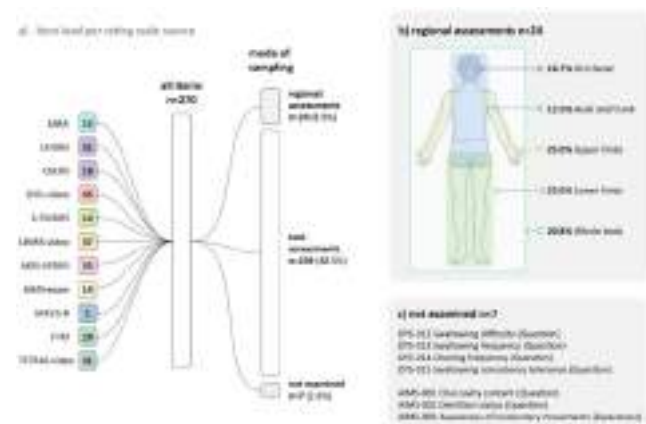


FIGURE 2 Overview of rating-scale items and regional assessments. (a) Classification of items by sampling mode. (b) Emphasis of body region(s) within region-based assessments. (c) Not examined items: patient-reported questions and qualitative judgements.

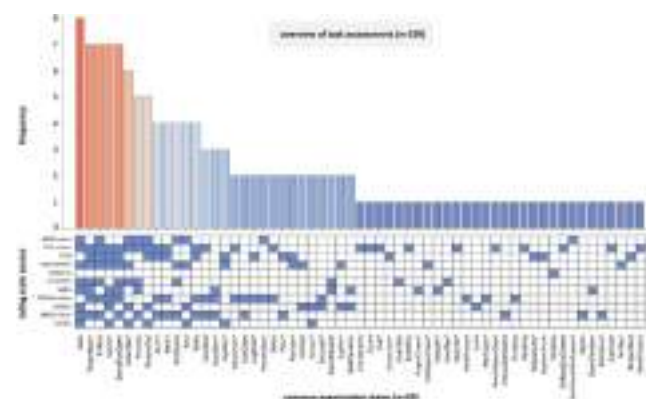


FIGURE 3 Overview of task-based assessments across movement disorder rating scales. Frequency plot displaying each task's occurrence across included rating scales, identifying core and scale-specific tasks. Grid plot indicates task inclusion by scale.

rating scales to identify common anchors and syndrome-defining phenomena.

Methods: Using a pragmatic, phenotype-centred strategy, we selected scales spanning ataxia, chorea, dystonia, functional movement disorders, myoclonus, parkinsonism, tardive syndrome, tic disorders, and tremor. We analysed content at two levels: (i) scale composition (motor vs functional/ADL vs non-motor) and (ii) item structure, classifying items by body region and task (Figure 1). Where available, structured video/examination protocols defined items; otherwise, items were extracted from text.

Results: Across scales we collated 270 discrete items (Figure 2). Direct motor examination dominated (median ~73%), and each phenotype carved out a distinct “examination space,” emphasising different regions and tasks. Item-frequency mapping revealed a shared backbone of cross-phenotype tasks (including gait, speech, and bilateral finger–nose testing), supporting a feasible harmonised core for digital capture (Figure 3). Other scale-specific items encode constructs clinicians use to distinguish syndromes, so a “core-only” approach could risk reducing sensitivity. Scales for disorders with prominent non-motor/behavioural/cognitive features allocated a smaller proportion to motor assessment.

Conclusion: Digital measurement will not replace rating scales; it should be built on them. By using scales to define clinically grounded targets we can develop harmonised, interpretable, and more sensitive next-generation outcomes.

Disclosure: Nothing to disclose.

EPO-0918 | Clinical and kinematic assessment of Parkinsonian features in young adults with 22q11.2 deletion syndrome

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Background and aims: 22q11.2 deletion syndrome (22q11.2DS) is associated with an increased risk of early-onset parkinsonism, yet its motor phenotype in young adults remains poorly defined. We aimed to characterize parkinsonian features in young adults with 22q11.2DS using clinical and kinematic assessments, and to compare them with Parkinson's disease (PD) patients and healthy controls (HC).

Methods: Seventeen consecutive young adults with 22q11.2DS were enrolled and compared with 14 HC (age- and sex-matched) and 35 PD patients. Motor assessment included UPDRS part III. Distinct bradykinesia features were objectively quantified using optoelectronic motion analysis of finger tapping. Cognition was assessed using the Montreal Cognitive Assessment (MoCA). Four patients were receiving antipsychotic treatment.

Results: All 22q11.2DS patients exhibited clinically detectable bradykinesia. Eight patients (47%) fulfilled criteria for parkinsonism, presenting with rigidity ($n = 5$), resting tremor ($n = 2$), or both ($n = 1$). UPDRS-III total scores were comparable between 22q11.2DS and PD. Kinematic analysis showed that 22q11.2DS

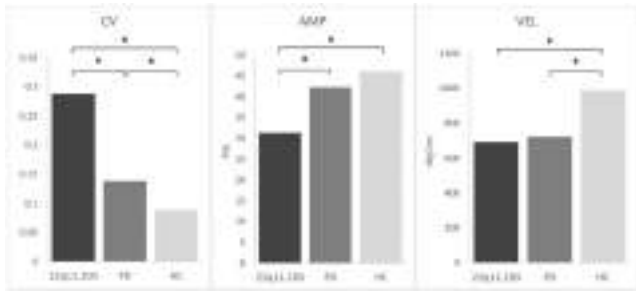


FIGURE 1 Kinematic Features of Bradykinesia in 22q11.2DS, PD, and HC. Bar plots show coefficient of variation (CV), movement amplitude (AMP), and velocity (VEL) during finger tapping. Asterisks indicate significant between-group differences.

patients performed finger tapping more slowly than HC but similarly to PD. Compared with HC and PD groups, movements in 22q11.2DS patients were smaller in amplitude and less regular. MoCA scores were significantly lower in 22q11.2DS compared with PD. No correlations were found between UPDRS-III or cognitive performance and any of the kinematic measures.

Conclusion: Young adults with 22q11.2DS show a parkinsonian motor phenotype partially overlapping with PD but with distinct additional features (i.e, hypokinesia and dysrhythmia). Movement abnormalities are independent of cognitive impairment. These findings highlight kinematic analysis as a valuable tool for assessing motor function in young adults with 22q11.2DS.

Disclosure: Nothing to disclose.

EPO-0919 | Clinical subtypes of essential tremor: Neurological soft signs versus age at onset

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Background and aims: Essential tremor (ET) subtypes may be defined by presence or absence of additional neurological signs (pure ET vs. ET-plus) or by age at tremor onset (early-onset ET, EOET vs. late-onset ET, LOET). Prospective studies with detailed clinical phenotyping comparing these concepts are scarce.

Methods: We prospectively enrolled 79 ET-patients (38.0% female) undergoing structured clinical assessment focused on predefined ET-plus signs, including resting tremor, impaired tandem gait, questionable dystonia, questionable bradykinesia, mild parkinsonian signs (hypomimia, reduced arm swing) and marked action tremor asymmetry. Patients were classified as pure ET or ET-plus and stratified by age at onset (40-year cut-off) into EOET and LOET. Tremor severity was assessed using TETRAS.

Results: Overall, 88.6% of patients were classified as ET-plus and 11.4% as pure ET. ET-plus-patients were older at baseline, but groups did not differ in disease duration, tremor severity or alcohol responsiveness (Table 1). 70% of ET-plus-patients had more than one soft sign (Figure 1). EOET and LOET accounted for 62.0% and 38.0% of cases, respectively. LOET were older and had shorter disease duration than EOET ($p < 0.001$). Alcohol responsiveness was reported by 80.0% of EOET vs. 33.3% of LOET

TABLE 1 Demographic and clinical parameters in essential tremor (ET)-plus vs. pure ET-patients. Statistical significance was assessed at the $\alpha=0.05$ level. Values with $p < 0.05$ are displayed in bold.

parameter	ET-plus	pure ET	p-value
Number of patients [n/N (%)]	70/79 (88.6)	9/79 (11.4)	
Median baseline age [years]	64.0	41.1	0.011
sex [% female]	35.7	55.6	0.240
Median age at onset [years]	28.7	19.3	0.099
Disease duration [years]	19.4	22.4	0.853
TETRAS [points]	17.5	11.5	0.085
Family history for ET [% positive]	85.7	66.7	0.247
Alcohol responsiveness [n/N (%)]	29/50 (58)	3/4 (75)	0.306

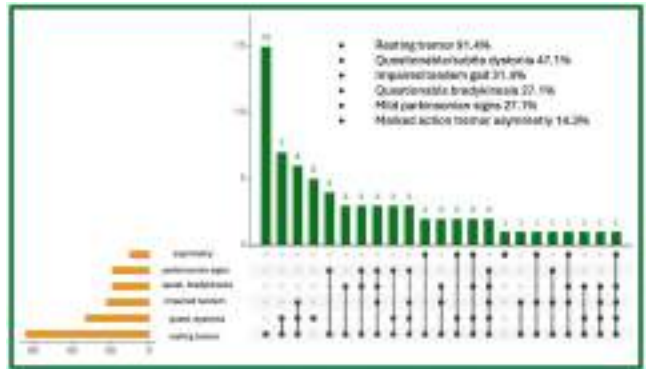


FIGURE 1 UpSet plot visualizing the frequency and distribution of additional neurological signs in patients with essential tremor plus.

TABLE 2 Demographic and clinical parameters in early-onset (EOET) vs. late-onset essential tremor (LOET)-patients. Statistical significance was assessed at the $\alpha=0.05$ level. Values with $p < 0.05$ are displayed in bold.

parameter	early-onset ET	late-onset ET	p-value
Number of patients [n/N (%)]	49/79 (62.0)	30/79 (38.0)	
Median baseline age [years]	45.9	74.0	<0.001
sex [% female]	35.7	46.6	0.773
Median age at onset [years]	18.0	57.1	<0.001
Disease duration [years]	25.6	25.3	<0.001
TETRAS [points]	16.0	23.8	<0.001
Family history for ET [% positive]	85.0	86.7	0.356
Alcohol responsiveness [n/N (%)]	24/30 (80)	8/24 (33.3)	<0.001
ET-plus [n/N (%)]	40/49 (81.6)	30/30 (100)	0.021

($p < 0.001$). Despite shorter disease duration, tremor severity was higher in LOET than EOET ($p < 0.001$; Table 2).

Conclusion: Additional neurological signs are common in ET, but ET-plus and pure ET do not differ in key aspects like alcohol responsiveness. In contrast, alcohol responsiveness is more frequent in EOET than LOET, while LOET appears characterized by faster tremor progression, suggesting different underlying pathophysiologies of these age-defined ET-subtypes.

Disclosure: Nothing to disclose.

EPO-0920 | Intrafamilial phenotypic heterogeneity in FBXO7-related PARK15 with compound heterozygosity: A case report of two siblings

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Background and aims: Biallelic pathogenic variants in the FBXO7 gene cause PARK15, a rare juvenile-onset parkinsonism characterized by motor and nonmotor features, with limited reports of intrafamilial phenotypic variability. This case report describes two Italian siblings with the same compound heterozygosity in the FBXO7 gene (p.Val233GlufsTer8 and p.Ile74Met), showing different clinical presentations despite identical genotypes. We also identified a potential novel pathogenetic FBXO7 variant.

Methods: Patient 1 (male, 21-years-old) had childhood-onset dysarthria and 2-year history of progressive slowness of movement. He was diagnosed with juvenile-onset parkinsonism and started dopaminergic medications with good levodopa response. Neurological examination showed mild left-predominant parkinsonism, and fragmentation of saccadic movements. Patient 2 (female, 27-years-old) presented at age 20 with verbal aggression and visual hallucinations, followed by a 2-year history of psychomotor slowing and gait impairment. Neurological examination showed mild-to-moderate left-predominant parkinsonism, fragmentation of saccadic movements and mild oculomotor apraxia. She also presented dysarthria and stimulus-induced myoclonus in the left hand. She shown poor levodopa tolerance due to behavioural side effects.

Results: Both patients exhibited MRI nigrosome-1 bilateral loss. Next-generation sequencing confirmed the presence of a compound FBXO7 heterozygosity with a pathogenic variant (p.Val233GlufsTer8) and a variant of unknown significance (p.Ile74Met). Mother and father carried the frameshift and missense variant respectively and were asymptomatic. A third sister was genetically wild-type and clinically healthy.

Conclusion: These cases highlight the possible intrafamilial heterogeneity in FBXO7-related PARK15 and support a potential pathogenic role for the p.Ile74Met variant (ACMG class 3), warranting functional studies for reclassification of variants in the FBXO7 gene.

Disclosure: Nothing to disclose.

EPO-0921 | Continuous subcutaneous foslevodopa/foscarbidopa improves nonmotor fluctuations in Parkinson's disease

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Background and aims: Non-motor fluctuations (NMFs) in Parkinson's disease (PD) are disabling features often related to pulsatile dopaminergic stimulation from oral levodopa.

Continuous subcutaneous foslevodopa/foscarbidopa infusion (CSFLI) provides stable 24-hour levodopa delivery, but real-world data on NMFs remain limited.

Methods: We evaluated the effects on motor and nonmotor fluctuations after 3 months of treatment with CSFLI. Sixty-one PD patients initiated CSFLI at our Center. Longitudinal data were available for 40 subjects. Motor fluctuations were assessed using the Unified Parkinson's Disease Rating Scale part IV (UPDRS-IV), non-motor fluctuations with the Nonmotor Fluctuation Assessment questionnaire (NoMoFa) and non-motor symptom burden with the Non-Motor Symptom Scale (NMSS). Sleep quality, behavioural features, autonomic dysfunction and quality of life (QoL) were also assessed using validated scales. Total levodopa equivalent daily dose (LEDD) was calculated at each timepoint. Within-subject longitudinal differences on clinical variables were assessed using repeated-measures ANOVA.

Results: After 3 months of treatment with CSFLI, significant changes were observed in the total NoMoFa score, mainly driven by a reduction in OFF-related NMFs, particularly within the sleep/fatigue, depression/anxiety and miscellaneous domains. Significant improvement in NMSS scores, sleep quality and QoL were also found. As expected, wake time spent in the OFF state decreased, with a concomitant increase in good ON time due to reduced troublesome dyskinesias. No significant changes were observed in total LEDD after 3 months of treatment.

Conclusion: Our data reveal that, along with motor fluctuation stabilization, treatment with CSFLI markedly reduces NMFs severity, with broad non-motor and motor benefits in PD.

Disclosure: Nothing to disclose.

EPO-0922 | Predicting atrophy in Parkinson's disease using the aggregation network diffusion model

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Background and aims: This study aimed to evaluate the Aggregation Network Diffusion (AND) model for predicting

individualized atrophy progression by simulating the spatiotemporal dynamics of misfolded α -synuclein spread and aggregation across functionally connected brain networks in PD.

Methods: Eighty-seven PD patients and 60 controls underwent 3D T1-weighted and resting-state functional MRI at baseline, with PD patients followed longitudinally over 7 years. A cohort of 34 PD patients from the Parkinson's Progression Markers Initiative with 4-year follow-up was included. The AND model simulated alpha-synuclein spread across the functional connectome through a system of ordinary differential equations modeling protein aggregation, and network diffusion based on parameters adaptable to individual patient dynamics. Patient-specific parameters were optimized using surrogate optimization to reproduce observed atrophy at first follow-up available. Model performance was assessed by Pearson's correlation between predicted and observed atrophy.

Results: The AND model successfully accurately predicted atrophy trajectories. Prefibrillar α -synuclein species combined with intermediate-weight protein aggregates most frequently yielded optimal atrophy predictions. Seed regions were predominantly localized to temporal and frontal lobes in PD patients. The model revealed two distinct atrophy progression patterns: one associated with low transmissibility, characterized by focal atrophy and gradual spread, and another linked to high transmissibility, marked by early, widespread bilateral involvement.

Conclusion: This study demonstrates the feasibility of using the AND model for personalized prediction of PD atrophy progression. By accounting for subject-specific patterns of protein spread and optimizing key model parameters, this approach provides new insights into the mechanisms driving neurodegeneration in PD.

Disclosure: Supported by EU-funding within NextGenerationEU - Mission 4, Component 2, Investment .3 - MUR-PNRR NeuroDETECT, MNESYS - A multiscale integrated approach to the study of the nervous system in health and disease, PE00000006, CUPB83C22004960002. R. Balestrino, S. Basaia, E. Sarasso, and E. Canu received research support from the Italian Ministry of Health. T. Cusolito, S. Pisano, F. Valtorta, E. Sibilla, V.S. Kostic, R. De Micco, A. Tessitore, F. Molinari, F. Esposito report no competing interests. M. Filippi is Editor-in-Chief of the Journal of Neurology, Associate Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received compensation for consulting services from Almirall, Biogen, Bristol-Myers Squibb, Eli Lilly, Merck, Novartis, Roche, Sanofi; speaking activities from Amgen, Bayer, Biogen, Bristol-Myers Squibb, Celgene, Chiesi Italia SpA, Eisai, Eli Lilly, Fujirebio, Genzyme, Janssen, Merck, Neopharm Gentili, Neuraxpharm, Novartis, Novo Nordisk, Roche, Sanofi, Takeda; participation in Advisory Boards for Alexion, Biogen, Bristol-Myers Squibb, Eli Lilly, GE Healthcare Ltd, Merck, Neuraxpharm, Novartis, Roche, Sandoz, Sanofi, Takeda; scientific direction of educational events for Biogen, Merck, Roche, Celgene, Bristol-Myers Squibb, Lilly, Novartis, Sanofi-Genzyme; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. F. Agosta is Associate Editor of NeuroImage: Clinical and the European Journal of Neurology; has received speaker honoraria from Biogen Idec, Bristol Myers Squibb, Eisai, Eli Lilly, GE Healthcare, Neuraxpharm, and Roche; and receives or has received research supports from the Italian Ministry of Health, the Italian

Ministry of University and Research, AriSLA (Fondazione Italiana di Ricerca per la SLA), the European Research Council (ERC), the EU Joint Programme – Neurodegenerative Disease Research (JPNDR), and Foundation Research on Alzheimer Disease (France).

EPO-0923 | CREXONT® (IPX203) significantly increases the duration of “Good on” intervals: Interim real-world results from the first 100 patients in ELEVATE-PD

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Background and aims: CREXONT is a novel extended-release carbidopa-levodopa formulation containing a mucoadhesive polymer, designed to improve levodopa delivery and absorption. We evaluated the impact of CREXONT on the duration of “Good On” intervals and daily motor fluctuations in patients with PD after switching from other levodopa-based therapies.

Methods: ELEVATE-PD is an ongoing real-world, open-label phase 4 study where PD patients are converted from prior levodopa-based therapies (IR CD/LD, IR+COMT inhibitors, Rytary) to CREXONT. Following a 5-week dose-optimization period and a 1-week stable dosing, patients are followed for 12 months. The average duration of continuous “Good On” intervals and daily number of motor fluctuations were assessed using data from patient PD diaries. This interim analysis includes 111 patients who completed Day 42/Visit 4.

Results: At baseline, mean duration of continuous “Good On” intervals across all treatment groups was 2.92 ± 1.38 hrs and average number of daily motor fluctuations was 5.86 ± 1.99 . CREXONT treatment increased mean duration of continuous “Good On” intervals to 6.65 hrs (+3.94 hrs increase) for patients switching from IR CD/LD ($n=74$), to 3.46 hrs (+0.99 hrs increase) from IR+COMT inhibitor ($n=9$), to 6.58 hrs (+3.09 hrs increase) from Rytary ($n=22$). Moreover, switching to CREXONT resulted in a reduction in the average number of daily motor fluctuations; it decreased to 2.96 (-3.25 [52.85%] reduction) in subjects converting from IR CD/LD, 5.15 (-1.11 [17.73%] reduction) from IR+COMT inhibitor and 2.79 (-1.92 [40.42%] reduction) from Rytary.

Conclusion: The longer duration of continuous “Good On” intervals and reduction of motor fluctuations after switching to CREXONT provide greater time and predictability for patients with PD “Off” fluctuations to perform their daily activities.

Disclosure: Supported by Amneal Pharmaceuticals.

EPO-0924 | The learning effect of smartphone-derived cognitive processing speed assessments as a proxy of cognitive functioning in multiple sclerosis

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Background and aims: The symbol digit modalities test (SDMT) score is crucial for assessing cognitive impairment in multiple sclerosis (MS), but is often influenced by a learning effect, regarded as unwanted variance. The goal of this study was to evaluate SDMT learning as a proxy of cognitive performance. **Methods:** Learning of people with MS and healthy controls (HCs) was evaluated using a two-part piecewise linear regression on daily smartphone SDMT (sSDMT) scores (see Figure 1). Cognitive impairment (CI) and preservation (CP) were defined using z-scores of baseline sSDMT relative to HC (CI: $z < -1.67$, CP: $z \geq -1.67$). sSDMT learning of HC/CP/CI was compared using ANCOVA and was correlated to baseline variables. Linear regression was performed to relate sSDMT learning to clinical outcomes and brain volumes.

Results: CP/CI: 66/19 MS and 20 HCs were analysed. A learning effect plateau was identified in HC/CI/CP: 80%/82%/100%, Figure 1 A higher baseline sSDMT was related to a higher plateau sSDMT ($\rho = 0.930$, $p < 0.001$) and a lower %-learned ($\rho = -0.266$, $p = 0.013$). The %-learned was higher for CI than CP (CP/CI = 15.52%/22.04%, adj. $p = 0.004$), but the Δ -learned and breakpoint were similar for HC/CI/CP, Figure 2 Relations of disability, cognition, and brain volumes with the plateau sSDMT were stronger when compared to the baseline sSDMT, Table 1.

Conclusion: Early-phase sSDMT learning was related to cognitive performance in MS. However, learning was not unique to disease status, and was not associated with disease severity. Moreover, plateau sSDMT showed stronger associations with disability and brain volumes than baseline sSDMT, underscoring the need to account for learning effects.

Disclosure: The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or

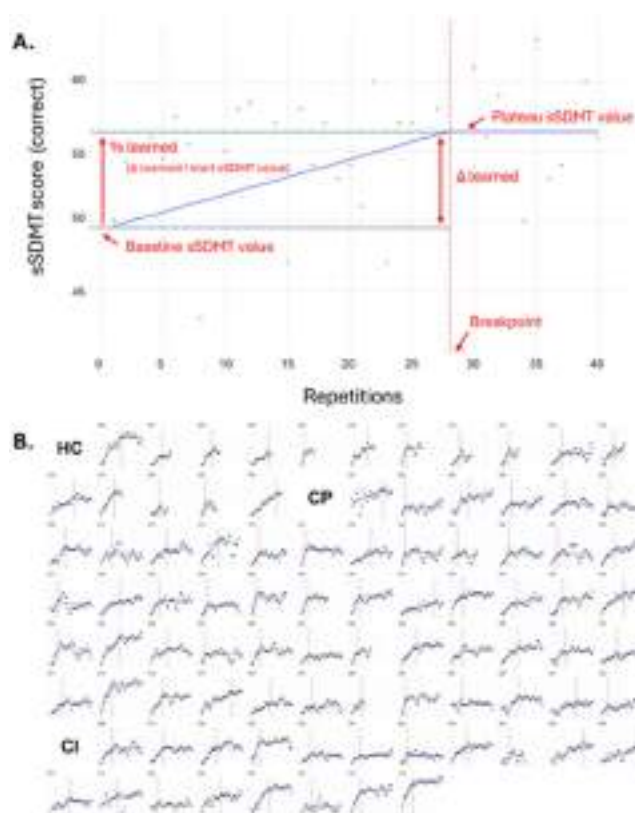


FIGURE 1: Depiction of variables extracted (A) from the sSDMT break-point analyses on the individual learning curves (B).

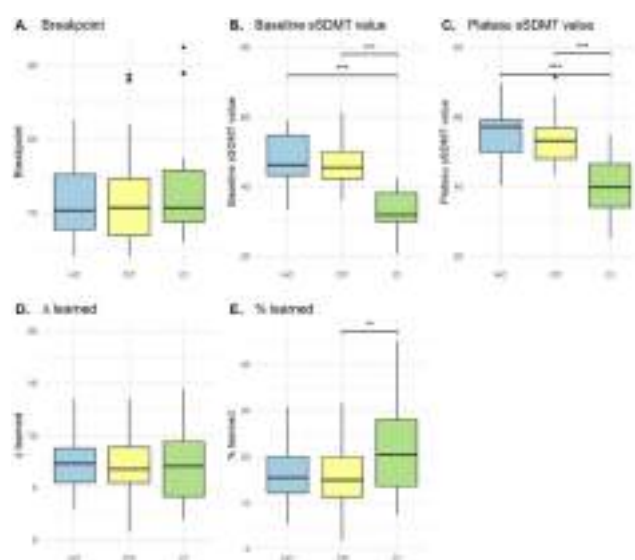


FIGURE 2 Extracted variables from the individual learning curves.

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[illegible]

EPO-0925 | Prognostic biomarkers of 10-year disease worsening in people with long-lasting multiple sclerosis

European Journal of Neurology, 2026

TABLE 1 Participant characteristics.

Test cohort, N=32	fit	missing	PU1	missing	PU2
Demographics					
Female sex (%)	113 (69.8%)	0	113 (69.8%)	0	113 (69.8%)
Age at visit (years, SD)	46.3 ± 10.4	0	46.3 ± 10.1	0	50.7 ± 9.9
Disease duration (days)					
Diagnose (years, SD)	11.9 (7.0–23.3)	0	17.0 (12.6–24.0)	0	23.1 (28.1–76.3)
IGRP (days)	3.0 (2.0–4.0)	2	4.0 (2.5–4.8)	1	3.6 (2.6–5.5)
IGRP (weeks, SD)	18.0 (17.4–17.1)	0	18.9 (18.0–17.7)	0	21.7 (18.8–24.8)
T2T (days, SD)	4.2 (3.7–5.2)	3	4.6 (3.0–5.7)	23	4.8 (4.2–5.2)
Treatment received (n, %)	111 (68.8%)	4	94 (58.8%)	0	108 (68.7%)
MR measures					
rTMS (S/T)	0.73 ± 0.041	0	0.71 ± 0.046	12	0.70 ± 0.045
sluTMS (S/T)	0.002 ± 0.013	0	0.001 ± 0.015	22	0.007 ± 0.014
sluTMS (S/T)	0.001 ± 0.0093	17	0.001 ± 0.0093	21	0.011 ± 0.0094
sluTMS (S/T)	0.009 ± 0.0013	0	0.008 ± 0.0013	12	0.008 ± 0.0013
sluTMS (S/T)	0.006 ± 0.0012	0	0.0060 ± 0.0012	12	0.0090 ± 0.0011
sluTMS (S/T)	0.00 ± 0.0020	0	0.02 ± 0.0020	12	0.01 ± 0.0019
Cortical thickness (mm, SD)	2.4 ± 0.088	0	2.5 ± 0.088	12	2.4 ± 0.085
sluTMS (S/T)	0.00 ± 0.007	0	0.09 ± 0.007	12	0.09 ± 0.007
sluTMS (S/T)	0.018 ± 0.0013	0	0.017 ± 0.0012	12	0.017 ± 0.0012
sluTMS (S/T)	0.007 ± 0.0001	0	0.0074 ± 0.0001	12	0.0070 ± 0.0001
sluTMS (S/T)	0.40 ± 0.004	10	0.40 ± 0.004	10	0.39 ± 0.004
sluTMS (S/T)	3.0 (2.0–7.0)	0	5.2 (3.0–9.0)	11	3.0 (1.8–12)
sluTMS (S/T)	0.0 (0.0–12.0)	13			
Seed-based biomarkers					
MR (g/100, SD)	11.0 (9.2–16.8)	11	12.2 (9.4–16.4)	17	14.1 (10.5–18.0)
GMF (g/100, SD)	84.2 (65.1–105.0)	51	84.6 (65.9–112.4)	35	87.5 (79.5–136.1)

Tails will follow characteristics of the included *p*-MSE [H(0)] at the significance level, α . See legend, FURTHER below.

1) and P(2) = follow-up p-Value; 2) Reported MSE measures are proportionally α -level or higher non-inferiority, Alternatives, Δ -IPTT = Non-Hole Plot Test; Brain-PAD = Brain Predicted Area Difference; EDG6 = Expanded Disability Status Scale; TSSW/F = Time-to-Fast Walk; Cortex Thickness = Mean Cortical Thickness; nCoVMM = Normalized Cerebral Gray Matter Volume; nCovGMV = Normalized Cerebral Gray Matter Volume; nCovWMV = Normalized Cerebral White Matter Volume; nCPMV = Normalized Cerebral Perivascular Space Volume; nCCMV = Normalized Deep Gray Matter Volume; nLVFV = Normalized Lateral Ventricular Volume; nLV = Normalized Lateral Volumes; nMUCCA = Normalized Mean Upper Cervical Cord Area; nTBV = Normalized Total Brain Volume; nThalVol = Normalized Thalamus Volume; nWMV = Normalized White Matter Volume; nSPAP = Oral Facility Audio Index; NSL = Neurocognitive Light Chain; * = Univariate; t = Multivariate; nMUCCA = Normalized Height in cm.



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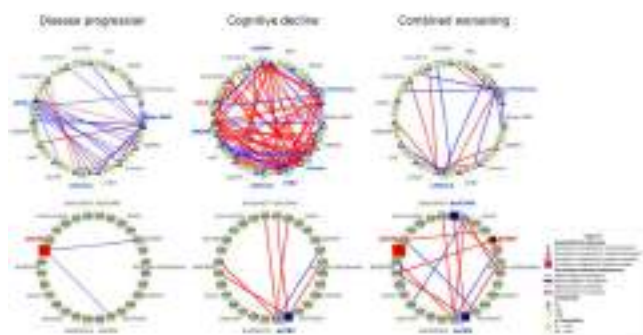


FIGURE 2 Network plot of relations between biomarkers related to worsening.

had lower cortical thicknesses at all timepoints and atrophy of nTBV and cortex, with an increase of nChPV and GFAP over follow-up. For inter-biomarker associations, see Figure 2. Baseline brain age (pooled OR=0.95, $p=0.020$) and high lesion volume (pooled OR=1.08, $p=0.049$) predicted disability progression (AUC=0.68), high cortical thickness decreased predicted the odds of cognitive decline (pooled OR=0.49, $p<0.001$, AUC=0.69) and combined worsening (pooled OR=0.57, $p=0.022$, AUC=0.65).

Conclusion: Our results indicate that 10-year disability progression and cognitive decline correlate with different biomarker patterns. Disability progression was associated inflammation (swelling of the choroid plexus), and the combination of low brain age with high lesional brain-burden, whilst cognitive decline was associated to cortical grey matter atrophy.

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Neuroimmunology & Neuroinflammation, and is editor of a *Neuromethods* book Springer. J. Killestein received consulting fees for F. Hoffmann-La Roche, Biogen, Teva, Merck, Novartis and Sanofi/Genzyme (payments to institution); reports speaker relationships with F. Hoffmann-La Roche, Biogen, Teva, Merck, Novartis and Sanofi/Genzyme (payments to institution); adjudication committee of MS clinical trials of Immunic (payments to institution). E. M. M. Srijbis reports speaker relations with Merck and Novartis (all payments to institution) and served on advisory board for Roche. M. Schoonheim serves on the editorial board of *Neurology* and *Frontiers in Neurology*, receives research support from the Dutch MS Research Foundation, Eurostars-EUREKA, ARSEP, Amsterdam Neuroscience, MAGNIMS and ZonMW (Vidi grant, project number 09150172010056) and has served as a consultant for or received research support from Atara Biotherapeutics, Biogen, Celgene/Bristol Meyers Squibb, EIP, Sanofi, MedDay and Merck.

EPO-0926 | Impact of OCT acquisition parameters on GCIPL thickness measurements in multiple sclerosis: A protocol comparison

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Background and aims: Retinal layer thickness measured by optical coherence tomography (OCT), particularly the ganglion cell and inner plexiform layer (GCIPL), is an established marker of neuroaxonal integrity in multiple sclerosis (MS). However, small longitudinal changes at the individual level often approach inter-scan variability, limiting clinical utility.

Methods: In this prospective study, participants with MS (pwMS) diagnosed according to the 2017 McDonald criteria and age- and sex-matched healthy controls (HC) underwent spectral-domain OCT at baseline and after two and four weeks. At each visit, five macular scan protocols were acquired: standard, high-ART, high-lines, high-resolution, and maximum. Reliability was assessed using intraclass correlation coefficients (ICC; two-way mixed-effects model, absolute agreement). Absolute test-retest reproducibility was quantified using mean absolute difference (MAD).

Results: Thirty-eight eyes from 9 pwMS (mean age 34.1 years [SD: 8.0], 44.4% female) and 10 HC (31.7 years [SD: 11.1], 50.0% female) were included. Mean baseline GCIPL thickness ranged from 70.8 to 71.5 μ m across protocols, demonstrating excellent inter-protocol agreement (ICC 0.99; 95% CI: 0.98–0.99; $p<0.001$). All protocols showed excellent test-retest reliability. High-ART and high-lines protocols demonstrated marginally lower absolute measurement variability (MAD 0.26–0.27; 95% CI: 0.21–0.32), while temporal agreement remained comparable across protocols. Mean acquisition time was shortest for the standard protocol (10.6s) and increased progressively for high-resolution, high-lines, high-ART, and maximum protocols.

Conclusion: All OCT acquisition protocols demonstrated excellent inter-protocol and test-retest reliability for GCIPL measurements. The high-lines protocol provides the most favorable

balance between measurement reliability and acquisition time, supporting its use for longitudinal GCIPL monitoring.

Disclosure: The authors declare no conflicts of interest related to this study.

EPO-0927 | Exploring genetically determined leukocyte telomere length in relation to age at onset in multiple sclerosis

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Background and aims: Telomere length is a marker of biological ageing and immune-cell senescence. In multiple sclerosis (MS), telomere shortening has been linked to disease severity, but its relationship with age at onset (AAO), a major source of phenotypic heterogeneity, remains incompletely understood. Genetically determined leukocyte telomere length (gLTL) provides an opportunity to investigate telomere biology independently of disease-related and environmental influences.

Methods: We analysed 2,824 relapsing–remitting MS patients with genome-wide genotype data imputed to the Haplotype Reference Consortium panel. Polygenic risk scores for gLTL (gLTL-PRS) were derived using summary statistics from a large genome-wide association study of UK Biobank participants. Eight PRS were constructed using a clumping-and-thresholding approach across increasing p-value thresholds. Associations between gLTL-PRS and AAO were assessed using linear regression models adjusted for relevant covariates.

Results: Across all PRS models, higher genetic predisposition to longer telomere length was associated with later AAO in MS. The strongest association was observed using the most inclusive PRS (p -value threshold <0.2 ; $\beta=0.57$, $SE=0.21$, $p=0.008$), consistent with a polygenic contribution of telomere-related variants to AAO variability.

Conclusion: These findings suggest that genetic variation underlying telomere biology may contribute to inter-individual differences in AAO in MS. The progressive strengthening of associations across increasingly inclusive PRS models supports the complex polygenic nature of this relationship and highlights telomere-related pathways as potentially relevant to MS phenotypic heterogeneity.

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EPO-0928 | Mitochondrial impact of fingolimod on peripheral immune cells in multiple sclerosis

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Background and aims: Fingolimod (FLM) limits lymphocyte egress from lymphoid organs. Mitochondrial dysfunction and metabolic reprogramming contribute to multiple sclerosis (MS) pathogenesis and disease progression. However, the impact of FLM treatment on mitochondrial activity and metabolism of peripheral immune cells (PBMCs) remains incompletely characterized. The aim of this study is to evaluate FLM effects on

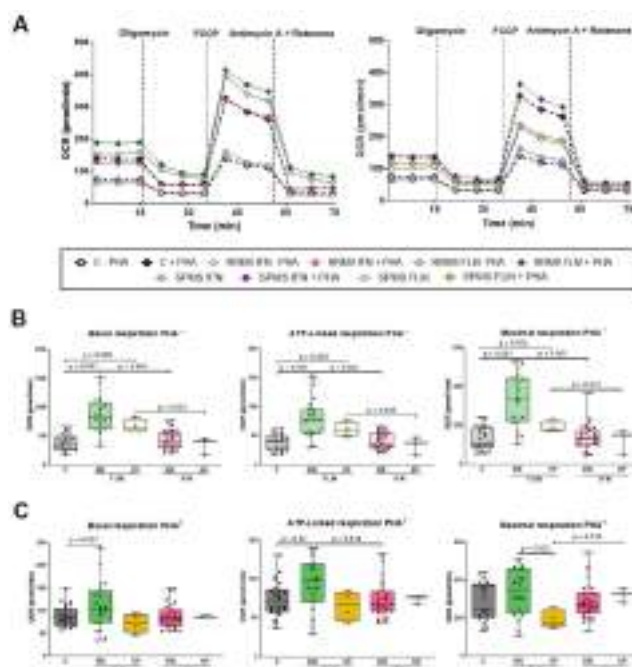


FIGURE 1 Effects of fingolimod on mitochondrial respiration of PBMCs stratified by MS clinical form. Oxygen consumption rate profiles and parameters of PBMCs from healthy controls and MS patients treated with IFN or FLM.

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Background and aims: Progressive multifocal leukoencephalopathy is a serious complication of natalizumab-treated multiple sclerosis, with anti-John Cunningham virus antibodies used for risk stratification. This study explores the role of the interferon- β -B cell activating factor axis in anti-John Cunningham virus antibody development.

Methods: This prospective study was conducted at Al-Azhar University Hospitals and Cairo Fatemic Hospital and included 186 patients with relapsing–remitting MS receiving natalizumab between October 2022 and November 2025. Serum anti-JCV antibody levels were assessed at baseline and after 12 and 24 months of treatment. Expression of type I and II IFN-inducible genes and BAFF was measured in peripheral blood using quantitative RT-PCR. BAFF gene polymorphisms (rs9514828, rs1041569, rs9514827) were analyzed using RFLP-PCR.

Results: No significant association was observed between type I or II IFN-inducible gene expression and serum anti-JCV antibody titers. In contrast, anti-JCV levels showed a significant correlation with BAFF gene expression. The BAFF TTT haplotype was significantly more prevalent among male anti-JCV–positive patients compared with anti-JCV–negative males at baseline, 12 months, and 24 months of natalizumab therapy. The BAFF TTT haplotype demonstrated 88% specificity, 70% sensitivity,

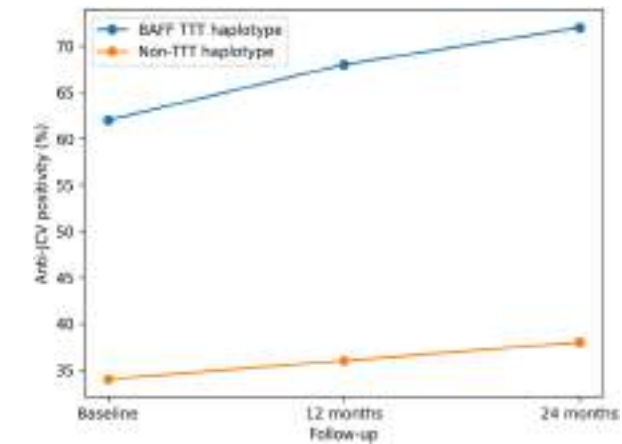


FIGURE 1 Anti-JCV seropositivity over time in male MS patients.

TABLE 1 Baseline characteristics.

Variable	Value
Total patients	186
Age (years), mean $\pm$ SD	34.6 $\pm$ 8.9
Male sex	82 (44.1%)
Female sex	104 (55.9%)
Anti-JCV positive	98 (52.7%)
Anti-JCV negative	88 (47.3%)
Natalizumab duration (months), median (IQR)	24 (12–35)

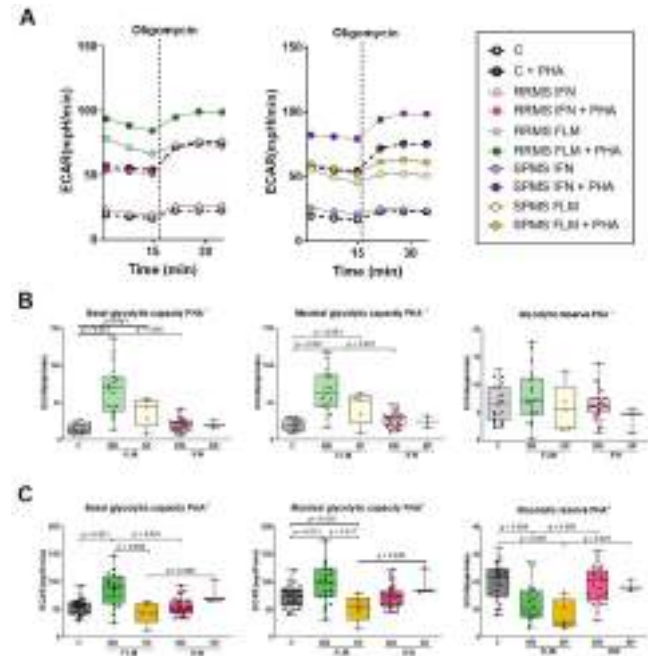


FIGURE 2 Effect of fingolimod on glycolysis of PBMCs stratified by MS clinical forms. Extracellular acidification rate profiles and parameters of PBMCs from healthy controls and MS patients treated with IFN or FLM.

mitochondrial function of PBMCs from MS patients, compared with interferon (INF)-treated patients and controls (HC).

Methods: Mitochondrial respiration and glycolytic activity were assessed by Seahorse XFP extracellular flux analyzer in MS patients treated with FLM ($n=24$) or IFN ($n=45$), and HC ($n=35$). Mitochondrial mass, membrane potential, reactive oxygen species (ROS) production, and glycolytic mediators were evaluated by flow cytometry. Mitochondrial electron transport chain (ETC) complexes were analyzed by Western Blot.

Results: In RRMS patients with FLM therapy, a pre-stimulated respiratory profile with significantly higher basal, maximal, and ATP-linked respiratory values were detected compared to IFN-treated patients and HC. Milder pre-stimulation, but also precluding further PHA-stimulation, was found in SPMS patients. In parallel, significant increases in unstimulated basal and maximal glycolytic capacities upon FLM-treatment led to a lower glycolytic reserve upon PHA-stimulation. FLM treatment produced lower fold-changes after PHA and significantly decreased mitochondrial depolarization with lower increase of complex II, leaving ROS and coupling at basal levels. After PHA-stimulation, decreased fold-changes of mitochondrial mass and lactate transporters (MCT1/4) were observed in FLM-treated compared to IFN-treated patients and HC, with no changes in PFKFB3.

Conclusion: Fingolimod modulates mitochondrial activity and PBMCs metabolism, which probably contribute to its therapeutic response in MS patients.

Disclosure: C.O.G. has received speaking and/or consultancy fees from Alexion, Biogen Idec, Bristol-Myers-Squibb, Janssen, Merck, Novartis, Roche, Sanofi-Genzyme, and Teva. Y.A. has received research grants, travel support, and lecturing and consulting fees from Bayer, Biogen, Roche, Merck, Novartis, Almirall, Sanofi-Genzyme, Janssen, and Bristol Myers Squibb. B.P. has received speaking, travel and/or training fees from Novartis, Almirall, Merck and Sanofi-Genzyme. The other authors nothing to disclose.

TABLE 2 BAFF TTT Haplotype performance.

Parameter	Value
BAFF TTT haplotype prevalence	Higher in anti-JCV (+) males
Specificity	88%
Sensitivity	70%
Positive predictive value	45%

and a 45% positive predictive value for identifying anti-JCV-positive male patients after 24 months of treatment.

Conclusion: These findings support a role for the BAFF pathway in regulating anti-JCV antibody production. The BAFF TTT haplotype may represent a novel genetic risk marker for anti-JCV seropositivity and, indirectly, for PML development in male MS patients treated with natalizumab.

Disclosure: Nothing to disclose.

EPO-0930 | Abstract withdrawn

EPO-0931 | Confirmed disability accrual in multiple sclerosis patients with expanded disability status scale score 5.5

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Background and aims: There is no consensus on the definition of disability accrual (DA) in patients with multiple sclerosis (MS) with a baseline Expanded Disability Status Scale (EDSS) score of 5.5. Indeed, progression has been variably defined as either a 1.0- or 0.5-point increase. This study aims to compare the proportion of DA and its confirmation at the end of the follow-up using the two definitions.

Methods: In this multicentric, retrospective, cohort study based on prospectively acquired data from the Italian Multiple Sclerosis Register, inclusion criteria were: diagnosis of Clinically Isolated Syndrome or relapsing-remitting MS, first visit on or after January 1, 2000, ≥ 3 EDSS evaluations, ≥ 5 years of follow-up. In patients with roving baseline EDSS of 5.5, confirmed DA (CDA) was defined as a 24-week confirmed disability increase of either 0.5- or 1-point. Proportions of CDA and sustained DA (absence of subsequent EDSS improvement until follow-up end) were calculated and compared.

Results: The whole sample included 30203 relapsing MS patients followed-up for 11.3 $\pm$ 4.3 years. A total of 228 (0.8%) patients had roving baseline EDSS = 5.5. Among those, CDA with 0.5- or 1-point EDSS increase occurred in 200 (87.7%) and 59 (25.9%) patients, respectively ($p < 0.001$). 104 out of 141 (73.8%) CDA with 0.5-point EDSS increase and 51 out of 59 (86.4%) CDA with 1-point EDSS increase were sustained at the end of the follow-up ($p = 0.05$).

Conclusion: In patients with MS and baseline EDSS score of 5.5, the 0.5-point CDA definition identifies a greater proportion of long-term disability worsening than the 1.0-point definition and should be preferred in this subgroup of patients.

Disclosure: E. Portaccio has received compensation for travel, advisory boards and/or speaking activities from Biogen, Merck Serono, Sanofi, Teva, Roche, BMS Celgene, Janssen, and Novartis, and serves on the editorial board of Frontiers in Neurology and Brain Sciences. L. Pastò received research support from Novartis and Biogen and speaker honoraria from Teva. A. Lugaesi has served on advisory boards and received honoraria or travel support from multiple companies including Biogen, Merck, Novartis, Roche, Sanofi/Genzyme, and others; her institutions received research grants from Novartis, Roche, and Sanofi/Genzyme. E. Cocco, F. Patti, V.L.A.M. Torri Clerici, D. Ferraro, V. Brescia Morra, G. Salemi, M. Vianello, R. Cerqua, P. Perini, S. Romano, C. Pozzilli, C. Tortorella, A. Di Sapio, P. Annovazzi, P. Iaffaldano, and M. Trojano received grants, honoraria, travel support, and/or served on advisory boards for pharmaceutical companies including Almirall, Alexion, Bayer, Biogen, BMS/Celgene, Janssen, Merck (Serono), Novartis, Roche, Sanofi/Genzyme, Teva, and others. M. Inglese received research grants from NIH,

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EPO-0932 | Satralizumab for the treatment of neuromyelitis optica spectrum disorders: A single center real life experience

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Background and aims: Neuromyelitis optica spectrum disorders (NMOSD) is a rare, inflammatory, central nervous system disease historically treated with rituximab (RTX). Satralizumab (SATRA), an anti-IL6 receptor monoclonal antibody, has recently been approved for AQP4-IgG-positive patients switching from RTX for inefficacy or adverse events. We report our real-world experience.

Methods: We retrospectively collected demographic, clinical and MRI data from all patients starting SATRA at our center between February 2023 and July 2025.

Results: 11 NMOSD patients (10 females) were enrolled. At SATRA initiation, median age was 48.5 years (25.7–71.8), median disease duration was 16.3 years (range: 1.0–34.1), median EDSS was 3.5 (0–6.0). Prior therapy was tocilizumab (TOCI) in 5/11 patients and RTX in 6/11. Among TOCI-treated patients, 3/5 had previously received RTX and 2/5 autologous hematopoietic stem-cell transplantation. TOCI was started for disease activity and the switch to SATRA was to shift to an on-label anti-IL-6R drug. All RTX switched for safety and among them 5/6 had hypogammaglobulinemia with recurrent infections. One started monthly intravenous immunoglobulin supplementation in February 2025. Over a median SATRA follow-up of 2.2 years (0.6–3.0) all patients remained clinically stable (median EDSS 3.5, 0–6.0) and showed no radiological disease activity. For patients switching from TOCI, global follow-up in anti-IL-6R therapy was 10.3 years (4.1–12.9).

Conclusion: In our small cohort, SATRA has proven to be effective and well tolerated, including long-term anti-IL-6R treated patients. Those who switched to SATRA to change mechanism of action stopping B-cell depletion because of worsening

hypogammaglobulinemia all experienced a reduction of adverse events in both frequency and severity.

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EPO-0933 | Multiple sclerosis and outpatient physician care in the last years of life: A population-based study

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Background and aims: Patients with MS require increasing healthcare support, particularly in the last year of life. Despite the benefits of palliative care in addressing symptom burden and improving quality of life, its integration into MS care remains inconsistent. This study aimed to characterize healthcare utilization and palliative care access among people with MS in the last years of life and to identify key factors associated with optimal care.

Methods: A retrospective cohort study was conducted using linked health administrative data. The cohort consisted of all adult decedents between 2016 and 2020, and we compared health care utilizations. Demographics, healthcare utilization, palliative care involvement, and medical interventions were analyzed. Predictors of palliative care receipt and hospital deaths were evaluated.

Results: MS decedents ($n=1975$) were younger and had fewer comorbidities than non-MS decedents ($n=500,904$). High percentage of MS decedents had outpatient neurology visits in the last five years of life but a steep decline closer to death. Palliative care use was similar but occurred earlier in MS. Multivariable regression showed that rural residence was associated with increased odds of hospital death and lower odds of receiving

palliative care, while receiving palliative care within the last five years of life reduced the odds of hospital death. Higher comorbidity and outpatient visits to urology were associated with a greater odd of receiving palliative care.

Conclusion: While palliative care receipt was comparable, rural disparities and high hospital deaths persist in MS. Earlier palliative integration, particularly through neuropalliative care, could improve quality of life and reduce hospitalizations.

Disclosure: Nothing to disclose.

Sleep-Wake Disorders 3

EPO-0934 | JU SLEEP WELL: A multiprofessional project designed to develop and test a digital CBT intervention to improve quality of life in patients with RLS

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Background and aims: Restless Legs Syndrome (RLS) is a difficult-to-treat chronic neurological condition. Cognitive Behavioral Therapy (CBT) can support coping and self-care in chronic diseases, yet no evidence-based digital CBT programs exist to complement medication for RLS. Therefore, this project aims to investigate, from the perspectives of patients, relatives, and healthcare professionals, gender- and age-related differences in experiences, symptom management, and treatment needs among patients with RLS, and to co-create a digital CBT-based intervention.

Methods: A four-step longitudinal multimethod design, including a randomized controlled trial (RCT) with long-term follow-up, will be used (Figure 1). Approximately 30 multiprofessional members, including people with RLS, researchers, and healthcare professionals (physicians, nurses, psychologists, and sociologists) specialized in RLS, sleep problems, and CBT, collaborate in the co-design of the digital CBT-based intervention.

Results: Step 1 (2022–2026) used various qualitative methods to explore and describe RLS-related care needs as experienced by patients, relatives, and healthcare professionals. Step

TABLE 1 Overview of the steps in JU SLEEP WELL study.

Number	Published articles	Steps in JU Sleep Well study
1	Benzer, A., Almoradi, Z., Lind, J., Ulander, M., Lundin, F., & Pakpour, A. (2023). Worldwide estimation of restless legs syndrome: a systematic review and meta-analysis of prevalence in the general adult population. <i>Journal of sleep research</i> , 32(3), e13783. https://doi.org/10.1111/jsr.13783	Systematic review and meta-analysis before step 1
2	Benzer, A., Almoradi, Z., Odzakovic, E., Kallio, V., Jemelita, S., Lind, J., Ulander, M., & Pakpour, A. (2024). Quality of life among patients with restless legs syndrome: A systematic review and meta-analysis. <i>Journal of Clinical Neuroscience: Official Journal of the Neurological Society of Australasia</i> , 122, 84–91. https://doi.org/10.1016/j.jocn.2023.07.071	Systematic review and meta-analysis before step 1
3	Park, M., Knutson, S., Odzakovic, E., Hellstrom, A., Sandlund, C., Chander, M., Lind, J., Pakpour, A. B., Brostrom, A., & members of the Jönköping University (JU) Sleep Well Research Group (2024). Validation of two brief instruments (the SJRL and CollabRAT2) to measure shared decision-making in patients with restless legs syndrome. <i>Journal of sleep research</i> , 33(6), e14071. https://doi.org/10.1111/jsr.14071	Step 2 (Quantitative design)
4	Odzakovic, E., Allgauer, M., Jonsson, L. L., Öberg, B., Fridlund, B., Ulander, M., Lind, J., & Brostrom, A. (2024). Experiences of facilitators and barriers for fulfillment of human needs when living with restless legs syndrome: a qualitative study. <i>International journal of qualitative studies on health and well-being</i> , 19(1), 2440824. https://doi.org/10.1080/17445019.2024.2440824	Step 1 (Qualitative design)
5	Knutson, S., Park, M., Odzakovic, E., Hellstrom, A., Sandlund, C., Chander, M., Lind, J., Fridlund, B., Pakpour, A., & Brostrom, A. (2024). The effect brief index-validation of a brief questionnaire to evaluate wellness based on a holistic perspective in patients with restless legs syndrome. <i>Sleep & Breathing - A European Journal</i> , 28(4), 1361–1371. https://doi.org/10.1007/s13253-024-01648-5	Step 2 (Quantitative design)
6	Hellstrom, A., Pakpour, A., Odzakovic, E., Bjork, M., Ulander, M., Knutson, S., Sandlund, C., & Brostrom, A. (2024). The psychometric properties of the Pains Visual Scale in persons living with restless legs syndrome. <i>Pain one</i> , 19(3), e011254. https://doi.org/10.1515/painone-2023-011254	Step 2 (Quantitative design)
7	Jonsson, L., Odzakovic, E., Ulander, M., Lind, J., & Brostrom, A. (2023). Primary healthcare nurses' experiences of symptoms and treatment needs of patients with RLS-associated symptoms at telephone nursing – an abductive analysis based on the Four Habits communication model. <i>International journal of qualitative studies on health and well-being</i> , 18(1), 243697. https://doi.org/10.1080/17445019.2023.243697	Step 1 (Qualitative design)
8	Odzakovic, E., Hellstrom, A., Jonsson, L., Lagerstedt, M., Fridlund, B., Jonsson, L. L., Ulander, M., Lind, J., & Brostrom, A. (2023). Perspectives for self-care actions in individuals with restless legs syndrome: a deductive qualitative analysis based on the COSE model. <i>Journal of health psychology</i> , 19(1), 4039–4076. https://doi.org/10.1177/14691463231151539	Step 2 (Quantitative design)
9	Odzakovic, E., Sandlund, C., Hellstrom, A., Ulander, M., Bjork, M., Jemelita, S., Kallio, V., Bjork, M., Knutson, S., Lind, J., Pakpour, A., & Brostrom, A. (2023). Self-care behaviors in patients with restless legs syndrome (RLS): development and psychometric testing of the RLS-Self-care behavior questionnaire. <i>Journal of sleep research</i> , 32(3), e14390. https://doi.org/10.1111/jsr.14390	Step 1 (Qualitative design)
10	Grosgaum, M., Odzakovic, E., Bjork, M., Kallio, V., Jemelita, S., Bjork, M., Ulander, M., Fridlund, B., Knutson, S., Sandlund, C., Pakpour, A., & Brostrom, A. (2023). Validation of the Health Literacy Scale Instrument in a Restless Legs Syndrome Population: Classical Test Theory and Rasch Analysis Study. <i>Journal of medical Internet research</i> , 27, e04474. https://doi.org/10.2196/16474	Step 2 (Quantitative design)
11	Odzakovic, E., Sjogblad Persson, A., Lindholm Larsson, E., Öberg, B., Jakobson, M., Bjork, M., Knutson, S., Fridlund, B., Jonsson, L. L., Ulander, M., Lind, J., & Brostrom, A. (2023). Experiences of sleep problems, subsequent daytime consequences, and self-care actions used to improve sleep among patients with restless legs syndrome: a qualitative content analysis. <i>Journal of research in nursing</i> : JRN, 13449871231184535. Advance online publication. https://doi.org/10.1177/17446871231184535	Step 1 (Qualitative design)

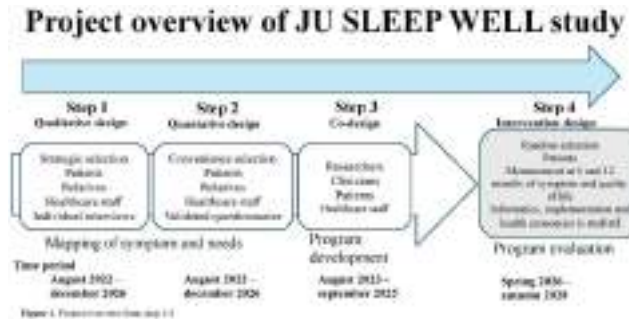


FIGURE 1 Project overview from step 1–4.

2 (2022–2026) validated five instruments for shared decision-making, mastery, wellness, e-health literacy, and self-care. Steps 1 and 2 have produced 11 publications to date (Table 1). In Step 3 (2026), the co-designed digital CBT-based intervention will be pilot-tested at one primary care center. In Step 4 (2026–2028), a

multicenter RCT conducted at eleven primary care centers will evaluate its long-term (12-month) effects.

Conclusion: The project's mapping of the experiences of patients, relatives, and healthcare professionals provides an evidence-based foundation for the co-created digital CBT-based intervention, which, if successful, can complement pharmacological treatment and improve quality of life for patients with RLS.

Disclosure: Nothing to disclose.

EPO-0935 | Pupillometric biomarkers of autonomic dysfunction in isolated REM sleep behaviour disorder

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Background and aims: Pupillary abnormalities are common in neurodegenerative disorders. In isolated REM sleep behaviour disorder (iRBD), increased pupil size and reduced pupillary light reflex have been reported, suggesting parasympathetic dysfunction. This study aimed to assess pupillary abnormalities in prodromal synucleinopathy using a bedside portable pupillometer.

Methods: Sixty individuals with iRBD (mean age=66.9 years, SD=9.6; 5 females) and 48 controls (mean age=67.0 years, SD=9.7; 5 females) underwent pupillometry using a portable infrared pupillometer (Neuro-Light, IDMed, France) after 5 minutes of dark adaptation. Five measurements were obtained from each eye following stimulation with a 320-lux white light flash lasting 1 second. Median values of maximum and minimum pupil diameter, their difference and variation were calculated for each eye. Maximum constriction velocity and latency to onset of constriction were calculated as median values from ten measurements across both eyes. Between-group comparisons were performed using ANCOVA with age as a covariate.

Results: After adjustment for age, the iRBD cohort showed significantly prolonged pupillary constriction latency compared with controls (239.3 ms, SD=34.3 ms vs. 215.6 ms, SD=37.9; $p=0.002$). No significant group differences were found in other parameters. Age significantly influenced most pupil size-related measures.

Conclusion: Pupillometry identified prolonged pupillary constriction latency in iRBD, consistent with early parasympathetic dysfunction. However, latency alone is unlikely to represent a robust marker. Further studies exploring its relationship with other clinical or laboratory biomarkers are warranted.

Disclosure: Supported by NW24-04-00456 and NW25-04-0079.

EPO-0936 | Association of endoplasmic reticulum stress and BDNF levels with disease severity and sleep quality in patients with restless legs syndrome

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Background and aims: Restless legs syndrome (RLS) is a neurological condition associated with a significant socioeconomic burden and reduced quality of life. Endoplasmic reticulum (ER) stress can trigger adverse cellular processes, including inflammation and cell death, and has been implicated in various neurological disorders. Brain-derived neurotrophic factor (BDNF) plays a crucial role in neuroplasticity, neuronal development, neuroprotection, and neuronal survival. However, the precise involvement of ER stress and BDNF in the molecular pathogenesis of RLS remains unclear. Therefore, this study aimed to evaluate the expression of ER stress-related proteins and BDNF in patients with RLS, assess their diagnostic potential, and examine their relationships with disease severity, quality of life, sleep quality, depression, and laboratory parameters.

Methods: This cross-sectional study included 35 patients with RLS and 25 healthy controls. Serum concentrations of ER stress-related proteins (ATF6, GRP78, and SERP1) and BDNF were measured using ELISA. Disease severity, sleep quality, quality of life, and depressive symptoms were assessed using validated scales.

Results: Serum levels of ATF6, GRP78, SERP1, and BDNF were significantly higher in patients with RLS compared to healthy controls. Correlation analyses revealed positive associations between these biomarkers and disease severity, as well as poorer sleep quality. ROC analyses indicated that ATF6 levels above 302.05 pg/mL, GRP78 above 1.24 ng/mL, SERP1 above 155.14 pg/mL, and BDNF above 6.50 ng/mL could predict the presence of RLS.

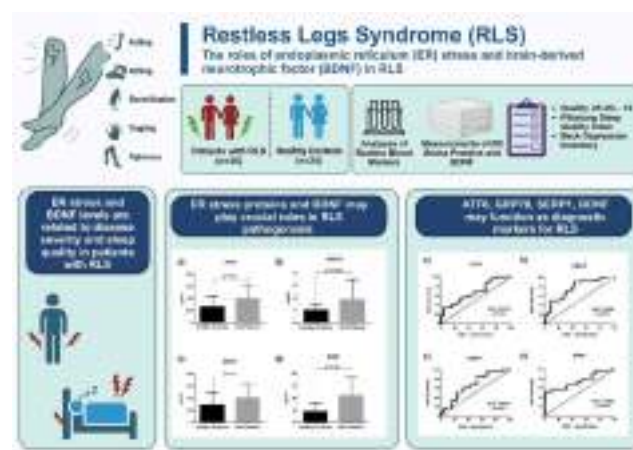


FIGURE 1 Elevated ER stress proteins (ATF6, GRP78, SERP1) and BDNF in Restless Legs Syndrome and their associations with disease severity and sleep quality, highlighting their potential role in RLS pathogenesis and diagnostic value.

Conclusion: These findings provide novel evidence suggesting that ER stress and BDNF may play a significant role in the pathophysiology of RLS and may represent potential biomarkers for disease identification.

Disclosure: Nothing to disclose.

EPO-0937 | Interplay between diagnostic features and treatment biomarkers in narcolepsy type 1

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Background and aims: Physiological features with diagnostic utility may also serve as disease-specific prognostic and treatment-response biomarkers. Here we study features emerging from recent work on multimodal diagnosis of narcolepsy type 1 (NT1), integrating sleep phenotyping, genetics, and artificial intelligence. We explore how these features change in individuals treated with oreporexton, an oral orexin receptor 2-selective agonist, in one phase 2 trial (NCT05687903) and two phase 3 trials (The First Light [FL]: NCT06470828; The Radiant Light [RL]: NCT06505031).

Methods: Participants with NT1 were randomized to receive oreporexton (dose varied by trial) or placebo. Nocturnal polysomnography (nPSG) was collected at baseline and at end of treatment. Maintenance of Wakefulness Tests (MWTs) were performed after each PSG night. In addition to manual scoring, nPSG and MWT data were analyzed to compute hypnodeltadensities from which overall sleep probability and NT1 diagnostic scores were calculated. Several NT1-relevant sleep features were also evaluated. Treatment effects were evaluated with baseline-adjusted models.

Results: Early-night rapid eye movement (REM) features known to be elevated in NT1 were reduced with oreporexton versus placebo, including first-quarter time in REM ($p < 0.001$ in all trials and treatment arms) and NT1-specific transitions (REM to/from Wake or N1). The nPSG-based NT1 diagnostic score was reduced in FL and RL 2 mg/2 mg arms versus placebo (both $p < 0.05$). In oreporexton-treated participants, MWT sleep probability was reduced versus placebo ($p < 0.05$, Phase 2), consistent with reduced daytime sleepiness.

Conclusion: Diagnostic NT1 features responded significantly to oreporexton treatment, reflecting reduced daytime sleepiness and fewer sleep abnormalities.

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EPO-0938 | Sleep biomarkers of stroke-related delirium: The STORM study

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Background and aims: Delirium is a frequent complication after acute stroke and is associated with worse outcomes. Sleep disturbances are common after stroke and may contribute to delirium, but evidence based on objective sleep measures is limited. The aim of this study is to investigate the association between sleep alterations and post-stroke delirium, and to assess their impact on 90-day functional outcome.

Methods: We retrospectively analyzed patients with acute ischemic stroke who underwent overnight bedside polysomnography during stroke unit hospitalization. Delirium was diagnosed using DSM-5 criteria. Functional outcome at 90 days was assessed using the modified Rankin Scale (mRS).

Results: The cohort included 128 patients (mean age 70.8 ± 12.6 years, 53.1% females); delirium occurred in 28.9% of patients. Patients with delirium were older and had more severe strokes. Delirium was associated with lower sleep efficiency (SE) ($p = 0.041$), higher wake after sleep onset ($p = 0.047$), and lower REM% ($p < 0.001$). In multivariate analysis, lower SE independently predicted delirium (aOR 0.964, $p = 0.010$), along with higher stroke severity. At 90 days, delirium was strongly associated with poor functional outcome (mRS > 2). Unfavorable outcome was also linked with reduced SE ($p = 0.032$), shorter total sleep time ($p = 0.015$), reduced REM% ($p < 0.001$), higher nocturnal heart rate ($p = 0.035$), and increased daytime sleep ($p = 0.002$). Delirium, diabetes, and daytime sleep (aOR = 0.984; $p = 0.037$) independently predicted poorer recovery.

Conclusion: Reduced sleep efficiency is an independent risk factor for post-stroke delirium, and sleep-wake disturbances are associated with worse long-term outcomes. Sleep assessment in acute stroke may help identify patients at risk for delirium and represents a potential target for interventions to improve stroke prognosis.

Disclosure: Nothing to disclose.

EPO-0939 | Sleep disorders in dementia with Lewy bodies: A prospective cohort study

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Background and aims: Sleep disturbances are highly prevalent in Dementia with Lewy Bodies (DLB), yet their role in the clinical phenotype remains under-investigated. This study

evaluates sleep disorders prevalence in DLB patients and their correlations with cognitive, and neuropsychiatric symptoms.

Methods: Consecutive patients with probable DLB or DLB-Mild Cognitive Impairment (McKeith criteria 2017/2020) were enrolled in a tertiary neurodegenerative diseases clinic. All participants underwent a standardized diagnostic protocol, including neuropsychological assessment, and Dopamine Transporter (DAT) scan. Sleep was assessed in all patients both subjectively with questionnaires and objectively with overnight in-lab video-polysomnography (VPSG).

Results: Thirty DLB patients (mean age 73.1 years; 60% males) were included. REM Behavior Disorder was diagnosed in 85.7% and insomnia in 50% of patients. Periodic limb movements (PLM > 15/h) were observed in 71.4%. Prevalence of Obstructive Sleep Apnea and Excessive Daytime Sleepiness was respectively 25% and 32.1%. Overall poor sleep quality was reported by 78.5% of patients. Worse performances in executive functions correlated with lower REM% ($p=0.008$), and higher PLM index ($p=0.024$), while worse performances in visuospatial functions correlated with higher N1% ($p=0.009$). Lower mean saturation during sleep correlated with higher severity of visual hallucinations ($p=0.038$). Lower DAT uptake in the caudate correlated with lower Sleep Efficiency ($p=0.022$), lower N3% ($p=0.028$), and higher PLM index ($p=0.045$). Finally, Insomnia Severity Index ($p=0.021$) and Epworth Sleepiness Scale ($p=0.018$) inversely correlated with putamen uptake.

Conclusion: Sleep disorders are highly prevalent in DLB, being associated with worse cognitive functions and more severe neuropsychiatric symptoms. Reduced striatal uptake is associated with worse quality sleep, higher insomnia and daytime sleepiness.

Disclosure: Nothing to disclose.

EPO-0940 | Effect of oreporexton (TAK-861) on cognitive symptoms in patients with narcolepsy type 1: Pooled data from two phase 3 trials

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Background and aims: Cognitive symptoms are frequent and disruptive in narcolepsy type 1 (NT1). Using pooled data from

two randomized, double-blind, phase 3 studies (The First Light: NCT06470828; The Radiant Light: NCT06505031) we report the effect of oreporexton (TAK-861) on cognitive symptoms in participants with NT1.

Methods: Participants aged 16–70 years diagnosed with NT1 were randomized to twice daily oral oreporexton 1 mg (The First Light only), 2 mg, or placebo, ≥ 3 hours apart for 12 weeks. Cognitive impairment was assessed with neuropsychological tests of attention (Psychomotor Vigilance Test [PVT] from 2 intraday sessions [9 am, 3 pm]), memory (Continuous Paired Associate Learning, CPAL) and executive function (International-Digit Symbol Substitution Test-Symbols, IDSST-S; One-Back Test, ONB). Patient-reported cognitive difficulties were assessed with the Functional Impacts of Narcolepsy Instrument Cognitive Function domain (FINI-CF) and British Columbia Cognitive Complaints Inventory (BC-CCI). For both trials, PVT and FINI-CF were secondary endpoints while the remainder were exploratory.

Results: Overall, 273 participants were randomized to oreporexton 1 mg/1 mg ($n=61$), 2 mg/2 mg ($n=136$), or placebo ($n=76$). Compared with placebo, 12-week of treatment with oreporexton (1 mg/1 mg or 2 mg/2 mg) resulted in improvement from baseline for each objective (PVT, CPAL, IDSST-s, ONB) and subjective (FINI-CF; BC-CCI) cognitive assessment. Group mean differences from placebo in change from baseline scores for each cognitive assessment are shown in Table 1.

Conclusion: The benefit to cognition based on both objective neuropsychological tests and subjective ratings of cognition, following 12 weeks treatment, indicates that oreporexton reduces cognitive symptoms in NT1.

Disclosure: This research was funded by Takeda Development Center Americas, Inc, who also provided funding to Envision Catalyst, an Envision Medical Communications agency, a part of Envision Pharma Group, to provide writing assistance under the direction of the authors. Fabio Pizza participated in an advisory board for Takeda and received support for congress participation and funding from Bioprojet. Yves Dauvilliers received funds for seminars, board engagements, and travel to conferences from Avadel, Bioprojet, Idorsia, Jazz Pharmaceuticals, Centessa,

TABLE 1 Group mean (least square and 95% CI) difference between oreporexton and placebo for endpoints from the objective and subjective cognitive assessments at week 12 in adults with NT1.

Cognitive Endpoint*	Oreporexton 1 mg/1 mg	Oreporexton 2 mg/2 mg
Objective neuropsychological tests		
Psychomotor Vigilance Test: number of lapses	-18.29 (-13.27, -23.32)	-10.87 (-12.86, -7.34)
P value	<0.0001	<0.0001
Continuous Paired Associate Learning: number of errors	-20.03 (-27.81, -12.14)	-18.77 (-26.76, -12.76)
P value	<0.0001	<0.0001
International-Digit Symbol Substitution Test: number of correct responses	14.37 (10.31, 18.42)	13.41 (12.20, 14.61)
P value	<0.0001	<0.0001
One-Back Test: speed of performance	-0.58 (-6.12, -0.04)	-0.18 (-6.12, -0.07)
P value	<0.0001	<0.0001
Subjective ratings of cognitive difficulties		
Functional Impacts of Narcolepsy: Impaired Cognitive Function domain	-30.42 (-37.73, -23.11)	-34.12 (-38.77, -29.47)
P value	<0.0001	<0.0001
British Columbia Cognitive Complaints Inventory	-4.44 (-5.65, -3.23)	-5.88 (-6.01, -4.15)
P value	<0.0001	<0.0001

*For all tests except the IDSST negative signs for group mean differences indicated a benefit of oreporexton versus placebo. For IDSST a positive sign indicates benefit of oreporexton versus placebo. Change from baseline is placebo-adjusted. In this analysis all P values are unadjusted.

Alkermes, Pharmanovia, and Takeda. Rafael Del Rio Villegas received consultancy fees from Alkermes, Bioprojet, and Takeda, and travel funds from Bioprojet, Jazz Pharmaceuticals, and Takeda. Gert J. Lammers has received consulting fees, grants, and travel funds from Alkermes, Bioprojet, Daiichi Sankyo, Eisai, Jazz Pharmaceuticals, and Takeda. Giuseppe Plazzi received consultancy fees from Bioprojet, Jazz Pharmaceuticals, Orexia, and Takeda. Thomas E Scammell received grants and personal fees from Takeda. Tathagata Banerjee, Alice Cai, Heather Romero, Ellie Stukalin, and Brian Harel are employees of Takeda Development Center Americas, Inc, and stockholders in Takeda Pharmaceuticals Company Limited. Paul Maruff is an employee of Cogstate Limited.

EPO-0941 | Improving automatic sleep stage scoring for narcolepsy through fine-tuning with data from central disorders of hypersomnolance

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Background and aims: Sleep stage classification is essential for narcolepsy diagnosis but remains time-intensive. Current deep learning models, trained primarily on healthy controls and patients with common sleep disorders have, show reduced performance on narcolepsy patients due to the condition's low prevalence and distinct sleep patterns. We investigated whether fine-tuning with data from central disorders of hypersomnolance (CDH) could improve automatic sleep stage classification in narcolepsy patients.

Methods: We employed a CNN-Transformer network predicting sleep stages from one EOG and three EEG channels from overnight polysomnography. The baseline model was trained on 58 healthy controls, 18 OSA patients and 78 insomnia patients. Fine-tuning used 15 insomnia patients, 7 OSA patients, 3 narcolepsy type 1 (NT1) patients, 4 narcolepsy type 2 (NT2) patients, 24 patients with other sleep disorders, and 23 narcolepsy borderland patients. Testing included 9 NT1, 6 NT2 and 8 narcolepsy borderland patients.

Results: The baseline model achieved Cohen's kappa of 0.72 and accuracy of 79%. Fine-tuning improved kappa to 0.75 ($p=0.002$) and accuracy to 81% ($p=0.002$). N1 detection improved with kappa increasing from 0.46 to 0.54 ($p<0.001$) and sensitivity from 45% to 56% ($p<0.001$). REM stage detection showed kappa improvement from 0.77 to 0.79 ($p<0.001$), and specificity from 93% to 97% ($p<0.001$).

Conclusion: Fine-tuning with rare sleep disorder data improved automatic sleep stage classification in narcolepsy patients. Enhanced N1 classification enables more precise sleep-onset and fragmentation assessment, while improved REM stage detection facilitates SOREMP identification. These results demonstrate that targeted fine-tuning can bridge performance gaps between common and rare sleep disorders in automated scoring.

Disclosure: Nothing to disclose.

EPO-0942 | Does age matter? Subjective and objective responses to hypoglossal nerve stimulation in older patients with obstructive sleep apnea

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Background and aims: Obstructive sleep apnea (OSA) is common in elderly and is associated with higher CPAP failure rates due to comorbid sleep disorders and cognitive decline. Hypoglossal nerve stimulation (HNS) has emerged as an alternative treatment for OSA; however, data on age-related treatment response remain limited.

Methods: We retrospectively analyzed 82 patients who underwent HNS implantation for OSA, stratified into ≤ 65 years ($n=29$) and >65 years ($n=53$). Subjective benefit was assessed at 12 months using a three-point scale: very satisfied (2), somewhat satisfied (1), and dissatisfied (0). Treatment success was defined as a $\geq 50\%$ reduction in apnea-hypopnea index (AHI) from baseline polysomnography to 12-month home sleep test, with a final AHI ≤ 20 events/hour.

Results: Baseline polysomnographic parameters did not differ between age groups. During the HNS titration study, patients >65 years showed higher periodic limb movement index, longer sleep latency, and shorter total sleep time, while apnea-hypopnea index (AHI) was similar between groups. At 12-month home sleep testing, total AHI did not differ by age; however, the >65 -year group demonstrated lower supine AHI and oxygen desaturation index, along with significantly greater subjective satisfaction. Overall treatment success was achieved in 42 patients (51.2%), with similar success rates in the >65 -year group (56.6%) and the ≤ 65 -year group (41.4%).

Conclusion: Despite less favorable sleep-related findings during titration, older patients reported greater subjective satisfaction after HNS, with objective treatment success comparable to younger patients. Advanced age should not be considered a limiting factor for HNS therapy.

Disclosure: Nothing to disclose.

EPO-0943 | Telemedicine consultation as diagnostic triage for adult and children with suspected narcolepsy: The TENAR reliability and diagnostic accuracy study

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TABLE 1 Baseline demographic and clinical characteristics of participants in the reliability and diagnostic accuracy studies.

		Reliability study N = 221	Diagnostic accuracy study N = 198
Sex, n (%)	F	129 (58.3)	108 (54.5)
Age, yrs	Mean (SD)	36.8 (17.1)	36.3 (17.2)
Region of origin, n (%)			
	North	161 (73.2)	145 (73.9)
	Centre	42 (19.1)	37 (18.8)
	South	18 (8.2)	9 (4.5)
	Islands	7 (3.2)	6 (3.0)
First consultation, n (%)			
	In-office consultation	115 (52)	97 (49)
	Telemedicine consultation	106 (48)	91 (46)
Epworth sleepiness scale, score	Mean (SD)	13.8 (4.8)	13.7 (4.5)
Excessive daytime sleepiness*, n (%)	Yes	219 (99)	187 (94.4)
Involuntary falling asleep*, n (%)	Yes	104 (47)	117 (59.1)
Cataplexy*, n (%)	Yes	72 (32.6)	69 (34.9)
Sleep paralysis*, n (%)	Yes	80 (36.2)	73 (37.1)
Hypnagogic hallucinations*, n (%)	Yes	76 (34.4)	79 (39.9)
Disrupted nocturnal sleep*, n (%)	Yes	99 (45)	88 (44.5)
Final diagnosis			
	Narcolepsy Type 1		32 (16.2)
	Narcolepsy Type 2		11 (5.6)
	Subjective excessive daytime sleepiness		42 (21.2)
	Major Depressive Disorder		33 (16.7)
	Obstructive Sleep Apnoea		27 (13.6)
	Smoking		9 (4.5)
	Circadian rhythm disorder		7 (3.5)
	Other		17 (8.6)

TABLE 2 Clinical characteristics of participants with narcolepsy in the diagnostic accuracy study.

	Narcolepsy type 1 N = 52	Narcolepsy type 2 N = 11
Sex, n (%)		
	Female	8 (54.5)
	Male	5 (45.5)
Age, yrs		
	Mean (SD)	27.6 (11.2)
	< 18 yrs, n (%)	1 (9.1)
Excessive daytime sleepiness*, n (%)	Yes	11 (100)
Cataplexy*, n (%)	Yes	0
Sleep paralysis*, n (%)	Yes	5 (45.5)
Hypnagogic hallucinations*, n (%)	Yes	5 (45.5)
Disrupted nocturnal sleep*, n (%)	Yes	4 (40.0)
Epworth sleepiness scale, score	Mean (SD)	13.3 (3.8)
MSLT, score	Mean (SD)	23.8 (3.6)
HIA DQ81796-82, n (%)	Yes	4 (40.0)
Cerebrospinal fluid orexinB, pg/mL	Mean (SD)	332.3 (39.2)
	< 110, n (%)	0
MSLT sleep latency, min	Mean (SD)	3.0 (4.0)
MSLT SOREMP, n	Mean (SD)	1.9 (1.3)

Background and aims: Sleep centres specialised in the care of narcolepsy are few and scattered in terms of countries and regions in countries distribution. Telemedicine consultation (TMC) could be important in guiding the diagnostic workup in cases where symptoms suggesting narcolepsy are present in people far from sleep centres. This study aimed at assessing the

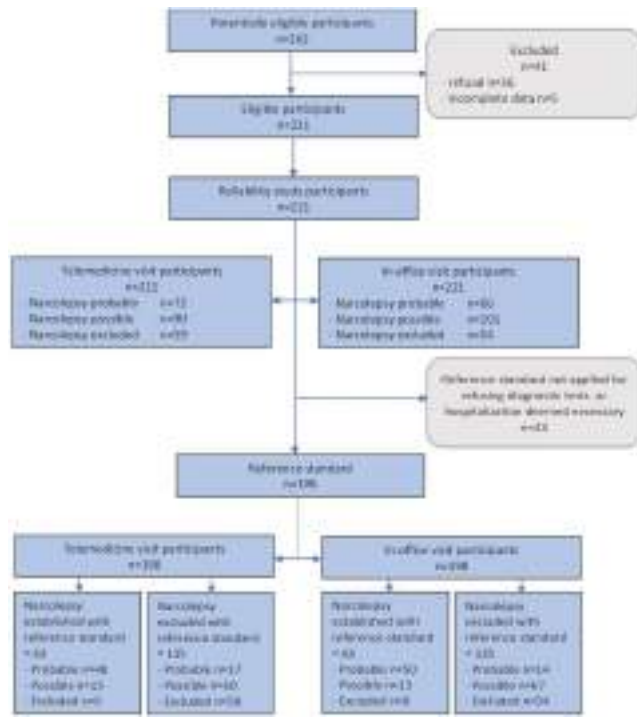


FIGURE Diagram of participant flow in the reliability and diagnostic accuracy studies.

inter-procedural reliability (compared to the in-office consultation) and the diagnostic accuracy of the TMC when excessive daytime sleepiness (EDS) due to narcolepsy is suspected.

Methods: Cross-sectional diagnostic study (2019–2023). Consecutive people of any age referred for EDS complaint to the Clinic for Narcolepsy in Bologna. The TMC was conducted via tablet for the patient, and via equipped personal computer in another room for the physician (interviewer). Interviewers expressed diagnostic orientation indicating narcolepsy as “probable”, “possible”, “excluded”. The final diagnosis was established by applying the ICSD-3 criteria. Inter-procedural reliability (Kappa statistics) and diagnostic accuracy measures were assessed.

Results: 221 participants were included in the reliability study, 198 in the diagnostic accuracy study. The agreement between the two consultation modalities on diagnostic orientation was “substantial” (Kappa 0.74, 95% CI 0.66–0.82). Agreement on the narcoleptic pentad symptoms was “substantial” (EDS, hypnagogic hallucinations, disrupted nocturnal sleep) or “almost perfect” (cataplexy, sleep paralysis). Telemedicine and in-office consultation had identical sensitivity (100%, 95% CI 94–100) and similar specificity (43%, 95% CI 35–52, vs 40%, 95% CI 32–49).

Conclusion: TMC is a reliable diagnostic triage procedure when EDS possibly due to narcolepsy is suspected, in particularly to exclude people who do not suffer from narcolepsy without the risk of false negatives.

Disclosure: Nothing to disclose.

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Background and aims: Narcolepsy (types 1 and 2) is a neurologic hypersomnolence disorder defined by a range of symptoms including excessive daytime sleepiness (EDS) and cataplexy (type 1 only). The study evaluated the incidence, prevalence and burden of narcolepsy in Germany.

Methods: Retrospective cohort study using anonymized health-care claims data involving 8.2 million people within the InGef database (2018–2023). Cohorts included pre-existing narcolepsy cases (PCN), incident narcolepsy cases (ICN) and controls (Figure 1).

Results: A total of 1,757 narcolepsy cases were identified; 1,163 PCN and 594 ICN. Mean (SD) age was 40.13 (19.52) for ICN and 48.11 (18.1) for PCN cohorts. 13% of ICN and 4% of PCN were children/adolescents (<18 years). Overall (2018–2023) prevalence per 100,000 people was 21.29 (95% CI: 20.30–22.31); annual incidence decreased from 1.93 to 1.18 per 100,000 people between 2018 and 2023 (Figure 2). Overall prevalence and annual incidence were higher in adults (≥18 years) vs. children/

adolescents (prevalence: 25.13 vs 7.10 per 100,000; annual incidence, 2018–2023: 1.27–2.08 vs 0.60–1.38 per 100,000, respectively), with comparable rates in males and females. Comorbidities were more frequent in ICN and PCN vs. controls, including non-narcolepsy sleep disorders (42.6% and 45.4% vs. 7.4%, respectively), psychiatric disorders (43.9% and 58.5% vs. 20.6%), and nervous system diseases (50.3% and 47.7% vs. 22.0%). The most frequently prescribed narcolepsy medications in ICN were modafinil (122 [24.95%] patients) and methylphenidate (43 [8.8%] patients).

Conclusion: This study provides robust estimates of incidence, prevalence and comorbidity burden associated with narcolepsy within the German population relative to controls.

Disclosure: Funding by Takeda Development Center Americas, Inc, who also provided funding to Envision Catalyst, an Envision Medical Communications agency, a part of Envision Pharma Group, to provide writing assistance under the direction of the authors.. InGef acted as subcontractor and received funding from WIG2 for execution of the analysis. Maureen Watt and Stephen Crawford are employees of Takeda Development Center Americas, Inc and stockholders of Takeda Pharmaceuticals Company Ltd. Itziar Ubillos, Belene Podmore and Aguenda Azpeitia are employees of OXON epidemiology, contracted by Takeda for this work. Jana Knop is an employee of Takeda Pharma Vertrieb GmbH & Co. Dominik Obermueller and Raeleesha Norris are employees of InGef, contracted by Takeda for execution of the analysis. Renate Almeida Schreck has nothing to disclose.

EPO-0945 | Abstract withdrawn

Neurological Manifestation of Systemic Diseases

EPO-0946 | Paroxysmal events in leptomenigeal carcinomatosis: The role of video-EEG in differentiating seizures from intracranial hypertension

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Background and aims: The objective of this study is to describe a case of leptomenigeal carcinomatosis (LC) presenting with recurrent transient neurological events (TNEs). We aim to emphasize the critical importance of a comprehensive diagnostic work-up, including video-electroencephalography (video-EEG), to correctly differentiate these paroxysms from focal epilepsy.

Methods: We report the case of a 41-year-old woman with a remote history of breast cancer on negative follow-up, presenting with recurrent episodes of intense headache and neck pain, tinnitus, upper limbs paraesthesia, right ptosis, and left hemiparesis with variable loss of consciousness (3–5 minutes). The work-up included brain computed tomography angiography (CTA), magnetic resonance imaging (MRI), video-EEG monitoring, and a lumbar puncture (LP).

Results: Brain imaging showed no pathological findings, while standard EEG showed focal slow anomalies. Thus, an initial suspicion of focal epilepsy was raised, and anti-seizure medications



FIGURE 1 Study design.

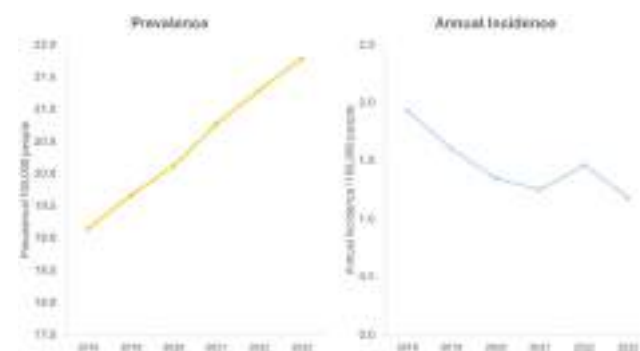


FIGURE 2 Prevalence and incidence.

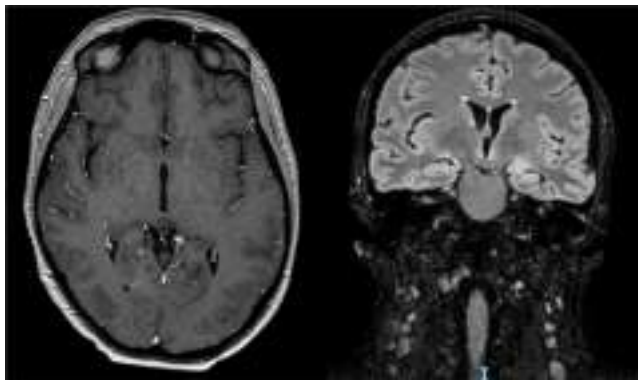


FIGURE 1

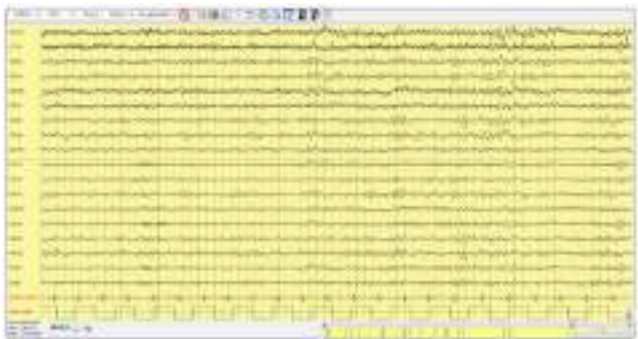


FIGURE 2

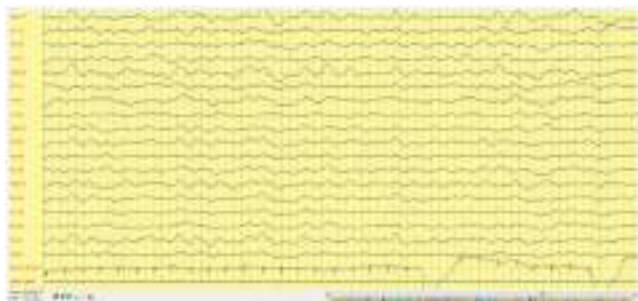


FIGURE 3

(ASMs) were started, which were ineffective. Video-EEG during an episode showed paroxysmal posterior bi-hemispheric monomorphic delta activity and subsequently generalized rhythmic delta activity (GRDA), without electrographic seizures. Subsequent LP revealed a high opening pressure of 32 cmH₂O and EpCAM-positive malignant cells. Body CT scan confirmed breast cancer recurrence. A diagnosis of LC with transient elevations in intracranial pressure (ICP) was made. Events resolved following CSF drainage and the introduction of acetazolamide.

Conclusion: Paroxysmal events in LC are not always epileptic; they may result from transient ICP increases known as “plateau waves”. These may cause brainstem dysfunction by reducing cerebral perfusion pressure. Video-EEG is essential to redefine the setting of recurrent, atypical TNEs, preventing inappropriate ASM use.

Disclosure: Nothing to disclose.

EPO-0947 | Presentation, treatment and outcome of a first stroke-like episode in a carrier of the compound heterozygous variants c.695G>A and c.2209G>C in POLG

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Background and aims: A patient with two heterozygous POLG variants in different exons, which manifested phenotypically as polyneuropathy and later with a first stroke-like episode (SLE) and seizures, has not yet been reported.

Methods: Case report

Results: The patient is a 19-year-old female who has suffered from POLG-related neuropathy since the age of 14 and was admitted for her first SLE, which manifested clinically as hemianopia and two days later led to impaired consciousness, hemiparesis, seizures and status epilepticus. Cerebral MRI showed a corresponding stroke-like lesion (SLL) in the right occipital lobe. The seizures were treated with levetiracetam, lacosamide and perampanel, but the treatment of status epilepticus required anesthesia with thiopental, ketamine and midazolam. In addition, glucocorticoids, NO precursors and antioxidants were administered. Under this treatment, the SLL initially increased in size and intensity before regressing. After extubation, a recurrent focal motor status epilepticus occurred, which could be stopped with midazolam. Since then, she has remained seizure-free and recovered completely

Conclusion: This case shows that POLG mutations can initially manifest phenotypically with polyneuropathy and SLE that SLE occurs before the onset of seizures, that status epilepticus due to POLG variants may require thiopental anesthesia and that levetiracetam, lacosamide and perampanel may have a favorable long-term effect on seizure activity in carriers of POLG mutations

Disclosure: Nothing to disclose.

EPO-0948 | Beyond the classic triad: Sudden hearing loss and demyelinating polyneuropathy in wernicke encephalopathy

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Background and aims: Wernicke encephalopathy (WE) is an acute neurological disorder caused by thiamine deficiency, classically defined by the triad of encephalopathy, ocular signs, and gait ataxia. However, this triad is present in a minority of patients, and several atypical manifestations have been described. Among them, sudden sensorineural hearing loss and peripheral neuropathy are uncommon but increasingly recognized features.

Methods: We report a single case of WE presenting with atypical neurological manifestations. Clinical findings, laboratory results, neuroimaging, electrophysiological studies, and response to treatment were retrospectively reviewed.

Results: A 46-year-old man with chronic alcohol abuse and pre-existing hypoacusis presented with sudden hearing loss of

one-week duration, associated with disorientation and progressive gait disturbance. Brain MRI showed characteristic lesions consistent with WE. Laboratory evaluation revealed hypertransaminasemia and multiple vitamin deficiencies. Paraneoplastic and autoimmune studies were negative. Electromyography demonstrated a demyelinating sensorimotor polyneuropathy, a finding that has been previously reported in WE and is likely related to severe thiamine deficiency and nutritional deficits. High-dose intravenous thiamine was promptly initiated, leading to marked clinical improvement, particularly in cognitive function and gait, although mild to moderate ataxia persisted at discharge.

Conclusion: This case highlights sudden sensorineural hearing loss and demyelinating polyneuropathy as atypical but documented manifestations of Wernicke encephalopathy. Thiamine deficiency can affect both central and peripheral nervous systems, including auditory pathways and peripheral nerves. Awareness of these non-classical features is essential to avoid diagnostic delay and to initiate timely thiamine treatment, thereby reducing the risk of permanent neurological damage.

Disclosure: Nothing to disclose.

EPO-0949 | Large vessel vasculitis and Ischemic stroke: A 10-year experience in Seville, Spain

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Background and aims: Ischemic stroke (IS) and transient ischemic attack (TIA) are rare but severe manifestations of large vessel vasculitis, including giant cell arteritis and Takayasu arteritis, with limited evidence guiding management.

Methods: Single-center retrospective database review of patients with GCA or TAK presenting with IS/TIA between 2014 and 2024 at a tertiary stroke center in Seville, Spain.

Results: Fifteen patients with LVV-related ischemic stroke were identified: 9 GCA-related, 5 TAK-related, and one with overlapping GCA/TAK. Median age was 64 years (range 40–86), and 66% (9/15) were female. In 13 cases, IS was the presenting manifestation leading to LVV diagnosis. Vertebral artery stenosis was observed in 67% of GCA patients, while all TAK patients had internal carotid artery (ICA) stenosis; the overlapping case showed both. Acute treatments included fibrinolysis (6.7%), thrombectomy (13%), and angioplasty with stenting (20%). For secondary prevention, 80% received dual antiplatelet therapy and 87% full-dose prednisone, with adjunctive immunosuppressors in four cases; two required treatment switch due to refractory disease. Tocilizumab was initiated after the first IS in one patient with prior TAK. At 90 days, 14 patients had mRS ≤ 3 , four experienced recurrent IS/TIA, and one TAK relapse was observed.

Conclusion: Although ischemic stroke secondary to LVV is uncommon, in our cohort, 87% of cases of GCA and TAK had IS as the initial presentation, highlighting the importance of having a high index of suspicion, which can guide clinical decision-making.

Disclosure: Nothing to disclose.

EPO-0950 | Neurological complications of infective endocarditis: Retrospective study

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Background and aims: Infective endocarditis (IE) is an infectious disease of the endocardial surface, mostly involving heart valves and cardiac devices. It presents with a wide spectrum of clinical manifestations and may affect multiple organ systems. Neurological complications are the most frequent extra-cardiac manifestations and are associated with significant morbidity and mortality.

Methods: We conducted a retrospective study reviewing electronic medical records of 158 patients diagnosed with IE at our hospital between 2017 and 2025.

Results: IE predominantly affected men (70.3%), the mean age of patients being 68.8 ± 14.6 years. The aortic valve was most frequently involved (46.2%), followed by the mitral valve (30.4%). Prosthetic valve involvement was observed in 30.4% of cases. *Staphylococcus aureus* was the most frequently identified pathogen (24%). Overall mortality rate was 28.5%. Neurological complications occurred in 24.1% of patients, most commonly ischemic stroke (57.9%), followed by encephalopathy (34.2%). Neurological symptoms were the initial manifestation in 50% of patients with neurological affection. A statistically significant association was found between the presence of neurological complications and mortality. Older age was associated with a higher frequency of neurological complications, whereas no association was observed with sex or the affected valve.

Conclusion: Neurological complications in the context of IE are fairly frequent, and often constitute the initial manifestation, with ischemic stroke being the most common manifestation. Their occurrence is significantly associated with mortality. Advanced age appears to be a predictive factor for neurological involvement. These findings emphasise the importance of early recognition and multidisciplinary management, particularly in older patients.

Disclosure: Nothing to disclose.

EPO-0951 | Cognitive function and sleep disturbances in rheumatoid arthritis: Clinical and inflammatory correlates

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Background and aims: This study aimed to evaluate cognitive function and sleep quality in RA and their associations with disease activity and inflammation.

Methods: This cross-sectional study included 68 RA patients (ACR/EULAR 2010). Cognitive function was assessed with MMSE and MoCA, sleep with the Munich Chronotype Questionnaire (MCTQ) and Epworth Sleepiness Scale (ESS). Disease activity and inflammation were measured using DAS28 and CRP. Patients were divided into two equal groups: with

cognitive impairment ($n=34$, 50%) and without ($n=34$, 50%). Sleep quality, cognitive scores, and inflammatory markers were compared between groups.

Results: Cognitive impairment was present in 50% of patients. Among these, 76% reported excessive daytime sleepiness (ESS > 10), 68% fatigue, 62% attention deficits, 59% memory complaints, and 53% poor sleep duration/quality (MCTQ midpoint > 03:00). Patients without cognitive impairment reported lower rates: 29% excessive sleepiness, 35% fatigue, 24% attention deficits, and 21% memory complaints. DAS28 and CRP were higher in the cognitively impaired group (DAS28 5.4 ± 0.6 vs 3.8 ± 0.5 ; CRP 23.1 ± 8.9 mg/L vs 9.2 ± 4.7 mg/L; $p < 0.001$). A significant positive correlation was observed between sleep disturbances (ESS, MCTQ) and cognitive impairment severity (MoCA $r = -0.48$, MMSE $r = -0.43$; $p < 0.01$) and with inflammatory activity.

Conclusion: Sleep disturbances are common in RA and closely associated with cognitive impairment and inflammation. Routine assessment may allow earlier identification of at-risk patients and support multidisciplinary management.

Disclosure: Nothing to disclose.

EPO-0952 | Universal neurological involvement in triple-A syndrome: Evidence from the largest genetically confirmed cohort

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Background and aims: Triple-A (AAAS, Allgrove) syndrome is a rare autosomal recessive disorder caused by biallelic mutations in the AAAS gene, which encodes ALADIN, a nuclear pore complex protein. It is classically defined by achalasia, alacrima, and adrenal insufficiency. While traditionally viewed as an endocrine disorder, increasing evidence suggests broader systemic involvement. Neurological symptoms such as neuropathy and dysautonomia have been described but remain poorly characterized and are often underrecognized. This study aimed to systematically define the neurological phenotype and underlying genetic landscape in the largest genetically confirmed AAAS cohort to date.

Methods: We analyzed 47 individuals with genetically ($n=43$) or clinically ($n=4$) confirmed AAAS from 13 international centers. All underwent standardized neurological assessment. Nerve conduction studies ($n=18$), electromyography ($n=10$), muscle MRI ($n=3$), and skeletal muscle proteomics ($n=2$) were performed. Genetic analysis identified variant types and frequencies; genotype-phenotype correlations were explored.

Results: Neurological manifestations were universal. Motor weakness (84%), hyperreflexia (80%), autonomic dysfunction (91%), and gait instability (92%) were predominant. Electrophysiology showed length-dependent axonal neuropathy; EMG confirmed chronic neurogenic changes. MRI revealed

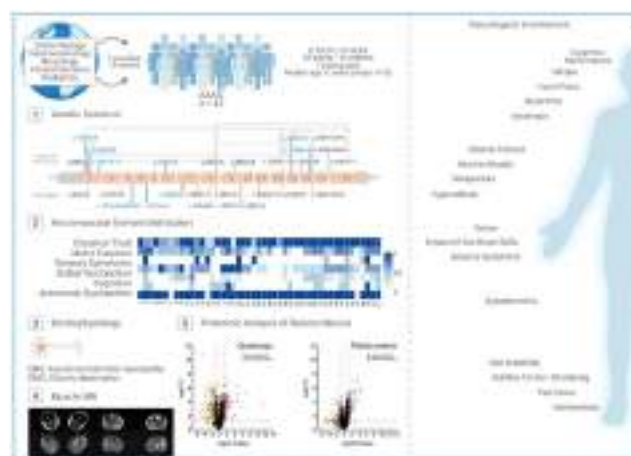


FIGURE 1 Graphical Abstract. Multisystem neurological involvement and genetic spectrum in Triple-A syndrome.

symmetric denervation. Proteomics identified consistent dysregulation of cytoskeletal, metabolic, and extracellular matrix pathways. Genetic testing revealed nine previously unreported AAAS variants. The most frequent variant, c.787T>C (p.Ser-263Pro), was found in 18 individuals, 17 of Serbo-Croatian origin. Truncating variants were associated with earlier and more severe phenotypes.

Conclusion: Neurological involvement in Triple-A syndrome is early, universal, and clinically defining. Our data support reclassifying the disease as a neurogenetic multisystem disorder, warranting systematic neurological assessment and integrated care.

Disclosure: Nothing to disclose.

EPO-0953 | Inflammation-mediated cognitive impairment following bone fracture

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Background and aims: Bone fracture is increasingly recognized as a risk factor for cognitive dysfunction beyond local tissue injury. However, the temporal characteristics of fracture-induced cognitive impairment and its underlying mechanisms remain unclear. This study investigated the effects of tibial fracture on hippocampus-dependent cognitive function in mice and explored the role of inflammation.

Methods: Male C57BL/6J mice were randomly assigned to fracture (Fx), sham (S), or control (C) groups. Behavioral and molecular assessments were conducted immediately and on postoperative days 7, 14, and 28. Locomotor activity was evaluated using the open field test, while contextual fear conditioning assessed hippocampus-dependent memory. Serum levels of IL-1 β , IL-6, and TNF- α were measured by ELISA. Hippocampal expression of BDNF and PSD95 was analyzed by Western blot, and microglial activation was assessed by Iba1 immunohistochemistry.

Results: Fx mice exhibited significant contextual memory impairment from day 7, peaking at day 14 and partially recovering by day 28, without motor deficits. These cognitive changes were accompanied by sustained peripheral inflammation, excessive hippocampal microglial activation, and downregulation of synaptic plasticity-related proteins.

Conclusion: Bone fracture induces persistent systemic and central inflammation, leading to hippocampus-dependent cognitive impairment. Inflammation may represent a key mechanistic link within the bone-brain axis and a potential therapeutic target.

Disclosure: Nothing to disclose.

EPO-0954 | Double trouble: Fungal brain abscess unmasking a lung malignancy

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Background and aims: Ring-enhancing brain lesions are commonly attributed to tuberculomas in tuberculosis-endemic regions, often leading to empirical treatment. The objective of this report is to highlight diagnostic challenges and adverse consequences of misattributing ring-enhancing lesions to tuberculosis when alternative etiologies coexist.

Methods: We describe the clinical presentation, neuroimaging findings, systemic evaluation, microbiological studies, and histopathological confirmation in a patient initially treated for presumed central nervous system tuberculosis and later reassessed at a tertiary care center.

Results: A 48-year-old woman presented with progressive headache followed by focal neurological deficits. She was empirically treated with antitubercular therapy and corticosteroids



FIGURE 1 CT Chest showing left lower lobe lung mass.

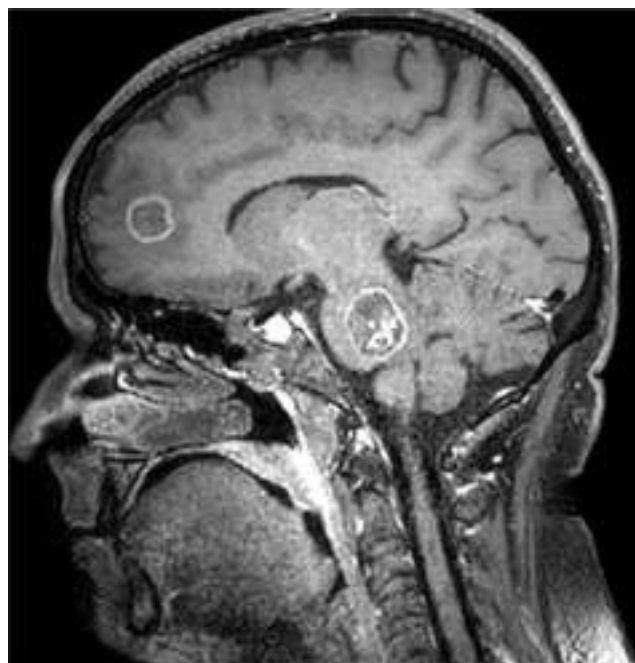


FIGURE 2 MRI Brain - T2 FLAIR sagittal section showing ring enhancing lesion with intracavitary projections in frontal lobe and brainstem.

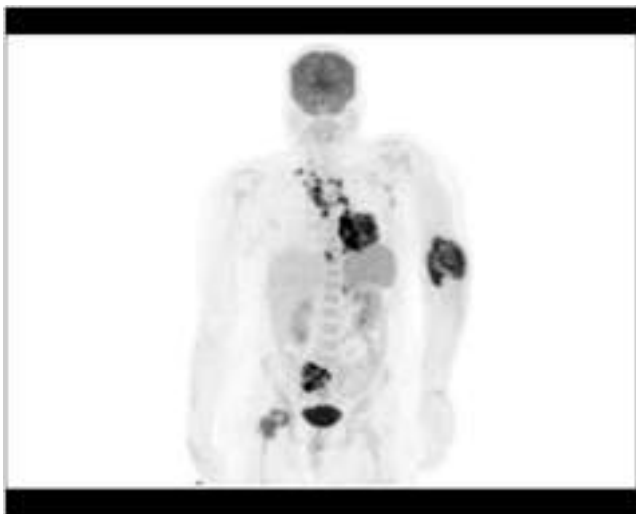


FIGURE 3 PET scan depicting multiple site metastasis.

prior to referral. Magnetic resonance imaging of the brain demonstrated multiple heterogeneous ring-enhancing lesions with diffusion restriction along the wall and intracavitary projections, suggestive of fungal abscess or metastasis. Systemic imaging revealed a left lower lobe lung mass with widespread metastatic disease. Endobronchial ultrasound-guided biopsy confirmed lung adenocarcinoma with epidermal growth factor receptor exon 19 deletion and concomitant *Aspergillus* colonization. Cerebrospinal fluid analysis and infectious workup were negative. Antitubercular therapy and corticosteroids were discontinued, and antifungal therapy with liposomal amphotericin B followed by voriconazole was initiated. Neurological deficits stabilized at three-month follow-up under palliative care.

Conclusion: This case emphasizes the need for cautious evaluation of ring-enhancing brain lesions in tuberculosis-endemic settings. Early tissue diagnosis, multidisciplinary assessment, and careful radiological interpretation are essential to avoid diagnostic delay and treatment-related harm.

Disclosure: Nothing to disclose.

EPO-0955 | Shunting, venous stenting, ONSF, and bariatric surgery for refractory IIH: A systematic review and meta-analysis

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Background and aims: Idiopathic intracranial hypertension (IIH) is characterized by elevated intracranial pressure (>25 mm H₂O) without an identifiable cause, mainly affecting obese

women of reproductive age. Patients present with headaches, papilledema, tinnitus, and visual disturbances, with potential vision loss. First-line treatment includes medical therapy and lifestyle modification, but optimal surgical management for medically refractory cases remains debated

Methods: A systematic review and meta-analysis were conducted. PubMed, Web of Science, Cochrane Library, and Scopus were searched until March 2024. Studies evaluating surgical interventions for IIH, including cerebrospinal fluid (CSF) diversion, dural venous sinus stenting (VSS), optic nerve sheath fenestration (ONSF), and bariatric surgery, were included. Data on clinical outcomes, visual parameters, complications, and re-intervention rates were pooled.

Results: A total of 119 studies were included: 2,561 CSF-diversion procedures, 888 VSS placements, 1,189 ONSF procedures, and 249 bariatric surgeries. Improvement rates for headaches were 38%, 47%, 36%, and 14%; papilledema 39%, 25%, 10%, and 14%; tinnitus 27%, 27%, and 14%; and visual field defects 32%, 43%, 33%, and 72% for CSF diversion, VSS, ONSF, and bariatric surgery, respectively. Visual acuity improved most with bariatric surgery (33%, 57%, 31%, and 71%). Bariatric surgery had the lowest rates of subsequent procedures (0%) and lowest minor and major complications (1% each)

Conclusion: Dural venous sinus stenting and bariatric surgery showed favorable clinical and visual outcomes with lower complication and reintervention rates than traditional surgical approaches. These interventions may represent among the most effective options for medically refractory IIH.

Disclosure: Nothing to disclose.

EPO-0956 | Brain health awareness among solid organ transplant candidates and recipients: A single centre survey

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Background and aims: Solid organ transplant candidates and recipients are a highly vulnerable population for brain disorders variably alongside their transplant history. This population would greatly benefit from brain health programs. In order to set up brain health multidimensional plans targeting patients affected with peripheral organ disease, including solid organ transplant candidates and recipients, we explored their view on brain health

Methods: This study employed a descriptive qualitative methodology to review the answers received through a pencil and paper survey conducted in a single surgical centre. Gender, age, and clinical status differences in brain health awareness were included in our analysis.

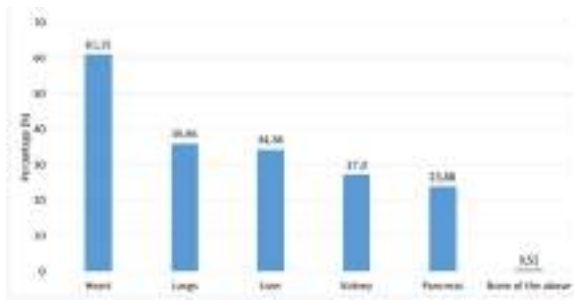


FIGURE 1 Peripheal organs influencing brain health on the view of the survey's participants.

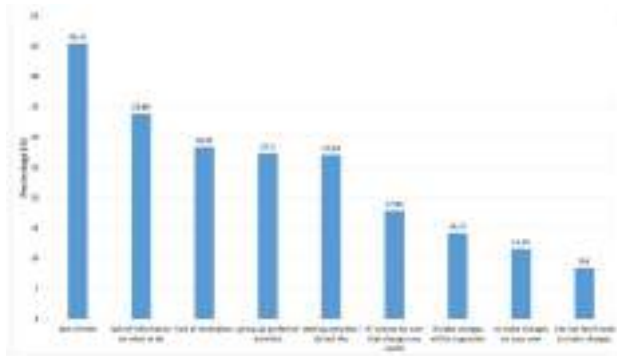


FIGURE 1 Barriers to make changes in lifestyle.

Results: A little over half of our respondents, above all women, reported always or often thinking about their brain health. Just over half of our patients exercise regularly and perform cognitively stimulating activities, with this figure being even lower for older patients and men. There is low health literacy on neurology and brain diseases. We have found full recognition that peripheal organ functioning, above all heart, may have an impact on brain health. Lack of time, lack of information on what to do, and lack of motivation emerged as main barriers to enforcing lifestyle changes. Most of our respondents were willing to undergo a cognitive screening test and receive educational information on brain health.

Conclusion: Our survey provided insights on brain health topic previously unexplored among patients affected by non-neurological disorders. The gathered results will be used to implement multidomain interventional brain health plans for transplant candidates and recipients.

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Background and aims: Cognitive impairment (CI) is common in chronic kidney disease (CKD) above all in end-stage phases as for kidney transplant (KTx) candidates. The pattern and etiology of CKD-related CI are not well understood, mainly related to mechanisms involving pathogenetic pathways typical of Alzheimer Disease (AD). The aim of this study was to assess the association between cognitive dysfunction and AD-related blood-based biomarkers (BBBs) profile in KTx candidates.

Methods: 119 end-stage CKD patients underwent a cognitive assessment through the Montreal Cognitive Assessment (MoCA) test, Trail Making Test (TMT) A and B and the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS). Enrolled participants also underwent Aβ40, Aβ42 and p-tau-217 assessment

Results: demographic and clinical features of our cohort are reported in Table 1 According to equivalent scores of MoCA, 18.5% of our CKD patients resulted cognitively impaired. RBANS detected a greater number of cognitively impaired patients (63.9% with a mild and 12.6% with severe impairment) when a stratification based on severity was performed. Table 2 shows the number and the type of RBANS pathological domains. Plasma Aβ40,

TABLE 1 Baseline characteristics of study participants overall and by cognitive impairment according to MoCA and RBANS.

Characteristic	Overall		MoCA < 26		MoCA ≥ 26	
	n	%	n	%	n	%
Demographics and Clinical						
Age (mean ± SD)	70.0 ± 10.0		70.0 ± 10.0		70.0 ± 10.0	
Gender (n)	119		119		119	
Male	65 (54.6%)		65 (54.6%)		65 (54.6%)	
Female	54 (45.4%)		54 (45.4%)		54 (45.4%)	
Education (years)	7.5 ± 3.5		7.5 ± 3.5		7.5 ± 3.5	
Time from CKD diagnosis (years)	10.0 ± 5.0		10.0 ± 5.0		10.0 ± 5.0	
Time from dialysis (years)	5.0 ± 3.0		5.0 ± 3.0		5.0 ± 3.0	
Time from transplant (years)	2.0 ± 1.0		2.0 ± 1.0		2.0 ± 1.0	
Time from transplant (months)	24.0 ± 12.0		24.0 ± 12.0		24.0 ± 12.0	
Time from transplant (days)	720.0 ± 360.0		720.0 ± 360.0		720.0 ± 360.0	
Time from transplant (weeks)	16.0 ± 8.0		16.0 ± 8.0		16.0 ± 8.0	
Time from transplant (months)	4.0 ± 2.0		4.0 ± 2.0		4.0 ± 2.0	
Time from transplant (years)	0.3 ± 0.2		0.3 ± 0.2		0.3 ± 0.2	
Time from transplant (decades)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (centuries)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (millennia)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (billions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (trillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (quadrillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (quintillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (sextillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (septillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (octillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (nonillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (decillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (hundredillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (thousandillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (millionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (billionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (trillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (quadrillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (quintillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (sextillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (septillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (octillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (nonillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (decillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (hundredillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (thousandillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (millionillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (billionillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (trillionillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (quadrillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (quintillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (sextillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (septillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (decillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (hundredillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (thousandillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (millionillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (trillionillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (septillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (quadrillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (sextillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (hundredillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (thousandillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (millionillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (billionillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (trillionillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (quadrillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (quintillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (sextillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (septillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (octillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (nonillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (hundredillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (billionillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (sextillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (septillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (octillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (nonillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (hundredillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (septillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (thousandillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (thousandillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (millionillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (billionillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
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Time from transplant (quintillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (sextillionillions of years)	0.0 ± 0.0		0.0 ± 0.0		0.0 ± 0.0	
Time from transplant (septillionillions of years)	0.0 ± 0.0		0.			

TABLE 2 Frequencies of number and type of RBANS pathological domains.

N of impaired domains	Total number of patients	Immediate Memory	Visuospatial/Executive abilities	Language	Attention	Delayed memory
0	11 (9.2 %)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
1	20 (16.8 %)	0 (0%)	0 (0%)	8 (40%)	2 (10%)	4 (20%)
2	22 (18.5 %)	16 (72.7 %)	3 (13.6 %)	11 (50 %)	9 (40.9 %)	5 (22.7 %)
3	24 (19.8 %)	13 (58.5 %)	5 (21.5 %)	21 (88.8 %)	24 (100 %)	7 (29.2 %)
4	11 (9.2 %)	29 (100 %)	9 (29 %)	27 (87.1 %)	29 (100 %)	30 (100 %)
5	9 (7.6 %)	9 (100 %)	9 (100 %)	9 (100 %)	9 (100 %)	9 (100 %)

TABLE 3 Biomarkers distribution by cognitive impairment according to RBANS and MoCA.

Biomarkers	RBANS - Cognitive impairment			MoCA - Cognitive impairment		
	No	Yes	P-value	No	Yes	P-value
Aβ42 (pg/ml, median [IQR])	402 (348 - 452)	461 (191 - 209)	0.2229	405 (358 - 446)	462 (241 - 496)	0.4922
Aβ42 (pg/ml, median [IQR])	150 (88 - 129)	133 (116 - 125)	0.1489	111 (88 - 126)	111 (88 - 125)	0.7146
Aβ42/ACβ42 (ratio, median [IQR])	0.1113 (0.0817 - 0.1312)	0.1102 (0.0808 - 0.1307)	0.9887	0.1113 (0.0817 - 0.1312)	0.1102 (0.0808 - 0.1307)	0.9194
p-tau-217 (pg/ml, median [IQR])	0.19 (0.16 - 0.49)	0.09 (0.24 - 0.94)	0.2007	0.18 (0.08 - 0.91)	0.18 (0.11 - 0.48)	0.8810

Aβ42 and p-tau-217 did not showed a significant association with CI as defined by either MoCA or RBANS. However, higher values for these biomarkers were found in cognitively impaired patients (Table 3).

Conclusion: In this exploratory cross-sectional study AD-related BBBs did not reliably discriminate cognitively impaired from unimpaired patients in a cohort of end-stage CKD patients, highlighting the strong influence of alternative pathways beyond amyloid pathology.

Disclosure: This work was supported by the Italian Ministry of University and Research under the National Recovery and Resilience Plan (NRRP), project HEAL ITALIA — Health Extended Alliance for Innovative Therapies, Advanced Lab research, and Integrated Approaches of Precision Medicine (Project code PE0000019, CUP B73D22000610004).

Tuesday, June 30 2026

Ageing and Dementia 5

EPO-0958 | Cataract surgery, cognitive trajectories, and blood–brain barrier integrity in ageing: A multimodal longitudinal study

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Background and aims: Sensory impairment is a modifiable contributor to cognitive decline and dementia risk. Cataracts cause progressive visual deprivation and increased perceptual effort, potentially accelerating neurocognitive and neurovascular ageing. Cataract surgery provides a human model of sensory restoration without direct brain intervention. This study examined whether cataract-related visual impairment and its correction are associated with changes in cognition, blood–brain barrier (BBB) integrity, brain structure, and brain-age trajectories in older adults.

Methods: 18 older adults undergoing bilateral cataract surgery were assessed at baseline, 3-months, and 9-months post-surgery. Each visit included assessments of visual function, cognition, neuroimaging, and blood sampling. Cognition was evaluated using the Repeatable Battery for Assessment of Neuropsychological Status-Update, with mood and anxiety monitored for affective confounding. Neuroimaging included dynamic contrast-enhanced MRI to quantify whole-brain BBB permeability and structural imaging for volumetric, cortical thickness, and brain-age analyses. Serum samples were collected for proteomic profiling.

Results: Cataract surgery induced significant visual improvement. Cognitive performance improved across domains, with greatest gains in immediate and delayed memory, independent of affective changes. BBB permeability decreased significantly by 9-months, while structural brain measures remained stable. Brain-age modelling demonstrated slowed ageing trajectories relative to chronological age. Proteomic analysis revealed modest changes in proteins related to cellular and metabolic regulation, without inflammatory activation or altered serum-BBB markers.

Conclusion: Visual restoration after cataract surgery was associated with improved cognition, reduced BBB permeability, and decelerated brain ageing, without structural brain change. These findings support sensory integrity as contributor to cognitive and neurovascular resilience and suggest cataract surgery may modify brain ageing trajectories.

Disclosure: Nothing to disclose.

EPO-0959 | Anti-amyloid drugs for patients with idiopathic normal pressure hydrocephalus and positive Alzheimer's disease biomarkers?

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Background and aims: Anti-amyloid therapy (AAT) has recently been approved for Alzheimer's disease (AD). Idiopathic normal pressure hydrocephalus (iNPH) frequently co-occurs with AD, yet the presence of degenerative comorbidities is considered an exclusion criterion for anti-amyloid therapy. Given the potential reversibility of iNPH and its frequent association with AD, a targeted evaluation of these patients is warranted. This study aimed to assess the prevalence and characteristics of iNPH patients with AD-positive biomarkers potentially eligible for (AAT).

Methods: We retrospectively identified iNPH patients according to international diagnostic criteria. Demographic, clinical, cerebrospinal fluid (CSF), and MRI data were collected. AD positivity was defined as A+T+, and eligibility for AAT was evaluated based on established exclusion criteria.

Results: Eighty-one iNPH patients (mean age 78.9 ± 5.1 years) referred from the Neurosurgery Service to the Leenaards

Memory Center (Lausanne University Hospital, Switzerland) between May 2019 and December 2023 were included. Eleven patients (13.6%) showed AD-positive biomarkers, and seven (8.6%) met eligibility criteria for AAT. Among ineligible patients, exclusion was due to >4 cerebral microbleeds on MRI (18%), Clinical Dementia Rating (CDR) >1 (9%), or active anticoagulant treatment (9%). No significant differences in clinical or radiological features were observed between eligible and ineligible patients.

Disclosure: Funding declaration: This study was supported by a joint grant of the Lausanne University Hospital Foundation and the Private Foundation of the Geneva University Hospital (Project #ROMENS 22). GB received fundings from Novartis and the UNIL Faculty of Biology and Medicine. GA received fundings from the Faculty of Biology and Medicine of the University of Lausanne, the Swiss National Science Foundation (#214855), the Solis Foundation, the Empiris Foundation and the Marina Cuennet-Mauvernay Foundation.

	100% AD positive monoclonal IgG1a n=17	100% AD positive/negative non-IgG1a n=14	p-value
Age (mean±SD)	59.6±3.4	59.8±3.8	990
Female (%/N)	10(58.8%)	10(71.4%)	877
Duration of disease (years)	24.0±3.0	48.9±9.9	292
Hydroxy-AP (nmol/L)	18.2±10.0	12.0±13.0	465
Alkaline phosphatase (%/N)	1.0±0.894	0.8±0.789	879
25(OH) vitamin D (nmol/L)	35.3±5.5	28.8±6.5	288
CRP	1.8±0.0	5.5±0.5	568
CRP<3	36±9%	25.4±7%	809
CRP>3	7.0±0	7.5±0	899
CRP<3	3.0±0	3.8±0	975
CRP>3	7.0±0	3.8±0	237
Hydroxy-AP	1.0±0	1.2±0	468
Alkaline phosphatase	0.0±0	0.8±0	288
25(OH) vitamin D	2.0±0	0.8±0	879
CRP<3	0.0±0	0.8±0	290
CRP>3	1.0±0	1.8±0	790

EPO-0960 | Multimodal characterization of subjective cognitive decline: Cognitive profiles, Alzheimer's disease blood biomarkers and brain connectivity

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Background and aims: Subjective cognitive decline (SCD) may represent a preclinical stage of Alzheimer's disease (AD), but its trajectory is highly heterogeneous. We compared cognitive profiles, AD blood biomarkers, and brain connectivity in SCD, amnesic mild cognitive impairment (aMCI) and healthy controls (HC).

Methods: Cognitive performance was assessed using the Italian Uniform Data Set Neuropsychological Battery, and metacognition (cognitive insight) with computerized tasks estimating metacognitive efficiency. Plasma p-tau217, GFAP, and NfL were measured using the Fujirebio Lumipulse platform ($n = 79$), and resting-state functional 3T MRI data were analyzed with independent component analysis ($n = 51$). Group comparisons and correlation analyses were performed.

Results: We enrolled 106 participants (36 HC, 39 SCD, 31 aMCI; age 55–85 years). Compared with HC, SCD showed lower global cognition ($p=0.008$) and metamemory efficiency ($p=0.002$). GFAP was increased in SCD ($p=0.01$) and aMCI ($p=0.001$)

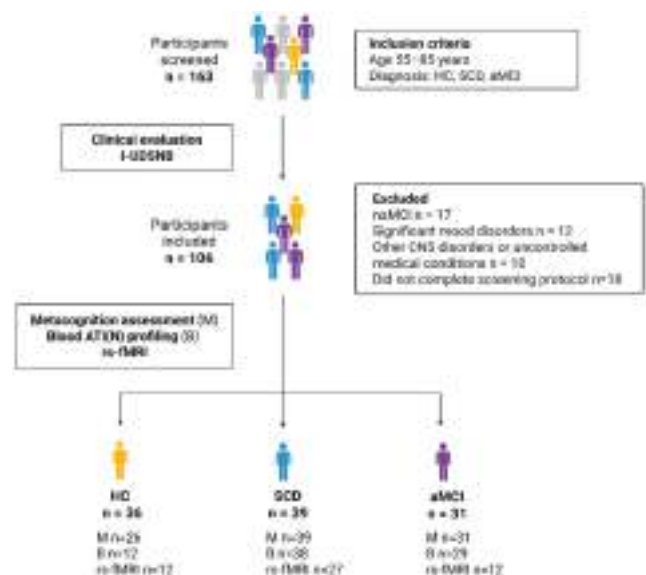


FIGURE 1 Study flowchart. Abbreviations: I-UDSNB, Uniform Data Set Neuropsychological Test Battery, Italian adaptation; ATI (N), amyloid, tau, neuroinflammation, neurodegeneration; rs-fMRI, resting-state functional MRI.

TABLE 1 Participant characteristics. Abbreviations: aMCI, amnesic mild cognitive impairment; DAN, dorsal attention network; DMN, default mode network; FPN, frontoparietal network; HC, healthy controls; SCD, subjective cognitive decline; VAN, ventral att.

	HC (n=58)	SCD (n=55)	aMCI (n=51)	p*	Post hoc test
Age, years, mean (SD)	68.1 (± 9.2)	68.5 (± 9.2)	73.9 (± 9.9)	0.001*	HC > SCD > aMCI
Female, n (%)	11 (19%)	21 (38%)	16 (31%)	0.07	
Education, years, mean (SD)	16.7 (± 1.6)	11.8 (± 1.7)	11.7 (± 1.2)	0.001*	HC > SCD > aMCI
Chastain Comorbidity Index, mean (SD)	0.3 (± 1.6)	0.4 (± 1.3)	4.1 (± 2.4)	0.46	
Braak neuropathology, mean (SD)		0.2 (± 0.4)	1.1 (± 0.3)	0.76	
MMSE, mean (SD)	28.5 (± 0.7)	28.5 (± 0.7)	26.3 (± 0.7)	<0.001*	HC > SCD > aMCI
Global cognition, z-score, mean (SD)	0.07 (± 0.7)	0.29 (± 0.3)	-0.87 (± 1.1)	<0.001*	HC > SCD > aMCI
Metacognitive efficiency score, mean (SD)	1.1 (± 0.4)	0.8 (± 0.4)	1.27 (± 0.3)	0.002*	HC > aMCI > SCD
Neurodegeneration efficiency score, mean (SD)	0.7 (± 0.4)	0.8 (± 0.4)	0.4 (± 0.3)	0.033	
NfL, pg/mL, mean (SD)	22.5 (± 1.8)	10.5 (± 0.8)	17.3 (± 0.9)	0.49	
GFAP, pg/mL, mean (SD)	19.8 (± 11.5)	10.2 (± 11.2)	41.9 (± 39.0)	<0.001*	SCD > aMCI > HC
p-Tau217, pg/mL, mean (SD)	0.08 (± 0.04)	0.18 (± 0.1)	0.22 (± 0.2)	0.002*	aMCI > HC > SCD
DMN connectivity, z-score, mean (SD)	4.0 (± 1.2)	4.0 (± 0.9)	4.0 (± 1.1)	0.71	
DAN connectivity, z-score, mean (SD)	4.07 (± 0.5)	4.04 (± 1.3)	4.1 (± 1.1)	0.47	
FPN connectivity, z-score, mean (SD)	4.06 (± 1.3)	3.9 (± 1.1)	4.2 (± 1.1)	0.17	
VAN connectivity, z-score, mean (SD)	4.0 (± 1.2)	4.15 (± 1.3)	3.9 (± 1.2)	0.38	

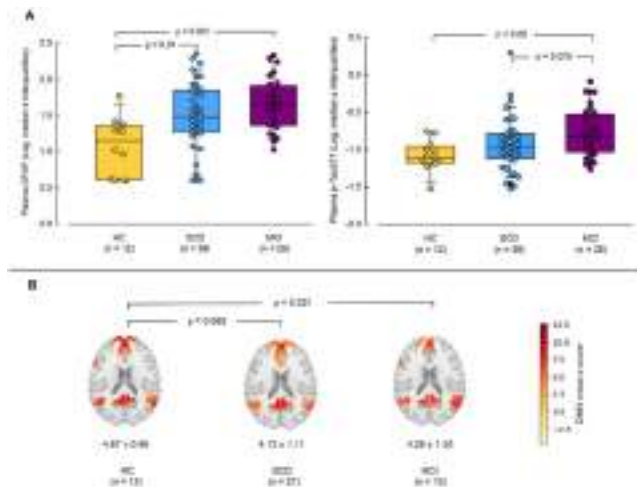


FIGURE 2 Plasma GFAP and p-Tau217 levels (A) and Default Mode Network connectivity (B) across diagnostic groups.

versus HC and remained significant after adjustment for age and education ($p=0.0039$). P-tau217 was higher in aMCI than HC ($p=0.01$), with a trend versus SCD ($p=0.078$), while NfL did not differ between groups. Default mode network connectivity was reduced in aMCI ($p=0.021$) and showed a trend toward reduction in SCD ($p=0.065$) compared with HC. Across the sample, global cognition correlated negatively with p-tau217 ($r=-0.34$, $p=0.007$), GFAP ($r=-0.23$, $p=0.05$), and NfL ($r=-0.25$, $p=0.04$). Nominal associations were observed between meta-cognitive efficiency and default mode and dorsal attention network connectivity ($\rho=0.35$; $p\approx 0.02$).

Conclusion: SCD showed subtle cognitive deficits, increased glial reactivity and connectivity changes, supporting its place along the AD continuum and the value of integrating multimodal biomarkers for early disease characterization.

Disclosure: Nothing to disclose.

EPO-0961 | Cerebrospinal fluid CCL25 as a biomarker for Alzheimer's disease: Associations with pathology, neurodegeneration, and cognitive decline

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Background and aims: Neuroinflammation plays a crucial role in Alzheimer's disease (AD) pathogenesis. We investigated the relationship between cerebrospinal fluid (CSF) C-C chemokine ligand 25 (CCL25), an inflammatory regulator, and AD pathology and progression.

Methods: We analyzed CSF CCL25, AD biomarkers (CSF β -amyloid [$A\beta$]42, phosphorylated tau [pTau]181, amyloid positron emission tomography [PET]), postmortem neuropathology, magnetic resonance imaging-based neurodegeneration, and cognitive function from 703 participants in the Alzheimer's Disease Neuroimaging Initiative cohort.

Results: We found that elevated CSF CCL25 levels were associated with cognitive impairment, abnormal $A\beta$ and tau pathology, greater brain atrophy, and worse cognitive performance (all $p<0.05$). CSF CCL25 exhibited nonlinear relationships with $A\beta$ and tau pathology, reaching a plateau as AD pathology increased. CSF CCL25 showed acceptable diagnostic accuracy in distinguishing amyloid-positive/negative ($A_{\pm}$) and tau-positive/negative ($T_{\pm}$) participants (area under the curve [AUC]=0.71–0.77) and autopsy-confirmed AD cases (AUC=0.77), with optimal performance in differentiating $A_{+}T_{+}$ from $A_{-}T_{-}$ participants (AUC=0.82–0.85 with age and sex adjustment). Longitudinally, higher baseline CSF CCL25 predicted accelerated amyloid accumulation, hippocampal atrophy, and cognitive decline. Mediation analyses revealed that CCL25 partially mediated associations between $A\beta$ pathology and tau pathology (mediating effect: 54.5%), neurodegeneration (18.2%), and cognitive decline (7.4%). Among 37 CSF CCL and CXCL chemokines examined, 28 were associated with at least one AD-related outcome, with CCL25 demonstrating the strongest associations

Conclusion: These findings suggest that CSF CCL25 is involved in early AD pathological progression and may serve as an inflammatory biomarker for diagnosis and monitoring disease progression in AD.

Disclosure: Nothing to disclose.

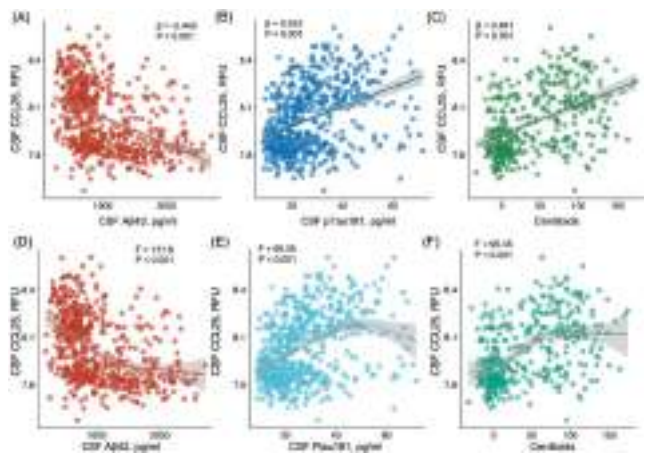


FIGURE 1 Association between CSF CCL25 and AD core pathology.

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Background and aims: Global aging drives increased dementia prevalence. While early detection is crucial for better prognosis and quality of life, diagnosis is often delayed because early symptoms are subtle. Since no widely available AI algorithm reliably identifies early dementia signs from digital behavioral patterns, our objective was to develop and validate such an AI-based tool for timely diagnosis and treatment.

Methods: We utilized the PreDEM system, an AI-based platform, for this investigation. Data was collected from 4,334 participants between September 2021 and March 2025, during which 171,577 cognitive tests were completed, resulting in 6,049,916 data points. A unified, normalized evaluation system was established for objective comparison across tests. The AI algorithm analyzed participants' digital behavioral patterns to identify early signs of dementia.

Results: Participants were categorized into "potentially treated" and "potentially healthy" groups. Analysis, including density function comparison and cluster analysis, showed clear differences between the groups. Within the affected group, a lower-performing subgroup was identified and subsequent neurological/neuropsychological examinations confirmed cognitive impairment. In the higher-performing affected subgroup, cognitive dysfunction was often linked to organic causes. Treating these underlying organic causes, combined with regular use of the PreDEM platform, led to significant cognitive improvement in these participants.

Conclusion: The study's platform is a promising new tool for the early detection and prevention of dementia. During pilot studies, the AI-based system reliably identified dementia patients, significantly surpassing the accuracy of traditional diagnostic methods. This method is therefore suitable for detecting the first, often unnoticed signs of dementia through AI-processed risk analysis.

Disclosure: Nothing to disclose.

EPO-0963 | When hearing loss is the first clue: An atypical onset of Creutzfeldt-Jakob disease with an isolated thalamic lesion

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Background and aims: Creutzfeldt-Jakob disease (CJD) is a rapidly progressive prion encephalopathy typically characterized by progressive dementia, myoclonus and ataxia. However, early presentations may be misleading, as atypical symptoms such as hearing loss and non-suggestive neuroimaging can pose a diagnostic challenge.

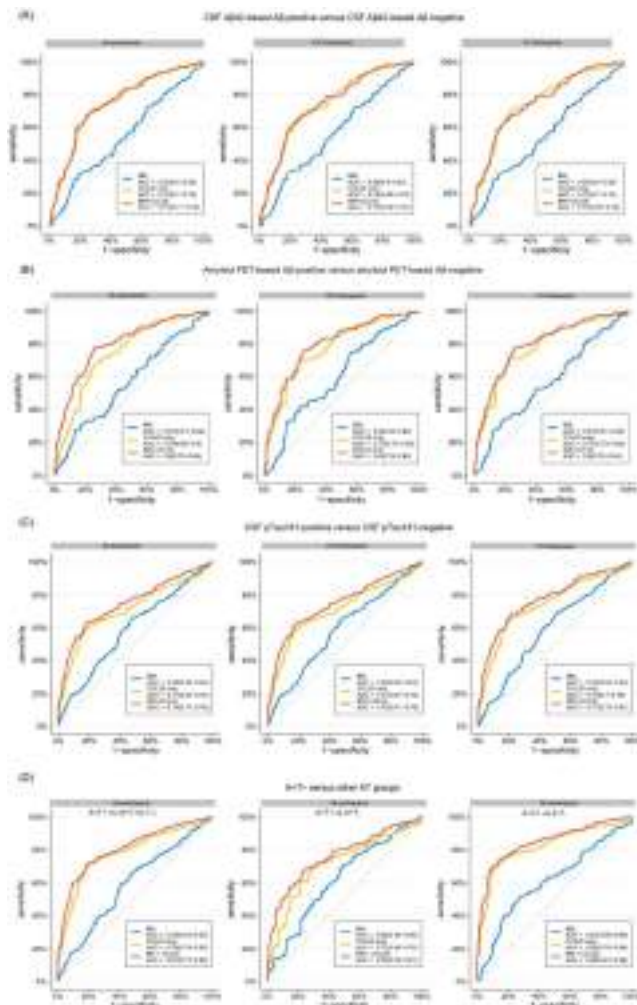


FIGURE 2 Accuracy of CSF CCL25 in predicting AD pathology.

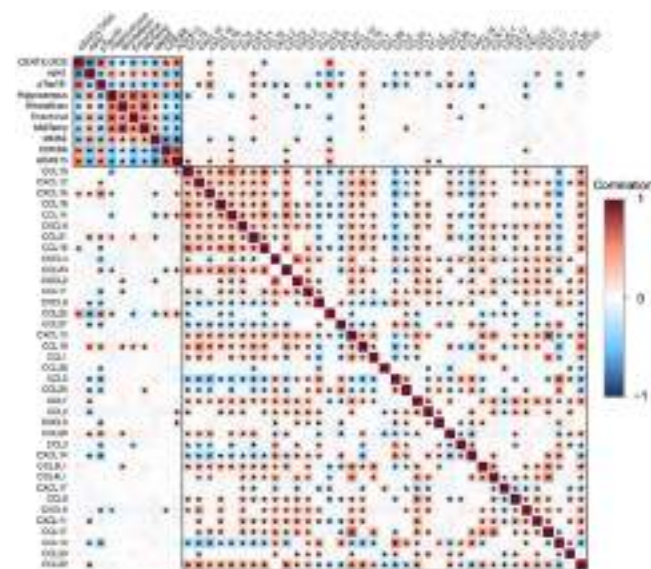


FIGURE 3 Correlation between chemokines and AD biomarkers.

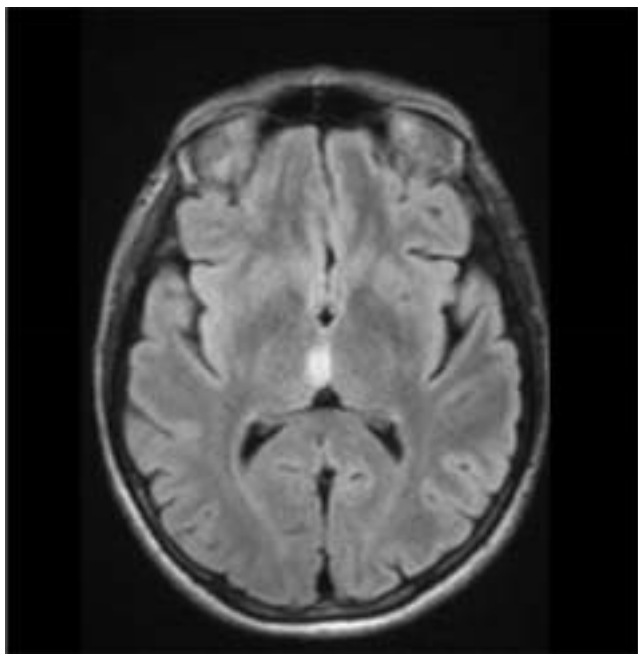


FIGURE 1 Axial T2-FLAIR MRI showing an isolated hyperintense lesion in the right thalamus.

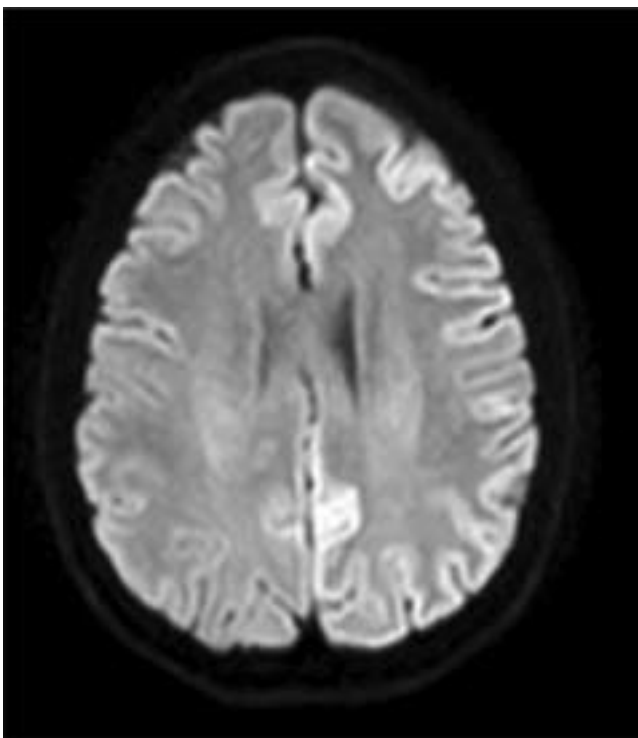


FIGURE 2 Axial diffusion-weighted MRI showing cortical ribboning in the frontoparietal regions.

Methods: We report the case of a 54-year-old woman with dyslipidaemia, ulcerative proctitis and chronic thrombocytopenia. In August 2025, she developed acute unilateral hearing loss, progressing to bilateral involvement within days, unresponsive to topical corticosteroids. In September 2025, she was assessed by otorhinolaryngology (ENT); audiometry showed normal hearing thresholds with impaired speech discrimination and

topical and oral corticosteroids were prescribed without improvement. In October 2025, following language impairment, she presented to the emergency department for neurological evaluation. Neurological examination revealed scanning dysarthria, mixed aphasia with paraphasias, slow reading with apraxia, nystagmus, myoclonus and a wide-based ataxic gait.

Results: Initial brain magnetic resonance imaging (MRI) in September 2025 showed an isolated unilateral hyperintense lesion in the right thalamus (T2/FLAIR) without diffusion restriction. Subsequent MRI in October 2025 demonstrated the evolution of classic “cortical ribboning” in frontoparietal regions and ipsilateral caudate nucleus involvement on diffusion-weighted imaging (DWI). Cerebrospinal fluid analysis revealed positive 14-3-3 protein with otherwise normal biochemistry. Electroencephalography showed diffuse encephalopathy with characteristic periodic sharp wave complexes.

Conclusion: This case highlights that CJD can masquerade as a primary ENT pathology. Hearing loss and atypical isolated thalamic lesions do not preclude a prion disease diagnosis. Recognizing these rare phenotypes is crucial for timely diagnosis in the absence of early “classic” imaging markers.

Disclosure: Nothing to disclose.

EPO-0964 | Acute and longitudinal effects of transcranial pulse stimulation on vascular, neuronal, and cognitive function in Alzheimer's models

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Background and aims: Transcranial Pulse Stimulation (TPS) is a non-invasive neuromodulation technique with growing therapeutic relevance for neurodegenerative disease. This work investigated both the acute physiological effects of TPS on cerebrovascular, neuronal, and network-level function, as well as the longitudinal impact of repeated TPS on Alzheimer's disease (AD) pathology and cognition.

Methods: Acute experiments were conducted in CD1, GCaMP-expressing, 5xFAD, and 3xTg-AD mice using multimodal imaging: real-time optoacoustic tomography, contrast-enhanced MRI, wide-field calcium imaging, c-Fos immunohistochemistry, and resting-state fMRI. Longitudinal study followed a clinical-analogous TPS protocol (six sessions over two weeks) to 3xTg-AD mice with sham controls. BBB integrity, amyloid and tau pathology (targeted AOI987 imaging, immunohistochemistry), cognition (Novel Object Recognition, Fear Conditioning), and proteomic signatures were assessed.

Results: Acute TPS induced robust, energy-dependent increases in cortical blood volume (~28–50%), with AD mice showing reduced vascular responsiveness consistent with vascular amyloidosis. BBB integrity remained intact across all modalities. TPS evoked strong neuronal activation, including immediate

calcium influxes and ~2-fold increases in c-Fos expression, and transiently reorganized functional connectivity within hypothalamic–hippocampal–limbic circuits. Longitudinal TPS caused no BBB disruption and produced modest reductions in amyloid burden and attenuated tau accumulation. Behaviorally, TPS improved short-term object recognition and enhanced forgetting of remote fear memory in 3xTg-AD females. Proteomics revealed upregulation of pathways related to calcium handling, energy metabolism, ion transport, and neurotransmission.

Conclusion: TPS safely modulates vascular, neuronal, and network dynamics while mitigating aspects of AD pathology and improving cognitive and molecular outcomes. These findings support TPS as a mechanistically grounded therapeutic strategy for neurodegenerative disease.

Disclosure: Nothing to disclose.

EPO-0965 | **Whole-brain DTI-ALPS: A novel voxel-based biomarker for white matter dysfunction in Alzheimer's and Parkinson's disease**

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Background and aims: The glymphatic system's role in neurodegenerative waste clearance is established, but non-invasive assessment remains challenging. This study compares the standard DTI-ALPS method with a novel whole-brain voxel-based DTI-ALPS approach to quantify perivascular flow in Alzheimer's (AD), Parkinson's disease (PD) and Dementia with Lewy Bodies (DLB).

Methods: Retrospective analysis included 25 PD patients with dementia (PDD), 58 with mild cognitive impairment (PD-MCI), 50 cognitively intact PD (PD-NC), 77 AD and 13 DLB patients, and 30 healthy controls (HC) (Table 1). Cognitive impairment was assessed by Mattis Dementia Rating Scale (DRS-2). DTI-ALPS was calculated using Frangi-filter-based perivascular space (PVS) segmentation on high-resolution T2-weighted MRI. A PVS orientation template enabled voxel-wise diffusivity assessment along/perpendicular to PVS. Mean whole-brain

TABLE 1 Demographics.

	HC (N=30)	PD-NC (N=50)	PD-MCI (N=58)	PDD (N=25)	DLB (N=13)	AD (N=77)
Age (mean)	63.4 (8.19)	62.5 (8.66)	63.5 (9.13)	68.2 (6.18)	69.9 (9.25)	67.8 (10.0)
Sex						
Female/Male	16/14	32/18	30/28	16/9	5/8	38/39
Hoehn/Yahr						
Median [Range]		2.00 [1.00-3.00]	3.00 [1.00-4.00]	3.00 [2.00-3.00]		
MoCA-LPDAS-III (OFF)						
Mean (SD)		26.3 (6.7)	46.2 (16.6)	32.9 (17.6)		
DRS-2						
Mean (SD)		142 (1.38)	136 (2.23)	128 (3.59)		
MMSSE						
Mean (SD)						23.9 (4.79)

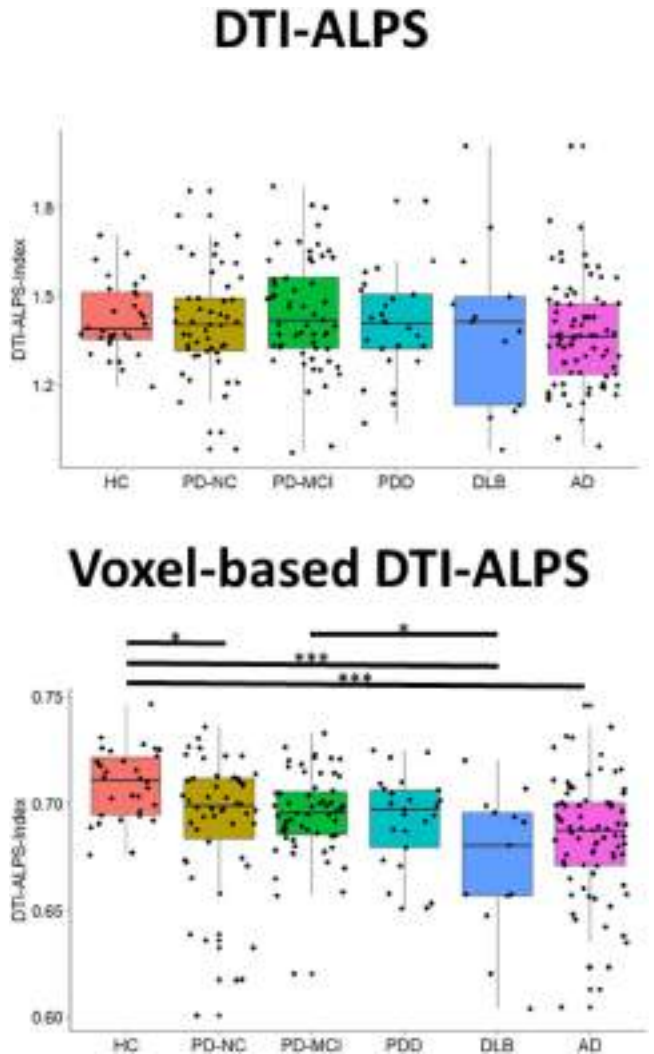


FIGURE 1 DTI-ALPS and voxel-based DTI-ALPS indices across HC, PD-NC, PD-MCI, PDD, DLB, and AD, highlighting group differences and statistical significance.

DTI-ALPS was extracted from white matter (tissue probability > 0.5). Group comparisons and clinical associations were analyzed.

Results: Conventional DTI-ALPS revealed no significant group differences. Voxel-based-DTI-ALPS was significantly lower in PD-NC ($p=0.040$, Cohen's $d=0.635$), DLB ($p<0.001$, Cohen's $d=1.599$) and AD ($p<0.001$, Cohen's $d=0.971$) compared with HC. Furthermore, DTI-ALPS was lower in DLB than PD-MCI ($p=0.026$, Cohen's $d=1.027$), Figure 1. A significant association was observed with initiation ($p=0.003$), but not with DRS-2 total- or subscores ($p>0.05$).

Conclusion: This novel whole-brain voxel-based approach, using a PVS template, demonstrates significant white matter dysfunction in AD, DLB, and cognitively intact PD, with the most pronounced impairment in DLB. The association with initiation deficits highlights its potential as a sensitive biomarker for early white matter dysfunction.

Disclosure: Nothing to disclose.

EPO-0966 | Development of the AO spinal metastasis staging (SMS) referral tool: An international multidisciplinary expert panel and survey study

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Background and aims: Spinal metastases progress from asymptomatic disease to debilitating pain, instability, and neurological deficits. Although timely recognition is essential, referral delays are common because patients often first present to non-spine clinicians and red flags rarely expedite referral. Existing

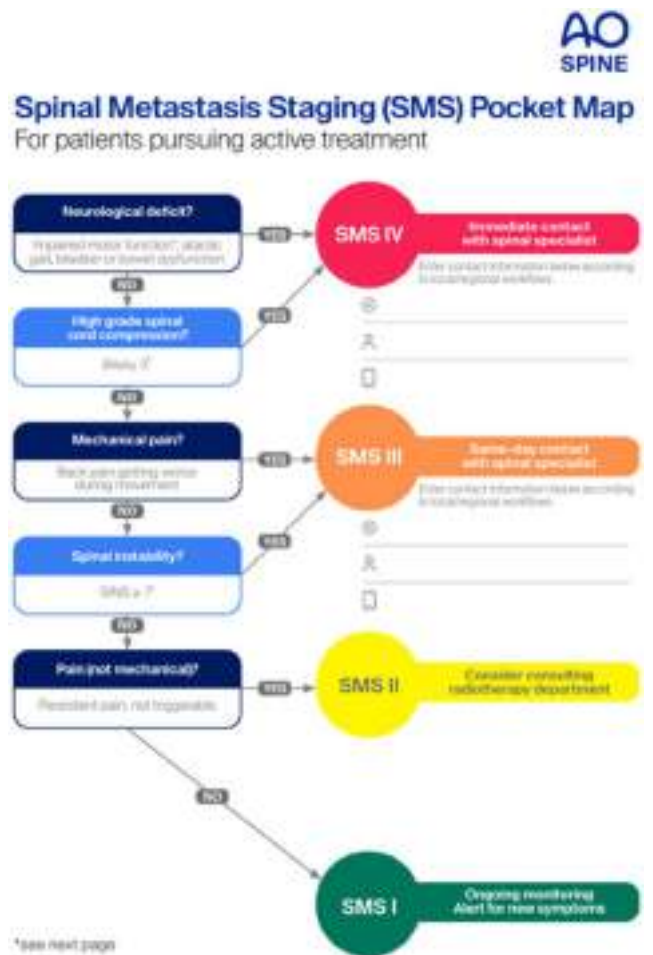


FIGURE 1 Front page of the SMS pocket map with the referral algorithm for patients with spinal metastases.

American Spinal Injury Association (ASIA) Impairment Scale

A The impairment is complete. There is no motor or sensory function below the level of injury.

B The impairment is incomplete. Sensory function, but not motor function, is preserved below the neurologic level (the key is preserved above the level of injury) and some sensation is preserved in the sacral segments S4 and S5.

C The impairment is incomplete. Motor function is preserved below the neurologic level and more than half of the key muscles below the neurologic level have a strength grade of 3 or more.

D The impairment is incomplete. Motor function is preserved below the neurologic level and at least half of the key muscles below the neurologic level have a strength grade of 3 or more.

E The patient's function is normal. All motor and sensory functions are undisturbed.

Bilsky grading (Best assessed with an MRI)

Grade 0: disc-only disease

Grade 1: spinal extension without cord compression (Bilalov)

Grade 2: spinal cord compression with cerebrospinal fluid (CSF) stubble around the cord

Grade 3: spinal cord compression with no CSF stubble around the cord

Spinal neoplastic instability score (SNS)

Element of SMS	SCORE	Element of SMS	SCORE
Location		Neurological to spinal alignment	
Junctional (cervical-C2, C2-C3, T11-T12)	3	Subluxation/translation present	4
L1-S1	2	De novo deformity (osteolysis/scoliosis)	3
Multiple (cervical-C2, C3-C4, L1-L2, L3-L4)	2	Normal alignment	0
Single (cervical-C2-C3, T11-T12)	1	Vertebral collapse	
Hybrid (C2-C3, T11-T12)	0	> 50% collapse	2
Plan relief with occiput/another plate with movement/loading of the spine		< 50% collapse	1
Yes	1	No collapse with > 50% body involved	0
No	0	More of the above	0
No mechanical pain but not mechanical	1	Postoperative involvement of the spinal elements (facet, pedicle of C7 post fracture or replacement with fusion)	
Plan free lesion	0	Isolated	1
Bone lesion		Unilateral	1
L1-L2	2	None of the above	0
Other (T11/T12)	1		
Blow	0		

NOTE: the total SNS score is the sum of all SNS elements

Remarks

- Neurological function/instability are different, discuss with your specialist who has experience in this field
- If unsure about SNS or Bilsky, please consult your local radiologist
- See QR code for minimum referral information, anatomy, and support with pain types, ASIA, SNS, and Bilsky

FIGURE 2 Back page of the SMS pocket map with explanation of items on the front and a QR link to further educational material.

guidelines target spine surgeons and radiation oncologists, leaving a gap for staging and referral tools for non-spine clinicians. We aimed to develop the Spinal Metastasis Staging (SMS) referral tool.

Methods: We defined the SMS system as four stages following natural progression of most spinal metastases: SMS I, asymptomatic; SMS II, inflammatory pain; SMS III, mechanical pain and/or instability; and SMS IV, neurological deficits and/or high-grade spinal cord compression. To aid clinical use, we translated the SMS stages into a referral algorithm, organized by urgency, presented in the form of a color-coded pocket map. The four stages and pocket map were revised by regional and international multidisciplinary expert panels, including spine and non-spine clinicians, and subsequently evaluated in an international online survey.

Results: Expert panels endorsed the four SMS stages. Among non-spine clinician survey respondents (32/120), 94% found the pocket map easy to understand, 91% judged the format suitable, and 91% expected improved referrals. Overall, 88% indicated at least occasional use, including 61% who would use the pocket map frequently or always.

Conclusion: The SMS referral tool was well received by spine specialists and non-spine clinicians, supporting that it is both expert-driven and user-friendly. A digital version with educational materials is provided for local implementation. Future work will focus on clinical validation to confirm the impact.

Disclosure: Nothing to disclose.

EPO-0967 | **Late transient post-radiotherapy enhancements in patients with high grade brain tumors – between pseudoprogression and radionecrosis: A case series**

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Background and aims: High-grade malignant brain tumors have a poor prognosis despite treatment. Pseudoprogression and radionecrosis are contrast-enhancing phenomena that usually appear before 3 months (post-radiotherapy) in a transient manner or between 3–12 months permanently, respectively. Distinguishing pseudoprogression and radionecrosis from true progression is of extreme importance, with variable treatment and prognostic implications. The aim of this work is to describe treatment related changes on MRI defined as late transient post-radiotherapy enhancements (LTPE) which do not fit the definition of pseudoprogression or radionecrosis and can be misdiagnosed as tumor progression.

Methods: Retrospective description of a case series of 7 patients with malignant high-grade brain tumors who showed late transient post-radiotherapy contrast-enhancing lesions that did not fit the definitions of pseudoprogression or radionecrosis and could be misdiagnosed as progressive disease. Demographic, tumor, treatment and LTPE characteristics (time of appearance and resolution, location), date of tumor progression, and MRI and methionine PET features were collected.

TABLE 1 Data collected from 7 patients. CT, chemotherapy; CV RF, cardiovascular risk factors; LTPE, late transient post-radical enhancements.

Patient	1	2	3	4	5	6	7
Age	45	52	55	51	52	47	55
CR-MR	No	Type 2 glioblastoma, IDHwt	Taken under surgery, IDHwt, anaplastic oligo, 10 to 100% Ki-67	No	No	Hypodense, IDHwt	Hypodense, IDHwt
Tumor	Glioblastoma	Glioblastoma	Glioblastoma	Medulloblastoma grade 4	Glioblastoma	Medulloblastoma grade 4	Medulloblastoma
MGMT status	MGMT methylated	MGMT methylated	MGMT methylated	MGMT methylated	MGMT methylated	MGMT methylated	MGMT methylated
RT status	RT negative	RT negative	RT negative	RT negative	RT negative	RT negative	RT negative
Complete or partial resection	Partial	Complete	Partial	Partial	Complete	Partial	Complete
Mark and use of radiotherapy	Median 10, 1000 Gy	Median 10, 1000 Gy	Median 10, 1000 Gy	Median 10, 1000 Gy	Median 10, 1000 Gy	Median 10, 1000 Gy	Median 10, 1000 Gy
RT Dose	45 Gy in 15 fractions	50 Gy in 15 fractions	50 Gy in 15 fractions	45 Gy in 15 fractions	50 Gy in 15 fractions	50 Gy in 15 fractions	50 Gy in 15 fractions
Concomitant CT	TMZ 75mg/m ²	TMZ 75mg/m ²	TMZ 75mg/m ²	TMZ 75mg/m ²	TMZ 75mg/m ²	TMZ 75mg/m ²	TMZ 75mg/m ²
Adjuvant CT	TMZ 150mg/m ²	TMZ 150mg/m ²	TMZ 150mg/m ²	TMZ 150mg/m ²	TMZ 150mg/m ²	TMZ 150mg/m ²	TMZ 150mg/m ²
Time of appearance LTPE	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)
Time of resolution LTPE	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)
Time of tumor progression	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)	10 months (1000 Gy)
Location of LTPE	Peritumoral	Peritumoral	Peritumoral	Peritumoral	Peritumoral	Peritumoral	Peritumoral

Table 1. Data collected from 7 patients. Age, cardiovascular risk factors, tumor diagnosis, MGMT and MMR status, complete or partial resection, date of start and end of RT, dose of concomitant and adjuvant CT, date of appearance and disappearance of LTPE, and tumor progression, location and previous fields of targeted RT field.

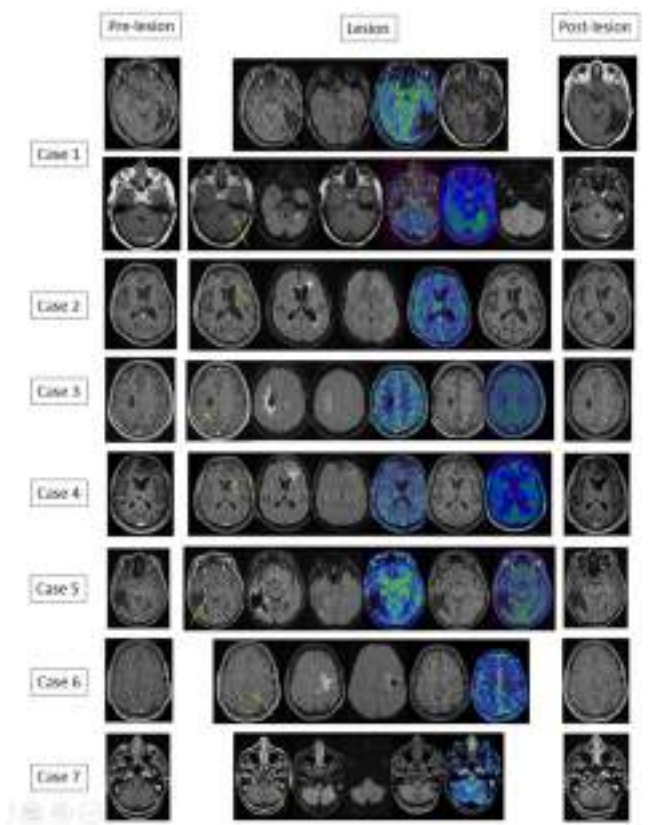


FIGURE 1 Representative MRI/PET images of the seven reported cases before, during, and after LTPE (Late transient post-radical enhancements).

Results: Seven patients aged 47–65 years were included: 4 had glioblastoma, 2 grade 4 IDH-mutated astrocytomas and 1 medulloblastoma. All patients underwent surgery followed by radio-chemotherapy. LTPE were located within the radiotherapy field, appeared between 11–30 months post-radiotherapy, and spontaneously resolved within 4–30 months after onset. Six LTPE showed frontotemporal distribution and four periventricular/ependymary.

Conclusion: The small size, late post-radiotherapy onset, and reversibility of these contrast-enhancing lesions highlight the overlap between LTPE, pseudoprogression and radionecrosis. Further studies are needed to better characterize this phenomenon and establish clear criteria to avoid misdiagnoses of tumor progression.

Disclosure: Nothing to disclose.

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Background and aims: More than half of glioblastoma patients experience epileptic seizures. Epilepsy–glioma interactions are linked to poor prognosis, yet predictors of epilepsy remain unclear. We examined associations with prognosis, as well as predictors of epilepsy and seizure control.

Methods: From a prospective database, 595 glioblastoma IDH-wildtype patients treated with standard therapy from 2016–2024 at Rigshospitalet were included and divided into training (2016–2021) and validation (2022–2024) datasets. Epilepsy diagnosis and medication use were extracted from medical records. As a surrogate marker of seizure control, we used dose burden of anti-seizure medication calculated as prescribed daily dose relative to WHO defined daily dose. Candidate genetic factors comprised pathogenic and likely pathogenic variants from genomic profiling. We used Cox regression and generalized linear models for survival and prediction analysis.

Results: Pre- and postdiagnostic epilepsy (HR: 1.41 [95% CI 1.06–1.87], $p=0.018$ and HR: 3.08 [2.34–4.05], $p<0.0001$, respectively) and higher dose burden at time of tumour diagnosis (HR: 1.06 [1.03–1.10], $p<0.0001$) were associated with poor overall survival. Multifocal disease and absence of RB1 alterations were associated with postdiagnostic epilepsy. Multifocal disease predicted higher dose burden in both pre- and postdiagnostic epilepsy ($p=0.009$ and $p=0.016$). In pre-diagnostic epilepsy, resection predicted lower dose burden than biopsy.

Conclusion: Epilepsy and lack of seizure control negatively impact prognosis in glioblastoma. They can be predicted using clinical and genomic factors. This can support clinical risk stratification and early epilepsy management if validated.

Disclosure: Benedikte Hasselbalch: paid member of an expert panel for Servier.

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Background and aims: In glioblastoma (GBM), fatigue is a widely understudied and may particularly impact patients. Several hypotheses may explain the development of fatigue, including direct effect of the tumor and its treatments. Moreover, concomitant depressive state and dysregulation of the autonomic nervous system may be observed. We postulate that a combined measure of physical and autonomic activities, acquired through a wearable device will provide biomarkers of the clinical and physical evolution.

Methods: We used a wearable device (Embrace+ smartwatch, Empatica) and an electronic diary to monitor patient seizures as well as daily the fatigue using a Visual Analog Scale (VAS). Every 2 months, at time of planned MRI, patients complete questionnaires about medications and validated fatigue and quality of life questionnaires.

Results: As of January 2026, 15/20 patients were enrolled. Daily values of the VAS have been weakly linked standardized fatigue questionnaires. However, findings suggests that patients tend to be less active during days in which they end up feeling more fatigued. Furthermore, machine learning models show promising results regarding prediction of fatigue and disease progression, based on parameters acquired by the wearable device. More results regarding interactions between fatigue and mood, impact of therapies and life expectancy are under investigation.

Conclusion: Our preliminary data suggest that fatigue measured by standardized questionnaires might be anticipated based on the completion of a simple VAS question completed daily by the patient and correlated to continuous activity measures obtained by a wearable device.

Disclosure: Nothing to disclose.

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Background and aims: Oculomotor disorders in oncology patients are usually caused by brainstem, orbital, metastases or meningeal infiltration, and may be the presentation of an occult tumor. Another etiology is paraneoplastic syndromes, such as myasthenia gravis or Eaton-Lambert syndrome (LES), showing variable clinical patterns. We report a case of progressive ophthalmoparesis evolving into One and a Half Syndrome (OAHS) as the initial manifestation of a lung carcinoma.

Methods: A 56-year-old woman, ex-smoker, presented with left ptosis and diplopia with limited bilateral abduction, unresponsive to pyridostigmine and corticosteroids. Diplopia progressively worsened over three weeks without fatigability. Examination only revealed a horizontal OAHS with left ptosis.

Results: TSH, antithyroid antibodies, anti-AChR, anti-MuSK, anti-P/Q-type VGCCs, ANA, onconeural antibodies were negative. Brain MRI and angio-TC: normal. Jitter and repetitive stimulation: normal. CSF: normal. Body CT: pulmonary mass in the right middle lobe. Biopsy: lung adenocarcinoma. High-dose

methylprednisolone followed by tapering and intravenous immunoglobulins produced partial improvement. One month after surgery the patient was asymptomatic. Adjuvant chemotherapy was added.

Conclusion: The absence of structural lesions and resolution after surgery suggest a paraneoplastic mechanism. LES with isolated ophthalmoplegia is rare and may even precede cancer diagnosis, more commonly small-cell lung carcinoma, but exceptionally other subtypes (including adenocarcinoma). Anti-P/Q-type VGCCs can be negative in 15% of LES associated with lung carcinoma, but the absence of neurophysiological abnormalities in our case suggests that progressive ophthalmoparesis may represent an independent paraneoplastic syndrome, already suggested in other cases with exophthalmos and non-infiltrative ophthalmoparesis. This presentation should prompt awareness and thorough search for occult tumors.

Disclosure: Nothing to disclose.

EPO-0971 | Delayed acute motor axonal neuropathy following BCMA-directed CAR-T therapy in relapsed/refractory multiple myeloma

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Background and aims: Chimeric antigen receptor T-cell (CAR-T) therapies targeting B-cell maturation antigen (BCMA) have transformed the treatment of relapsed/refractory multiple myeloma (MM). However, compared with CD19-directed constructs, BCMA-directed CAR-T therapies are associated with a distinct and still incompletely characterized spectrum of neurological complications.

Methods: A patient treated with BCMA-directed CAR-T therapy developed delayed neurological symptoms. Clinical, neurophysiological, neuroimaging, and cerebrospinal fluid (CSF) studies were performed.

Results: A 65-year-old woman with relapsed/refractory IgA kappa MM and high tumor burden received fourth-line BCMA-directed CAR-T therapy (ciltacabtagene autoleucel). On day +25, she developed grade 2 cytokine release syndrome without immune effector cell-associated neurotoxicity syndrome (ICANS). One month after infusion, disease assessment showed a partial response, and she developed a sixth nerve palsy with normal CSF findings, which resolved after corticosteroid treatment. Two months post-infusion, she presented with subacute progressive symmetric tetraparesis causing severe disability, without sensory loss or dysautonomia. Neurological examination revealed global areflexia and marked weakness, followed by encephalopathy. CSF analysis showed marked lymphocytic pleocytosis (685 cells/ μ L; 96% CAR-T cells) and thiamine deficiency, without other abnormalities. Electromyography demonstrated features

consistent with acute motor axonal neuropathy. Brain and spinal MRI excluded central nervous system involvement. Treatment included intravenous immunoglobulins, corticosteroids, thiamine supplementation, and anakinra. Encephalopathy resolved rapidly, but motor recovery was limited. The patient died four months after CAR-T infusion due to respiratory infection.

Conclusion: BCMA-directed CAR-T therapy may cause severe delayed peripheral neurotoxicity, highlighting the need for prolonged multidisciplinary neurological monitoring beyond the acute post-infusion period.

Disclosure: Nothing to disclose.

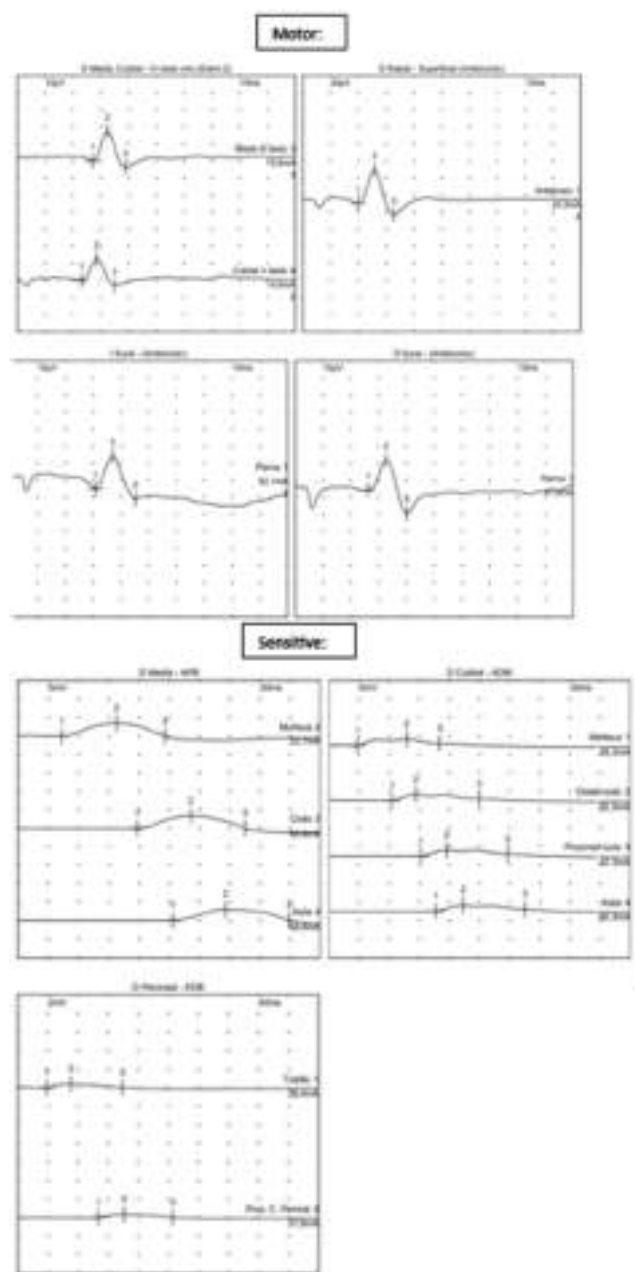


FIGURE 1 EMG findings: Marked reduction in compound motor action potential amplitudes in the bilateral median and ulnar nerves, and the right common peroneal nerve. Below, preserved sensitive responses.

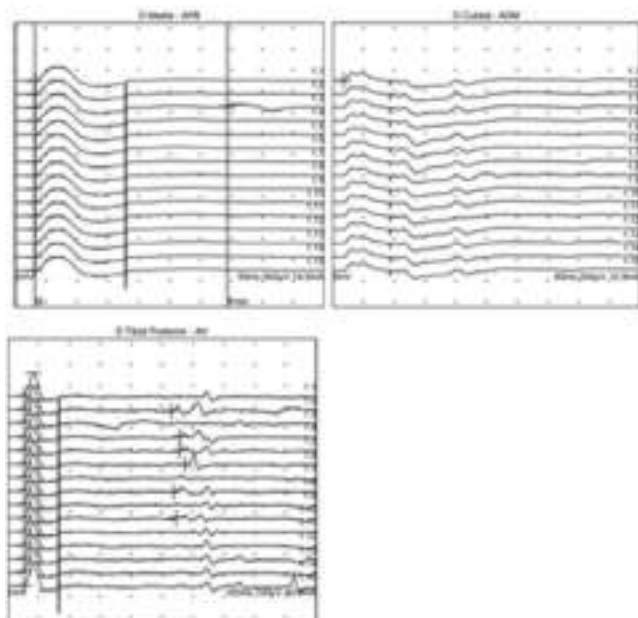


FIGURE 2 EMG findings: normal F-waves in the right median nerve, absent F-waves in the ulnar nerve, and delayed F-waves in the right posterior tibial nerve.

EPO-0972 | Optimized parameters in image-guided focused ultrasound enable safe and reproducible blood–brain barrier opening in preclinical glioma models

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Background and aims: Focused ultrasound (FUS) in combination with systemically administered nanobubbles is an emerging, non-invasive strategy to transiently disrupt the blood–brain barrier (BBB) and enhance drug delivery to the central nervous system. Despite its promise, defining acoustic parameters that ensure both efficacy and safety remains challenging, particularly in pathological settings such as brain tumors.

Methods: We systematically optimized key FUS parameters—including acoustic pressure, burst length, pulse repetition frequency, and number of bursts—to achieve consistent and safe BBB opening in healthy mice. The optimized protocol was then evaluated in several GL261 based glioma mouse models. BBB disruption was assessed using contrast-enhanced MRI, Acoustic Feedback Mapping, and Evans Blue dye extravasation, with histological validation. Safety was evaluated using FLAIR and T2*-weighted MRI to detect hemorrhage or tissue damage.

Results: An optimized set of FUS parameters (1 MPa, 10 ms bursts, 0.5 Hz, 60 bursts) reliably opened the BBB in healthy mice without parenchymal injury, confirmed by T1- and T2*-weighted MRI (Figure 1). The same protocol effectively opened the BBB in tumor and peritumoral regions in GL261 based-glioma models (Fig. 2). Higher pressures or increased burst number or frequency caused hemorrhage, establishing a safety threshold. Acoustic Feedback Mapping showed subharmonic and ultraharmonic emissions correlated with contrast

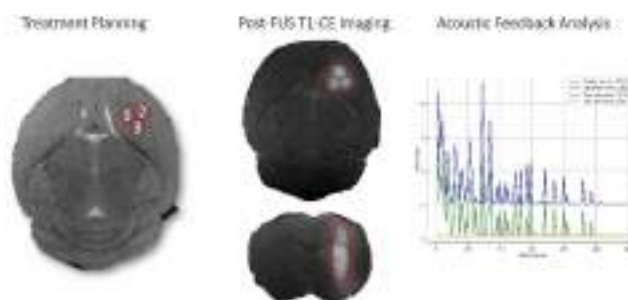


FIGURE 1 Successful BBBO achieved using an optimized MRI-guided FUS protocol, as evidenced by post-FUS T1 contrast-enhanced imaging and subharmonic/ultraharmonic frequency analysis.



FIGURE 2 Successful BBBO in GL261 tumor-bearing mice demonstrated by T1 contrast-enhanced MRI after FUS, with no evidence of hemorrhage on T2* post-FUS Imaging.

enhancement, and histology confirmed Evans Blue extravasation supporting successful BBB disruption.

Conclusion: We establish a reproducible and safe FUS protocol for BBB opening and demonstrate its successful application in preclinical glioma models. These findings provide an important foundation for the clinical translation of image-guided, FUS-mediated drug delivery strategies in neuro-oncology.

Disclosure: Nothing to disclose.

EPO-0973 | Cognitive decline as a prognostic marker of disease activity in patients with glioblastoma

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Background and aims: In patients with glioblastoma, cognitive impairment is influenced by treatment- and patient-related factors, but is strongly driven by tumor-related pathological processes. There is evidence that cognitive decline has predictive value for disease progression. However, existing studies are limited by small sample sizes, heterogeneous tumor entities, insufficient control of important covariates, and the lack of a direct comparison between neuropsychological outcomes and the Karnofsky Performance Status Scale (KPS).

Methods: The present study investigated the predictive value of a neuropsychological test battery comprising eight subtests (NeuroCogFX) in comparison with the KPS for predicting tumor progression according to the RANO criteria. A Cox regression model with time-dependent covariates was applied, including the control variables age, education, sex, MGMT

promoter methylation status, extent of resection, tumor hemisphere, and the use of steroids, anticonvulsants and psychotropic medication. The sample consisted of 99 patients with a total of 309 assessment time points.

Results: Cognitive decline, defined as deterioration in three tests relative to baseline, was associated with a fivefold increased risk of radiologically confirmed tumor progression (HR= 5.00; $p < 0.001$). In contrast, decline in KPS was not a significant predictor. Among the covariates, MGMT promoter methylation (HR=0.42; $p = 0.005$) and a higher number of years of education (HR=0.87; $p = 0.019$) emerged as protective factors.

Conclusion: These results highlight the value of continuous neuropsychological monitoring as a sensitive approach for detecting disease progression. It provides clinically meaningful information, particularly in cases of equivocal radiological findings or for treatment-related decision-making.

Disclosure: Nothing to disclose.

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EPO-0974 | Predicting intracranial aneurysm rupture risk from DSA images: Automated segmentation, multimodal prediction, and validation

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Background and aims: Subarachnoid hemorrhage (SAH) is a highly fatal acute cerebrovascular event, most commonly caused by the rupture of an intracranial aneurysm (IA). Current methods for predicting IA rupture are limited in accuracy and consistency. These methods often rely heavily on clinician experience and carry a non-negligible risk of oversight, falling short of the requirements for precise clinical management.

Methods: To address these limitations, we conducted a retrospective study using digital subtraction angiography (DSA) images from 890 patients with IAs. Initially, the aneurysm regions of interest (ROIs) were annotated. We then developed a U-KAMA segmentation algorithm to achieve fully automated IA segmentation. From these segmented regions, 1834 radiomic features were extracted. Following a feature selection process, 57 key imaging features were retained and subsequently integrated with clinical characteristics to build a multimodal model for predicting IA rupture.

Results: The model demonstrated robust performance. For automated IA segmentation, it achieved a mean Dice similarity coefficient of 0.815. The process was 92.62% faster than manual segmentation, reducing the average time per case from 8.36 minutes to 0.617 minutes. For rupture prediction, the multimodal model achieved an area under the curve (AUC) of 0.893, significantly outperforming models based on single data modalities.

Conclusion: This study demonstrates high performance in both fully automated IA segmentation and rupture prediction. Our work provides a reliable, artificial intelligence-driven solution to advance the automated and precise assessment of IA rupture risk, showing significant potential for clinical translation and application.

Disclosure: Nothing to disclose.

EPO-0975 | Platelet function-defined clopidogrel inactivity as an independent contributor to recurrent ischemic stroke: A confounder-adjusted analysis

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Background and aims: Recurrent ischemic stroke despite antiplatelet therapy remains a significant clinical challenge. Reduced or absent clopidogrel activity, reflected by reduced inhibition of platelet aggregation, may represent an under-recognised and potentially modifiable contributor to recurrence. We aimed to evaluate clopidogrel antiplatelet activity as an independent factor in recurrent ischemic stroke, accounting for major confounders

Methods: Patients admitted with recurrent ischemic stroke between 2019 and 2025 were retrospectively analysed. Of 254 screened patients, 105 were included based on aggregometry test (assessed using adenosine diphosphate-induced platelet aggregation) being done, compliance to medication and availability of records. Platelet activity was categorized and clopidogrel inactivity was evaluated as an independent contributor to recurrence after excluding confounders, including atrial fibrillation, poorly controlled diabetes (HbA1c >8), uncontrolled LDL, large vessel occlusion, and coagulopathies.

Results: Among 105 patients, 40 were on clopidogrel alone and 43 on dual antiplatelet therapy. In the clopidogrel-only group,

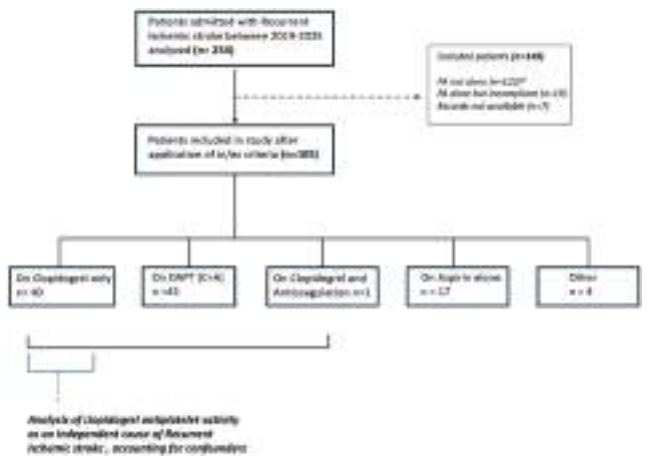


FIGURE 1 Study design and patient allocation across anti-thrombotic therapy groups.



FIGURE 2 Distribution of platelet inactivity and competing mechanisms of recurrent ischemic stroke across antiplatelet strategies.

suboptimal activity was observed in 31/40 patients (77.5 %) with poor activity in 16/40 patients (40%). After exclusion of all confounders, clopidogrel inactivity alone plausibly contributed to recurrence in 17 patients/105 (16.1%, 8.5 % [Poor activity]). Comparatively this cohort had only 17 patients on aspirin alone of which 4 patients developed recurrence (3.8%) after exclusion of all confounders.

Conclusion: Suboptimal clopidogrel antiplatelet activity is especially common in south Asians and Indians may independently contribute to recurrence in almost one-fifth of the even after accounting for major confounders. Platelet function-guided tailoring of antiplatelets may represent an important strategy for secondary stroke prevention.

Disclosure: Nothing to disclose.

EPO-0976 | Prior stroke or transient ischaemic attack history and intensive care unit outcomes: A propensity-matched cohort study

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Background and aims: Prior cerebrovascular disease may increase vulnerability during critical illness through reduced cerebral reserve and impaired autoregulation. Whether prior stroke or transient ischaemic attack (TIA) independently predicts intensive care unit (ICU) mortality remains unclear. We evaluated whether prior stroke/TIA history affects ICU mortality and

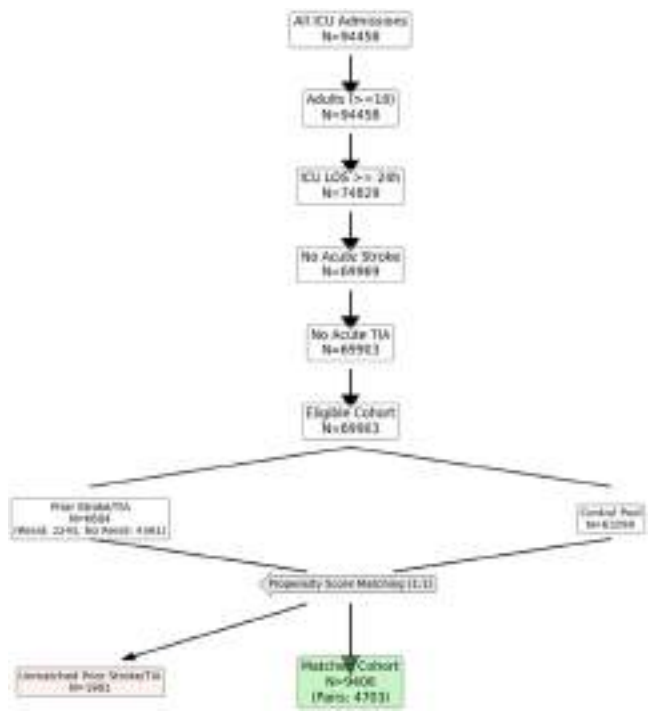


FIGURE 1

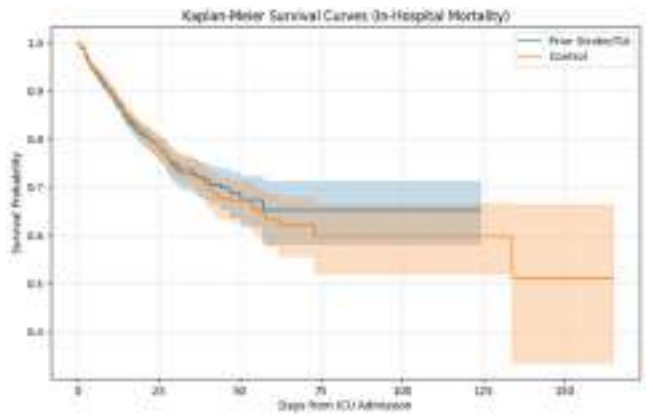


FIGURE 2

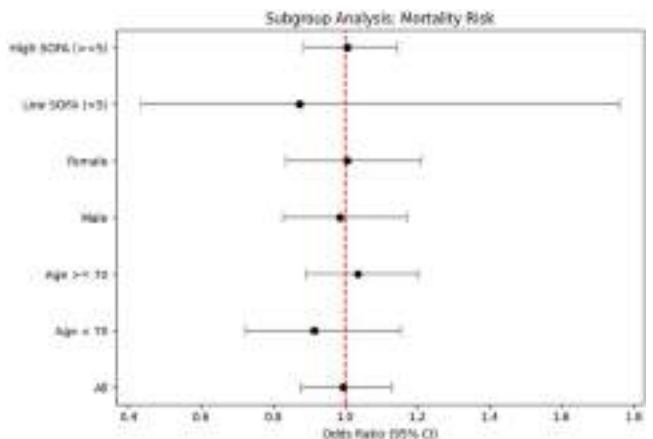


FIGURE 3

recurrent cerebrovascular events independent of cardiovascular comorbidity burden.

Methods: Retrospective propensity-matched cohort study using the Medical Information Mart for Intensive Care IV (MIMIC-IV) database (2008–2022). Adults with ICU stay ≥24 hours were included; those admitted for acute stroke or TIA were excluded. Prior stroke/TIA was identified using International Classification of Diseases codes. One-to-one propensity score matching balanced cardiovascular risk factors and comorbidities. Primary outcome was in-hospital mortality; secondary outcomes included recurrent stroke, seizures, and delirium.

Results: Among 69,903 eligible ICU stays, 6,604 (9.4%) had prior stroke/TIA history. After matching (4,703 pairs), groups were well-balanced (all standardized mean differences <0.1). In-hospital mortality was similar between prior stroke/TIA patients (11.4%) and controls (11.5%; matched odds ratio 0.99, $p=0.95$). Kaplan-Meier analysis showed no survival difference (log-rank $p=0.54$). However, prior stroke/TIA patients experienced higher rates of recurrent ischemic stroke (4.3% vs 2.6%), seizures (16.2% vs 8.0%), and delirium (8.8% vs 6.3%). On multivariable analysis, prior stroke/TIA independently predicted recurrent stroke (odds ratio 1.36, 95% confidence interval 1.09–1.71, $p=0.007$).

Conclusion: After controlling for cardiovascular comorbidities, prior stroke/TIA history does not independently increase ICU mortality. However, these patients experience significantly higher rates of recurrent cerebrovascular events and

neurological complications, warranting enhanced neurovascular monitoring during critical illness.

Disclosure: Nothing to disclose.

EPO-0977 | **Mortality in stroke survivors during a global health crisis: The Geneva population-based study**

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Background and aims: We examined 5-year mortality among stroke survivors and whether the impact of the COVID-19 pandemic on mortality was higher in stroke patients compared to the general population.

Methods: We evaluated 5-year mortality in all patients who suffered from a first-ever stroke in the canton of Geneva (504'257 inhabitants) in 2018-2019. Mortality was ascertained through official records. Predictors of mortality were identified using Cox proportional hazards models, and mortality among stroke survivors (alive ≥90days post-stroke) was compared with that of the general population using standardized mortality ratios (SMRs).

Results: In 2018 and 2019, there were 1123 first-ever stroke patients (1,000 ischemic strokes [IS] and 123 intracerebral hemorrhages [ICH]). Five-year mortality was 40.3% in IS and 56.1% in ICH. In IS, mortality was associated with age (HR=1.06), premorbid disability (mRS, HR=1.28), stroke severity (NIHSS, HR=1.09), and coronary heart disease (HR=1.54; all $p < 0.001$). In ICH, mortality was associated with age (HR=1.07) and stroke severity (NIHSS, HR=1.10; both $p < 0.001$). During follow-up, mortality was consistently higher among stroke survivors than in the general population. However, no excess mortality was observed during the pandemic period (2020–2021) compared with the post-pandemic years 2022–2024 (SMR 1.88 [1.56–2.25] vs 1.94 [1.62–2.30], $p = 0.82$).

Conclusion: While stroke survivors have sustained excess mortality compared with the general population, no increase was observed during the pandemic years (2020–2021) compared with the post-pandemic years (2022–2024).

Disclosure: Nothing to disclose.

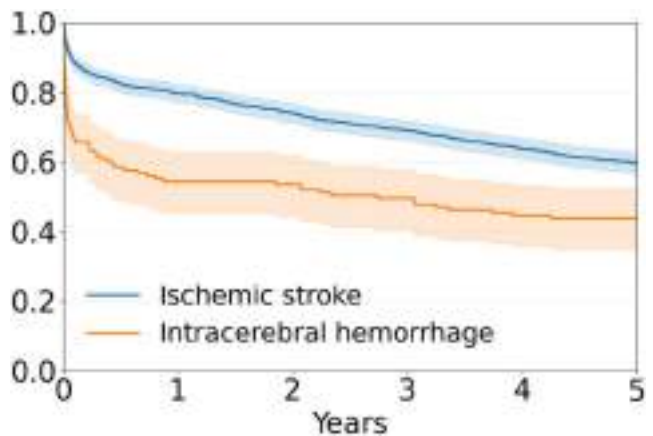


FIGURE 1 Kaplan Meier curves.

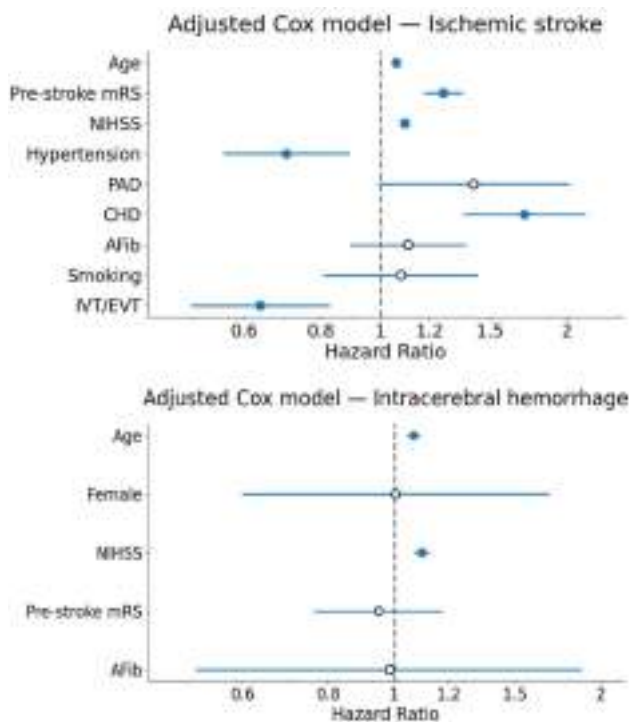


FIGURE 2 Multivariable Cox proportional hazards models for 5-year mortality after ischemic and hemorrhagic stroke. Variables with $p < 0.05$ in univariable Cox regression were entered into the multivariable model.

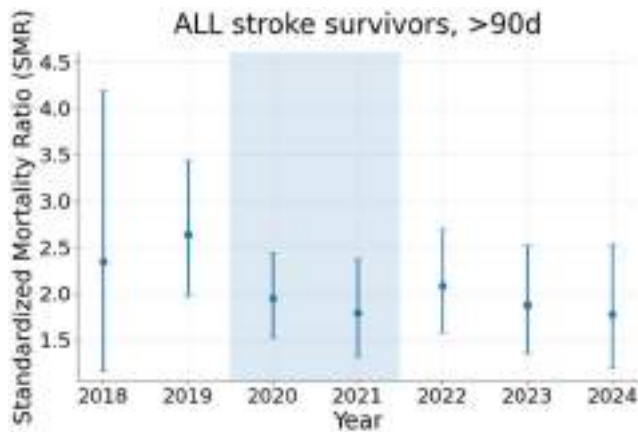


FIGURE 3 Standardized mortality ratios (SMR) of stroke survivors. Ischemic and hemorrhagic strokes are combined.

EPO-0978 | **Epidemiological study, evaluation of clinical, genetics and neuroimaging aspects in a cohort of Brazilian patients with Cadasil**

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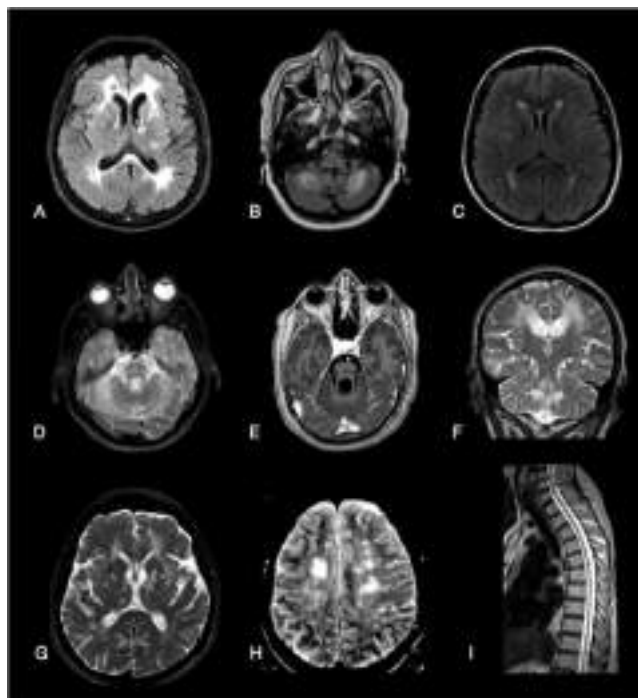


FIGURE 1 MRI scans from 9 cases analyzed in this study.



FIGURE 2 Topography of brain lesions in the cohort of patients with CADASIL.

Background and aims: CADASIL is a dominant autosomal disease caused by mutations at the NOTCH 3 gene. This study aims to evaluate demographic aspects and clinical symptoms of patients with CADASIL in a region with a high prevalence of this disease in Rio de Janeiro/Brazil, analyze morphometrically white matter in cerebral MRI, and study variants of NOTCH3 gene.

Methods: Clinical evaluation and follow-up of patients at a Specialized Center in CADASIL in the north of the State of Rio de Janeiro/Brazil (2018–2025). Research on mutations in NOTCH3 gene. Brain morphological evaluation by 1.5-Tesla MRI.

Results: 33 patients from 27 different families. 19 (58%) men and 14 (42%) women; 70% white and 30% Afro-descendant. Mean age at first symptom: 42.3 (± 10.6); 19 (58%) presented migraine, 7 (21%) cognitive impairment. 32 patients heterozygous for the pathogenic allele variant c.457C>T from the ten probably unrelated kindreds. One patient is heterozygous for the pathogenic allele c.328C>T. All patients with pathogenic allele had

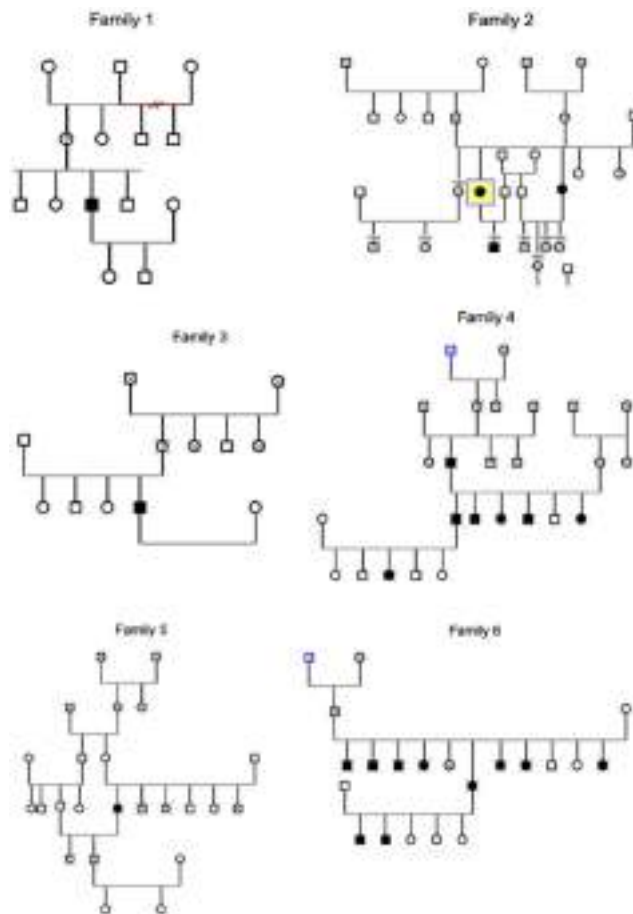


FIGURE 3 Pedigree of six families with NOTCH3 gene mutations or variants. Square symbols, men; circular symbols, women; filled symbols, CADASIL affected.

MRI lesions; MRI analyses: 61% presented bilateral white matter lesions, 48% bilateral temporal pole lesions, 55% parietal lobe, 27% basal ganglia, and 24% cerebellar lesions.

Conclusion: CADASIL is a rare and neglected disease. In this study, conducted in a region of Rio de Janeiro with a high prevalence of the disease, there was a predominance of men and white individuals, and migraine was the main neurological symptom. Regarding the changes in MRI, the parietal and temporal lobes were the most affected regions; bilateral temporal lesion (a hallmark of the disease) was observed in 48% of cases.

Disclosure: Nothing to disclose.

EPO-0979 | Simulating and augmenting clinical workflows in emergency stroke triage: A multi-center comparative study of three artificial intelligence paradigms

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Background and aims: Early diagnosis and rapid identification of ischemic stroke and LVOs are demanding and challenging. This study aimed to simulate three typical emergency work

paradigms using a large model in order to explore the robustness of different levels of information integration in real-world multi-center settings.

Methods: We developed three paradigms: Paradigm 1 (Clinical-Only): Relies solely on medical records (simulating preliminary expressions after a clinical consultation and examination). Paradigm 2 (Report-Guided): Medical records plus radiology reports (obtained from brain non-contrast CT, simulating decisions based on the reports). Paradigm 3 (Holistic Review): Medical records, radiology reports, and original CT images (simulating high-level physicians making comprehensive decisions by combining reports and personally reviewing the images).

Results: Paradigm 1 performs excellently in the central hospital but fails in external centers due to heterogeneity in medical record documentation (F1 score 93.6 vs. 57.4), revealing the limitations of relying solely on empirical inquiries. Paradigm 2 improves the stability of general diagnosis but is limited in identifying large vessel occlusion (LVO) due to missed descriptions of subtle signs in reports (Information Loss). Paradigm 3, by introducing direct interpretations of the original images, the model achieves the best performance in LVO tasks across all centers (F1 score 68.8), significantly outperforming Paradigm 2.

Conclusion: Although radiology reports provide important references, in high-risk emergency tasks, AI (and doctors) must be able to read the original images directly (Paradigm 3) in order to overcome the heterogeneity of medical records and the limitations of report information, achieving a satisfactory diagnosis and treatment.

Disclosure: Nothing to disclose.

EPO-0980 | Long term outcomes of symptomatic intracranial atherosclerotic stenosis: A 3 year follow up study

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Background and aims: Large artery intracranial atherosclerotic stenosis (ICAS) is often described to have high rates of complications such as stroke recurrence and mortality. Though latest guidelines recommend maximum medical therapy as the mainstay of treatment for symptomatic ICAS, there is insufficient

TABLE 1 Baseline characteristics.

Variables	Did not achieve maximal medical therapy	Achieved maximal medical therapy	Total	P-value
Age, Mean (SD)	65.9 (11.4)	63.8 (12.5)	66.1 (13.5)	0.589
Male sex, N (%)	78 (65.8)	159 (81.8)	237 (81.3)	0.000
Ethnicity, N (%)				0.333
Chinese	89 (77.2)	179 (88.8)	274 (71.4)	
Malay	54 (45.8)	44 (23.1)	58 (15.1)	
Indian	11 (8.3)	37 (18.5)	38 (9.8)	
Concatori	4 (3.1)	10 (3.9)	14 (3.8)	
Myocardial, N (%)	87 (73.7)	174 (70.2)	261 (70.4)	0.879
Hypertension, N (%)	102 (78.7)	200 (77.8)	302 (76.2)	0.627
Hyperlipidemia, N (%)	90 (75.8)	205 (79.8)	301 (76.2)	0.296
Diabetes mellitus, N (%)	54 (42.2)	115 (45.3)	169 (44.2)	0.586
Chronic kidney disease, N (%)	11 (8.8)	42 (16.4)	53 (15.8)	0.044
Duration of follow up in months, Mean (SD)	29.7 (16.6)	23.9 (16.8)	26.1 (16.4)	0.871
Location of stenosis, N (%)				0.373
Anterior circulation	104 (82.5)	209 (81.1)	316 (81.6)	
Posterior circulation	20 (15.8)	37 (14.6)	57 (15.3)	
Both	2 (1.6)	11 (4.3)	13 (3.4)	
Patterns of infarcts, N (%)				0.788
Perforator	44 (36.8)	85 (32.6)	129 (36.1)	
Embolic	23 (19.4)	57 (22.2)	80 (23.9)	
Hypoperfusion	34 (28.1)	83 (27.5)	97 (26.6)	
Unknown	12 (9.8)	21 (8.3)	33 (9.7)	
HaCRP, Mean (SD)	8.5 (21.4)	10.4 (26.3)	10.1 (25.2)	0.643
Homocysteine, Mean (SD)	11.7 (4.6)	13.1 (10.2)	12.6 (8.9)	0.115

TABLE 2 Multivariate analysis of factors affecting mortality rates in patients with ICAS.

Variable	OR (95% CI)	P-value
Achieved maximal medical therapy	8.41 (1.08 – 65.36)	0.042
Age	0.98 (0.92 – 1.01)	0.086
Diabetes	2.61 (0.87 – 7.85)	0.087
Chronic kidney disease	5.25 (1.14 – 24.6)	0.027

analysis regarding the effectiveness of this in reducing rates of mortality, recurrent strokes and cardiovascular events.

Methods: This multicentre prospective cohort study included patients who were treated for symptomatic ICAS between 2020 and 2022. Patients were stratified into two groups; those who achieved maximal medical therapy and those who did not. The rates of adverse outcomes such as recurrent strokes, mortality and myocardial infarction were compared between the two groups via univariate and multivariate binary logistic regression.

Results: Among 399 patients, 33.2% achieved maximal medical therapy. Mortality occurred in 4.9% of patients, recurrent stroke in 17.0% and myocardial infarction in 2.3%. On multivariate analysis, achieving maximal medical therapy was associated with lower rates of mortality (OR 8.41; CI 1.08–65.36, $p=0.042$) and a longer duration of survival (Chi-square=9.871; $df=1$; $p=0.002$). Achieving maximal medical therapy was not associated with a significant reduction in rates of recurrent strokes and myocardial infarction.

Conclusion: ICAS is a serious condition which carries high rates of complications. Aggressive medical therapy to achieve optimum control of cardiovascular risk factors remains crucial to improving outcomes.

Disclosure: Nothing to disclose.

EPO-0981 | Is the mini-cognitive evaluation superior to MMSE and MOCA to identify mild post stroke cognitive impairment?

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Background and aims: The diagnosis of post-stroke cognitive impairment (PSCI) is imperative for its effective management. However, the sensitivity and specificity of screening tests are inadequate for the diagnosis of mild PSCI. The objective of this study was to assess the discriminatory capacity of a newly developed screening test, the Mini-cognitive evaluation (MCE), in patients with at most mild PSCI (defined by an MMSE score ≥ 20). The primary objective was to ascertain the most accurate screening test (MMSE, MoCA, MCE) for discriminating stroke from controls. The secondary objectives were (1) to determine the most accurate test for identifying patients with PSCI, and (2) to examine the associations between MCE and the location of cerebral atrophy using VBM analysis.

Methods: We included 122 patients referred in the acute stroke unit (ASU) of Amiens University Hospital (62 at the ASU and 60 at post-stroke visit) and 436 controls (comprehensive assessment performed in 89 patients, 44 with PSCI).

Results: The MCE demonstrated superiority ($p=0.0001$) to the MMSE and MoCA in (1) discriminating between patients and controls (ROC AUC of 0.791 [0.731, 0.851]) and (2) discriminating patients with PSCI (ROC AUC=0.896 [0.838-0.954]) ($p=0.001$). MCE score was associated with overall brain atrophy ($p=0.01$) and using VBM analysis with atrophy of numerous hemispheric structures, predominantly on the left side ($p\leq 0.001$, uncorrected).

Conclusion: This study demonstrated that MCE is a promising diagnostic tool for identifying PSCI. The potential benefit of its use as a first line test in the management of stroke patients has now to be determined.

Disclosure: Y.Lesnard: Nothing to disclose O. Godefroy: during the last two years has served on scientific advisory boards and speaker (GE Healthcare, ESAI SAS), and did not received funding for travel and meetings from pharmaceutical companies S. Wannepain : Nothing to disclose A. Routier-Thorel : Nothing to disclose S.Canaple: funding for travel from MEDTRONIK C.Lamy: Nothing to disclose C.Leclercq : Nothing to disclose A.Courselle: Nothing to disclose M. Roussel : Nothing to disclose.

EPO-0982 | Prominent vessel sign on susceptibility-weighted imaging is associated with CTP perfusion-core mismatch in anterior circulation ischemic stroke

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Background and aims: Perfusion imaging can identify salvageable ischemic brain tissue but requires use of contrast agents and professional post-processing software. This study aims to evaluate the role of prominent vessel sign (PVS) on acute susceptibility-weighted imaging (SWI).

TABLE 1 Baseline characteristics between patients with and without prominent vessel sign.

	All (N=303)	Without PVS (N=203)	With PVS (N=100)	p value
Clinical characteristics				
Age	66 (57 – 75)	66 (57 – 75)	67.5 (57 – 82)	0.753
Male sex	101 (66.3)	114 (66.7)	67 (65.7)	0.864
Stroke onset type				
Witnessed	134 (77.2)	138 (78.6)	76 (76.5)	0.422
Unwitnessed	69 (22.8)	43 (21.4)	26 (25.5)	
Pre-morbid mRS	0 (0 – 0)	0 (0 – 0)	0 (0 – 0)	0.933
Baseline NIHSS	4 (2 – 7)	3 (2 – 5)	5 (3 – 11)	<0.001
Medical history				
Hypertension	231 (76.2)	159 (79.1)	72 (70.8)	0.190
Diabetes	78 (25.7)	63 (31.3)	15 (14.7)	0.002
Hypercholesterolemia	126 (41.6)	88 (43.8)	38 (37.3)	0.276
Atrial fibrillation	31 (10.2)	15 (7.5)	16 (15.7)	0.028
Previous stroke	65 (21.5)	49 (24.4)	16 (15.7)	0.082
Imaging characteristics				
Onset or last known well to baseline MRI, hours	7.2 (2.9 – 13.7)	7.4 (3.1 – 13.6)	6.6 (2.8 – 14.5)	0.688
Time from baseline MRI to CTR, days	50 (38 – 75)	51 (39 – 69)	58 (36 – 76)	0.794
CTP core infarct volume	0 (0 – 0)	0 (0 – 0)	0 (0 – 4)	<0.001
CTP penumbra volume	3 (0 – 52)	0 (0 – 7)	73 (28 – 130)	<0.001
Mismatch volume	3 (0 – 48)	0 (0 – 7)	70 (28 – 130)	<0.001
Presence of LVO	82 (27.1)	23 (11.4)	59 (57.8)	<0.001
Presence of CTP mismatch	124 (40.9)	41 (20.4)	83 (81.4)	<0.001

CTP core infarct volume was defined as the area with rCBF<30%. CTP penumbra volume was defined as the area with Tmax>6s. Presence of CTP mismatch was identified according to the imaging criteria of the EXTEND trial: core infarct volume < 70ml, mismatch volume > 10ml, and mismatch ratio > 3.2.

TABLE 2 Univariable and multivariable logistic regression analyses of factors associated with presence of CTP perfusion-core mismatch.

	Univariable		Multivariable	
	OR (95% CI)	p value	OR (95% CI)	p value
Age	1.01 (0.99 – 1.03)	0.124	-	
Female sex	1.30 (0.80 – 2.10)	0.269	-	
Time from onset or last known well to baseline MRI	1.01 (0.97 – 1.04)	0.783	-	
Hypertension	0.77 (0.45 – 1.31)	0.333	-	
Diabetes	0.51 (0.10 – 0.99)	0.018	0.84 (0.41 – 1.68)	0.615
Hypercholesterolemia	0.87 (0.54 – 1.30)	0.543	-	
Atrial fibrillation	1.04 (0.35 – 6.30)	0.006	2.11 (0.77 – 5.75)	0.144
Baseline NIHSS	1.11 (1.06 – 1.17)	<0.001	1.01 (0.95 – 1.07)	0.846
Previous stroke	0.95 (0.54 – 1.67)	0.864	-	
Presence of large vessel occlusion	9.54 (5.23 – 17.40)	<0.001	3.94 (1.89 – 8.21)	<0.001
Prominent vessel sign	17.05 (9.31 – 31.22)	<0.001	10.25 (5.32 – 19.74)	<0.001

Methods: We included consecutive patients with anterior circulation acute ischemic stroke (AIS) admitted within 24 hours from onset or last known well from a prospective observational cohort between June 2023 and April 2025 in China. Emergency MRI was performed as the initial imaging modality for stroke diagnosis. PVS was defined as a focal increase in the number or diameter of hypointense vessels compared with the contralateral hemisphere. Perfusion-core mismatch on CT perfusion was obtained according to the Extending the Time for Thrombolysis

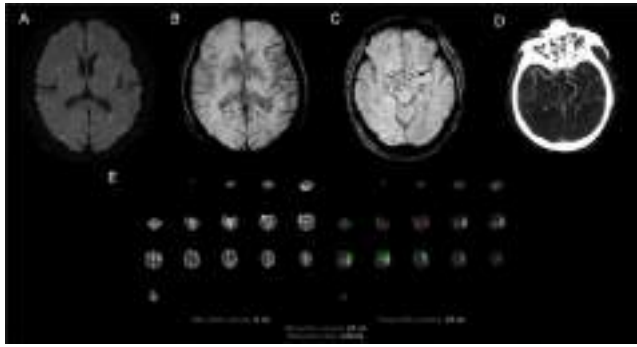


FIGURE 1 Representative image of prominent vessel sign on SWI with MCA occlusion and perfusion-core mismatch.

in Emergency Neurological Deficits (EXTEND) trial immediately after baseline MRI scan.

Results: Among 303 patients (median age 66 [IQR 57–75] years, 33.7% females), 102 (33.7%) had PVS on SWI at admission. The sensitivity of the PVS for identifying CTP mismatch was 66.9%, specificity 89.4%, positive predictive value 81.4%, and negative predictive value 79.6%. The presence of PVS on SWI remained associated with CTP mismatch (OR 10.25, 95% CI 5.32–19.74, $p < 0.001$) after adjusting for diabetes, atrial fibrillation, baseline stroke severity, and presence of large vessel occlusion.

Conclusion: PVS on SWI is frequently observed and associated with CTP mismatch among patients with anterior circulation AIS within 24 hours from onset. The MRI marker may serve as a readily available tool for rapid identification of patients who may benefit from acute therapeutic intervention.

Disclosure: Nothing to disclose.

MS and Related Disorders 7

EPO-0983 | Elevated serum BAFF in patients with neuromyelitis optica spectrum disorder after rituximab treatment

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Background and aims: Despite achieving complete peripheral B cell deletion after over six months of rituximab therapy, a clinically significant minority of neuromyelitis optica spectrum disorder (NMOSD) patients experience breakthrough relapses, yet the underlying mechanism remains unclear.

Methods: From January 2024 to April 2025, this study enrolled 55 NMOSD patients and 15 healthy controls (all negative for autoimmune antibodies) at The First Affiliated Hospital of Fujian Medical University. Among the patients, 32 were in the remission phase under RTX treatment, and 23 were in the acute phase of the disease (16 were not receiving RTX treatment, and 7 were experiencing relapse despite RTX treatment). Serum BAFF protein concentrations were measured using ELISA. The non-parametric Kruskal-Wallis H test was used to assess overall differences among groups, followed by Dunn's test with Bonferroni correction for pairwise comparisons.

Results: Results showed that RTX-treated patients in remission had significantly higher serum BAFF levels compared to healthy controls ($p < 0.0001$). In contrast, there was no significant difference in BAFF levels between the overall acute phase group and controls. Notably, one RTX-treated patient in the stable group exhibited an exceptionally high BAFF level (1771.26 pg/mL) and experienced a clinical relapse one month later.

Conclusion: NMOSD patients treated with rituximab exhibit persistently high expression of BAFF. Extremely high BAFF levels may predict relapse in RTX-treated patients, serving as a potential early biomarker for relapse.

Disclosure: Nothing to disclose.

EPO-0984 | Menopause in multiple sclerosis: Treatments and quality of life

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Background and aims: Menopausal symptoms may overlap with multiple sclerosis (MS) manifestations and contribute to reduced quality of life (QoL). To characterize the use of menopausal hormone therapy (MHT) and symptomatic pharmacological treatments during menopause and to assess the effect of menopause on quality of life (QoL) in women with MS

Methods: Cross-sectional, observational study including postmenopausal women with MS. QoL was measured with the Short Cervantes Scale (SF-16). Demographic and clinical data, as well as current MHT and symptomatic drug use, were recorded. Suitability for MHT was evaluated using a standardized algorithm derived from Spanish Society of Gynecology recommendations.

Results: Ninety-eight women were enrolled (mean age 52 years), with a mean interval of 4 years since the final menstrual period. Anxiety was present in 65%, fatigue in 76%, and urinary symptoms in 51%. Only 7.1% were on MHT, despite a large proportion fulfilling eligibility criteria (56.5% for combined oral MHT and 83% for combined transdermal MHT). Compared with the premenopausal phase, menopause was associated with increased prescriptions of symptomatic medications: antidepressants +33%, anxiolytics +42%, hypnotics +33%, and drugs for urinary symptoms +48%. Higher symptomatic drug use correlated with poorer baseline QoL. The worst scores were found in the menopause/health and psychological domains. The mean global Cervantes score was 37.6 (SD 19.0), and 50% of participants showed impaired QoL (29.6% mild impairment, 20.4% QoL could be improved).

Conclusion: Menopause increases the QoL burden in women with MS and worsens shared “invisible” symptoms. Optimizing menopause care, including appropriate MHT, may reduce polypharmacy and improve well-being

Disclosure: Celia Oreja-Guevara has received speaker and consultation fees from Alexion, Amgen, Biogen Idec, BMS, Juvise, Merck, Novartis, Roche, Sanofi-Genzyme, Sandoz, Viartis and Neuraxpharm. The rest of authors nothing to disclose.

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Background and aims: Neurofilament light chain (NfL) is an important serological biomarker with prognostic relevance in multiple sclerosis (MS). This study aimed to investigate the presence of humoral immunity against NfL in patients with MS.

Methods: Patients fulfilling the 2017 MS diagnostic criteria with a minimum follow-up of three years were included. Baseline cerebrospinal fluid (CSF) NfL levels, serum NfL levels at the last visit, anti-NfL antibodies, and the levels of interleukin-10, IL-27, IL-35, C-X-C motif chemokine ligand 13 (CXCL13) and transforming growth factor-beta were measured. Associations between antibody and cytokine levels and disability accumulation were analyzed.

Results: The study included 47 patients with relapsing remitting MS, nine with primary progressive MS (PPMS), and 20 healthy controls. The median age of MS patients was 33 (IQR: 26–48), and the median disease duration was 5.4years (IQR: 4.4–8.7). No association was found between CSF anti-NfL antibody levels and the time to reach an Expanded Disability Status Scale (EDSS) score of 3 ($p=0.95$) or the time to the second relapse ($p=0.6$). Serum CXCL13 levels were lower in PPMS patients compared with healthy controls ($p=0.03$). No correlation was observed between NfL levels and anti-NfL antibodies in serum/CSF. Serum NfL was better at distinguishing MS subgroups than CSF NfL. CSF IL-27 levels were lower in samples obtained during relapse compared with remission ($p=0.02$).

Conclusion: Anti-NfL antibody presence was not associated with prognostic outcomes in MS. Serum NfL levels were more sensitive than CSF NfL levels in differentiating MS subgroups.

Disclosure: The present work was supported by the Research Fund of Istanbul University and the “Geleceğimsin” Project Support Program of the Turkish Multiple Sclerosis Society. Project No: 41971.

EPO-0986 | Clinical and MRI correlates of overactive bladder symptoms in relapsing–remitting multiple sclerosis: A preliminary study

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Background and aims: Overactive bladder (OAB) is a common but underrecognized condition in relapsing–remitting multiple sclerosis (RRMS). Its associations with clinical characteristics, disability, magnetic resonance imaging (MRI) findings, and symptom-specific contributors remain unclear. This study evaluated factors associated with OAB symptoms and the contribution of individual OAB Questionnaire–V8 items.

Methods: Between February 2023 and February 2024, 197 patients assessed with the OAB-V8 and with MRI performed

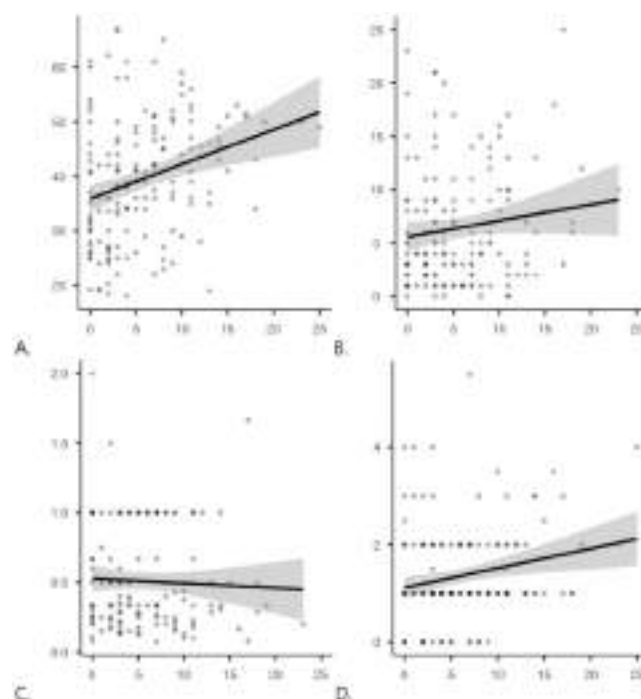


FIGURE 1: Correlation analyses between overactive bladder status and age, disease duration, annualized relapse rate, and EDSS.

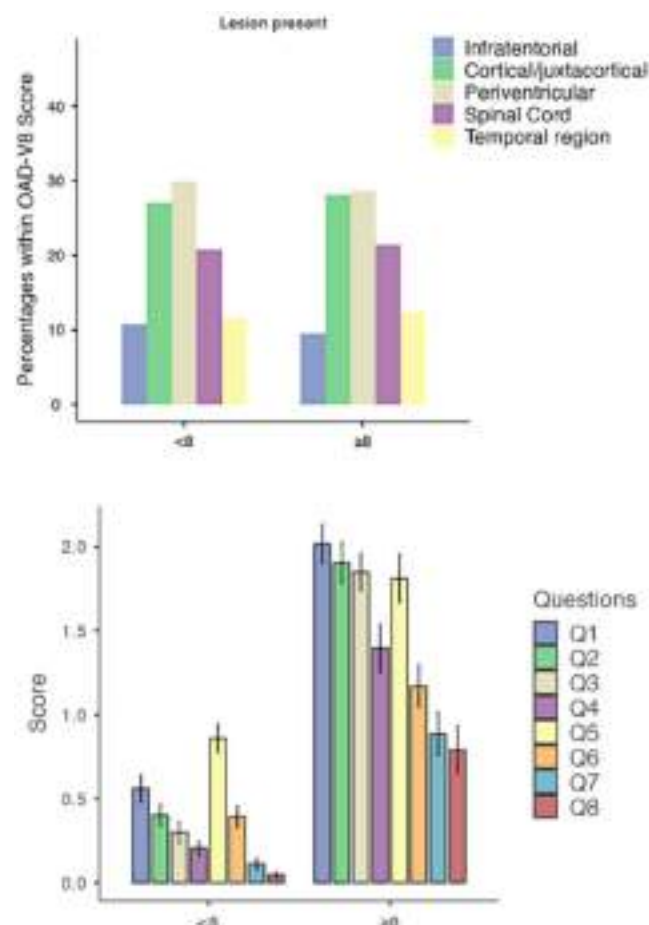


FIGURE 2 Proportional distribution of lesion localization according to overactive bladder status and contribution of individual OAB-V8 questionnaire items to the total score

TABLE 1 Comparison of demographic, clinical, and radiological characteristics according to OAB-V8 score.

		OAB Score <8	OAB Score ≥8	P value
Sex	Female, n=112	46(37.7%)	76(62.3%)	0.028
	Male, n=58	7(18.4%)	51(81.6%)	
Age		45.33±10.01, 45 (19–85)	37.7 (11.38, 37 (18–67)	0.003
Disease duration		7.55±5.66, 8 (0–25)	5.91±5.42, 4 (0–25)	0.058
ARR		0.67±0.35, 0.33 (0.08–1.87)	0.53±0.37, 0.34 (0.07–2)	0.323
EDSS		1.63±1.06, 1 (0–5.5)	1.21±0.85, 1 (0–4)	0.011
Periventricular	Present	51(32.9%)	194(67.1%)	0.741
	Absent	3(40%)	3(60%)	
Cortical/subcortical	Present	50(34.7%)	94(65.3%)	0.615
	Absent	3(18.8%)	13(12.1%)	
Temporal	Present	22(35.5%)	46(41.9%)	0.199
	Absent	31(31.6%)	67(68.4%)	
Spinal Cord	Present	58(34.5%)	73(65.5%)	0.622
	Absent	5(41.7%)	5(38.3%)	
Infratentorial	Present	17(31.5%)	57(68.5%)	0.753
	Absent	26(34%)	70(66%)	
Cerebellum	Present	4(33.3%)	18(66.7%)	1
	Absent	44(33.3%)	88(66.7%)	
Cervical	Present	18(35.2%)	70(64.8%)	0.378
	Absent	15(30.6%)	34(69.4%)	
Thoracic	Present	9(29%)	18(75%)	0.131
	Absent	14(37.8%)	25(62.2%)	

within the preceding year were screened; after exclusions, 160 patients with RRMS were included. Patients were grouped by OAB-V8 score (<8 vs. ≥8). Clinical and radiological characteristics were analyzed, and correlations between OAB-V8 scores and clinical parameters were assessed.

Results: Patients with OAB-V8 scores ≥8 were older and had higher Expanded Disability Status Scale (EDSS) scores than those with scores <8. OAB-V8 scores correlated moderately with age ($\rho=0.327$, $p<0.001$) and weakly with EDSS ($\rho=0.177$, $p=0.025$). No significant associations were found with disease duration, annualized relapse rate, or MRI lesion localization. Sub-question analysis showed that sudden urgency (Q3; $r=0.786$), bothersome urgency (Q2; $r=0.773$), and daytime frequency (Q1; $r=0.753$) were the strongest contributors to the total OAB-V8 score.

Conclusion: Although lesion localization—particularly spinal cord involvement—has been linked to urinary dysfunction in some studies, evidence remains inconsistent, with disability and overall disease burden emerging as stronger determinants of OAB symptoms. Urgency-related symptoms primarily drive OAB severity, highlighting the importance of focused symptom assessment.

Disclosure: Nothing to disclose.

EPO-0987 | MS-FLOWER, an ongoing U.S. multicenter pilot study to identify challenges to MSCopilot®'s integration into multiple sclerosis standard clinical care

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Background and aims: MSCopilot®, a clinically validated software-as-medical-device (SaMD), and its digital biomarkers measure Multiple Sclerosis (MS) impact on walking, vision, cognition, and dexterity enabling remote symptoms' self-assessment. While prior studies demonstrated that MSCopilot® scores correlate with the Extended Disability Scale Score (EDSS) and the 4-item MS functional composite, integrating the tool into routine care remains challenging. MS-FLOWER (NCT06922942) is a U.S. pilot study evaluating the feasibility, usability, and integration of MSCopilot® in routine care.

Methods: This 6-month prospective study follows people with MS and their healthcare providers (HCPs). Patients remotely complete MSCopilot® monthly digital tests and questionnaires: Patient Health Questionnaire (PHQ-8) and Modified Fatigue Impact Scale-5 (MFIS-5). HCPs review results through a web dashboard. Questionnaires evaluating training, onboarding process, ease of use of MSCopilot® at-home and clinical workflows integration are administered to both patients and HCPs (Figure).

Results: Eight MS specialists recruited 62 patients (75.8% female, mean age 45.3±11.2 years, median EDSS 2.0). The population included 92.0% relapsing-remitting MS, 3.2% active secondary progressive MS (SPMS), 1.6% non-active SPMS and 3.2% primary progressive MS. Following onboarding, all HCPs felt confident introducing MSCopilot® to patients with 75% doing so in 5 minutes or less. All patients surveyed after inclusion ($n=28$) reported MSCopilot® was easy to use.

Conclusion: Clinical integration of MSCopilot® could enhance disease monitoring, optimize clinical workflows, and improve patient engagement. This study paves the way for broader adoption of digital biomarkers in routine care, supporting personalized, data-driven disease management.



FIGURE 1 MS-FLOWER study design.

Disclosure: Freeman L.: Hoffmann La Roche, Genentech, EMD Serono, Sanofi, Horizon Therapeutics and TG Therapeutics—consultant and/or advisory board member; Medscape, Inc and the MS Association of America— speaking fees for participation in educational programs; MS Association of America, Genentech, and Merck— speaking fees; NIH/NINDS, PCORI, Genentech, Sanofi and EMD Serono— research support/grants. Pardo G.: Biogen Inc, EMD Serono, TG Therapeutics, Horizon Therapeutics, Novartis Pharmaceuticals, Sanofi Genzyme, BMS, Roche/Genentech, and Amgen—speaking fees; Biogen Inc, Roche/Genentech, Sanofi Genzyme, Novartis Pharmaceuticals, EMD Serono, BMS, TG Therapeutics, ViewMind, Ad Scientiam, Continuum Therapeutics, and CorEvitas— research support/grants. Feng J.: EMD Serono, Genentech, TG Therapeutics, Novartis, BMS, Horizon—advisory board member. Brochu T, Carment L, Gorin C, Klaeylé L, Zinaï S.: Ad Scientiam— employees; may hold shares or stock options. Graves J.: Sanofi, Genentech, Ad Scientiam, Octave and EMD Serono— research grants; Octave, TRIX and Google— consulting fees. Naismith R.: Alexion Pharmaceuticals, Astoria, Biogen, Bristol Myers Squibb, Celltrion, Genentech, Genzyme, EMD Serono, Horizon Therapeutics, ImmPACT-Bio, Kyverna, Novartis, Sandoz, TG Therapeutics—consultant and/or advisory board member and/or research grants.

EPO-0988 | **Reduced hippocampus–amygdala transition area (HATA) volume in progressive multiple sclerosis: A cross-sectional MRI study**

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Background and aims: MRI biomarkers of progression remain limited in multiple sclerosis. Progressive multiple sclerosis (PMS) is characterized by neurodegeneration and disability accumulation. Mesotemporal substructures, including the hippocampus–amygdala transition area (HATA), may undergo phenotype-related atrophy. We examined whether bilateral HATA volume differs between PMS and relapsing–remitting MS (RRMS).

Methods: Cross-sectional cohort of 106 MS patients (RRMS $n=68$; PMS $n=38$) underwent T1-weighted MRI. HATA and amygdala nuclei volumes were derived with FreeSurfer. The primary endpoint was bilateral HATA volume (mm³). Phenotype effects were estimated using multivariable OLS with HC3-robust standard errors, adjusting for age, sex, disease duration, and intracranial volume (ICV; per 100,000 mm³). Secondary ROI tests used Benjamini–Hochberg FDR.

Results: Bilateral HATA volume was lower in PMS than RRMS (mean [SD] 88.49 [17.85] vs 106.74 [18.49] mm³). In the adjusted model, PMS was associated with reduced bilateral HATA (beta = −10.11 mm³; 95% CI −17.62 to −2.60; $p=0.008$). The phenotype effect was significant for left HATA (beta = −6.59; $p=0.003$) but not right (beta = −3.52; $p=0.145$). No secondary ROI survived FDR correction. An HATA-by-phenotype interaction suggested a stronger HATA–MSFC association in PMS ($p=0.035$).

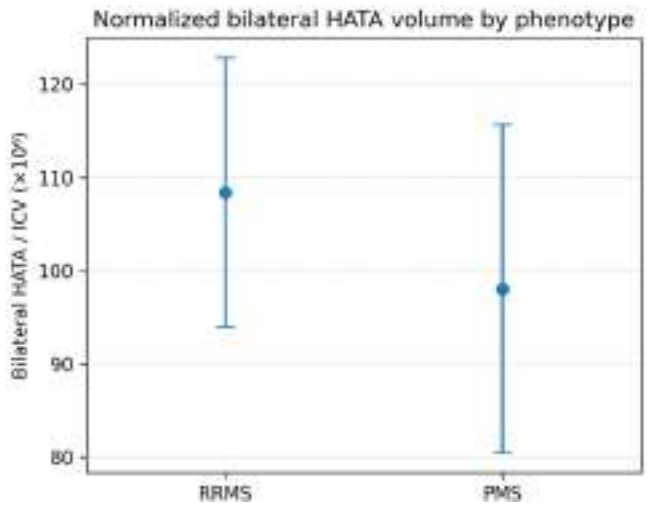


FIGURE 1 Normalized bilateral HATA volume is lower in PMS than RRMS (normalization: (HATA/ICV) × 10⁶).

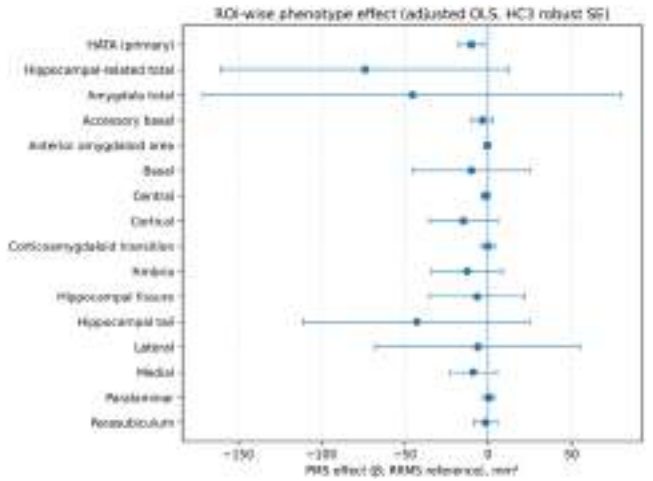


FIGURE 2 ROI-wise PMS effect (β, 95% CI) from HC3-robust OLS models adjusted for age, sex, disease duration and ICV

TABLE 1 Primary model (bilateral HATA): PMS associated with lower HATA volume after adjustment (β = −10.11 mm³; 95% CI −17.62 to −2.60; $p=0.008$)

Primary model: Bilateral HATA volume (mm ³)			
Predictor	β (mm ³)	95% CI	p
PMS (vs RRMS)	−10.11	[−17.62, −2.60]	0.008
Age (per year)	−0.02	[−0.38, 0.34]	0.995
Male sex	−3.44	[−9.74, 2.87]	0.283
Disease duration (per year)	−0.12	[−0.64, 0.41]	0.651
ICV (per 100,000 mm ³)	−0.79	[−0.42, −1.15]	<0.001

Conclusion: Bilateral HATA volume is reduced in PMS after adjustment for intracranial volume and clinical covariates, consistent with HATA as a candidate mesotemporal marker of progressive phenotype. Longitudinal, lesion-aware validation is warranted.

Disclosure: Nothing to disclose.

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Background and aims: Visual onset (VO) is common in multiple sclerosis (MS) and is associated with a better prognosis. Cerebrospinal fluid (CSF) biomarkers of intrathecal immunoglobulin synthesis are increasingly investigated for early prognostic stratification; however, the prognostic role of lambda-free-light-chain (L-FLC, Lambda) index remains unclear. This pivotal study investigated whether VO is associated with a distinct CSF biomarker profile, focusing on Lambda index.

Methods: This monocentric retrospective observational study included 217 MS-patients (2017 McDonald criteria) at diagnostic lumbar puncture, classified as VO ($n=50$) or non-VO (brainstem, cerebellar, sensory, motor, sphincteric, or spinal). Age at onset (AAO), disease duration (DD), and MRI features (lesion count, spinal cord involvement, and gadolinium-enhancing lesions) were collected. CSF/serum IgG, kappa and LFLC were collected to calculate IgG, Kappa, and Lambda indexes. Group comparisons (Student's, Mann-Whitney U and Chi-square test) and multivariable linear regression analyses were performed; sensitivity analyses used log-transformed Lambda values.

Results: VO-patients were younger than non-VO (mean $\pm$ SD: 36.0 ± 11.3 vs 42.0 ± 12.2 years), with no differences in MRI burden. Lambda index was significantly lower in VO (median [IQR]: 4.9 [10.4]) than non-VO (8.3 [17.0]; $p=0.019$). This difference remained after adjustment for AAO and DD, which were not independently associated with Lambda index, and persisted in sensitivity analyses. No differences were observed for other biomarkers.

Conclusion: In this pivotal study, VO is associated with a lower Lambda index despite similar radiological burden, suggesting a distinct intrathecal humoral responses across onset phenotypes. The Lambda-index may refine early prognostic stratification in MS and warrants validation in larger cohorts.

Disclosure: Nothing to disclose.

EPO-0990 | Quantitative gait analysis for the detection and longitudinal monitoring of progressive multiple sclerosis: Relevance of the semiogram

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Background and aims: In progressive multiple sclerosis (pMS), gait deterioration is a key marker of disability. To detect progression, conventional clinical scales such as the EDSS remains limited in sensitivity and granularity. Inertial sensors are relevant tools for quantifying gait quality in clinical settings. It may enable the development of digital movement biomarkers for detection and follow-up of progression, and to provide information for functional rehabilitation and therapeutic adaptation.

Methods: Twenty-two patients with pMS (11 women, mean age 58 years, mean EDSS 5.5 [3.5–6]) and 19 control subjects (12 women, mean age 51 years) performed 10-meter walking tests with u-turn every 6 months over 1 to 2 years, equipped with 3 inertial sensors. Parameters computed from accelerometry and gyrometry time-series were aggregated according to semiological criteria, compared to healthy population, and compiled into a visual radar named “semiogram” (see Figure). Correlations between these scores and established functional scales such as the EDSS were analyzed.

Results: The reproducibility of the parameters was excellent. All studied parameters, except stability, were able to discriminate patients from healthy subjects. A significant correlation was demonstrated between gait parameters and clinical scores, particularly the EDSS and motor subscores. It also support a better sensibility to progression compared with the EDSS.

Conclusion: Sensor-based gait analysis through a multidimensional score is an objective and sensitive biomarker of motor disability in pMS detection and follow-up. Sensors time-series provide clinically relevant information for personalized monitoring and rehabilitation, which will be crucial in coming years to evaluate new treatments for progressive forms of multiple sclerosis.

Disclosure: Nothing to disclose.

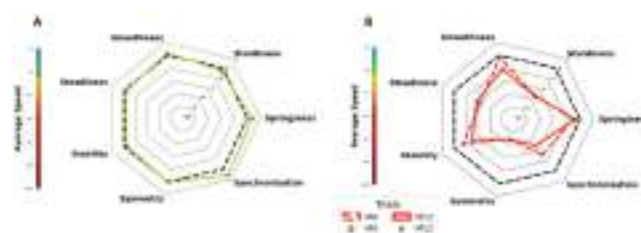


FIGURE 1 Semiograms. Each criterion is compared with the healthy population (dark dashed lines). (A) healthy subject. (B) patient with progressive multiple sclerosis at M0 and M12 (EDSS 5.5 unchanged, however notable motor deterioration).

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Background and aims: Multiple sclerosis (MS) is an inflammatory demyelinating disease of the central nervous system, in which sleep disorders are frequent but often underrecognized, contributing to disability and reduced quality of life.

Methods: We conducted a cross-sectional, descriptive, and analytical study including patients with MS at Habib Bourguiba University Hospital between January 2023 and September 2024. Sleep disorders were assessed using the Insomnia Severity Index (ISI), the Epworth Sleepiness Scale (ESS), the Pittsburgh Sleep Quality Index (PSQI), and the STOP-BANG questionnaire. Restless legs syndrome and parasomnias were investigated through clinical interviews. Clinical, radiological, and therapeutic data were extracted from medical records.

Results: Eighty patients with MS were included; 63.8% had at least one sleep disorder. Poor sleep quality (PSQI > 5) was found in 57.5%, moderate to severe insomnia in 27.5%, parasomnias in 25%, excessive daytime sleepiness in 28%, and restless legs syndrome in 21.3%. Sleep disorders were significantly associated with unemployment ($p=0.037$), longer diagnostic delay ($p=0.045$), higher EDSS score ($p=0.048$), and symptoms such as spasticity, neuropathic pain, and decreased libido ($p=0.026$, $p=0.018$, and $p=0.043$, respectively). Radiologically, lesion localization in the corona radiata was associated with the presence of sleep disorders ($p=0.006$). Therapeutically, baclofen ($p=0.004$) and hypnotics ($p=0.045$) were associated with these disorders.

Conclusion: Sleep disorders are commonly observed in patients with MS and are influenced by clinical, psychological, and radiological factors. Systematic screening and multidisciplinary management are essential to improve quality of life.

Disclosure: No

EPO-0992 | Clinical phenotype at onset predicts early functional outcomes in pediatric acquired demyelinating syndromes

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Background and aims: We assessed whether onset phenotype predicts functional severity at presentation and outcome at discharge in pediatric acquired demyelinating syndromes (ADS).

Methods: We retrospectively analyzed pediatric ADS ($n=65$), including myelin oligodendrocyte glycoprotein antibody-associated disease, classified at onset as encephalitic (acute disseminated encephalomyelitis/cortical/cerebral deficits), optic neuritis, brainstem/cerebellar, or myelitis. Functional severity was measured with the Pediatric Cerebral Performance Category (PCPC). PCPC at onset was compared using Kruskal–Wallis

TABLE 1 Demographic and clinical characteristics of the overall cohort and by onset phenotype in pediatric acquired demyelinating syndromes (ADS). Data are reported as n (%) or median (interquartile range).

Variable	Total (n=65)	Optic neuritis (n=16)	Encephalitic (n=23)	Brainstem/Cerebellar (n=15)	Myelitis (n=11)
Demographics (n (%))					
Age at onset (years; median (IQR))	7 (4-11)	6 (4-10)	6 (4-11)	6 (3-10)	6 (4-11)
Female:male ratio (n (%))	33 (50.8):32 (49.2)	16 (100):0 (0)	12 (52.2):11 (47.8)	8 (53.3):7 (46.7)	5 (45.5):6 (54.5)
Median time to diagnosis (months; median (IQR))	12 (3-20)	10 (3-18)	12 (3-20)	12 (3-20)	12 (3-20)
Length of hospital stay (days; median (IQR))	10 (3-15)	10 (3-15)	10 (3-15)	10 (3-15)	10 (3-15)
PCPC at onset (median (IQR))	3 (2-4)	3 (2-4)	3 (2-4)	3 (2-4)	3 (2-4)
PCPC at discharge (median (IQR))	4 (3-5)	4 (3-5)	4 (3-5)	4 (3-5)	4 (3-5)
Discharge to rehabilitation (%)	12 (18.5)	0 (0)	7 (30.4)	1 (6.7)	4 (36.4)
Relapse rate (%)					
Positive	12 (18.5)	0 (0)	7 (30.4)	1 (6.7)	4 (36.4)
Negative	53 (81.5)	16 (100)	16 (69.6)	14 (93.3)	7 (63.6)
Total (n=65)					

IQR, Interquartile Range; n, absolute; PCPC, Pediatric Cerebral Performance Category.

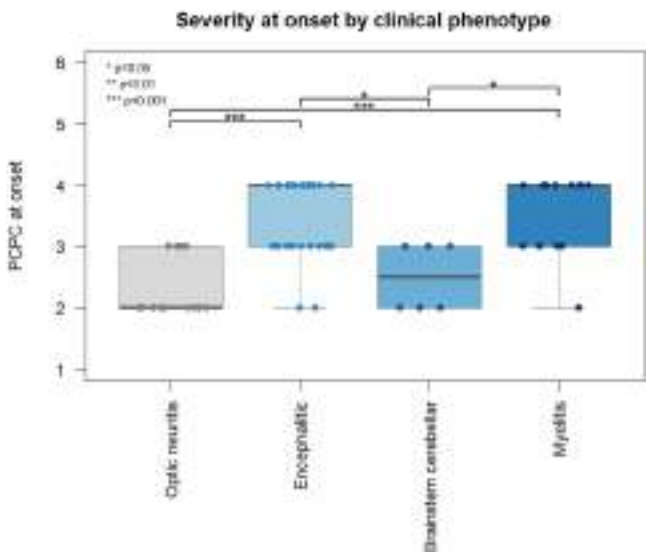


FIGURE 1 PCPC severity at onset by clinical phenotype in pediatric acquired demyelinating syndromes (ADS). Boxplots show median and interquartile range. Pairwise differences are based on Dunn's post-hoc test with Bonferroni correction.

with Dunn post-hoc tests. Ordinal logistic regression assessed associations between phenotype and PCPC at onset and discharge, adjusting for age, sex, and preceding infection. Length of stay was analyzed using linear regression.

Results: PCPC at onset differed across phenotypes ($p<0.001$); Dunn post-hoc showed higher PCPC in encephalitic vs optic neuritis and myelitis vs optic neuritis (both $p<0.001$). In adjusted ordinal models, encephalitic phenotype (OR 48.7, 95% CI 9.33–341; $p<0.001$) and myelitis (OR 58.0, CI 9.49–474; $p<0.001$) were associated with worse PCPC at onset; older age (OR 1.17, CI 1.02–1.37; $p=0.034$) and preceding infection (OR 3.10, CI 1.03–9.89; $p=0.048$) were also associated with higher PCPC. At discharge, encephalitic phenotype remained associated with worse PCPC (OR 9.01, CI 2.34–39.3; $p=0.002$); myelitis showed a trend (OR 3.96, CI 0.92–18.4; $p=0.069$); age remained associated (OR 1.17 per year, CI 1.02–1.34; $p=0.027$). Encephalitic phenotype was associated with longer hospital stay (CI 1.67–19.7; $p=0.021$).

Conclusion: In our cohort, onset phenotype strongly predicts functional severity and early outcome. Encephalitic

presentations carry the highest acute burden, persistently worse discharge status, and longer hospitalization, supporting phenotype-informed risk stratification at admission.

Disclosure: Nothing to disclose.

EPO-0993 | **Optical coherence tomography (OCT)-based retinal nerve fibre layer thickness associations with radiological data in lithuanian patients with MS**

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Background and aims: Longitudinal monitoring of multiple sclerosis (MS) relies mainly on repeated magnetic resonance imaging (MRI), which can be limited by cost, availability, and the need for contrast agents. The 2024 McDonald criteria now include the optic nerve as a fifth location for dissemination in space, enabling assessment with optical coherence tomography (OCT), a non-invasive and accessible alternative for tracking disease progression.

Methods: We conducted a retrospective medical documentation review of patients diagnosed with MS according to the 2010/2017 McDonald criteria, treated at the Department of Neurology of Kauno Klinikos, Lithuanian University of Health Sciences (LUHS) Hospital.

Results: A total of 84 MS patients were included, 85 % have relapsing-remitting disease course. Spearman's bivariate correlation analyses and odds ratio estimations using ordinal logistic regression were performed (Table 1). A marginally significant association was observed between changes in pRNFL thickness over time and the appearance of new periventricular lesions. An ordinal logistic regression analysis was performed to determine whether a single measurement of pRNFL thickness correlated with MRI lesion load, adjusted for age and gender. pRNFL thickness was a significant prognostic factor for MRI lesion load, with each 1 µm reduction in pRNFL thickness associated with a 5 % higher probability of being classified into a higher lesion category (Table 2).

TABLE 1 Correlation between MRI changes and peripapillary retinal nerve fiber layer (pRNFL) thickness changes over time in OCT measurements.

Lesion region	Spearman's ρ coefficient	Spearman correlation p-value	Odds ratio	95% confidence interval	p-value (OR)
Subcortical	-0.075	0.499	0.830	0.633-1.090	0.180
Periventricular	0.216	0.049	1.381	0.837-2.277	0.205
Infratentorial	0.056	0.613	1.051	0.936-1.181	0.395
Spinal cord	-0.018	0.869	0.992	0.923-1.065	0.818
Signs of activity	0.078	0.481	1.029	0.971-1.092	0.818

Spearman's ρ coefficients and p-values were obtained from bivariate correlation analysis. Odds ratios (OR), 95% confidence intervals (CI), and corresponding p-values were derived from ordinal logistic regression (PLUM method).

TABLE 2 Correlation between MRI changes (number of lesions) and pRNFL variation in a single OCT measurement.

Variables	B	SE	p-value	OR (Exp(B))	95% CI (OR)
Single OCT pRNFL	-0.052	0.023	0.023	0.95	[0.91, 0.99]
Gender	0.968	0.510	0.050	2.69	[1.00, 7.30]
Age	0.013	0.025	0.596	1.01	[0.96, 1.06]

Ordinal logistic regression predicting baseline MRI lesion load (dependent variable: MRI_total_1; higher values indicate a greater number of lesions). Dependence of MRI lesion changes on the predictor variables. SE – standard error; OR – odds ratio, CI – confidence interval.

Conclusion: No significant associations were found between single-timepoint pRNFL thickness and individual MRI lesion locations, while a weak borderline correlation with paraventricular lesion dynamics was considered a statistical artefact. Reduced pRNFL thickness was an independent predictor of higher overall MRI lesion burden.

Disclosure: Nothing to disclose.

EPO-0994 | **sNfL and sGFAP as prognostic biomarkers in pediatric multiple sclerosis**

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Background and aims: Pediatric multiple sclerosis (PedMS) is characterized by high inflammatory activity and neuroaxonal and glial damage. Serum biomarkers able to detect subclinical disease activity are relevant in this population. This study assessed the prognostic value of serum neurofilament light chain (sNfL) and glial fibrillary acidic protein (sGFAP) in predicting clinical, radiological, and cognitive outcomes in PedMS.

Methods: This study included PedMS followed at two Italian referral centers. All patients underwent cognitive assessment using the Symbol Digit Modalities Test (SDMT), physical disability evaluation using EDSS, as well as brain MRI scans and sNfL and sGFAP measurement by SIMOA and the Ella systems, at baseline and every 6 months for 24 months. Correlation analysis was performed using Spearman nonparametric test.

Results: Fifty-three PedMS [71.7% females; median age at onset: 14 (IQR 13.0–16.0) years; median EDSS: 2.0 (IQR 1.0–3.0); median SDMT scores: 49.0 (IQR 40.0–55.0)] were included. Baseline median levels of sNfL were 19.9 (IQR 7.5–37.0) pg/mL and those of sGFAP were 103.3 (IQR 86.6–131.3) pg/mL. Higher baseline sNfL levels were significantly ($p < 0.05$) associated with younger age at onset and, although not statistically significant

(range $p=0.05$ – 0.1), with increased physical and cognitive impairment and a treatment switch during follow-up. Elevated baseline sGFAP levels were significantly ($p<0.05$) associated with EDSS score at 24 months and higher number of clinical relapses ($p<0.05$) during the follow-up.

Conclusion: The integration of sNfL and sGFAP levels with clinical, radiological and cognitive assessments can improve early risk stratification and support personalised therapeutic strategies in PedMS.

Disclosure: Nothing to disclose.

EPO-0995 | Functional neurological symptoms in multiple sclerosis: A scoping review

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Background and aims: Functional neurological symptoms (FNS), including functional motor and sensory disturbances or non-epileptic seizures, may co-occur with multiple sclerosis (MS) and complicate both diagnosis and management. This scoping review aimed to synthesize available literature on the prevalence, clinical features, diagnostic approaches, and management strategies for FNS and functional overlay in individuals with MS.

Methods: Following the PRISMA-ScR guidelines, we searched PubMed, Embase, and PsycINFO from inception to July 2025. Studies were eligible if they reported FNS in people with MS. Two reviewers independently screened records, extracted data, and charted results. Data were summarized narratively across domains of prevalence, symptom types, diagnostic pathways, and management interventions.

Results: The search retrieved 1,042 records, of which 47 met inclusion criteria (15 observational studies, 20 case series, 12 single case reports). Reported prevalence of FNS varied widely, ranging from 3% to 21% across clinical samples. Functional motor symptoms (paresis, gait disturbance) and functional sensory loss were the most frequently described; psychogenic non-epileptic seizures and functional tremor were less common but

often led to diagnostic delay. Neuroimaging and neurophysiological testing were frequently used to distinguish functional from demyelinating symptoms, though misdiagnosis was reported in up to 15% of cases. Interventions most often involved multidisciplinary rehabilitation, psychological therapies, and patient education. Evidence for treatment effectiveness was limited, with most data from uncontrolled studies.

Conclusion: FNS are under-recognized but clinically relevant in MS. They may contribute to increased disability, healthcare utilization, and patient distress if not appropriately identified. This review highlights the need for high-quality prospective studies to evaluate targeted interventions.

Disclosure: Nothing to disclose.

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EPO-0996 | Reassessing PML risk in multiple sclerosis: The impact of anti-JCV antibody Assay selection

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Background and aims: Natalizumab (NTZ) treatment increases the risk of progressive multifocal leukoencephalopathy (PML) due to JC virus (JCV) reactivation in people with Multiple Sclerosis (pwMS). PML risk has been assessed for several years via the Stratify Test (ST), a reference anti-JCV antibody assay. A new assay, Immunowell JCV Test (IT), has recently entered clinical use with the introduction of biosimilar NTZ. The aim of our study is to reassess PML risk using the IT in comparison with the established assay ST.

Methods: We enrolled 169 NTZ-treated pwMS at Tor Vergata MS Center. Paired serum samples were collected and tested with both tests on the same day. Mean and median JCV index values were compared. Patients were categorized into four risk categories based on test-specific cut-offs, and frequency distributions were analyzed.

Results: Mean and median JCV index values were higher for IT (0.85 and 0.32) than for ST (0.63 and 0.00). Risk class distributions differed significantly between the two tests (McNemar test, $p<0.001$), especially at risk extremes. A discordant risk class was observed in 113 patients (66.9%). Sign and Wilcoxon tests confirmed a significant statistical difference, with IT assigning higher risk categories than ST in 107 cases, compared with only 6 cases in which ST assigned a higher class ($p<0.001$).

Conclusion: The new IT assay tends to reclassify patients into higher PML risk categories, which may influence clinical decisions. Further validation is needed to assess IT's specificity and correlation with JCV replication, to support accurate risk stratification and avoid unnecessary therapy changes.

Disclosure: Polovina J.C. received consultation fees for Sandoz. Landi D. received travel funding from Biogen, Merck Serono, Sanofi, Teva, Bristol Myers Squibb, Mylan, Neuraxapharm, speaking or consultation fees from Sanofi, Merck Serono, Teva, Biogen, Roche, Novartis, Bristol Myers Squibb, Janssen,

Alexion, Amgen Nicoletti C.G. received travel funding from Biogen, Merck Serono, Sanofi-Genzyme, Roche, Teva, Novartis, Bristol Mayer Squibb, Janssen, Almirall, Horizon, speaking and consultation fees from Sanofi, Almirall, Merck-Serono, Roche, Novartis, Biogen. She is a sub-investigator in clinical trials for Biogen, Merck Serono, Roche, Sanofi, Novartis, Teva, Bristol Mayer Squibb. Dionisi C. received travel fundings from Biogen, Merck Serono, Bristol Mayer Squibb, Almirall, Viatrix. Mataluni G. is an Advisory Board member of Argenx. Received honoraria for speaking, consultation fees or travel fundings from Almirall, Biogen Idec, Merck, Novartis, Sanofi, Roche, CSL Behring, Horizon, Kedrion, She is a sub-investigator in clinical trials for Biogen, Merck Serono, Novartis. Di Gianvito V: received travel fundings from Novartis, Biogen, Roche. G.A. Marfia is an Advisory Board member of Almirall, Amgen, Alexion, Neuraxpharm, Sandoz, Sanofi Genzyme, Merck-Serono, Novartis, Roche and received honoraria for speaking or consultation fees from Almirall, Bayer Schering, Biogen, Merck Serono, Neuraxpharm, Novartis, Sanofi-Genzyme, Roche, Amgen, Alexion and BMS. She is the principal investigator in clinical trials for Biogen Idec, Merck Serono, Novartis, Roche, Sanofi-Genzyme, Merck Serono and BMS. Napoli F, Miscioscia A, Campobasso M: nothing to disclose.

EPO-0997 | Changing paradigms in the placement of autologous stem cell transplantation for multiple sclerosis: A single-centre experience

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Background and aims: Autologous haematopoietic stem cell transplantation (aHSCT) has historically been considered a rescue therapy for multiple sclerosis (MS) due to safety concerns. Improved transplant strategies and better patient selection are expanding its use, particularly in treatment-naïve patients.
Methods: In this retrospective single-centre observational study, all patients with MS treated with aHSCT per clinical practice (Muraro et al, Nature Reviews Neurology 2025) since 2022, were included.
Results: Fourteen patients (7F), all with ‘aggressive MS’, were included (Table 1): 13 with relapsing-remitting and one with active-progressive MS. Median age was 26.8 years (range 20.2–33.8), median EDSS 3.3 (range 1.0–6.0), and median disease duration 1.5 years (range 0.2–12.5). Patients were treatment-naïve (6/14) or early switchers (7/14). Only one patient, with paediatric onset, displayed longer disease duration and several previous treatments. Follow-up was more than 24 months in one patient, 12 months in six patients, six months in two, three months in

TABLE 1 Baseline characteristics of patients.

Tot.	14 patients
Female sex – no. (%)	7 (50.0)
Disease course	
RR – no. (%)	13 (92.9)
AP – no. (%)	1 (7.1)
Previous treatments	
Naïve – no. (%)	6 (42.9)
Early switchers – no. (%)	7 (50.0)
Non-early switchers – no. (%)	1 (7.1)
Last DMD before transplant	
None – no.	6 (42.9)
NAT – no.	2 (14.3)
OCR – no.	2 (14.3)
OFA – no.	2 (14.3)
CLAD – no.	1 (7.1)
ALEM – no.	1 (7.1)
Age at enrolment – median (range), y	26.8 (20.2–33.8)
Disease duration at enrolment – median (range), y	1.5 (0.2–12.5)
EDSS at enrolment – median (range)	3.3 (1.0–6.0)
ARR at enrolment (95% CI)	1.7 (1.0–2.4)
Spinal cord involvement – no. (%)	13 (92.9)

Abbreviations: tot. = total; no. = number; RR = relapsing remitting; AP = active progressive; DMD = Disease Modifying Drugs; NAT = natalizumab; OCR = ocrelizumab; OFA = ofatumumab; CLAD = cladribine; ALEM = alemtuzumab; y = years; EDSS = expanded disability status scale; ARR = annualized relapse rate.

TABLE 2 Mobilization and conditioning regimens.

Tot.	14 patients
aHSCT completed – no. (%)	11 (78.6)
Apheresis completed – no. (%)	3 (34.3)
Mobilization completed – no. (%)	1 (7.1)
Mobilization regimens (12 patients)	
CYC 4 gr/m ² + G-CSF – no. (%)	11 (91.7)
CYC 4 gr/m ² + Hydrocortisone (relapse pre-mobilization) – no. (%)	1 (7.1)
CYC 4 gr/m ² + Plerixafor (poor mobilizer) – no. (%)	1 (7.1)
Steroids + G-CSF + Plerixafor (chemo-free regimen)* – no. (%)	1 (7.1)
Conditioning regimens (10 patients)	
BEAM+ATG – no. (%)	9 (81.8)
miniBEAM+ATG* – no. (%)	1 (9.1)
CYC+ATG† – no. (%)	1 (9.1)
Duration of follow-up – median (range), y	1.5 (1.3–2.2)

Abbreviations: tot. = total; aHSCT = autologous Hematopoietic Stem Cell Transplantation; CYC = cyclophosphamide; G-CSF = Granulocyte Colony Stimulating Factor; BEAM = busulfan, etoposide, cytarabine, melphalan; ATG = anti-thymocyte globulin.
*Chemo-free regimen was chosen due to previous CYC liver injury. In the same patient a conditioning regimen with mini-BEAM + ATG was chosen.
†CYC + ATG conditioning regimen was chosen in the patient with paediatric onset MS.

two, while three were still completing the procedure (2 post-apheresis, 1 post-mobilization). Mobilization and conditioning regimens are listed in Table2. No clinical relapses or MRI activity were observed after transplant. EDSS improved in 9/11 evaluable patients, one patient remained stable, whereas the progressive one worsened without inflammatory activity. Febrile neutropenia occurred in 9/11 patients and mild anti-thymocyte globulin reactions in 4/11. No late toxicities were reported.
Conclusion: Our findings support a paradigm shift toward earlier use of aHSCT in selected MS patients. Young age, short disease duration and low disability characterized our cohort. Larger studies with longer follow-up are needed to confirm efficacy and long-term safety.
Disclosure: IG received honoraria from Roche, Merck, Novartis, Biogen. GO has nothing to disclose. AG received compensation

for speaking activities from Novartis. MCA and VM have nothing to disclose. MAR received honoraria from Biogen, Bristol Myers Squibb, Eli Lilly, Janssen, Roche, AstraZaneca, Bromatech, Celgene, Genzyme, Horizon Therapeutics Italy, Merck Serono, Novartis, Sanofi and Teva; she receives research support from the MS Society of Canada, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla; she is Associate Editor for Multiple Sclerosis and Related Disorders. LL and AB have nothing to disclose. FC has nothing to disclose. RG has nothing to disclose. MF is Editor-in-Chief of the Journal of Neurology, Associate Editor of Human Brain Mapping, Neurological Sciences, and Radiology; received honoraria from Alexion, Almirall, Biogen, Novartis, Roche, Sanofi Bayer, Celgene, Chiesi Italia SpA, Eli Lilly, Genzyme, Janssen, Merck-Serono, Neopharmed Gentili, Novo Nordisk, Sanofi, Takeda, and TEVA, Bristol-Myers Squibb; he receives research support from Biogen Idec, Merck-Serono, Novartis, Roche, the Italian Ministry of Health, the Italian Ministry of University and Research, and Fondazione Italiana Sclerosi Multipla. LM received compensations for speaking activities and/or for participating to advisory board from Merck, Celgene, Biogen, Sanofi, Novartis, Roche, Alexion, and Amgen.

EPO-0998 | Clinical course, safety, and pregnancy outcomes in multiple sclerosis patients treated with cladribine: Single center real-world experience

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Background and aims: To evaluate clinical, hematological, and pregnancy outcomes of relapsing-remitting multiple sclerosis (RRMS) patients treated with cladribine in a real-world setting.

Methods: We retrospectively analyzed 145 patients (screened from 190 admissions) with active follow-up and regular data. Demographics, EDSS scores, relapse rates, laboratory results (Years 1–2), MRI findings, adverse events, and pregnancy outcomes were evaluated.

Results: The mean age was 42.4 years (range 23–67), and 67% were female ($n=97$). Mean disease duration was 13.6 years. Relapses occurred in 26 patients (18%) during treatment, observing a higher frequency in females. Clinical and radiological stabilization was achieved in the majority. Regarding safety, Grade 3 lymphopenia ($<500/\text{mm}^3$) occurred in approximately 6–7% of patients, while Grade 4 ($<200/\text{mm}^3$) severe lymphopenia was rare (1%). Common adverse events included fatigue, herpes zoster, and alopecia. A total of 14 pregnancies were recorded (1 paternal). Outcomes included healthy live births and some pregnancy losses (ectopic/chemical/stillbirth); no developmental issues were reported in infants.

Conclusion: Cladribine demonstrated high efficacy and a manageable safety profile in this expanded real-world cohort ($n=145$). The incidence of severe lymphopenia was consistent with current literature. While healthy live births were achieved, pregnancy planning and monitoring remain crucial due to potential risks.

Disclosure: Nothing to disclose.

EPO-0999 | Exploratory CSF metabolomic profiling across oligoclonal band types 1–5 using ^1H -NMR spectroscopy

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Background and aims: Oligoclonal band (OCB) patterns in cerebrospinal fluid (CSF) represent distinct intrathecal immunological states and are routinely classified into five types (OCB Types 1–5). While OCB classification provides important diagnostic and immunological information, the associated biochemical heterogeneity within the CSF has not been systematically explored. To investigate whether CSF metabolomic profiles differ across OCB Types 1–5 using proton nuclear magnetic resonance (^1H -NMR) spectroscopy, focusing on spectral-level variability rather than definitive metabolite identification.

Methods: To investigate whether CSF metabolomic profiles differ across OCB Types 1–5 using proton nuclear magnetic resonance (^1H -NMR) spectroscopy, focusing on spectral-level variability rather than definitive metabolite identification.

Results: Distinct differences in CSF spectral profiles were observed across OCB-defined groups, with partial separation between specific OCB types. Several ppm regions demonstrated statistically significant or trend-level differences across OCB classifications. Exploratory pairwise evaluations suggested that comparisons involving OCB Types 4 and 5 tended to display more pronounced spectral variability, with selected ppm regions contributing disproportionately to group-wise patterns.

Conclusion: This exploratory analysis indicates that CSF metabolomic profiles vary across OCB Types 1–5, reflecting biochemical heterogeneity associated with distinct intrathecal immunological states. Although evaluated at the spectral level without definitive metabolite identification, these findings support the potential of CSF-based ^1H -NMR metabolomics as

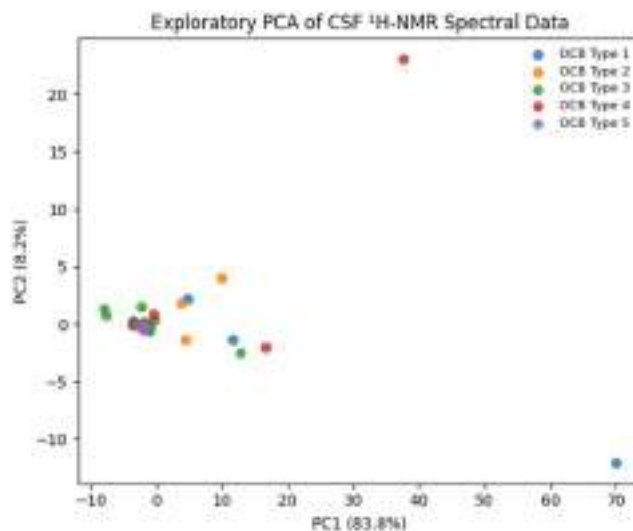


FIGURE 1 Exploratory PCA score plot based on CSF ^1H -NMR spectral data, with samples colored according to OCB Types 1–5. PCA was performed at the ppm level without targeted metabolite annotation.

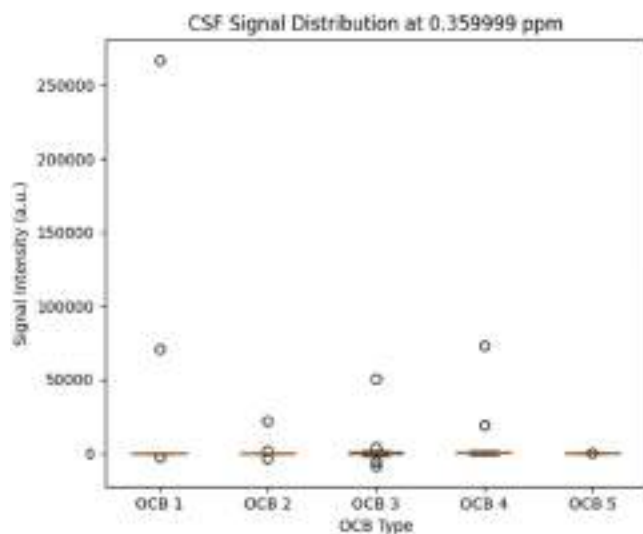


FIGURE 2 Distribution of CSF signal intensity at a representative ppm region across OCB Types 1–5.

a complementary approach to conventional OCB assessment. Further studies integrating targeted metabolite annotation and longitudinal clinical data are warranted.

Disclosure: Nothing to disclose.

EPO-1000 | From pregnancy to disability in multiple sclerosis: Can machine learning predict the course?

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Background and aims: Pregnancy reduces relapse activity in relapsing–remitting multiple sclerosis (RRMS), yet postpartum disability evolution is heterogeneous. We aimed to predict postpartum disability trajectories using pregnancy-related clinical and obstetric variables with machine-learning models.

Methods: We retrospectively analyzed 662 women with RRMS contributing 909 pregnancies. Predictors included pre-/postpregnancy

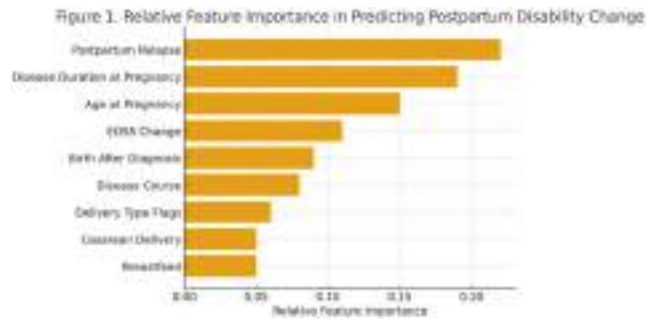


FIGURE 1 Relative feature importance in predicting postpartum disability change top predictors of postpartum disability change (Δ EDSS) in women with multiple sclerosis. Feature importance values were averaged across Random Forest and XGBoost models.

TABLE 1 Performance of classification models for Δ EDSS categories.

Model	Train Acc	Test Acc	Train Precision	Test Precision	Train Recall	Test Recall	Train F1	Test F1
Random Forest	0.9794	0.8681	0.9804	0.8485	0.9794	0.8881	0.9794	0.8581
XGBoost	0.9986	0.8516	0.9986	0.8305	0.9986	0.8516	0.9986	0.8388
Logistic Regression	0.8459	0.8571	0.8712	0.8501	0.8459	0.8571	0.8453	0.8532
Elastic Net	0.8459	0.8681	0.8732	0.8626	0.8459	0.8681	0.8434	0.8645
Support Vector Classifier	0.8583	0.8846	0.8565	0.8651	0.8583	0.8846	0.8527	0.8748

TABLE 2 Top 15 predictors of Δ EDSS across models.

Rank	Feature	P value
1	Postpartum Relapse	0.4911
2	Disease Duration at Pregnancy (years)	0.1050
3	Age	0.0770
4	Age at Pregnancy	0.0695
5	Pre-pregnancy EDSS	0.0560
6	Target EDSS	0.0559
7	Pregnancy Number	0.0317
8	Total Pregnancies	0.0253
9	Term Delivery	0.0203
10	RRMS Course	0.0170
11	Elective Termination	0.0163
12	SPMS Course	0.0138
13	Miscarriage	0.0110
14	Breastfeeding	0.0102
15	Birth After Diagnosis	0.0000

EDSS, disease duration, maternal age, postpartum relapse, pregnancy number, delivery type, and breastfeeding. After multiple imputation and standard preprocessing, regression (Δ EDSS) and 3-class classification (improved/stable/worsened) models were trained with an 80/20 split and 5-fold cross-validation (Random Forest, XGBoost, elastic net, and support vector methods).

Results: Classification generalized better than regression (test accuracy 85–88%, F1 0.84–0.87 vs test R^2 0.31–0.39; MAE 0.41–0.48 EDSS points). Postpartum relapse was the strongest predictor, followed by disease duration, age at pregnancy, and pre-pregnancy EDSS; obstetric variables showed smaller but relevant contributions. Performance was highest in women with multiple pregnancies (classification accuracy > 92%).

Conclusion: Machine learning can stratify postpartum disability risk in RRMS using routinely collected reproductive and clinical data, with categorical risk prediction outperforming continuous Δ EDSS prediction. Postpartum relapse remains the dominant driver of disability change, highlighting the postpartum period as a key therapeutic window; external validation is warranted.

Disclosure: Said Alizada: Nothing to disclose, Murat Emec: Nothing to disclose, Ergi Kaya: Nothing to disclose, Yasemin Simsek: Nothing to disclose, Mehmet Hilal Ozcanhan: Nothing to disclose, Serkan Ozakbas: Nothing to disclose.

EPO-1001 | Functional connectivity of sensorimotor network as function of EDSS: Preliminary resting state fMRI study in patients with secondary progressive MS

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Background and aims: Demyelinating lesions in multiple sclerosis (MS) cause injury to neuronal paths and therefore lead to poorer communication between distant brain regions. This communication may be measured with functional magnetic resonance at rest (r-fMRI) and visualized as synchronization of neuronal activity between these regions. Sensorimotor network links regions responsible for processing sensory input and executing motor output; these regions are interconnected and their activity is synchronized even at rest. This preliminary analysis evaluated if higher EDSS scores are associated with sensorimotor network connectivity with other brain regions.

Methods: Seventeen patients with a diagnosis of secondary progressive MS (EDSS: 6.47 ± 1.42 (range: 3.5–8.5), age: 54.1 ± 1.4 , 7 males) without enhancing lesions on MRI were included. High resolution T1-w images and functional magnetic resonance data at rest were obtained at 3T and processed with CONN software.

Results: Higher scores on EDSS were related with poorer connectivity of sensorimotor regions with multiple regions in occipital cortex and cuneal cortex that is associated with visual processing, visual attention, spatial processing and object

recognition (blue color on FIGURE 1). However, higher EDSS scores demonstrated increased connectivity in the right crus of the cerebellum (contains motor tracts traveling from the cerebral cortex; red color on FIGURE 1).

Conclusion: Higher EDSS scores are associated with poorer connectivity with regions involved in visual processing. However, the increased connectivity with crus of the cerebellum suggests some compensatory mechanisms.

Disclosure: Nothing to disclose. This study was supported by Medical Research Agency of Poland, grant 2021/ABM/02/00002 – 00.

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EPO-1002 | Brain volumetric MRI abnormalities associated with dysphagia in multiple sclerosis

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Background and aims: Multiple sclerosis (MS) patients may present dysphagia. The neuroanatomical alterations underpinning dysphagia in MS are debated.

Methods: In this cross-sectional study enrolling both MS patients and healthy controls (HCs), subjects underwent clinical assessment through Expanded Disability Status Scale (EDSS), Brief International Cognitive Assessment for MS (BICAMS), Swallowing Disturbance Questionnaire (SDQ), Beck Anxiety Inventory (BAI). Subjects performed brain MRI (T1/T2 sequences) at three sites. We assessed T2 lesions volume [lesion segmentation tool, SPM12, brain and gray matter volume (SIENAX), brainstem volume (Freesurfer), cerebellar volume (SUIT toolbox)]. We performed a multivariable linear regression using gray matter, T2 lesions, brainstem and cerebellar volume, BAI scores as factor of interest, SDQ as dependent variable and age, gender and sites as covariates.

Results: We enrolled 41 patients [30 females; age: 38.4 ± 10.4 years, 37 relapsing-remitting; disease duration: 8.5 (0–26) years; EDSS: 2.5 (1–5.5)] and 20 HCs (8 females; mean age 38.7 ± 13.4 years). Compared with HCs, MS patients had higher T2 lesion volumes (coeff. 6.97; $p=0.02$), lower normalized brain volume (coeff. -87.56 ; $p<0.01$) and reduced volumes of gray matter (coeff. -43.8 ; $p=0.001$), midbrain (coeff. -0.49 ; $p=0.003$), pons (coeff. -1.47 ; $p=0.002$) and medulla (coeff. -0.60 ; $p=0.003$) volume. Higher SDQ scores correlated with reduced volumes of pons (coeff. -0.93 ; $p=0.001$) and anterior cerebellar lobules (coeff. -1.12 ; $p=0.006$).



FIGURE 1 Regions in blue depict decreased functional connectivity with sensorimotor network in patients with higher EDSS scores, whereas the red regions in cerebral crus demonstrate better connectivity in patients with higher EDSS scores.

Conclusion: Dysphagia in MS may be caused by neurodegenerative processes affecting the infratentorial regions of the brain, leading to dysfunction of the cranial nerves that emerge from the pons and reduced motor coordination of the swallowing system related to anterior cerebellar damage.

Disclosure: Nothing to disclose.

EPO-1003 | Knowledge gaps in reproductive health among women with MS (KNOWwMS): An international survey

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Background and aims: Multiple sclerosis (MS) has typical onset in women during their reproductive years. Since reliable and consistent information about reproductive health is of pivotal importance for women with MS (WwMS), the KNOWwMS project was established to develop a core information set. As a first step, a global survey was conducted to assess access to information, knowledge gaps and misinformation concerning reproductive health in WwMS, focusing on limited resources settings.

Methods: The survey included 18 closed questions informed by a scoping review and refined by a multi-stakeholder Scientific Advisory Group, and one open-ended question. Target respondents of the online survey were WwMS aged ≥18 years. The survey was available from June to December 2025, in 9 languages.

Results: The questionnaire was completed by 480 WwMS (mean age 37.1 years, SD 9, 92% <50 years) from 31 countries (Figure 1: 23.5% from low- and middle- income countries), mostly from urban settings (91.9%). Gaps identified included: reproductive health issues never even discussed by their neurologist (42.6%), and absence of counselling about pregnancy while

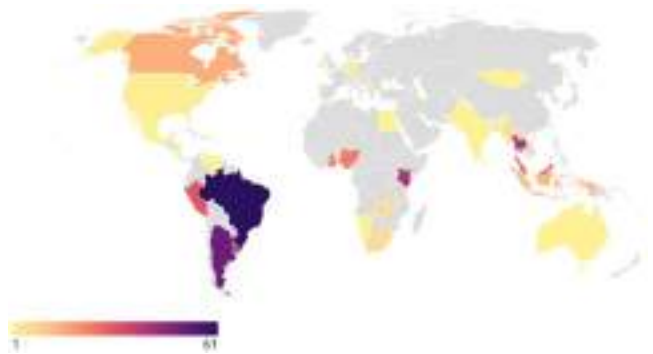


FIGURE 1 Geographic distribution of survey responses.

TABLE 1 Survey responses- Items 1 to 3.

1. When did you first or were often exposed to a health issue?	N (%)	
At diagnosis	186 (38.1)	
Before leaving MS treatment	184 (37.5)	
When planning pregnancy	94 (19.3)	
While pregnant	11 (2.3)	
Never	204 (42.6)	

2. Did you ever receive counselling about the following reproductive health topics?	No (%)	Yes (%)
Contraception while you have MS	186 (38.1)	94 (19.3)
Recording pregnant and having MS	340 (70.8)	140 (29.2)
Taking medications for MS and becoming pregnant	333 (69.3)	147 (30.7)
How MS and its medications may affect pregnancy	330 (68.3)	150 (31.7)
Reproductive and having MS	430 (89.5)	50 (10.5)
Reproductive while taking medications for MS	427 (88.7)	63 (13.3)

3. What is your main source of information on reproductive health issues (contraception, pregnancy, breastfeeding)?	Not selected (%)	Selected (%)
Neurologist	226 (47.1)	254 (52.9)
General practitioner	430 (89.6)	50 (10.4)
Other specialist	397 (82.7)	63 (13.3)
Family	401 (83.7)	79 (16.3)
Friends/family	429 (88.9)	51 (10.7)
Healthcare provider	429 (88.9)	51 (10.7)
Online media	430 (89.6)	50 (10.4)
Checklist	430 (89.6)	50 (10.4)
Internet	330 (68.3)	150 (31.7)
Friends/family	430 (89.6)	50 (10.4)
None	103 (21.4)	177 (36.9)

*For multiple response questions, participants could select more than one option. Therefore, percentages may not add up to 100%.

TABLE 2 Survey responses- Items 4 to 18.

Survey item	No (%)	Yes (%)	Not sure (%)	Not applicable (%)
4. Do you have access to contraception?	186 (38.1)	194 (40.4)	50 (10.4)	50 (10.4)
5. Do you have any concerns about contraception affecting reproduction of your MS?	186 (38.1)	94 (19.3)	136 (28.3)	50 (10.4)
6. Do you think MS will affect your ability to become pregnant and have children?	299 (62.1)	140 (29.2)	40 (8.3)	50 (10.4)
7. Do you think that if you have a diagnosed MS, you might become more pregnant?	406 (84.6)	144 (29.8)	40 (8.3)	110 (22.9)
8. Do you think pregnancy will worsen your MS symptoms?	131 (27.3)	140 (29.2)	180 (37.5)	50 (10.4)
9. Have you decided against having children due to concerns about MS?	300 (62.5)	140 (29.2)	50 (10.4)	50 (10.4)
10. Do you think that having MS while being pregnant affects the safety of the baby?	300 (62.5)	140 (29.2)	136 (28.3)	50 (10.4)
11. Do you think MS medications are safe for children?	186 (38.1)	140 (29.2)	140 (29.2)	50 (10.4)
12. Have you ever received counselling about pregnancy while taking medication for MS?	307 (63.8)	140 (29.2)	50 (10.4)	50 (10.4)
13. Do you think that if you get pregnant you will have to stop taking MS medications?	71 (14.6)	270 (56.3)	140 (29.2)	50 (10.4)
14. Have you ever heard of "pregnancy support"?	131 (27.3)	194 (40.4)	50 (10.4)	50 (10.4)
15. Do you have any concern that breastfeeding could worsen your MS symptoms?	147 (30.6)	140 (29.2)	136 (28.3)	50 (10.4)
16. Do you think that if you have MS you will have to stop taking MS medications?	186 (38.1)	140 (29.2)	50 (10.4)	50 (10.4)
17. Do you think that if you have MS you will have to stop taking MS medications?	131 (27.3)	140 (29.2)	50 (10.4)	50 (10.4)
18. Do you think that if you have MS you will have to stop taking MS medications?	131 (27.3)	140 (29.2)	50 (10.4)	50 (10.4)

NA, not applicable for the item.

taking medications for MS (44.1%). Most women believed, or were not sure, that MS would affect their ability to become pregnant and have children (57.9%); responses to MS being passed on

to children showed limited knowledge of familial risk (69% no, or unsure); 34.5% decided against having children due to MS-related concerns (Tables 1 and 2).

Conclusion: This global survey identified significant knowledge gaps among WwMS on key aspects of fertility, childbearing, and treatment safety. These findings will inform the core information set for WwMS.

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EPO-1004 | Cancer unleashes multiple sclerosis activity: An Italian multicenter matched cohort study

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Background and aims: Despite being an established autoimmunity driver, the potential impact of cancer on MS activity remains largely unexplored. This study aims to compare the risk of MS reactivation in the 12 months before and after cancer diagnosis with that observed in a matched cohort of MS patients without cancer.

Methods: We conducted a retrospective cohort study including MS patients with cancer from four Italian MS centers, matched 1:1 by sex and age to pwMS without cancer. Index date corresponded to cancer diagnosis date in cases and was aligned by MS disease duration in controls. MS activity was assessed longitudinally collecting relapses and new T2/Gd+ lesions during the

24 months peri-index date. To account for repeated MRI scans within individuals, generalized estimating equation (GEE) negative binomial models were fitted, adjusting for DMT class at enrollment and MRI region, and including an interaction term between cancer status and time.

Results: We included 148 patients with cancer and 148 controls. During the peri-index period at least one clinical relapse occurred in 20.7% (30) of cancer patients, while no relapses were observed among controls ($p < 0.001$). At least one new T2 and/or Gd+ lesion was detected in 29.7% (78/263) of MRI scans from cases, compared with 6.7% (17/254) from control ($\chi^2 = 41.2, p < 0.001$). Adjusted GEE models showed greater increase in T2 and/or Gd+ lesion accrual over time among cases ($p = 0.019$).

Conclusion: Cancer development appears to trigger disease reactivation, possibly reflecting interactions between immunosurveillance and autoimmunity, warranting tailored MS management strategies and oncological vigilance in unexpected MS reactivations.

Disclosure: V. DI GIANVITO received travel fundings from Novartis, Biogen, Roche. M. PONZANO nothing to disclose J. FRAU scientific advisory board and honoraria as a speaker and consultant from Alexion, Amgen, Biogen, Bristol, Novartis, Merck, Sanofi, Sandoz. M. LUCCHINI has received honoraria from Biogen, Merck Serono, Novartis, Roche, Sanofi-Genzyme, Bristol-Myers Squibb, Horizon for consulting services and/or speaking honoraria E. SIGNORIELLO received personal compensation from Almirall, Biogen, Sanofi, Novartis, Roche, Horizon, Alexion, Merck, Mylan and Teva for traveling and advisory boards. L. VENTURA nothing to disclose E. FEDELE nothing to disclose F. NAPOLI nothing to disclose C.G. NICOLETTI received travel funding from Biogen, Merck Serono, Sanofi-Genzyme, Roche, Teva, Novartis, Bristol Mayer Squibb, Janssen, Almirall, Horizon, speaking and consultation fees from Sanofi, Almirall, Merck-Serono, Roche, Novartis, Biogen. She is a sub-investigator in clinical trials for Biogen, Merck Serono, Roche, Sanofi, Novartis, Teva, Bristol Mayer Squibb. G. MATALUNI is an Advisory Board member of Argenx. Received honoraria for speaking, consultation fees or travel fundings from Almirall, Biogen Idec, Merck, Novartis, Sanofi, Roche, CSL Behring, Horizon, Kedrion, She is a sub-investigator in clinical trials for Biogen, Merck Serono, Novartis. M. MIRABELLA received honoraria for speaking, advisory board/consulting from Biogen, Novartis, Merck Serono, Roche, Almirall, Sanofi Genzyme, Janssen, Bristol-Myers Squibb, Viartis, Alexion. He is principal investigator in clinical trials for Biogen, Merck-Serono, Novartis, Roche, Sanofi Genzyme, Argenx. G. LUS received speaker honoraria and/or consultancy from Biogen, Teva, Genzyme, Merck, Novartis, Almirall and Roche E. COCCO serves on scientific advisory boards and received honoraria for speaking from Alexion, Biogen, BMS, Janssen, Merck, Novartis, Roche, and Sanofi Genzyme. D. CENTONZE acted as an Advisory Board member of Almirall, Bayer Schering, Biogen, GW Pharmaceuticals, Merck Serono, Novartis, Roche, Sanofi-Genzyme, Teva, Protagon, Sandoz, Bristol-Myers Squibb and Alexion and received honoraria for speaking or consultation fees from Almirall, Bayer Schering, Biogen G.A. MARFIA is an Advisory Board member of Almirall, Amgen, Alexion, Neuraxpharm, Sandoz, Sanofi Genzyme, Merck-Serono, Novartis, Roche and received honoraria for speaking or consultation fees from Almirall, Bayer Schering, Biogen, Merck

Serono, Neuraxpharm, Novartis, Sanofi-Genzyme, Roche, Amgen, Alexion and BMS. She is the principal investigator in clinical trials for Biogen Idec, Merck Serono, Novartis, Roche, Sanofi-Genzyme, Merck Serono and BMS. D. LANDI received travel funding from Biogen, Merck Serono, Sanofi, Teva, Bristol Myers Squibb, Mylan, Neuraxapharm, speaking or consultation fees from Sanofi, Merck Serono, Teva, Biogen, Roche, Novartis, Bristol Myers Squibb, Janssen, Alexion, Amgen

EPO-1005 | Dynamics of choroid plexus volume in multiple sclerosis patients treated with hematopoietic stem cell transplantation

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Background and aims: Choroid plexus volume (ChPv) is a potential magnetic resonance imaging (MRI) biomarker of immune cell trafficking into the central nervous system (CNS). Increased ChPv is associated with focal and chronic inflammation in multiple sclerosis (MS). While some therapies reduce ChPv in patients with recent inflammation, data regarding autologous hematopoietic stem cell transplantation (AH SCT) are lacking. We therefore investigated longitudinal ChPv changes in MS patients after AH SCT.

Methods: Consecutive relapsing MS patients undergoing AH SCT (BEAM-ATG conditioning) underwent standardized 3T MRI at baseline, 6–12 months post-AH SCT, and then yearly. ChPv and total brain volume (TBV) were segmented using FreeSurfer, T2-lesion volume (T2LV) manually by an expert operator.

Results: Twelve patients (92% female; 42% relapsing-remitting MS) were included. At baseline, 92% lacked recent focal inflammatory activity. During a median 26-month follow-up, two patients relapsed (months 8 and 15) with new spinal cord lesions only. Post-AH SCT, a median 7% ChPv reduction occurred at 6 months; the patients who relapsed both showed ChPv increase preceding disease activity. TBV decreased by 3% at 6 months, stabilizing at 12 months. No new brain lesions were detected after AH SCT, except for one case showing a mild increase in T2LV at month 6 without changes in ChPv.

Conclusion: ChPv decreased post-AH SCT regardless of recent focal inflammation, suggesting an effect on CNS cell trafficking independent from this latter and supporting AH SCT effect on both peripheral and compartmentalized inflammation. ChPv increases preceded spinal cord-only relapses, supporting the role of the choroid plexus as an entry point for pathogenic lymphocytes regardless of lesion location.

Disclosure: Nothing to disclose.

EPO-1006 | Multimodal assessment of optic nerve involvement in multiple sclerosis

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Background and aims: The 2024 McDonald criteria for multiple sclerosis (MS) have formally recognized the optic nerve as a fifth anatomical site for demonstrating dissemination in space. This study aimed to assess the diagnostic accuracy of visual evoked potentials (VEPs), magnetic resonance imaging (MRI), and optical coherence tomography (OCT) in detecting optic neuritis (ON), and to identify the most effective test combinations.

Methods: We performed a cross-sectional analysis including 33 patients with relapsing–remitting MS (pwRRMS) and unilateral ON (ON+), 33 age- and sex-matched patients with other inflammatory neurological diseases (ONIND), and 107 pwRRMS without a clinical history of ON. All participants underwent VEP, MRI, and OCT. OCT peripapillary and macular scans followed OSCAR-IB QC4 and APOSTEL 2.0 standards. Diagnostic performance was evaluated using receiver operating characteristic (ROC) curve analysis.

Results: Compared with ONIND, VEPs showed the highest diagnostic accuracy in ON+ patients (AUC 0.9072, 93.94% sensitivity and 87.5% specificity). OCT demonstrated high specificity (92.19%) but lower sensitivity (69.7%; AUC 0.8094), while MRI reached 100% specificity with 63.64% sensitivity. The most accurate strategy was MRI or VEP plus OCT (AUC 0.931; sensitivity 84.85%; specificity 96.88%). The “one-out-of-three” and “two-out-of-three” rules also performed well (AUC 0.914 and 0.8935). Among pwRRMS without clinical ON, 9.8% fulfilled the MRI or



FIGURE 1 Multimodal characterization of optic neuritis. DIR MRI demonstrates acute right optic nerve involvement (A, red arrow). OCT reveals peripapillary retinal nerve fiber layer thinning consistent with left optic neuritis sequelae (B), while VEPs show delayed.

Diagnostic test	AUC	P-value	Sensitivity (%)	Specificity (%)	Optimal trade-offs	Maximal Index
VEP	0.9072	<0.0001	93.94	87.5	7.316	83.44
OCT	0.8094	<0.0001	69.7	92.19	8.021	81.89
MRI	0.8182	<0.0001	63.64	100	7.316	83.44
VEP + OCT	0.9317	<0.0001	84.85	96.88	7.316	83.44
VEP + MRI	0.9298	<0.0001	84.85	100	7.316	83.44
MRI + OCT	0.9204	<0.0001	63.64	100	7.316	83.44
1 out of 2	0.9142	<0.0001	100	91.91	8.021	83.44
1 out of 3	0.8935	<0.0001	81.82	96.88	8.021	76.7
2/3	0.9178	<0.0001	84.85	100	7.316	83.44
MRI or VEP + OCT	0.931	<0.0001	84.85	96.88	7.316	83.44

FIGURE 2 ROC-based comparison of diagnostic strategies for optic neuritis. ROC curves compare single-modality and combined diagnostic approaches. Optimal trade-offs between sensitivity and specificity are highlighted, with red indicating highest sensitivity.

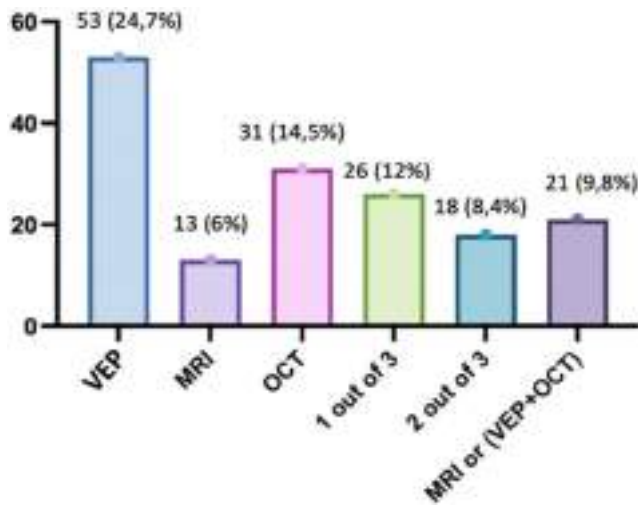


FIGURE 3 Subclinical optic nerve involvement in RRMS patients without optic neuritis. Bar graph depicting the number and percentage of eyes testing positive on individual and combined diagnostic tests in 107 RRMS patients (214 eyes) with no prior optic neuritis.

(VEP + OCT) criterion, 8.4% were positive on at least two tests, and 37.8% on at least one.

Conclusion: A multimodal assessment improves detection of optic nerve involvement and may identify subclinical damage, with different criteria applicable according to clinical context.

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EPO-1007 | Assessment of dietary habits in patients with multiple sclerosis in Opole and Silesian Province, Poland

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Background and aims: The aim of the study was to assess dietary habits in patients with multiple sclerosis (MS) in relation to psychosocial factors and quality of life.

Methods: The study included 91 patients with MS (68 women and 23 men) with a mean age of 49.8±11.0 years. Dietary patterns were assessed using the Pro-Healthy Diet Index-10 (pHDI-10) and the Non-Healthy Diet Index-14 (nHDI-14), derived from the Dietary Habits and Nutrition Beliefs Questionnaire (KomPAN). Additionally, a nutritional interview was conducted, and the Hospital Anxiety and Depression Scale (HADS), Visual Analogue Scale (VAS), and the EQ-5D-5L quality of life questionnaire were administered.

Results: The intensity of both health-promoting (pHDI-10) and health-adverse (nHDI-14) dietary characteristics was low in examined MS patients. In 60.4% of participants, the daily energy intake was lower than the total metabolic rate. The majority of respondents had a below-average intake of dietary fiber (72.5%), vitamin D (93.4%), and iron (70.3%). Higher intakes of protein and saturated fatty acids were associated with higher nHDI-14 scores, whereas higher intakes of dietary fiber, calcium, magnesium, and vitamin B12 were associated with higher pHDI-10 scores. Men demonstrated significantly higher nHDI-14 levels than women. Neither disease duration nor disability level had a significant effect on dietary habits. Dietary habits were associated with pain intensity and quality of life, but not with depressive or anxiety symptoms.

Conclusion: Nutritional education should be an integral component of the comprehensive management of MS patients, as it may contribute to improving dietary habits and quality of life.

Disclosure: Nothing to disclose.

EPO-1008 | Abstract withdrawn

EPO-1009 | Characterization of natural killer cell aging and terminal differentiation in multiple sclerosis

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Background and aims: Insights into immune aging and related functional impairment are essential for advancing understanding of multiple sclerosis (MS) course and pathophysiology and may inform individualized treatment decisions. CD57 expression on CD56dim natural killer (NK) cells and its proportion within the total NK cell population is a well-established indicator of mature NK cells. Here, we compared immunosenescence in patients with MS (pwMS) versus healthy controls (HCs) and evaluated influential covariates.

Methods: From an ongoing study, peripheral blood mononuclear cells from 69 pwMS and 10,437 HCs in Austria, Switzerland, and Germany were analyzed using flow cytometry, gated on CD3, CD56, CD57, NKG2D, DNAM-1, NKG2A, and TIGIT. Age-adjusted z-scores for CD57+CD56dim NK-cell proportions were estimated using a generalized additive model based on the HCs.

Results: Among 69 pwMS, 65.2% were female with a mean age of 40.5 years (SD 10.2), mean disease duration of 10.5 years (SD 9.47), and median EDSS of 2 (IQR 0–3). Age-adjusted z-scores for

CD57 on CD56dim NK cells were comparable between pwMS and HCs. No differences were observed by sex or disease course. Notably, the proportion of the immunoregulatory CD56bright cells was significantly reduced in pwMS.

Conclusion: CD57+CD56dim NK cells are a robust marker for NK-cell aging and may serve as a reliable surrogate of immunosenescence. Age-adjusted z-scores for the proportions of CD57+CD56dim NK cells can be estimated in pwMS to assess accelerated immune-cell aging. In future studies, this may be useful to assess the impact of immunosenescence on disease activity and progression.

Disclosure: Nothing to disclose.

Neurorehabilitation and Neurotraumatology

EPO-1010 | When copeptin and vasopressin diverge: Neuroendocrine dissociation and diffuse axonal injury in traumatic brain injury

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Background and aims: Traumatic brain injury (TBI) can disrupt hypothalamic–pituitary regulation, leading to neuroendocrine alterations that may reflect injury severity. Copeptin and arginine vasopressin originate from the same precursor and are expected to show parallel regulation under physiological conditions. This study evaluated the relationship between copeptin and vasopressin levels in TBI and their association with injury severity and diffuse axonal injury (DAI).

Methods: Eighty-six adult patients admitted with TBI to a tertiary neurotrauma center were included. Clinical severity was assessed using the Glasgow Coma Scale (GCS). Serum copeptin and vasopressin levels were measured, and neuroimaging data from computed tomography and magnetic resonance imaging were reviewed to identify traumatic lesions, including DAI.

Results: Copeptin and vasopressin levels demonstrated a significant inverse correlation (Spearman $\rho = -0.45$, $p < 0.01$), indicating dissociated regulation following TBI. DAI was identified in 9.3% of patients and was associated with significantly higher copeptin levels compared with those without DAI ($p = 0.045$). Higher copeptin concentrations were observed in patients with lower GCS scores. In contrast, vasopressin levels showed a positive correlation with GCS ($\rho = 0.366$, $p = 0.003$) and were significantly lower in patients with unfavorable outcomes, including mortality ($p = 0.006$) and the need for neurosurgical intervention ($p = 0.048$).

Conclusion: TBI is associated with altered copeptin and vasopressin regulation, resulting in a dissociated neuroendocrine profile. Within this context, elevated copeptin levels and their association with DAI suggest that copeptin may reflect axonal damage severity, supporting its further evaluation as a biomarker in neurotrauma research.

Disclosure: Nothing to disclose.

EPO-1011 | Dynamic EEG markers of confusional state and cognitive outcome after moderate-to-severe traumatic brain injury

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Background and aims: Cognitive impairment is a frequent and disabling consequence of moderate-to-severe traumatic brain injury. Post-traumatic amnesia (PTA) duration is a well-established prognostic marker of global disability, but its specificity for long-term cognitive outcome is limited. After emergence from PTA, recently conceptualized as part of the post-traumatic confusional state (PTCS), overt confusional symptoms typically resolve, while residual cognitive deficits persist. EEG may provide complementary prognostic information across this transition.

Methods: Forty-two patients with moderate-to-severe TBI admitted to inpatient rehabilitation underwent resting-state EEG at admission and discharge. EEG abnormalities were graded using the semi-quantitative Grand Total EEG score (GTE), with exploratory analyses of slow wave activity and background activity subdomains. Cognitive outcome at six months was assessed using the Montreal Cognitive Assessment (MoCA), while disability was evaluated using the Disability Rating Scale (DRS).

Results: At admission, GTE was associated with PTA/PTCS severity. At discharge, SWA showed substantial normalization, consistent with resolution of PTCS, whereas Background Activity abnormalities persisted in a subset of patients, suggesting ongoing cortical disorganization. In multivariate logistic regression models predicting MoCA impairment at 6 months, discharge GTE score was an independent predictor (OR = 2.61, 95% CI 1.47–4.66, $p = 0.002$). In contrast, PTA/PTCS duration was strongly associated with DRS ($p < 0.001$).

Conclusion: EEG provides prognostic information complementary to PTA/PTCS duration after severe TBI. While PTA/PTCS duration remains a robust predictor of global disability, EEG—particularly background organization at T1—better captures vulnerability to persistent cognitive impairment. EEG thus emerges as a dynamic prognostic tool in the subacute phase of TBI.

Disclosure: Nothing to disclose.

EPO-1012 | Effects of localized muscle fatigue on dynamic balance in healthy young adults: A randomized crossover study

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Background and aims: Muscle fatigue is traditionally associated with impaired postural control; however, accumulating evidence suggests that exercise performed to the point of fatigue may also elicit short-term adaptive responses within the neuromuscular system. The effects of exercise-induced localized muscle fatigue, particularly when applied to different body segments, remain insufficiently understood. This study aimed to

investigate the effects of exercise protocols designed to induce localized muscle fatigue of the upper and lower limbs on dynamic balance in healthy young adults.

Methods: A randomized, controlled crossover study was conducted in 32 healthy young adults. Dynamic balance was assessed using the Y Balance Test before and immediately after exercise protocols targeting either the upper or lower limbs and designed to induce localized muscle fatigue. Sessions were separated by at least 48 hours to minimize carry-over effects. Heart rate and perceived exertion were monitored to ensure that the physiological response was predominantly muscular rather than cardiorespiratory.

Results: Following the exercise protocols, significant increases in reach distance were observed in the Y Balance Test across most directions, indicating short-term changes in dynamic balance performance. No significant differences were found between upper- and lower-limb exercise protocols. No significant associations were observed between self-reported physical activity levels and balance changes.

Conclusion: Exercise protocols designed to induce localized muscle fatigue were associated with short-term improvements in dynamic balance, regardless of the body segment involved. Although conducted in healthy individuals, these findings may inform the design of training and rehabilitation strategies incorporating controlled exposure to exercise-induced fatigue.

Disclosure: Nothing to disclose.

EPO-1013 | SPInal CSF leaks in chronic subdural hematoma (SPICE): Interim results of a prospective observational trial

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Background and aims: Chronic subdural hematoma (cSDH) is the most common intracranial hemorrhage, characterized by slowly expanding subdural fluid collections that ultimately cause brain compression and neurological impairment. The underlying pathophysiology remains incompletely understood. An increasingly recognized and directly treatable cause are spontaneous spinal cerebrospinal fluid (CSF) leaks. This study aimed to estimate the prevalence of spinal CSF leaks in patients presenting with cSDH.

Methods: This interim analysis derives from a prospective, monocentric observational study of patients admitted with cSDH to the Department of Neurosurgery, University Medical Center Freiburg (07/2024–12/2025; NCT06323434). Standard cSDH treatment was followed by MRI of the brain and spine according to standard-workup in spontaneous intracranial hypotension. Patients with intermediate leak risk (Bern Score ≥ 3) or orthostatic headache underwent dynamic myelography. The primary outcome was the proportion of confirmed CSF leaks.

Results: Of 159 eligible patients, 132 consented; 22 withdrew. Six patients are awaiting myelography, and 20 were lost to follow-up before myelography. Among the 84 patients who underwent myelography, a spinal CSF leak was identified in 16.7%



FIGURE 1 Pipeline and interim results of the SPICE study: prevalence of underlying spontaneous spinal CSF leaks in patients with chronic subdural hematoma.

($n = 14$, 95% CI 10–26%), with a majority of CSF–venous fistulas ($n = 12$, 14.3%, 95% CI 8–23%). A more restrictive analysis yields a CSF leak prevalence of 13.5% (95% CI 8.2–21.3%) among the 104 primarily included patients.

Conclusion: Spinal CSF leaks represent a relevant and directly curable cause in a substantial proportion of patients with cSDH. These findings challenge prevailing assumptions regarding the relationship between spinal CSF leaks and cSDH and have important implications for future diagnostic pathways.

Disclosure: none

EPO-1014 | Daytime dysfunction in military personnel following blast injury

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Background and aims: Blast injuries (BI) are a leading cause of trauma among Ukrainian military personnel (MP), often resulting in mild traumatic brain injury (mTBI) and sleep disturbances, particularly daytime dysfunction (DD). Both mTBI and anxiety may contribute to DD, but their relative impact remains unclear.

Methods: 136 male MPs with BI (median 36.0; range 22–56) were enrolled. Participants were divided into two groups: BI with mTBI ($n = 62$; 45.6%) and without mTBI ($n = 74$; 54.4%). Only acute mTBI cases (≤ 21 days) were included. Anxiety was assessed using the Hospital Anxiety and Depression Scale. DD was measured using component 7 of the Pittsburgh Sleep Quality Index and dichotomized: 0 = no disturbance (score < 2), 1 = DD (score ≥ 2). Binomial logistic regression models evaluated associations between DD, mTBI and anxiety.

Results: The model demonstrated good fit (AIC=151; McFadden $R^2 = 0.201$). Anxiety severity was significantly associated with DD ($\beta = 0.209$, SE=0.047, OR=1.23, 95% CI 1.13–1.35, $p < 0.001$). mTBI was also linked to higher DD risk ($\beta = 1.066$, SE=0.407, OR=2.90, 95% CI 1.31–6.45, $p = 0.009$). In Model 2, the anxiety $\times$ mTBI interaction did not improve fit. Anxiety remained a significant predictor of DD (OR=1.23, $p = 0.002$), whereas mTBI status (OR=2.64, $p = 0.327$) was not statistically significant.

Conclusion: Final analyses indicated that anxiety was the only statistically significant predictor of DD irrespective of mTBI status.

Disclosure: Nothing to disclose.

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Background and aims: Voice onset time (VOT) is an auditory-based acoustic feature used to distinguish stop consonants in various languages. Children with sensorineural hearing impairment (HI) often struggle with discriminating between voiced and voiceless speech sounds. The role of this auditory ability in cochlear implant recipients has not been well understood. This study aimed to compare VOT contrast perception among Cochlear Implant (CI), Hearing Aid (HA) recipients, and Normal Hearing (NH) children.

Methods: A total of 60 subjects aged 6 to 12 years, divided into NH, HA, and CI groups, were selected. Test stimuli included 180 tokens of consonant-vowel-consonant combinations of non-sense Hindi stop phonemes with varying initial positions. These stimuli were presented in an ABX paradigm to assess identification scores.

Results: Statistical analysis revealed significant differences in VOT scores among all groups. There was no overlap in identification scores between NH & CI and HA & CI groups. CI children performed better than HA recipients but poorer than NH children. Significant differences were observed in scores between NH and both HA and CI groups, as well as between HA and CI groups. These findings align with previous research.

Conclusion: Children with CI and HA demonstrated different VOT discrimination abilities compared to NH children, likely due to limitations of CI and HA devices in processing speech signal characteristics. Older CI children with speech and language training and longer device use performed better than the HA group, suggesting that CI may enhance access to speech information and improve perception.

Disclosure: Nothing to disclose.

EPO-1016 | Neuroeducation and mindfulness-based meditation as novel group-based neurorehabilitation approach to improve cognition in multiple sclerosis

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Background and aims: To evaluate the efficacy of a novel group-based neurorehabilitation approach, combining neuroeducation with mindfulness-based meditation, in reducing self-perceived but also objective cognitive deficits in people with relapsing-remitting multiple sclerosis (pwRRMS).

Methods: In this ongoing study, pwRRMS aged 18–50 years with EDSS <4 and disease duration ≤10 years are enrolled. Participants attend a group-based program (5–8 per group) consisting of seven MS-specific neuroeducation sessions (90 minutes, weekly) led by a trained neuropsychologist, each followed by 10–15 minutes of mindfulness-based meditation supported by home practice via an in-house app. Objective neuropsychological and patient-reported outcomes are assessed before

and after the 7-week intervention, with the Perceived Deficits Questionnaire (PDQ-20) as the primary outcome measure.

Results: Currently, 57 participants have been included. The PDQ-20 showed a significant reduction in perceived cognitive deficits ($d=0.39$). Change scores showed a significant increase in information processing speed (IPS) with moderate effect size (SDMT, $d=0.63$). More pronounced cognitive impairment at baseline tended to be associated with larger IPS gains. Moreover, significant improvements were observed in fatigue (FSMC, $d=0.47$), anxiety (HADS-A, $d=0.54$), and depression (HADS-D, $d=0.61$), together with increased coping self-efficacy (CSES, $d=0.61$), general self-efficacy expectation (SWE, $d=0.66$), and reduced perceived stress (PSS, $d=0.53$). Further, neuropsychological testing showed improved working memory.

Conclusion: These interim findings indicate that the combination of neuroeducation and mindfulness-based meditation significantly enhances well-being and additionally shows promising effects on cognitive functioning in pwRRMS.

Disclosure: Naomi Gyger: nothing to disclose. Tobias Monschein: has participated in meetings sponsored by, received speaker honoraria or travel funding from Biogen, BMS/Celgene, Merck, Novartis, Roche, Sanofi, Genzyme and Teva. Tania Geiger: nothing to disclose. Fritz Leutmezer: has participated in meetings sponsored by, received speaker honoraria or travel funding from Actelion, Almirall, Biogen, BMS/Celgene, Johnson&Johnson, MedDay, Merck, Novartis, Roche, Sanofi-Genzyme and Teva, and received honoraria for consulting Biogen, BMS/Celgene, Merck, Novartis, Roche, Sanofi-Genzyme and Teva. Gisela Pusswald: nothing to disclose. Iris-Katharina Penner: Almirall, Biogen, BMS, Celgene, Genzyme, Janssen, Merck, Novartis, Roche, Teva // speakers bureau or advisory board, consulting fees; The German MS Society, Celgene, Novartis, Roche, Teva // research grants.

EPO-1017 | Effect of rTMS on neuropsychological outcomes in mild to moderate traumatic brain injury: A prospective cohort study

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Background and aims: Traumatic brain injury (TBI) is a major global public health problem and a leading cause of long-term cognitive, behavioural, and psychological disability, particularly among the productive age group. Disruption of fronto-cortical networks and underlying white matter tracts has been strongly implicated in post-TBI neuropsychological deficits. Recent advances and international evidence have highlighted repetitive transcranial magnetic stimulation (rTMS) as a promising non-invasive neuromodulatory intervention in TBI rehabilitation.

Methods: Adult patients with mild to moderate TBI (GCS > 10) underwent MRI brain with Diffusion Tensor Imaging (DTI) and were classified based on white matter tract deviation and cerebral edema. Neuropsychological assessment using the Montreal Cognitive Assessment (MOCA) and Hamilton Depression Rating Scale (HDRS) was performed at baseline and follow-up. Patients with persistent deficits received high-frequency rTMS (10 Hz) over the right/left dorsolateral prefrontal cortex at 110% of individual resting motor threshold, delivered in 10 sessions over 5 days.

Results: Sixty patients (40 males, 20 females; mean age 36.24 years) were analyzed. Mean resting motor threshold reduced from 75.35% at baseline to 70.61% at follow-up, with a corresponding reduction in stimulus intensity (110% RMT) from 82.81% to 77.84%, indicating enhanced cortical excitability. Cognitive outcomes improved significantly, with mean MOCA scores increasing from 19.22 at baseline to 23.82 post-intervention. Mean HDRS scores decreased from 5.56 to 4.70, reflecting improvement in depressive symptoms.

Conclusion: rTMS resulted in sustained improvement in cortical excitability, cognitive performance, and psychological well-being in mild to moderate TBI patients, particularly those with white matter tract involvement.

Disclosure: The Study was funded by UP Counsel for Science and Technology (UP CST). Nothing else to disclose

EPO-1018 | Predicting long-term outcome in prolonged disorders of consciousness: Post-acute prognostic factors from the nationwide DOCTOR study

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Background and aims: Prolonged disorders of consciousness (PDOC) following acquired brain injury are characterised by major prognostic uncertainty, complicating clinical decision-making and ethical deliberation. While acute-phase predictors have been studied extensively, less is known about prognostic factors available at admission to specialised neurorehabilitation. This study aimed to identify demographic and clinical predictors for recovery of consciousness, level of functioning, and independence in PDOC.

Methods: Prospective cohort study including PDOC patients ≥ 16 years admitted to the nationwide Dutch PDOC chain of care (2019–2023). Potential predictors at rehabilitation admission: demographic and clinical characteristics, time post-injury (TPI), level of consciousness, aetiology, Coma Recovery Scale-Revised (CRS-R). Outcomes: recovery of consciousness, level of functioning, and functional independence measured with the Functional Independence Measure (FIM) up to 104 weeks. Multivariable Generalized Estimating Equations were used, applying Bonferroni correction.

Results: Of 129 enrolled patients (mean age 38 years; 52% male), 62% regained consciousness. A non-smoking history was the only independent predictor for regaining consciousness (OR 2.46, 95% CI 1.33–4.52). Shorter TPI ($B = -0.26$, $p < 0.001$) and higher CRS-R at admission ($B = 3.15$, $p < 0.001$) independently predicted higher functional outcome. Higher CRS-R also predicted functional independence at 2 years (OR 1.38, 95% CI 1.13–1.67).

Conclusion: Prediction of long-term recovery in PDOC at the start of specialised rehabilitation remains challenging. Simple clinical variables, particularly smoking history and CRS-R score, provide limited but relevant prognostic information. These findings underline the need for integrated longitudinal prognostic models combining clinical, neurophysiological, and imaging data to better support personalised and ethically informed care pathways.

Disclosure: Nothing to disclose.

Headache 6

EPO-1019 | Post-traumatic headache: Evaluating repetitive transcranial magnetic stimulation (rTMS) as a novel treatment strategy

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Background and aims: Post-traumatic headache (PTH) is a disabling sequela of mild traumatic brain injury (mTBI), and currently there is no established effective treatment for PTH. Recently, repetitive transcranial magnetic stimulation (rTMS) has emerged as a promising treatment modality. Aim: To evaluate the effect of rTMS in adults with PTH 4–6 months after mTBI.

Methods: A randomised, sham-controlled clinical trial in adults with PTH was conducted from August 2023 to August 2024. Participants received 5 sessions of active rTMS ($n = 30$) or sham rTMS ($n = 31$) over a 2-week period, 4–6 months after mTBI. The primary outcome was defined as the mean change in the number of headache days of moderate to severe intensity per month before versus after intervention. Primary outcome data were collected via a prospective, daily headache diary. Follow-up was conducted at 1-month after end of treatment (EOT).

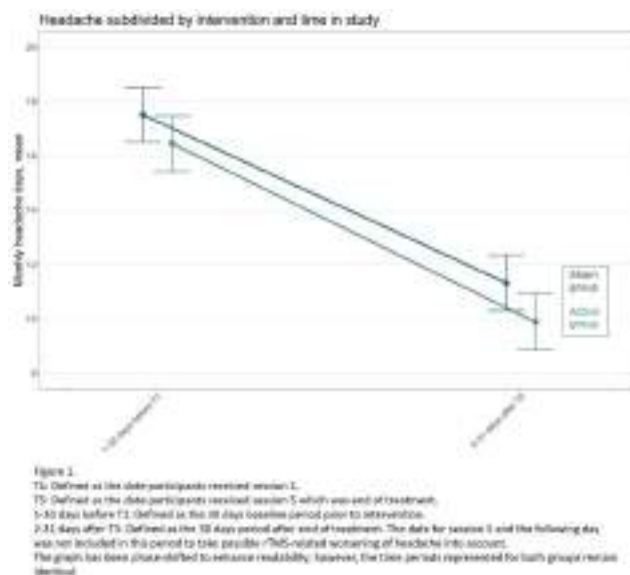


FIGURE 1 Headache subdivided by intervention and time in study.

Results: At 1-month EOT, preliminary results show no significant difference in the mean change of monthly headache days of moderate to severe intensity between the two groups. The mean reduction was 6.6 days in the active group versus 6.2 days in the sham group.

Conclusion: rTMS did not significantly reduce the number of monthly headache days of moderate to severe intensity compared with sham-rTMS in adults with PTH 4–6 months post-mTBI. Consequently, this study does not provide evidence to support rTMS as an effective treatment of PTH. Further research is needed to determine whether rTMS can improve long-term outcomes in patients with PTH.

Disclosure: The authors declare no conflicts of interest related to the manuscript. The study was funded by The Health Research Fund of Central Denmark Region. The funder was not involved in the study design; collection, analysis, or interpretation of data; and in the writing of the article.

EPO-1020 | Distinguishing migraine with aura from acute ischemic events in emergency settings: A cross-sectional study

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Background and aims: Differentiating migraine with aura (MA) from transient ischemic attack (TIA) is challenging because both conditions can present with negative neurological symptoms. Here, we aimed to evaluate two venous imaging markers: “the venous asymmetry index” and “index vein sign” on susceptibility-weighted images (SWI) in MA and TIA groups, and to derive a simple, clinically applicable score to distinguish MA from TIA.

TABLE 1 Venous imaging findings on acute SWI in patients with TIA and migraine with aura.

Variable	TIA (n=209)	MA (n=21)	p-value
Venous Asymmetry Index			
2 (marked)	2 (0.9%)	9 (42.9%)	<0.001
1 (mild)	42 (20.1%)	18 (85.7%)	
0 (none)	225 (107.6%)	2 (9.5%)	
Venous Asymmetry location			
Central	42 (20.1%)	19 (90.5%)	0.001
Subcortical	2 (1.0%)	0 (0%)	
Venous Asymmetry pattern			
Focal	23 (11.0%)	7 (33.3%)	0.20
Diffuse	21 (10.1%)	12 (56.2%)	
Venous Asymmetry Distribution			
Frontal	8 (3.8%)	9 (42.9%)	0.02
Parietal	44 (20.9%)	17 (80.5%)	
Temporal	11 (5.3%)	16 (76.2%)	<0.001
Occipital	11 (5.3%)	11 (52.4%)	
Index Vein Signs			
Laterality – Right / Left	12 (5.7%) / 16 (7.7%)	9 (42.9%) / 9 (42.9%)	<0.001

Values are n (%) unless otherwise specified. For each row, the p-value refers to the central comparison between groups. For each row, the p-value refers to the central comparison between groups. For each row, the p-value refers to the central comparison between groups. For each row, the p-value refers to the central comparison between groups.

TABLE 2 Multivariate logistic regression analysis results and symptom-concordant venous imaging (SVI) scores.

Predictor	SE	aOR (Exp(B))	Lower cut-off of 95% CI for aOR	B (log-odds)	Score points	p
Age below 60	1.11	10.70	1.21	2.4	2	0.01
Venous Asymmetry Index						
Mild (1) vs None	0.84	10.47	2.01	2.3	2	0.005
Marked (2) vs None	1.98	785.3	16.97	6.7	7	0.001
Index vein sign	0.73	5.8	1.4	1.8	2	0.02
Speech impairment	0.96	9.38	1.71	2.23	2	0.01
Hypoesthesia	1.134	23.59	2.61	3.361	3	0.002

Model variables shown in figure. Regression model including age, gender, speech impairment, and index vein sign. B (log-odds) and aOR (Exp(B)) are presented with 95% confidence intervals (CI). Standard scores (SE), and corresponding log-odds coefficients (B). Points are based on comparison of each variable to the reference category in the model. The SVI score is the sum of the points for each variable. All p-values are two-tailed.

Methods: In this cross-sectional study, patients presented with acute stroke-like symptoms (speech disturbance, facial asymmetry, hemianopia, hemiparesis, or hypoesthesia) between January 2023 and December 2024, were prospectively evaluated. Primary analyses tested symptom-concordant venous asymmetry index and index vein sign, evaluating their relationship to the final diagnosis. The Symptom-concordant Venous Imaging (SVI) score was constructed.

Results: A total of 290 diffusion weighted image (DWI)-negative patients were eligible (TIA n=269; MA n=21). The symptom-concordant venous asymmetry index was present in 90.5% and the index vein sign in 85.7% of migraine with aura however these findings were observed in only 16.4% and 10.4% of the TIA group, respectively (Table-1). On follow-up MRIs, venous imaging signs frequently resolved in migraineurs. Multivariable analysis models revealed that symptom-concordant venous imaging findings were associated with MA (Table-2). The SVI score demonstrated a discriminatory performance with an AUC of 0.969 (95% CI: 0.949–0.990). At a cut-off value of ≥8 points, sensitivity was 71.4% and specificity was 95.9%.

Conclusion: In patients presented with acute stroke-like symptoms, marked venous asymmetry and index vein sign concordant with symptoms and SVI score of >8 were strongly associated with MA.

Disclosure: Nothing to disclose.

EPO-1021 | Effective migraine prevention improves cognitive complaints

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Background and aims: Cognitive complaints, such as memory and concentration difficulties, are increasingly recognized as part of the clinical spectrum of migraine. We aimed to evaluate whether subjective cognitive complaints improve with effective preventive treatment for migraine acting outside the central nervous system.

Methods: We conducted a prospective observational study including adults with migraine diagnosed according to the ICHD-3 criteria, initiating preventive treatment with either anti-CGRP monoclonal antibodies (mAbs) or OnabotulinumtoxinA (BT). Responders were defined as those who achieved $\geq 50\%$ reduction in monthly migraine days for episodic migraine and $\geq 30\%$ reduction for chronic migraine from baseline to 3–6 months follow-up. Cognitive complaints, assessed using the Subjective Memory Complaints scale (SMC) and depressive symptoms (Hospital Anxiety and Depression Scale (HADS-D)), were compared between the two evaluations according to responder status.

Results: Participants ($N=100$, mean age 42.27 years, 98% women, 71% with chronic migraine), received either mAbs ($N=66$) or BT (34), and 57 responded to treatment. Responders showed a significant improvement in subjective memory complaints ($B=-1.46$, $p=0.037$), regardless of the type of preventive treatment ($p=0.857$). The improvement was also significant in patients without depressive symptoms ($p=0.027$) but related to improvement of HADS scores in participants with higher scores.

Conclusion: Cognitive complaints are a frequent and burdensome manifestation of migraine, reflecting its broader ictal and interictal impact beyond pain. This study shows that effective preventive treatment is associated with significant improvement in these cognitive symptoms, reinforcing their role as part of the migraine spectrum.

Disclosure: Nothing to disclose.

EPO-1022 | Assessment of cortical excitability in migraine patients using the paired-pulse transcranial magnetic stimulation technique

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Background and aims: Thalamocortical dysrhythmia, observed in migraine, is often linked to cortical hyperexcitability. This pattern also appears in fibromyalgia, etc. This study examines neurophysiological markers in migraine via short-interval intracortical inhibition assessed with the threshold tracking technique (TT-SICI).

Methods: Participants included 15 patients with chronic migraine (CM; mean age 37.8 years; 86.6% female), 9 with episodic migraine (EM; mean age 34.5 years; 77.3% female), and 16 healthy controls (C; mean age 28.3 years; 68.8% female). Bilateral paired-pulse TMS targeted the primary motor cortex. Motorevoked potentials were recorded from hand muscles to evaluate threshold dynamics at interstimulus intervals (ISIs) of 2–7 ms using TT-SICI. Validated tools included PHQ-9, PDS (Personality Disorder Severity), clinical interviews for headache/psychiatric status, and the Edinburgh Handedness Inventory (EHI).

Results: A significant TT-SICI reduction occurred in CM versus C only in the left hemisphere at ISIs of 3.5 and 4 ms ($p<0.05$). No differences appeared at other ISIs, during right-hemisphere stimulation, or in EM versus controls. All subjects were right-handed (EHI). In CM, positive correlations emerged between

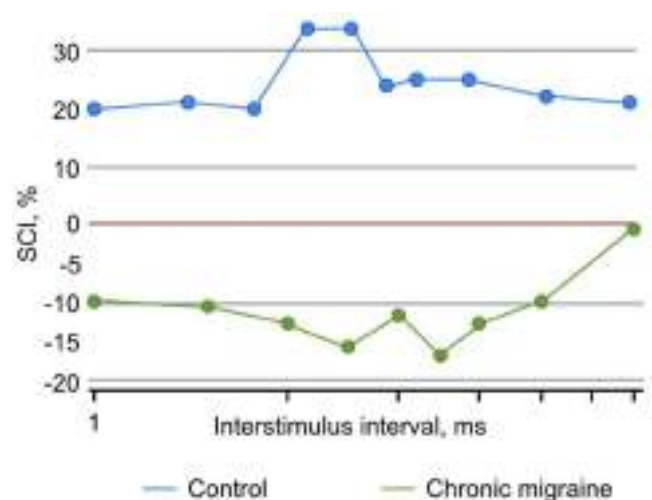


FIGURE 1 Individual inhibition curves (CM vs C).

TT-SICI and headache intensity at ISIs 3, 3.5, and 7 ms ($r=0.415$, 0.596 , 0.438); with PHQ-9 at 3.5 ms ($r=0.430$); and with PDS at 5 ms ($r=0.420$).

Conclusion: Reduced TT-SICI at 3.5–4 ms in the left hemisphere CM patients indicates intracortical hyperexcitability and GABAergic inhibition deficit. Positive correlations with depression (PHQ-9), personality disorder severity (PDS), and headache intensity suggest shared mechanisms linking emotional disturbances and chronic headache. These results reinforce central sensitization in CM and highlight TT-SICI as a potential biomarker for personalized assessment and treatment monitoring.

Disclosure: Nothing to disclose.

EPO-1023 | Inflammatory markers and white matter lesions on brain MRI in patients with migraine

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Background and aims: Brain magnetic resonance imaging (MRI) frequently reveals white matter lesions (WML) in patients with migraine. Low grade systemic inflammation has been proposed as a contributing factor in migraine pathophysiology and may be associated with structural brain changes on MRI.

Methods: We performed a retrospective observational study including patients diagnosed with migraine with available brain MRI and blood analysis. Patients were classified as migraine with aura (MA) or migraine without aura (MWO), episodic migraine (EM) or chronic migraine (CM). Brain MRI

were reviewed to evaluate the presence of white matter lesions. Laboratory parameters obtained from routine blood analysis included platelet count, neutrophils, lymphocytes, monocytes, C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR). Inflammatory ratios were calculated, including platelet-to-lymphocyte ratio (PLR), neutrophil-to-lymphocyte ratio (NLR), and lymphocyte-to-monocyte ratio (LMR). Descriptive and comparative analysis were performed.

Results: A total of 62 patients were included, 23/62 (37%) presented WML on brain MRI. WML were significantly more frequently present in patients with MA than in those with MWO [19/23 (83%) vs 15/39 (38%), $p < 0.001$]. Patients with WML showed significantly higher CRP levels compared to those without WML [0.28 (0.21–0.48) vs 0.10 (0.09–0.18), $p < 0.001$]. No significant differences were observed in other inflammatory parameters.

Conclusion: Our results show that WML are more prevalent in patients with MA and are associated with higher systemic inflammation, as reflected by elevated CRP levels. These findings may suggest the presence of inflammatory activity in patients with WML, highlighting the potential role of immune biomarkers in the underlying pathophysiology of WML which warrants further research.

Disclosure: ISCIH, EU and FSE funding through JR23/0005;PI24/01085.

EPO-1024 | Headache outcomes on the first day of fasting with once-daily Rimegepant: A post-hoc exploratory analysis

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Background and aims: Rimegepant was evaluated previously for short-term prevention of fasting-triggered headache during Ramadan, with outcomes assessed over weekly intervals. This post-hoc analysis examined daily outcomes over the initial week of fasting, focusing on day 1, to evaluate rimegepant's potential for prevention of headache triggered by single-day fasting for religious or medical reasons.

Methods: Participants were randomized to open-label once-daily rimegepant 75 mg from weeks 1–4 (Immediate Start; $n = 52$) or 2–4 (Staggered Start; $n = 53$) of the fast. The proportion of participants with headaches by pain intensity (none, mild, moderate, severe), the proportion with moderate-to-severe headaches, and the duration of moderate-to-severe headaches was compared between groups using nominal p -values.

Results: Headache intensity on day 1 was lower in the Immediate Start arm than the Staggered Start arm ($p = 0.015$), with more participants reporting no headache (50.6% vs 27.8%) and fewer participants reporting moderate (21.0% vs 33.3%) or severe (8.0% vs 18.9%) headache. Fewer participants experienced moderate-to-severe

headache on day 1 in the Immediate Start arm than the Staggered Start arm (difference [95% CI]: -28.8% [-49.1% , -8.5%]; $p = 0.009$). Moderate-to-severe headache duration on day 1 was shorter in the Immediate Start arm than the Staggered Start arm (difference [95% CI]: -1.9 [-4.3 , 0.5] hours), although the difference was not significant ($p = 0.117$). Similar trends were observed across the initial week of fasting (not shown).

Conclusion: Rimegepant improved headache-related outcomes on day 1 in individuals with fasting-triggered headache. These exploratory findings support further evaluation of rimegepant's potential for prevention of headache triggered by single-day fasting.

Disclosure: This study was conducted as a collaboration between the American Center for Psychiatry and Neurology (ACPN; Abu Dhabi, UAE) and Pfizer. The ACPN funded the study and is the study sponsor. Rimegepant was supplied by Pfizer. Tauofik Alsaadi has served on an advisory board for Pfizer. Reem Suliman and Ibrahim Al Qaisi have no conflicts of interest to declare. Gal Ifergane has received consulting fees and honoraria from Teva, Novartis, Eli Lilly, Pfizer, AbbVie, Organon, and Lundbeck; and has received research support from Teva and Pfizer. Jiyue Yang, Terence Fullerton, Denise Chou, Ed Whalen, Nora Vainstein, Danna Davidovici, Matanya Tirosh, and Anthony Chan are (or were at the time of the study) employees of Pfizer and may own stock/options in Pfizer. Ifra Rehman is a post-doctoral fellow at Pfizer. Azhar Hussain is a student at Touro College of Pharmacy.

Clinical Neurophysiology 1

EPO-1025 | Neurovascular reactivity during mental stress in narcolepsy with cataplexy: A microneurographic study

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Background and aims: Mental stress induces an arousal reaction and a subsequent response on various physiological parameters such as blood pressure (BP). Previous studies with microneurography, a technique allowing for in vivo nerve registration of muscle sympathetic nerve activity (MSNA), have shown that individuals with inhibited MSNA after arousal (“responders”) showed a lesser BP response when compared to “non-responders”. In narcolepsy with cataplexy (NC), microneurography has shown a deranged sympathetic activation during REM sleep: the aim of the study is to assess whether a similar occurrence is found in wake patients.

Methods: 15 male patients with NC (aged 37 ± 11 years) and 15 male healthy controls (aged 36 ± 11 years) were subjected to microneurography of the peroneal nerve and concomitant recording of respiratory movements, blood pressure and electrodermal activity. Responders and non-responders were defined by significant MSNA inhibition after electrical skin stimulation. All individuals were then recorded at rest, after mental arithmetic stress and after cold stimulation. BP variation was then analyzed between NC patients and healthy controls, as well as responders vs non-responders between groups.

Results: 3 NC patients and 3 healthy controls could be classified as responders, thus showing no difference. As expected, stress induced a BP increase which was less elevated in responders, both in the NC and in the healthy control group.

Conclusion: In this study, wake NC patients showed no difference in sympathetic response to stress, which differed in responders vs non-responders resulting in a different impact on blood pressure.

Disclosure: Nothing to disclose.

EPO-1026 | Direct current stimulation shapes metabolic phenotype of human i3 cortical neurons

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Background and aims: Transcranial direct current stimulation (DCS) is a promising disease-modifying strategy in neurodegenerative disorders, offering a non-invasive alternative to deep brain stimulation (DBS) and a complementary approach to pharmacological therapies. However, clinical outcomes remain heterogeneous, with responders and non-responders, highlighting the need for mechanistic insight. At cellular level, we previously showed that DCS in neuroblastoma cells induces lipid metabolic remodeling, reduces inflammation, enhances mitochondrial oxidative metabolism and energy availability, and promotes extracellular vesicle release. We here aim at investigating the biological effects of DCS on human i³-neurons using a multi-omic approach.

Methods: Human i³-neurons, generated from healthy donor iPSCs via doxycycline-induced expression of an NGN2-containing cassette, were stimulated with DCS (0.5 μ A; $\approx$ 18 nA/cm²; 10 min), once or three times. Phenotypic modulation was assessed by mass spectrometry-based lipidomic and proteomic analyses and RNA sequencing.

Results: DCS induced a metabolic-nuclear response in i³-neurons, affecting lipid mobilization and persistent modulation of the gene expression machinery, with proteomic enrichment of RNA processing, transcriptional control, chromatin remodeling, and genome stability pathways. A single stimulation elicited a transient response at 24 h, characterized by membrane lipid remodeling and stress-metabolic proteins, that largely resolved by 72 h. In contrast, repeated stimulations stabilized a state marked by membrane reorganization, involving modulation of ether-linked lipids and fatty acid unsaturation, mitochondrial-lipid metabolic coupling, and transcriptional regulation impacting cellular signaling activity.

Conclusion: Our integrated multi-omics data show DCS induces biological changes on neuronal metabolism, signaling, and plasticity, supporting its clinical potential as a mechanism-based neuromodulation strategy.

Disclosure: SI, nothing to declare MG, nothing to declare VB, nothing to declare EB, nothing to declare EA, nothing to declare MP, nothing to declare ADG, nothing to declare TB, nothing to declare AP, is founder and stakeholder of Newronika srl (Milan, Italy), a Company that develops and produces tDCS devices. MM, nothing to declare AR, nothing to declare PS, nothing to declare.

EPO-1027 | Towards a neurophysiological biomarker of neurotoxicity in Charcot-Marie-tooth disease

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Background and aims: Demyelinating Charcot-Marie-Tooth (CMT) is characterized by progressive demyelination with secondary axonal degeneration. Although axonal loss is the primary cause of neurological disability, marked phenotypic variability exists and the underlying neurotoxic mechanisms remain poorly understood. We explored the relationship between CMT severity and motor axonal properties preceding degeneration.

Methods: Patients with CMT1A (N=11), CMT1B (N=5), and CMTX1 (N=4) underwent conventional nerve conduction studies supplemented with median motor axon excitability testing at the wrist using threshold-tracking (TRONDNF protocol). Axonal excitability parameters were correlated with the Charcot-Marie-Tooth Examination Score version 2 (CMTESv2) using Spearman correlation and linear regression.

Results: Across groups, several axonal excitability parameters correlated with CMTESv2, with subgroup-specific regression patterns. For instance, refractoriness at 2ms of the recovery cycle was positively associated with CMTESv2 in CMT1A and negatively in CMT1B, with regression slopes differing significantly ($t = -2.906$, $p = 0.013$). Temperature did not differ across subgroups ($p = 0.46$).

Conclusion: Refractoriness at 2ms primarily reflects nodal voltage-gated sodium channel (VGSC) function, which is independent of passive membrane properties. Translational studies in CMT1B show that dysmyelination is associated with VGSC dysregulation, increasing motor axon vulnerability to activity-dependent degeneration. Notably, refractoriness changes occurred in opposite directions in CMT1A and CMT1B, enabling differentiation between these conditions, yet both indicate altered nodal VGSC function, supporting VGSC dysregulation as a shared pathophysiological mechanism across CMT subtypes. Recovery cycle refractoriness therefore reflects neuropathy

severity in demyelinating and intermediate CMT and may serve as a neurophysiological biomarker of neurotoxicity, with implications for prognostication and assessment of therapeutic efficacy in future disease-modifying trials.

Disclosure: Nothing to disclose.

EPO-1028 | Diagnostic ultrasound-induced changes of motor cortical excitability in humans

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Background and aims: This proof-of-concept study investigates whether certified diagnostic ultrasound effectively modulates cortical excitability. By targeting primary motor cortex, we demonstrate that conventional clinical protocols induce neuroplastic changes, potentially bypassing the experimental hardware hurdles and making transcranial ultrasound (TUS) immediately accessible for hospital applications.

Methods: Sixteen healthy volunteers (31.9 ± 11.0 years) participated in a sham-controlled crossover study. Using a certified DWL Multi-Dop X4 clinical system, 30 minutes sonication was delivered through the right transtemporal window via a 2.0 MHz linear probe, targeting the Abductor Pollicis Brevis (APB) hotspot at a depth of 45–58 mm. Cortical excitability was mapped via paired-pulse Transcranial Magnetic Stimulation (TMS), measuring Short-Interval Intracortical Inhibition (SICI), Intracortical Facilitation (ICF), and Long-Interval Intracortical Inhibition (LICI). Resting Motor Threshold (RMT) and Breath-Holding Index (BHI) monitored mechanical or vascular artifacts. Non-parametric tests and Bayesian inference (Bayes Factor, BF) quantified the evidence.

Results: Post-insonation, SICI was significantly reduced in the stimulated hemisphere (T0: 65.0%, T1: 67.5%; $p=0.016$, $BF_{10}=7.65$), indicating increased local excitatory drive. Conversely, the control hemisphere showed a robust LICI increase (T0: 54.0%, T1: 86.0%; $p=0.010$, $BF=7.88$). Inter-hemispheric comparisons confirmed that the reduction in inhibition was specific to the stimulated side ($p=0.046$, $BF=3.90$). No significant changes occurred in ICF, RMT, or BHI.

Conclusion: Diagnostic ultrasound elicits focal neuroplastic changes, likely via Gamma-Aminobutyric Acid-B (GABA-B) pathways. This represents the first evidence that standard clinical Doppler system can selectively modulate the motor cortex, traditionally requiring high-costly experimental hardware, establishing standard sonography as a viable neuromodulation tool ready for immediate clinical translation.

Disclosure: The authors declare that this research was conducted in the absence of any commercial or financial relationships that could be constructed as a potential conflict of interest.

EPO-1029 | Electrocortical changes due to vibro-tactile stimulation in patients with cervical dystonia

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Background and aims: Cervical dystonia (CD) involves involuntary, sustained, or intermittent neck muscle movements. Although its pathophysiological remain unclear, increasing evidence points to altered sensory processing, particularly in the tactile and proprioceptive domains. Vibro-tactile stimulation (VTS) has been proposed as a non-invasive neuromodulatory intervention. This study investigated the clinical effects of VTS in CD and the associated cortical and cortico-muscular correlates.

Methods: 10 CD patients underwent 64-channel EEG and surface EMG recordings from bilateral sternocleidomastoid and trapezius muscles. Head kinematics were measured using a wireless inclinometer, with the head angle index (HAI) as outcome. During a preliminary session, 1-minute VTS was applied to each of four muscles; the one producing maximal HAI improvement was selected. Patients then completed a randomized cross-over protocol with three 5-minute conditions: rest with inactive VTS, active VTS, and sham VTS (5 seconds per minute). EEG and EMG were analyzed for spectral power (theta, alpha, beta), cortico-muscular coherence (CMC), and intermuscular coherence (IMC).

Results: Active VTS progressively reduced HAI relative to baseline, significantly more than sham. During active-VTS EEG over the contralateral sensorimotor cortex showed increased alpha power and a trend toward reduced theta. Theta CMC decreased and alpha CMC increased during VTS. IMC between homologous muscles showed a non-significant trend toward increased theta coherence.

Conclusion: VTS applied to cervical muscles improves dystonic posture in CD, associated with modulation of cortical alpha/theta rhythms and cortico-muscular and intermuscular coupling. These findings support VTS as a promising non-invasive neuromodulatory strategy targeting sensory dysfunction in cervical dystonia.

Disclosure: The authors declare no conflict of interest

EPO-1030 | Pupillary light reflex index in insomnia disorder: An index of daytime autonomic arousal

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Background and aims: The present study investigates whether dynamic Pupillary Light Reflex (PLR) metrics can serve as day-time physiological markers of arousal dysregulation in Insomnia Disorder (ID).

Methods: To evaluate diurnal autonomic nervous system activity, this study used the PLR as a noninvasive index of arousal. A Psychomotor Vigilance Test (PVT) was performed as a behavioral measure of objective daytime sleepiness, hypothesizing that, compared to Healthy Controls ($n=24$; mean (M)=49.87; standard deviation (SD)=11.75; 15F), ID patients ($n=29$; $M=49.03$; $SD=11.07$; 21F) would show objective signs of altered daytime arousal.

Results: The non-parametric t-test showed no significant group differences in PVT performance. Considering PLR metrics, ID patients showed a faster pupil re-dilation after constriction, alongside a slower return of the pupil to 50% of its original diameter. Spearman's correlations were performed to evaluate the association between the PLR indices and clinical features. Results demonstrate positive associations between pupil re-dilation after constriction and time for the pupil to return to 50% of its original diameter, more severe ID symptoms, higher pre-sleep arousal, and more dysfunctional beliefs about sleep.

Conclusion: The results confirm increased daytime arousal state in individuals with insomnia, supporting the use of pupil-ometry to assess physiological alterations in this condition.

Disclosure: Nothing to disclose.

EPO-1031 | Propofol anesthesia induces a brain state of intermediate consciousness: Insights from EEG-based time-evolving functional brain networks

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Background and aims: Consciousness depends on the brain's ability to balance excitability and stability [1, 2]. While non-human primate studies suggest propofol disrupts cortical dynamics [3], its impact on dynamic network stability in humans remains unclear. We investigated how propofol alters cortical network stability and dynamics relative to sleep and wakefulness using EEG-derived time-evolving functional brain networks (TEFBNs), capturing dynamic changes in brain organization associated with consciousness [4].

Methods: We recorded EEG time-series (10–20 system) from adults across three groups: healthy controls during wakefulness ($n=51$), nighttime sleep ($n=14$), and propofol anesthesia ($n=10$). EEG in the anesthesia group was acquired prior electroconvulsive therapy (ECT) in depressed patients. Baseline network organization did not differ from controls, indicating no clinical influence. TEFBNs were constructed, and network-based measures of stability and dynamics were analyzed.

Results: Under propofol anesthesia, subjects occupied an intermediate brain state that was neither fully awake nor asleep. Although slow-wave activity resembled NREM sleep, TEFBNs revealed a distinct network organization. Bilateral cortical regions showed equal temporal importance, indicating increased

local synchronization and reduced functional differentiation. Network metrics further demonstrated subtle destabilization of cortical dynamics not observed during natural sleep or wakefulness, suggesting that propofol induces a state of symmetric local coherence despite disrupted global integration.

Conclusion: These findings extend primate work showing that propofol impairs consciousness by destabilizing cortical networks. To our knowledge, this is the first human evidence that propofol produces an intermediate state that is awake-like yet exhibits sleep-like slow waves, reflecting a mechanism distinct from natural sleep, not merely a deeper stage of it.

Disclosure: This work was supported by the Marga und Walter Boll-Stiftung Nr. 2020-03.01–21.

EPO-1032 | Pathophysiology of fatigue in chronic inflammatory demyelinating polyradiculoneuropathy: The importance of proximal demyelination

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Background and aims: Fatigue is a complex symptom that has multiple mechanisms from central nervous system to immunological processes. We hypothesize that fatigue's more pronounced in patients with proximal conduction blocks and proximal demyelinating and the involvement of cranial nerves.

Methods: CIDP patients were evaluated for fatigue and underwent electrophysiological investigations. Bilateral routine NCS, proximal assessments of median (wrist-elbow-axilla) and ulnar nerve (wrist-elbow-above-elbow-axilla), motor recordings on nasalis and trapezius muscles (stimulating from facial and spinal-accessory nerve), blink-reflex testing were performed. Conduction-blocks, conduction-slowing, F/A-waves in proximal-distal segments were evaluated. Fatigue was assessed with Krupp-Fatigue-Severity-Scale (FSS) (<2.8:no fatigue, >6.1:chronic, 2.8-6.1:mild).

Results: Ten CIDP patients were evaluated (mean age 49.8 ± 8.3). The mean FSS score was 4.32 ± 1.3 . The mean INCAT score was 2.2 ± 1.8 . Chronic fatigue was present in 20%, 10% had no fatigue. The mean number of conduction blocks was 3.9 ± 1.8 (min-max 1–6). In patients with chronic fatigue, conduction blocks in the median and ulnar nerves were more frequently located in proximal segments compared with the non-fatigued/mildly fatigued groups. There were no significant differences in nasalis-trapezius recordings. Blink reflex latencies tended to be longer in fatigued patients.

Conclusion: These findings suggest that fatigue in CIDP may be associated with the topographic distribution of conduction blocks. Although no marked differences were detected in cranial nerve studies, the tendency toward prolonged blink reflex latencies in fatigued patients is notable in terms of central-peripheral interactions. These results highlight the need to consider the distribution of conduction blocks to better understand the pathophysiology of fatigue in CIDP.

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Background and aims: Novel 2024 McDonald criteria included the kappa free light chain (KFLC) index as an additional cerebrospinal fluid (CSF) biomarker representing intrathecal inflammation as well as evoked potentials (EPs) demonstrating functional central nervous system pathway dysfunction. Aim of this study was to evaluate association of these biomarkers reflecting inflammation and neurodegeneration in early relapsing–remitting multiple sclerosis (RRMS).

Methods: Analysis included 52 consecutive treatment-naïve people with newly diagnosed RRMS (34 females; mean age 37.7 ± 11.1 years; median Expanded Disability Status Scale [EDSS] 2.0). All patients underwent standardized diagnostic work-up including brain and cervical spinal cord MRI, CSF analysis with KFLC index calculation and multimodal EP testing (visual EP [VEP], brainstem auditory EP [BAER], and median and tibial somatosensory EP [mSSEP, tSSEP]). Pathological findings of EP were summarized calculating an EP score.

Results: Higher KFLC index correlated with shorter VEP P100 latency and higher VEP amplitude on left-eye stimulation, shorter BAER wave V latencies bilaterally, and with higher tSSEP amplitudes. Consistent with these findings, KFLC index values were significantly higher in participants with lower EP pathological burden (EP score classification; $p=0.01$), and similarly across VEP- and tSSEP-based pathological groupings ($p=0.015$ and $p=0.008$, respectively). No significant associations were observed between KFLC index and clinical or neuroradiological parameters in our cohort.

Conclusion: In newly diagnosed RRMS, higher intrathecal inflammatory activity was paradoxically associated with less neurophysiological pathway dysfunction. These findings suggest that KFLC index and EP scores represent different aspects of early MS neuropathology emphasising their complementary utilisation in fulfilment of new MS diagnostic criteria.

Disclosure: Nothing to disclose.

EPO-1034 | The effect of demyelination patterns on clinical course and prognosis in CIDP

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Background and aims: Chronic inflammatory demyelinating polyneuropathy (CIDP) is a heterogeneous immune-mediated neuropathy characterized by multifocal demyelination affecting different nerve segments. Our study aimed to investigate the association between demyelination distribution patterns, clinical features, and prognosis in CIDP.

Methods: We retrospectively evaluated initial and follow-up electrophysiological studies of 42 patients with CIDP (2000–2024) according to the 2021 EFNS/PNS criteria. Patients were classified as distal (distal latencies $>150\%$ of the upper limit of normal), intermediate (conduction velocity $<70\%$ of the lower limit of normal and/or conduction block or abnormal temporal dispersion), or diffuse (combined distal and intermediate abnormalities). Patients were categorized according to electrophysiological patterns, and longitudinal changes were analyzed in relation to clinical prognosis.

Results: Forty-two patients were analyzed longitudinally. At baseline, electrophysiological patterns were classified as intermediate in 61.9%, diffuse in 28.5%, and distal in 9.6% of patients; 12.5% showed normalization at follow-up. Distal pattern was

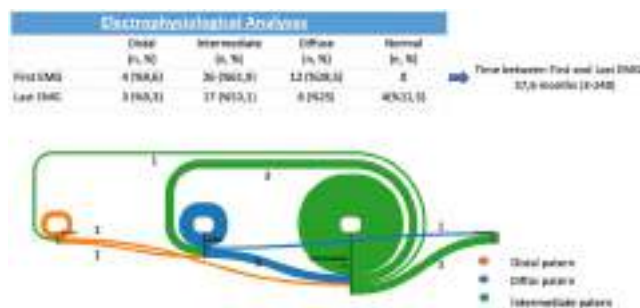


FIGURE 1 Electrophysiological analysis: comparison between initial and final EMG results.

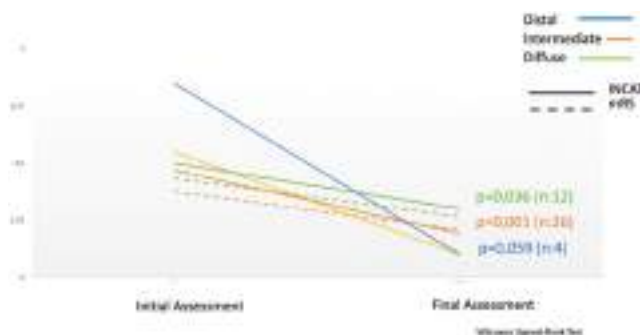


FIGURE 2 Based on electrophysiological patterns after initial assessment, comparison of INCAT and mRS scores before and after treatment.

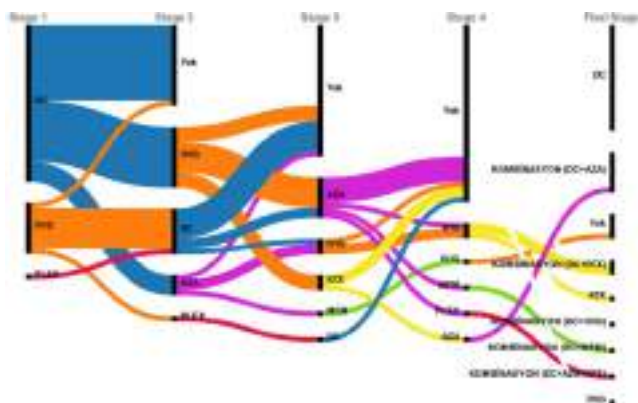


FIGURE 3 Treatment records during initial treatment and follow-up period.

associated with shorter symptom duration, higher INCAT and mRS scores, more frequent triggering events, and a higher rate of subacute onset (all $p \leq 0.036$). Treatment was initiated 24.1 months earlier, partial remission lasted 51 months longer, and total disease duration was 11.6 years shorter in distal compared with intermediate pattern (all $p \leq 0.03$). Monophasic course was more frequent with temporal dispersion ($p = 0.023$), whereas axonal degeneration predicted lower remission rates ($p = 0.011$).

Conclusion: Demyelination patterns significantly influence the clinical course and prognosis of CIDP, highlighting the prognostic value of electrophysiological pattern analysis.

Disclosure: Nothing to disclose.

EPO-1035 | Transient loss and recovery of dorsal rootlet excitability during selective dorsal rhizotomy

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Background and aims: Selective dorsal rhizotomy (SDR) is an established treatment for lower-limb spasticity in children with cerebral palsy (CP). Intraoperative neurophysiological monitoring (IONM) guides the procedure, yet its protocols are debated. This study aimed to assess the dynamics of dorsal root (DR) excitability during SDR.

Methods: A retrospective analysis of IONM data from 15 children with CP (mean age 7.1 ± 4.3 years, GMFCS III-V) was conducted. Surgery involved single-level laminectomy (L1/L2) under total intravenous anesthesia. The IONM protocol determined the threshold stimulation intensity (TSI) using 3-Hz pulses, followed by 50-Hz train stimulation for each whole DR and its subsequently subdivided rootlets.

Results: Analysis of 150 DRs (L2-S1) and 684 rootlets revealed significant inter-root differences. The median TSI (in mA) for whole DRs was lowest at S1 (0.48, IQR 0.28–0.61) and highest at L4 (0.98, IQR 0.60–1.21). Subdivision into rootlets increased median TSI markedly, peaking at L4 (2.03, IQR 1.05–2.72). A key finding was transient loss of EMG response in 8 patients despite stimulation up to 5 mA, which was reversed by irrigation

with warm saline during 5 min, leading to a significant decrease in recalculated TSI. Statistical analysis confirmed significant inter-root differences at each procedural stage (Kruskal-Wallis, $p < 0.05$) and a significant overall reduction in thresholds over time (Friedman test, $p = 0.007$), with the most pronounced decrease at L4 (from 2.03 to 1.22, $p = 0.004$).

Conclusion: The observed reduction in rootlet excitability may be attributed to specific surgical aspects of SDR. Thus, proper IONM interpretation is crucial to determine individual rootlet function at each segmental level.

Disclosure: Nothing to disclose

EPO-1036 | Ischemia resistance in pathophysiology of early diabetic motor and sensory polyneuropathy

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Background and aims: Nerve excitability testing has shown resistance to ischemia in motor nerves after 10–30 minutes of compression as an early and specific finding in diabetic neuropathy. Sensory nerves have not previously been investigated. We hypothesize that resistance to ischemia is seen in motor and sensory nerves before clinical and electrophysiological signs of neuropathy.

Methods: We tested diabetic patients (10 sensory, 13 motor measurements, age 30–60 yrs) and compared them with healthy controls (12 sensory, 9 motor measurements). The following protocol was applied at two separate sessions, one for the motor and one for the sensory nerves: short TRONDNF including strength/duration time constant (SDTC), multitasking: 5 min pre-ischemia, 5 min ischemia (induced with a pressure cuff), 10 min post-ischemia. We have tracked 4 parameters: threshold decrease (in multitask protocol), recovery cycle (RC) at 6 ms, TE_{d100}, and TE_h overshoot of threshold electrotonus.

Results: People with diabetes showed statistically highly significant less loss of axonal super-excitability in both sensory and motor axons (SupEx isc motor –10.7 % vs. 1.3%, SupEx isc sensory –3.5 % vs. 13.9 %; $p < 0.001$). Preliminary results showed also that they differed from normal controls in ischemia-induced changes in strength/duration time constant and in the time course of ischemia-induced changes in threshold current.

Conclusion: Resistance to compression-induced nerve ischemia in diabetic patients can be detected by the threshold current in the recovery cycle. The time course of ischemia-induced changes in threshold current and an abnormal strength/duration

time constant indicate that tissue acidification may contribute to nerve excitability parameters.
Disclosure: Nothing to disclose.

EPO-1037 | **Functional relationship between large and small fibers in diabetic neuropathy**

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Background and aims: Diabetic neuropathy (DN) has two different phenotypes, painful DN (PDN) and painless DN (PLDN). Small fiber (SF) damage has been suggested in pain production, however, role of large fibers (LF) remains unclear; We electrophysiologically investigated LF and SF functions in PDN and PLDN.

Methods: In 140 diabetics, we scaled SF and LF functions by electrodermal skin conductance (ESC) of SUDOSCAN and by nerve conduction study (NCS). NCS scale is defined as follows; NCS-0: No NCS anomalies, NCS-1: Prolonged F-wave latency with normal SNAP & CMAP amplitudes, NCS-2: Fall in SNAP less than 5 microvolts with normal CMAP, NCS-3: Fall in CMAP amplitude to between 2mV and 5 mV, NCS-4: Decrease in CMAP amplitudes less than 2mV.

Results: Fall in ESC and increase in the NCS-scale showed positive correlation with coefficient 0.55. Among the cases with ESC lower than 60 micro-siemens, 20% of the cases with NCS-0 and NCS-1 had pain, while 48% of the NCS-2, -3, and -4 groups ($p < 0.05$) complained of pain. Among the NCS-0 and -1 cases with lower ESC, PDN showed sural SNAP of $6.9 \pm 1.2 \mu V$, which was lower than $10.6 \pm 4.1 \mu V$ of PLDN ($p < 0.01$).

Conclusion: In diabetes, electrophysiological functions of LF and SF generally decline in parallel. Even if the ESC decreases, pain is unlikely to occur if LF function is maintained normally. LF activate collateral inhibitory neurons in the dorsal horn of the spinal cord. Reduced impulse input from the LF might facilitate pain impulse transmission from the damaged SF.

Disclosure: None

EPO-1038 | **Neurophysiological investigation of the Bereitschaftspotential in healthy subjects during simple and complex, clonic and tonic movements**

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Background and aims: Recent evidence questions the clinical applicability of the Bereitschaftspotential (BP) due to limited

TABLE 1 Morphological analysis of the BP from the recorded traces for each individual task. Data are presented as mean $\pm$ standard deviation and as counts.

	BP			Onset Early (ms)	Amplitude Early (μV)	Onset Late (ms)	Amplitude Late (μV)	Max Amplitude (Electrodes)	Distribution of TE
	BC+HB	BC	HB						
HC	5	5	5	2415.3 ± 254.3	5.8 ± 1.7	4514.4 ± 185.9	8.7 ± 3.7	C1-C3	PTP on
HCC	0	3	0	2037.8 ± 858.0	5.2 ± 1.1	741.5 ± 351.4	9.4 ± 1.8	C1-C3	PTP on
HTS	0	0	0	2226.7 ± 733.0	2.3 ± 1.7	491.8 ± 117.3	8.1 ± 3.5	C1-C3	PTP on
FC	0	0	1	3170.4 ± 692.7	3.9 ± 1.7	396.0 ± 82.0	8.2 ± 5.5	Fc2-C3	FC on
BC	0	2	0	2602.0 ± 898.0	3.8 ± 1.8	816.0 ± 374.8	8.7 ± 2.7	Fc2-Fc3	FC on

TABLE 2 Results of the within-subject comparison of recorded traces across trials versus the simple clonic hand movement. Data are presented as mean difference $\pm$ standard deviation.

	Onset Early (ms)	P-value	Amplitude Early (μV)	P-value	Onset Late (ms)	P-value	Amplitude Late (μV)	P-value
HC vs HC	-190.3 $\pm$ 822.4	0.947 ⁺	1.0 ± 1.3	0.124	-190.1 $\pm$ 411.0	0.940	1.8 ± 2.7	0.231 ⁺
HC vs HCC	-700.0 $\pm$ 806.0	0.815 ⁺	0.0 ± 2.5	0.419	-179.6 $\pm$ 180.0	0.007 ⁺	1.4 ± 2.8	0.180
HC vs HTS	-417.0 $\pm$ 738.1	0.880 ⁺	-6.4 ± 1.2	0.075	-53.1 $\pm$ 188.9	0.440	3.1 ± 5.2	0.140
HC vs FC	-295.2 $\pm$ 448.1	0.230	0.0 ± 3.0	0.404	-65.6 $\pm$ 23.3	0.027	1.9 ± 3.3	0.001

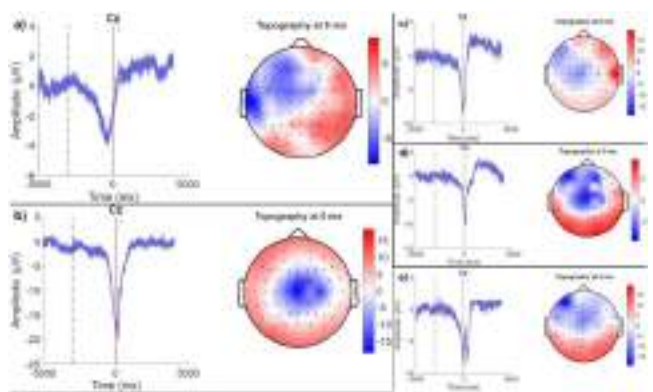


FIGURE 1 BP recordings and colorimetric maps for: (a) simple clonic hand movement, (b) foot movement, (c) complex clonic hand movement, (d) tonic hand movement and (e) blink movement.

study generalizability, multiple factors affecting BP morphology, and methodological heterogeneity, which collectively hinder definitive conclusions on BP onset timing and spatial distribution across body regions.

Methods: We cross-sectionally examined the BP during voluntary hand, foot, and eye movements in healthy participants. Simple and complex hand movements, foot dorsiflexion, and eyelid contraction were recorded, including brisk (< 0.5 s) and sustained (~ 1 s) actions. EMG onset at 50% maximal voluntary contraction served as the trigger, and BP was recorded via 64-channel DC EEG. A 7-second epoch window was applied, artifact-free trials averaged, and BP morphology, distribution, and lateralization were qualitatively assessed using scalp maps and waveforms.

Results: Fifteen healthy participants (mean age 28.0 ± 4.5 years; 9 females) underwent BP recordings. A contralateral centro-frontoparietal BP was observed during simple and complex clonic and tonic hand movements, whereas foot movements

elicited a bilaterally distributed central BP with ipsilateral diffusion, and blink movements showed a bilateral frontal distribution (Figure 1; Table 1). Within-subject analyses revealed an earlier onset of the early phase for complex hand and foot movements, and both earlier early phase onset and delayed late phase onset for tonic hand movements. No amplitude differences were observed (Table 2)

Conclusion: Several factors may influence differences in BP elicited by various movements. Studying normative models across healthy subjects is crucial to characterize BP topography, optimize electrode placement and analysis protocols, and provide a non-invasive window into movement characteristics and potential pathological alterations.

Disclosure: Nothing to disclose.

EPO-1039 | **Simulation of freezing of Gait in a virtual reality environment: An electrophysiological approach in Parkinson's disease**

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Background and aims: This study investigated electrophysiological changes in motor and brainstem pathways under virtual reality (VR) conditions mimicking freezing of gait (FoG)-triggering situations to better understand the pathophysiology of FoG in idiopathic Parkinson's disease (iPD).

Methods: In this prospective study (May 2024–August 2025), patients aged 40–80years with iPD with FoG (iPD-FoG), PD without FoG (iPD-nFoG), and age- and sex-matched healthy individuals were included. iPD diagnosis was based on Movement Disorder Society 2015 criteria, and FoG was assessed using established criteria. Clinical evaluation included the Unified Parkinson's Disease Rating Scale, Timed Up and Go test, auditory startle reflex, resting motor threshold, motor evoked potentials, and H-reflex assessment. All evaluations were performed in the off-medication state. Neurophysiological measurements were obtained at rest and during a VR condition designed to replicate the clinical setting of the Timed Up and Go test.

TABLE 1 Demographic and clinical findings of the study groups: iPD-FoG, iPD-nFoG, and healthy individuals.

Parameter	iPD-FoG n = 8	iPD-nFoG n = 18	Healthy individuals n = 10	P ^a
Age (years)	60.8±8.0	61.9±10.2	60.1±4.2	0.803
Sex, males (%)	5 (62.5%)	7 (38.9%)	6 (60.0%)	0.846
Side of onset (right/left, n %)	4 (50.0%)	7 (38.9%)	—	0.633
LEDS (avg±sd)	11.22±4.29±4.4	4.68±3.02±2.2	—	0.000**
Hoehn-Yahr stage	1 (25.0)	1 (5.6)	—	0.018*
UPDRS Part I	11.7±7.1	4.2±4.0	—	0.021*
UPDRS Part II	19.2±13.8	6.9±6.4	—	0.011*
UPDRS Part III	14.9±21.9	12.3±11.0	—	0.808**
FoG-Q score	11.8±2.1	—	—	—
MMSE score	28.2±1.0	28.3±1.8	29.1±0.0	0.590
TUG time (s)	42.3±14.5	13.4±3.2	10.3±0.0	0.002**

Abb. iPD = idiopathic Parkinson's disease, FoG = freezing of gait, LEDS = Levodopa equivalent daily dose, UPDRS = Unified Parkinson's Disease Rating Scale, Part I = Screening of total UPDRS score, Part II = Motor-related UPDRS subscore, TUG = Timed Up and Go test.
*p < 0.05, **p < 0.01, group differences between iPD-FoG and iPD-nFoG as per Kruskal-Wallis test.

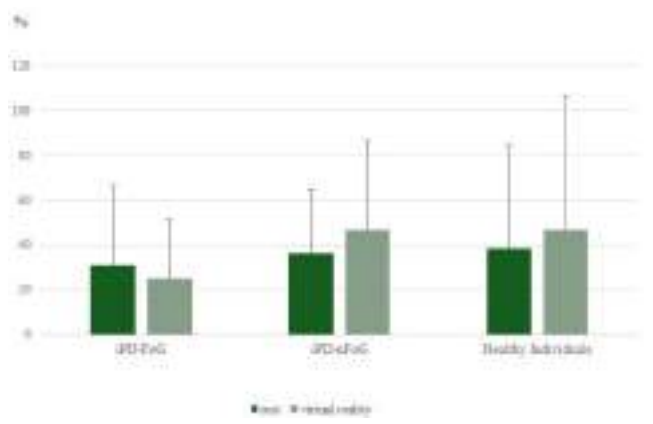


FIGURE 1 Graphics of mean H/M ratios between rest and virtual reality conditions across the groups.

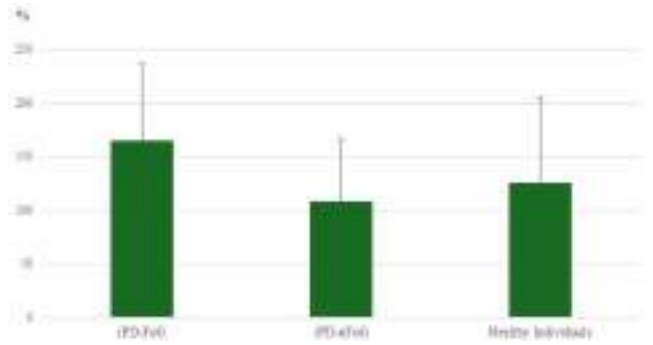


FIGURE 2 MEP amplitude change (% of rest) in iPD-FoG, iPD-nFoG, and healthy individuals.

Results: Eight iPD-FoG patients, ten iPD-nFoG patients, and ten healthy individuals were included. Mean ages were comparable across groups. Resting motor threshold was lower in both iPD groups than in healthy individuals. During VR exposure, mean H/M ratios were higher in healthy individuals and iPD-FoG patients than in iPD-nFoG patients. Percentage increases in motor evoked potential amplitudes were greater in iPD-FoG patients and healthy individuals than in iPD-nFoG patients. Auditory startle reflex amplitudes decreased under VR conditions in all groups.

Conclusion: VR exposure induces corticospinal facilitation while suppressing brainstem pathways. Spinal excitability is facilitated in iPD-nFoG patients, suppressed in iPD-FoG patients, and remains unchanged in healthy individuals.

Disclosure: Nothing to disclose.

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Background and aims: High-quality reference limits for nerve conduction studies (NCS) are essential for accurate diagnosis of neuromuscular disorders. Traditionally, reference limits are derived from examinations of healthy individuals. This is expensive and time-consuming, and it is challenging to include children and elderly. Alternative methods – extrapolated norms (E-norms), extrapolated reference values (E-Ref), and multivariate extrapolated reference values (MeRef) – estimate reference limits from historical hospital data. However, the performance of the current versions of these methods varies across different NCS measurements. We aimed to improve these methods by modifying them using changepoint detection.

Methods: The changepoint algorithm detects significant changes in a data sequence by evaluating different ways of segmenting the data based on segment mean and variance. The modified methods were used to calculate reference limits for common NCS measurements using a historical database of 24,816 patients. The results were compared with reference limits derived from 680 healthy subjects, and evaluated using the F1 score.

Results: The changepoint-modified methods achieved greater F1 scores on average than the original methods for lower-extremity NCS measurements (0.74 vs 0.64), but lower scores for upper-extremity measurements (0.56 vs 0.66). In most cases, good performance required at least 500 measurements and datasets containing fewer than 20% abnormal values.

Conclusion: Changepoint detection improves the performance of E-norms, E-Ref, and MeRef methods for lower-extremity nerves. These improvements are promising and may allow for more accurate diagnostics of neuromuscular disorders, in particular for children, elderly and people living in areas where obtaining local reference values from healthy subjects is unrealistic.

Disclosure: None of the authors has any conflict of interest to disclose. This work received financial support from the South-Eastern Norway Regional Health Authority and the Norwegian Medical Association.

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Background and aims: Reference limits are essential for correctly interpreting nerve conduction studies (NCS). Traditionally, these limits are established using large groups of healthy individuals which is expensive, logistically demanding, and raises ethical concerns. Alternatively, reference limits can be estimated indirectly using historical hospital data containing a mixture of normal and abnormal measurements. Most current alternative methods assess each NCS measurement separately when trying to distinguish normal from abnormal results. In this study, we introduce a novel method – MultiNorm – that instead identifies healthy nerves.

Methods: We combined different types of historical NCS measurements from individual nerves into multidimensional maps and identified regions most likely to represent healthy nerves. Measurements from the presumed healthy nerves were then used to establish reference limits for commonly used NCS and compared with traditional reference limits (from $N=680$ healthy individuals) and four recently published indirect methods (E-norms, E-Ref, MeRef, MMC) using F1 score.

Results: MultiNorm achieved a total average F1 score of 0.81, 0.13 higher than the second-best performing method, extrapolated norms. Reliable performance required datasets containing at least 500 measurements. Unlike the other methods, the performance of MultiNorm remained stable until the proportion of abnormal measurements exceeded 50%, 30% higher than the tolerance of the other approaches.

Conclusion: The MultiNorm approach performs favorably compared with existing indirect methods and shows improved robustness in datasets containing more than 20% abnormal values. It allows accurate estimation of NCS reference limits using historical hospital data and represents a practical and reliable complement to traditional, directly derived reference limits.

Disclosure: None of the authors has any conflict of interest to disclose. This work received financial support from the South-Eastern Norway Regional Health Authority and the Norwegian Medical Association.

EPO-1042 | **Change in functional impact of dyskinesias and motor fluctuations with foslevodopa/foscarbidopa in Parkinson's disease**

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Background and aims: Foslevodopa/foscarbidopa (LDp/CDp), a continuous subcutaneous infusion of levodopa (LD) and carbidopa (CD) prodrugs, reduces dyskinesias and Off times in adults with advanced Parkinson's disease (aPD). Here, we investigated the functional impact of this reduction.

Methods: This post hoc analysis included two phase 3 aPD clinical trials: a 12-week, double-blind, double-dummy, randomized controlled trial (RCT) of LDp/CDp vs oral LD/CD (NCT04380142) and a 52-week open-label safety trial (OLST) of LDp/CDp (NCT03781167). Change from baseline was analyzed for items 4.2 (functional impact of dyskinesias) and 4.4 (functional impact of motor fluctuations) on the Movement Disorder Society-Unified Parkinson's Disease Rating Scale.

Results: In the RCT, the functional impact of dyskinesias significantly worsened from baseline to week 12 with oral LD/CD (mean=0.2, standard deviation [SD]=0.7, $p=0.045$), whereas it significantly improved with LDp/CDp (mean=-0.5, SD=1.0, $p=0.003$, between-group difference $p=0.001$). In the OLST, this functional improvement for dyskinesias with LDp/CDp was -0.6 at week 52 (SD=1.1, $p\leq 0.001$). The functional impact of motor fluctuations in the RCT did not change significantly with oral LD/CD ($p=0.344$), while it significantly improved

TABLE 1 RCT.

RCT			
Change from baseline	Oral LD/CD	LDp/CDp	Between-group difference, P value
Change from baseline in functional impact of dyskinesias, mean (SD), within-group P value			
Week 12, n = 108 ^a	0.2 (0.7), P = .045 [*]	-0.5 (1.0), P = .003 [*]	.001 [*]
Final visit, ^b n = 141 ^a	0.2 (0.6), P = .025 [*]	-0.4 (1.0), P = .003 [*]	.003 [*]
Change from baseline in functional impact of motor fluctuations, mean (SD), within-group P value			
Week 12, n = 108 ^a	-0.1 (1.2), P = .344	-1.0 (1.3), P < .001 [*]	.003 [*]
Final visit, ^b n = 141 ^a	-0.2 (1.2), P = .223	-0.6 (1.5), P = .001 [*]	.253

CD, carbidopa; CDp, foscarbidopa; LD, levodopa; LDp, foslevodopa.
RCT, randomized controlled trial; SD, standard deviation.
^aThis is the total sample size: the sum of participants in the oral LD/CD group and LDp/CDp group.
^bFinal visit is the last nonmissing postbaseline data available for the participant.
^{*}Indicates statistical significance, $P < .05$.

TABLE 2 OLST.

OLST		
Change from baseline	LDp/CDp	Within-group difference from baseline, P value
Change from baseline in functional impact of dyskinesias, mean (SD)		
Week 52, n = 133	-0.6 (1.1)	< .001 [*]
Final visit, ^a n = 244	-0.6 (1.1)	< .001 [*]
Change from baseline in functional impact of motor fluctuations, mean (SD)		
Week 52, n = 133	-1.2 (1.4)	< .001 [*]
Final visit, ^a n = 244	-0.9 (1.5)	< .001 [*]

CD, carbidopa; CDp, foscarbidopa; LD, levodopa; LDp, foslevodopa.
OLST, open-label safety trial; SD, standard deviation.
^aFinal visit is the last nonmissing postbaseline data available for the participant.
^{*}Indicates statistical significance, $P < .05$.

with LDp/CDp (mean = -1.0, SD = 1.3, $p \leq 0.001$, between-group difference $p = 0.003$). In the OLST, motor fluctuation impairment remained improved at 52 weeks (mean = -1.2, SD = 1.4, $p \leq 0.001$).

Conclusion: Functional impairment from dyskinesias and motor fluctuations significantly improved after LDp/CDp treatment in patients with aPD.

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EPO-1043 | Cumulative symptoms improvement with foslevodopa/foscarbidopa vs oral medication in advanced Parkinson's disease

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Background and aims: This post hoc analysis evaluated cumulative symptom improvements with foslevodopa/foscarbidopa

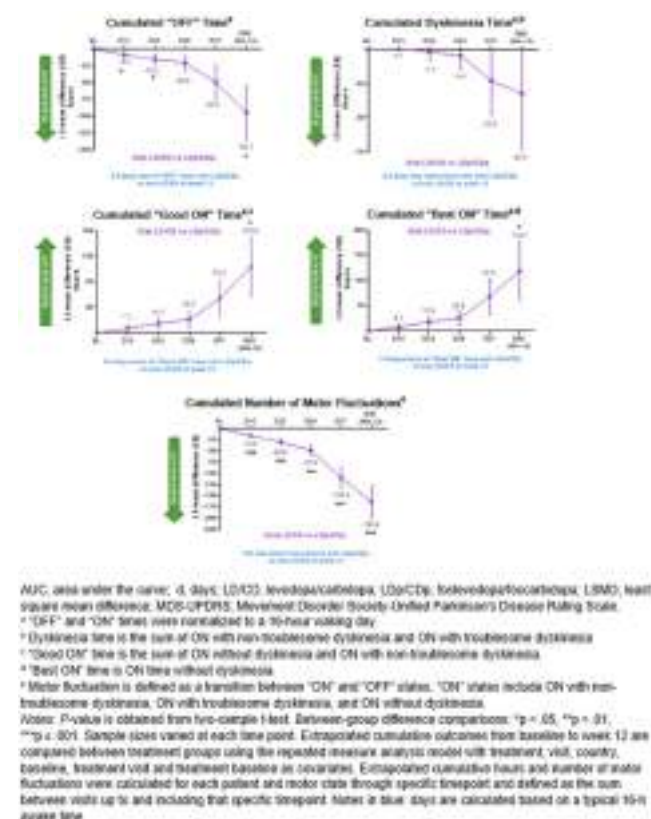


FIGURE 1: Least squares mean differences between treatment groups of cumulated outcomes in the randomized controlled trial.

(LDp/CDp) vs oral medication in advanced Parkinson's disease (aPD).

Methods: Analyses included data from a 12-week, phase 3, double-blind, randomized controlled trial (RCT, NCT04380142) of LDp/CDp vs oral levodopa/carbidopa (LD/CD), and a 52-week open-label trial (OLT, NCT03781167) plus 96-week open-label extension (OLE, NCT04379050) of LDp/CDp vs oral standard of care (SoC) from a 24-month observational study (PROSPECT). Outcomes imputed between visits included change from baseline to week 12 in extrapolated cumulative hours in OFF, ON time with dyskinesia, "good" and "best" ON times from PD diaries (data normalized to 16-h awake time), and number of motor fluctuations. Additional outcomes were assessed using area under the curve. Extrapolated cumulative outcomes were defined as the sum between visits and were reported as least squares mean differences between treatment groups using repeated-measure analyses.

Results: In the RCT, over 85 days, participants receiving LDp/CDp experienced 5.9 fewer accumulative OFF days ($p=0.0219$) and 7.4 more normalized "best ON" days ($p=0.0438$) vs oral LD/CD. LDp/CDp also led to 163 fewer cumulative number of motor fluctuations ($p\leq 0.001$) vs oral LD/CD (Figure 1). When comparing MDS-UPDRS Part II scores, there were 3.5 times more cumulative improvements in motor activities of daily living (ADL) with LDp/CDp vs oral LD/CD ($p=0.3109$). Similar results were obtained from the OLT/OLE vs SoC analysis.

Conclusion: These findings highlight that a sustained and consistent response to LDp/CDp provides meaningful benefits in people with aPD, resulting in improvements in motor symptoms and ADL compared to optimized oral medication.

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EPO-1044 | EEG microstates as predictors for cognitive decline in prodromal DLB

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Background and aims: EEG Microstates (MS) provide temporal and spatial characteristics of whole brain networks. We previously showed that changes in MS temporal parameters (occurrence, time coverage and mean duration) differ between healthy controls and subjects with mild cognitive impairment with Lewy bodies (MCI-LB), such that MCI-LB subjects display hyperexcitability, particularly in MS A (arousal network) and B (visual network). Here we studied whether baseline parameters of MS A and B predict cognitive decline in a prospective longitudinal study.

Methods: Altogether 85 participants on the prodromal spectrum of dementia with Lewy bodies (DLB) who were followed for up to 4 years were analysed: 38 cognitively normal subjects with core clinical features of DLB (CN-CCF), and 26 subjects with mild cognitive impairment with Lewy bodies (MCI-LB). The baseline MS topographies and their temporal parameters were calculated from 5-minute, 204-channel EEG recordings. Linear mixed models were used to predict cognitive decline both longitudinally and cross-sectionally while accounting for age, sex, and the interval between the visits.

Results: Our results show that higher occurrence of MS A tended to predict deterioration of executive functions with time ($p=0.053$). Higher occurrence of MS B predicted visual memory decline at baseline ($p=0.025$).

Conclusion: We demonstrated that increased excitability of the MS representing the arousal network tends to predict changes (decline) in executive functions within up to 4 years of follow-up.

Higher excitability of the MS representing the visual network predicted visual memory impairment at baseline.

Disclosure: Nothing to disclose.

EPO-1045 | Beyond advanced therapies: Sublingual apomorphine as rescue treatment for refractory freezing of gait in LCIG-treated Parkinson’s disease

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Background and aims: Freezing of gait (FoG) is a disabling motor symptom in advanced Parkinson’s disease (PD) and may persist despite device-aided therapies such as levodopa-carbidopa intestinal gel (LCIG). In some patients, FoG episodes remain refractory to additional LCIG bolus doses, representing an unmet therapeutic need. Sublingual apomorphine provides rapid dopaminergic stimulation through a non-invasive route; however, evidence of its use in patients already receiving advanced therapies is scarce.

Methods: We report two male patients with advanced PD receiving stable LCIG therapy who presented recurrent FoG episodes not responding to additional LCIG boluses. The first patient was a 70-year-old man with 18 years of disease duration, treated with LCIG as first device-aided therapy, who received sublingual apomorphine 10mg as rescue treatment. The second patient was a 59-year-old man with 14 years of disease duration, previously treated with continuous subcutaneous apomorphine infusion and currently on LCIG due to suboptimal motor control. Sublingual apomorphine was introduced for persistent FoG. Clinical response, functional impact, and tolerability were assessed based on clinical evaluation and patient-reported outcomes.

Results: Both patients experienced a clear clinical improvement in FoG episodes after sublingual apomorphine administration, allowing recovery from motor blocks refractory to LCIG boluses. Treatment was well tolerated, with no reported adverse effects.

Conclusion: Sublingual apomorphine may represent a useful and well-tolerated rescue option for refractory FoG in complex PD patients treated with LCIG. These cases suggest that rescue strategies using different dopaminergic mechanisms should also be considered in advanced disease stages.

Disclosure: Nothing to disclose.

EPO-1046 | Seizures or Spells? What should the “S” stand for in FBDS associated with LGI1 encephalitis?

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Background and aims: Anti-leucine-rich glioma-inactivated 1 (LGI1) encephalitis is a rare antibody-mediated neurological disease affecting the central nervous system. Its main feature

is facio-brachial dystonic seizures (FBDS), defined as brief, paroxysmal, stereotyped movements affecting facial, cervical and proximal upper limb muscles. The pathophysiological origin of these movements is still unclear.

Methods: We performed a narrative review of the available literature on LGI1 encephalitis, focusing on the clinical and instrumental characterization of FBDS. In parallel, we describe the clinical course, diagnostic work-up, and therapeutic management of a 77-year-old man with LGI1 encephalitis, analyzing clinical and instrumental findings to assess which features supported an epileptic versus dystonic nature.

Results: In our patient, frequent FBDS-like episodes occurred despite persistently normal prolonged video-EEG recordings. The clinical course was atypical, with a prolonged isolated dystonic phase and no limbic seizures. These findings are in contrast to a purely epileptic genesis and suggest a prominent dystonic phenotype. In line with literature reports describing heterogeneous LGI1 presentations, our case is consistent with the existence of different phenotypes, with variable contributions of both epileptic and dystonic mechanisms.

Conclusion: Available evidence suggests that FBDS lie along a continuous spectrum, with one extreme representing a purely epileptic phenotype and the other a purely dystonic phenotype. This model may explain the heterogeneity observed in both clinical and instrumental findings, reflecting distinct patterns of cortical and subcortical network involvement. This perspective highlights why the meaning of the “S” in FBDS warrants reconsideration.

Disclosure: Nothing to disclose.

EPO-1047 | Frozen in Laughter: A curious case of paroxysmal facial pain

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Background and aims: Hemimasticatory spasm (HMS) is a rare movement disorder with paroxysmal, unilateral contractions of jaw-closing muscles. Because it can mimic mandibular dystonia, temporomandibular joint disorders, trigeminal neuralgia, or hemifacial spasm, HMS is often under-recognized. Most reported cases are associated with facial hemiatrophy; idiopathic HMS without hemiatrophy is uncommon and diagnostically challenging.

Methods: We report the clinical, neurophysiological, imaging, and treatment features of HMS without facial hemiatrophy. A single-patient case report was conducted at a movement disorders clinic. Triggers and phenomenology were documented. Interictal and ictal needle EMG and trigeminal electrophysiology (blink reflex, masseter reflex, masseteric silent period) were performed, and brain/face MRI reviewed for structural lesions and masticatory asymmetry.

Results: A 34-year-old school teacher had 2 years of painful right masseter–temporalis spasms triggered by laughter/eating, sometimes causing brief jaw locking and teeth chattering, with occasional nocturnal episodes. Medications (clonazepam, haloperidol, cyclobenzaprine) were ineffective. Examination showed right masseter/temporalis hypertrophy with episodic contractions; cranial nerves and jaw jerk were normal and no sensory tricks were present. Autoimmune/inflammatory tests



FIGURE 1 EMG (Masseter): Normal at rest (no spontaneous activity; normal MUPs). During spasms, brief clonic bursts (10–200 ms) with variable inter-burst intervals (20–200 ms), sometimes merging into continuous activity; peak firing up to ~200 Hz.



FIGURE 2 Paroxysmal, painful involuntary spasms of the right masseter and temporalis muscles.

were normal. Interictal EMG was normal; ictal EMG showed clonic bursts (10–200 ms) with variable intervals and firing up to 200 Hz. Blink reflex was normal; during attacks, masseter reflex latency was delayed with reduced amplitude and an attenuated masseteric silent period. MRI showed right masseter enlargement only. Botulinum toxin produced marked improvement.

Conclusion: HMS should be considered in trigger-sensitive unilateral jaw-closing spasms with pain and hypertrophy even without facial hemiatrophy; attack-related impairment of inhibitory silent periods supports diagnosis and botulinum toxin is an effective first-line therapy.

Disclosure: Nothing to disclose.

EPO-1048 | Deep brain stimulation in KMT2B-related dystonia: Electrophysiological and clinical parameters during a 12 month-follow-up period in two children

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Background and aims: Pathogenic variants in the KMT2B gene lead to childhood-onset, generalized dystonia (DYT-KMT2B).

Deep brain stimulation (DBS) to the internal globus pallidus (GPi) is an effective therapy. With sensing-enabled neurostimulators, electrophysiological biomarkers can be recorded chronically. Previously, pallidal theta activity correlated with symptom severity, and entrained gamma may indicate effective DBS. However, longitudinal dynamics of electrophysiological biomarkers in KMT2B-related dystonia are unknown.

Methods: Clinical and electrophysiological data were recorded regularly in the first 12-months follow-ups after GPi-DBS (COMEDD-trial, NCT07244549) in two children with DYT-KMT2B (age at surgery 5 and 9 years) with the Percept neurostimulator. Dystonia was quantified using the Burke-Fahn-Marsden-Dystonia-Rating-Scale motor score (BFMDRS-M). Pallidal electrophysiology during rest and movement was recorded ON/OFF DBS and analyzed using short-fourier-transformation and spectral parametrization. Peak activity in theta band and beta or gamma band was recorded out-of-hospital and analyzed for chronic biomarker changes and circadian periodicity.

Results: Both girls were DBS responders ($54 \pm 3\%$ improvement in BMFDRS-M at 12 months-follow-up). In both patients, entrained gamma occurred at clinically used amplitudes, and there was theta suppression over time. In one of the two patients, chronic recordings revealed increased peak biomarker activity especially around times of symptom deterioration. Circadian periodicity analysis suggests distinct patterns for theta, gamma and beta band activity.

Conclusion: DBS is an effective therapy in KMT2B-related pediatric dystonia. Theta suppression and occurrence of entrained gamma seem to be associated with clinical improvement and may be used to guide electrophysiology-based programming in future. Further, chronic biomarkers could be used to understand out-of-hospital symptom fluctuations.

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EPO-1049 | Diagnostic sensitivity of a shortened accelerometry-only functional tremor battery

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Background and aims: Electrophysiologically supported identification of positive signs is important in confirming the diagnosis of functional tremor. In this respect, the functional tremor

battery proposed by Schwingenschuh et al. is a valuable diagnostic tool but requires surface electromyography, specialised equipment and expertise, limiting its routine clinical use. As most battery tests rely on accelerometric data, we aimed to evaluate the diagnostic sensitivity of a shortened, accelerometry-only version of the functional tremor battery.

Methods: Thirty patients with clinically diagnosed functional tremor were included. All patients underwent the full functional tremor battery (maximum score 10 points). A proposed cutoff score of ≥ 3 points was used as a standard to ensure a homogeneous cohort. Among patients meeting this cutoff, we examined whether the cutoff would still be met after excluding surface electromyography-based tests (coactivation sign and tremor coherence). Subsequently, the weight loading test was also excluded to evaluate its redundancy in the overall diagnostic sensitivity of the shortened test battery.

Results: Twenty-four of 30 patients (80%) scored ≥ 3 points on the full battery and were included in further analyses. After exclusion of surface electromyography-based tests, all 24 patients (100%) continued to score ≥ 3 points. Following additional exclusion of the weight loading test, 23 of 24 patients (95.8%) still met the cutoff.

Conclusion: A shortened accelerometry-based functional tremor battery demonstrates diagnostic sensitivity comparable to the full battery that includes surface electromyography. This simplified approach reduces technical demands, examination time, and required expertise, potentially enabling wider clinical implementation.

Disclosure: Nothing to disclose.

EPO-1050 | Opicapone for management of sleep disturbances in Parkinson's disease: Post-hoc analysis of the OASIS trial in patients with and without probable RBD

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Background and aims: Rapid eye movement sleep behavior disorder (RBD) is prevalent in Parkinson's disease (PD). Opicapone improved sleep disturbances in fluctuating PD patients with PD-related sleep disturbances in the exploratory Phase IV Opicapone in Sleep dISorder (OASIS) trial. This OASIS post-hoc analysis evaluates opicapone's effect on sleep disturbances, motor/non-motor symptoms and quality of life in PD patients with and without probable RBD, as determined by their medical history.

Methods: The 6-week, single-arm OASIS trial evaluated opicapone 50 mg in levodopa-treated fluctuating PD patients with sleep disturbances. Assessments included PD Sleep Scale-2 (PDSS-2), Movement Disorder Society-sponsored Non-Motor Symptoms rating scale (MDS-NMS), Patient and Clinician Global Impressions of Change, Parkinson's Fatigue Scale-16 (PFS-16), Parkinson's Disease Questionnaire-8 (PDQ-8).

whether efficacy could be achieved in patients with lower baseline symptom burden (MG-ADL scores: 6–9).

Methods: Analysis included seropositive patients treated with nipocalimab or placebo with baseline MG-ADL score below cohort median score of 9. Efficacy was evaluated by changes in MG-ADL (meaningful clinical improvement [MCI]: ≥ 2 -point improvement; substantial clinical improvement [SCI]: ≥ 3 -point improvement), and QMG (MCI: ≥ 3 -point improvement; SCI: ≥ 4 -point improvement) scores at week (W) 24. Two-sample *t*-tests compared changes-from-baseline between groups.

Results: In this population, baseline demographics were generally balanced between nipocalimab and placebo-treated patients (Table). At W24, MG-ADL reductions (mean [SD]) from baseline were greater with nipocalimab (-4.5 [2.64]) versus placebo (-2.3 [2.37]); difference: -2.23 (standard error [SE]: 0.588); 95%CI (-3.41 ; -1.06); $p < 0.001$. QMG reductions (mean [SD]) were also greater with nipocalimab (-5.2 [4.45]) versus placebo (-1.9 [3.69]); difference: -3.38 (SE: 0.986); 95%CI (-5.34 ; -1.41); $p = 0.001$. A greater proportion of patients receiving nipocalimab met MCI and SCI criteria for MG-ADL and QMG at W24 versus placebo (Table).

Conclusion: In patients with baseline MG ADL scores 6–9, nipocalimab demonstrated greater efficacy versus placebo, achieving MCI and SCI in MG ADL and QMG at W24. These results support the benefit of nipocalimab for sustained disease control in patients with gMG with lower baseline MG-ADL scores.

Disclosure: Carlo Antozzi: received funding for travel, meeting attendance, and advisory board participation from Alexion, Momenta, Sanofi, argenx, UCB, Janssen Pharmaceuticals and Johnson & Johnson. Wim Noel, Wissam Karmous, Marie Fitzgibbon, Kavita Gandhi, Ibrahim Turkoz, and Sindhu Ramchandren: are employees of Johnson & Johnson and may hold stock or stock options in the company. Michael Kutch: is an employee of Cytel Inc, which derives profits from interactions with pharmaceutical sponsors.

EPO-1052 | Corticosteroid use in nipocalimab-treated Japanese patients with generalized myasthenia gravis: Phase 3 vivacity-MG3 open-label extension study

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Background and aims: In seropositive adults with generalized myasthenia gravis (gMG), nipocalimab treatment (nipocalimab+standard-of-care [SOC]) vs placebo+SOC achieved sustained disease control during the 24-week VIVACITY-MG3 study (NCT04951622) double-blind phase. Sustained disease control was maintained through open-label extension (OLE) week 60. During OLE, 45% of patients receiving nipocalimab ($N = 89$) reduced/discontinued corticosteroids; a substantial proportion demonstrated minimum symptom

TABLE 1 Patient baseline and efficacy endpoint results.

	Japanese (N=12)	Non-Japanese (N=34)
Patients Receiving Steroids at OLE Baseline (N)	11	78
Baseline Prednisone Equivalent (mg/day), mean (SD)	9.2 (2.46)	19.6 (16.27)
Endpoint Prednisone Equivalent (mg/day), mean (SD)	6.2 (2.92)	14.3 (11.51)
Prednisone Equivalent Change (mg/day), mean (SD)	-3.0 (2.74)	-5.3 (12.38)
Patients with Steroid Reduction* at data cutoff (N)	7	33
Baseline Prednisone Equivalent (mg/day), mean (SD)	10.4 (2.32)	25.1 (20.53)
Endpoint Prednisone Equivalent (mg/day), mean (SD)	5.6 (2.82)	11.2 (10.50)
Prednisone Equivalent Change (mg/day), mean (SD)	-4.8 (1.68)	-13.9 (13.32)
Patients with ≤ 5 mg prednisone mg/day at data cutoff, n (%)	5 (71.4%)	12 (36.4%)
MSE† achieved in first 48 weeks, n (%)	4 (57.1%)	14 (42.4%)

The data (n=off date: August 24, 2024).
*Reduction defined as a decreased dose or discontinuation of steroid.
†Minimum symptom expression (MSE) is defined as MG-ADL total score of 9 or 1.
‡Population size, n=number in group; OLE=open-label extension; SD=standard deviation

expression (MSE; MG-Activities of Daily Living [MG-ADL] score=0/1) and sustained for ≥ 8 weeks. In Japan, achieving minimal gMG symptoms with low corticosteroid doses (target: ≤ 5 mg/day) is an important treatment goal. We characterized the impact of nipocalimab on corticosteroid use in Japanese patients in the VIVACITY-MG3 OLE phase.

Methods: Corticosteroid doses (mg/day prednisone-equivalent) were evaluated through OLE data cut-off (August 2024). Among patients who reduced corticosteroid dose (≤ 5 mg/day), the proportion achieving MSE was evaluated.

Results: Of 11 Japanese patients receiving corticosteroids at OLE baseline, 7 (64%) receiving nipocalimab in OLE reduced/discontinued corticosteroids at data cutoff; of these, 5 (71%) reached ≤ 5 mg/day. Among steroid reducers, mean (SD) steroid dose reduced from 10.4 (2.32) to 5.6 (2.82) mg/day prednisone-equivalent in Japanese patients, while in remaining population dose reduced from 25.1 (20.53) to 11.2 (10.50) mg/day prednisone-equivalent. Among those reducing corticosteroids, 4/7 (57%) patients achieved MSE within 48 weeks (Table 1).

Conclusion: In OLE, over half of Japanese patients reduced/discontinued corticosteroids with substantial proportions tapering to low-dose corticosteroid (≤ 5 mg/day) and achieving MSE within 48 weeks. These results are consistent with the overall population where almost half of patients receiving corticosteroids at baseline, reduced/discontinued steroids and showed sustained disease control with nipocalimab.

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Background and aims: Autoantibody-mediated complement activation damages neuromuscular junctions in gMG, resulting in variable muscle weakness. The phase 3 NIMBLE trial evaluated subcutaneous cemisiran (siRNA inhibiting hepatic C5 production) and subcutaneous pozelimab (monoclonal antibody targeting C5) as monotherapies or in combination in patients with gMG.

Methods: NIMBLE is a double-blind, placebo-controlled trial. A total of 288 patients with gMG were randomised to cemisiran (600mg Q12W), pozelimab (200mg Q4W), combination (both 200mg Q4W), or placebo. We present key results for the primary efficacy endpoint, change from baseline in MG-ADL at Week 24, based on August 2025 database lock ($n=239$), and the cumulative rate of TEAEs based on December 2025 database lock ($n=281$).

Results: NIMBLE met its primary endpoint. In the cemisiran ($n=64$) and combination ($n=67$) arms, the least squares mean (SE) of change from baseline at Week 24 in MG-ADL total score was -4.5 (0.4) and -4.0 (0.4), with placebo-adjusted differences of -2.3 ($p=0.0005$) and -1.7 ($p=0.0086$); achieving 76.6% ($n=75$) and 99.6% ($n=75$) terminal complement inhibition, respectively. TEAEs occurred in 74.7% (59/79), 81.6% (40/49), 81.5% (66/81), and 81.9% (59/72) in the cemisiran, pozelimab, combination, and placebo arms, respectively. No meningococcal, serious infections or deaths occurred in the cemisiran arm during the double-blind treatment period (DBTP); SAEs of infections in other arms were low. There were 2 deaths (pneumonia [cemisiran], septic shock [combination]) in patients on concomitant immuno-suppressive therapies and comorbidities after the DBTP.

Conclusion: Quarterly, subcutaneous cemisiran was generally well-tolerated and achieved significant improvement in symptoms with partial complement inhibition among patients with gMG.

Disclosure: This study was funded by Regeneron Pharmaceuticals, Inc. SJ has served as an international advisory board member for Alexion, Alnylam, Argenx, Immunovant, Johnson and Johnson, Merck, Novartis, Regeneron and UCB pharmaceuticals, is currently an expert panel member of Myasthenia

Gravis consortium for Argenx pharmaceuticals and has received speaker fees from Argenx, Eisai, Merck, Terumo BCT and UCB pharmaceuticals. He is also a board member (trustee) of the UK myasthenia patient charity, Myaware. TV has served as the USF Site Principal Investigator for MG clinical trials sponsored by Alexion/AstraZeneca Rare Disease, Amgen, argenx, Cartesian Therapeutics, COUR, Dianthus Therapeutics, EMD Serono, ImmunAbs, Immunovant, Johnson & Johnson, NMD Pharma, Regeneron Pharmaceuticals, Inc, VOR, and UCB. He has served as a speaker for Alexion/AstraZeneca Rare Disease, argenx, CSL Behring, and Johnson & Johnson. He served on advisory boards and/or performed consulting work for Alexion/AstraZeneca Rare Disease, argenx, Amgen, Dianthus Therapeutics, Johnson & Johnson, ImmunAbs, NMD Pharma, and VOR. He also participated in CME events sponsored by AcademicCME, CMEO, PER, and MedLive. HM has served as a consultant for Alexion, argenx, and UCB, and has received speaker honoraria from Novartis, Johnson & Johnson and Merck, and research support from the Ministry of Health, Labour and Welfare, Japan. AM received speaker or consultancy honoraria or financial research support from Alexion Pharmaceuticals, Argenx, Amgen, Axunio, Desitin, Grifols, Janssen, Merck, Novartis, Octapharma, Sanofi and UCB. He serves as chairmen of the Association for Research in Myasthenic Syndromes in Germany (VEMSID e.V.). RP, UC, NJ, MD, KAM, SS, DP, AS, and LP are employees and stockholders of Regeneron Pharmaceuticals, Inc. JFH Jr has received research funding from Ad Scientiam, Alexion AstraZeneca Rare Disease, argenx, Cartesian Therapeutics, Centers for Disease Control and Prevention, Merck EMD Serono, MGFA, Muscular Dystrophy Association, NIH, NMD Pharma, and UCB Bioscience; honoraria/consulting fees from AcademicCME, Alexion AstraZeneca Rare Disease, Amgen, argenx, Biohaven Ltd, Cartesian Therapeutics, CheckRare CME, CoreEvitas, Curie.bio, H. Lundbeck A/S, Japan Tobacco Company, Kyverna Therapeutics, Merck EMB Serono, NMD Pharma, Novartis Pharma, PeerView CME, Physicians' Education Resource (PER) CME, PlatformQ CME, Regeneron Pharmaceuticals, Inc, Seismic Therapeutics, TG Therapeutics, Toleranzia AB, UCB Bioscience and Vor Bio; non-financial support from Alexion AstraZeneca Rare Disease, argenx, Biohaven Ltd, Cartesian Therapeutics, Toleranzia AB, and UCB Pharma. AH, RM, JV, AS, TL, and YH have nothing to declare.

EPO-1054 | Corticosteroid and biologic use in a real-world population with generalized myasthenia gravis in the United States and Europe

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Background and aims: Generalised myasthenia gravis (gMG) is a neuromuscular condition manifesting as fluctuating muscle weaknesses and fatigue. Treatments include corticosteroids and targeted biologic treatments. This study describes corticosteroid and biologic use in a multi-country sample of gMG patients.

Methods: Data were drawn from the Adelphi MG Disease Specific Programme (DSP)[™], a cross-sectional survey of neurologists and their patients with gMG in the France, Germany, Italy,

TABLE 1 Patient age, sex, MG-ADL score and time since gMG diagnosis, split by country.

	All Patients	France	Germany	Italy	Spain	UK	USA
Age, mean (SD)	61.7 (11.3)	59.7 (10.8)	60.4 (11.1)	58.5 (11.8)	61.7 (11.3)	60.1 (10.8)	61.7 (11.3)
Female sex, n (%)	391 (50.2)	111 (51.8)	101 (51.8)	91 (51.8)	111 (51.8)	101 (51.8)	101 (51.8)
MG-ADL score at time of survey, mean (SD)	4.5 (3.3)	4.5 (3.3)	4.5 (3.3)	4.5 (3.3)	4.5 (3.3)	4.5 (3.3)	4.5 (3.3)
Time since gMG diagnosis, mean (SD)	1.8 (2.8)	1.8 (2.8)	1.8 (2.8)	1.8 (2.8)	1.8 (2.8)	1.8 (2.8)	1.8 (2.8)
Time since gMG diagnosis, n (%)	627 (80.7)	127 (60.5)	127 (60.5)	127 (60.5)	127 (60.5)	127 (60.5)	127 (60.5)

TABLE 2 Proportion of gMG patients prescribed maintenance treatment of corticosteroid and biologic treatments, split by country.

	All Patients	France	Germany	Italy	Spain	UK	USA
Patients prescribed maintenance treatment at time of survey, n (%)	391 (50.2)	111 (51.8)	101 (51.8)	91 (51.8)	111 (51.8)	101 (51.8)	101 (51.8)
Patients prescribed corticosteroids at time of survey, n (%)	337 (86.2)	98 (48.1)	98 (48.1)	98 (48.1)	98 (48.1)	98 (48.1)	98 (48.1)
Patients prescribed biologics at time of survey, n (%)	101 (25.8)	101 (48.1)	101 (48.1)	101 (48.1)	101 (48.1)	101 (48.1)	101 (48.1)

Spain, UK and United States, from January 2024 - November 2025. Physicians provided patient-level demographics, clinical characteristics and treatment.

Results: Overall, 146 neurologists reported on 917 gMG patients, patient demographics are shown by country in **TABLE 1** Physicians reported a mean (Standard Deviation; SD) Myasthenia Gravis - Activities of Daily Living (MG-ADL) score of 4.5 (3.3) and 59.8% of patients were in Myasthenia Gravis Foundation of America class II. At time of survey, 88.3% of patients were prescribed a gMG treatment. Of those, 40.4% were prescribed corticosteroids, most commonly prednisone (35.6%). Patients had been prescribed their current regimen of corticosteroids for a mean (SD) 1.8 (2.8) years and 43.1% were prescribed a dosage ≥ 20 mg. Among patients on treatment, 37.2% were prescribed biologics. Patients had been prescribed their current biologic treatment regimen, for a mean (SD) 1.1 (1.2) years at survey and the biologic was administered a mean (SD) 2.3 (4.1) times per month.

Conclusion: Corticosteroid and biologic therapies were frequently prescribed in the six countries included in this analysis. Reducing steroid burden and frequent administrations of biologic therapies whilst maintaining efficacy could benefit this population.

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Lauretta Birija is an employee at Adelphi Real World, which received research funds from Amgen for this work. Beth Poirrette is an employee at Adelphi Real World, which received research funds from Amgen for this work. Amy Foster is an employee at Adelphi Real World, which received research funds from Amgen for this work. Ankur Bhambri is an employee of Amgen and receives salary and/or stocks/shares in Amgen.

EPO-1055 | Healthcare costs of generalized myasthenia gravis in Germany: A real-world retrospective cohort study

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Background and aims: Generalized myasthenia gravis (gMG) is a chronic autoimmune neuromuscular disorder characterized by muscle weakness and fatigue affecting daily activities. This study describes patient characteristics and healthcare costs among patients with gMG in Germany.

Methods: We conducted a retrospective cohort study using anonymized secondary healthcare claims data from the InGef database from 2017 to 2024. Patients aged ≥ 18 years with ≥ 1 inpatient or ≥ 2 outpatient diagnoses and ≥ 12 months of continuous enrollment before and after the index date were included. Follow-up was variable length with a minimum of 12 months. Annualized healthcare costs per patient per year (PPPY) were calculated across outpatient, inpatient, emergency department, and pharmacy services.

Results: A total of 4,025 patients with gMG were identified (mean age 61.2 years, standard deviation 16.7 years; 49.3 percent female). Median total healthcare costs were 6,115.06 euros (interquartile range 2,942.54 to 13,011.55 euros) PPPY, with gMG-related costs of 1,279.06 euros (interquartile range 346.74 to 3,238.00 euros). Costs were higher among patients with ≥ 1 exacerbation ($N=2,144$; median total costs 7,757.87 euros PPPY; median gMG-related costs 2,262.18 euros PPPY) and among patients with ≥ 1 myasthenic crisis ($N=93$; median total costs 34,605.52 euros PPPY; median gMG-related costs 14,928.98 euros PPPY).

Conclusion: This real-world study demonstrates the substantial economic burden of gMG in Germany. Higher costs among patients with exacerbations and myasthenic crises emphasize the impact of disease severity and the need for improved disease management.

Disclosure: Christiane Schneider-Gold receives research funding from Amgen. Seth Anderson is an employee of Amgen and

TABLE 1 All-cause and gMG-related healthcare costs by disease severity.

	All-cause healthcare costs during follow-up				gMG-related healthcare costs during follow-up			
	All-cause costs	gMG-related costs	All-cause costs	gMG-related costs	All-cause costs	gMG-related costs	All-cause costs	gMG-related costs
All	1001	1001	1001	1001	1001	1001	1001	1001
Male	1001	1001	1001	1001	1001	1001	1001	1001
Female	1001	1001	1001	1001	1001	1001	1001	1001
Median	1001	1001	1001	1001	1001	1001	1001	1001
IQR	1001	1001	1001	1001	1001	1001	1001	1001

receives salary and/or stocks/shares in Amgen. Ina Bucholz is an employee of Amgen and receives salary and/or stocks/shares in Amgen. Vukašin Višković is an employee at InGef, which received research funds from Amgen for this work. Anna Marzà-Florensa is an employee at PHARMO, which received research funds from Amgen for this work. Marion Ludwig is an employee at InGef, which received research funds from Amgen for this work. Abigail Postema is an employee at PHARMO, which received research funds from Amgen for this work. Ankur Bhambr is an employee of Amgen and receives salary and/or stocks/shares in Amgen.

EPO-1056 | Diagnostic utility of anti-SRP54 antibody ELISA in immune-mediated necrotizing myopathy: Experience from a real-world cohort

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Background and aims: Immune-mediated necrotizing myopathy (IMNM) associated with anti-signal recognition particle (SRP) antibodies typically presents with rapidly progressive proximal weakness and markedly elevated creatine kinase levels. In clinical practice, line-blot immunoassays are commonly used to detect anti-SRP antibodies, but their reliability and reproducibility are sometimes questioned. We examined the performance of an enzyme-linked immunosorbent assay (ELISA) targeting anti-SRP54 antibodies and explored its clinical relevance in a real-world IMNM cohort.

Methods: Patients evaluated for idiopathic inflammatory myopathies between 2002 and 2023 were retrospectively reviewed. Eighty-seven patients with inflammatory myopathies and 107 healthy controls were included. Anti-SRP IMNM was defined based on line-blot immunoassay results, and the diagnostic performance of anti-SRP54 ELISA was assessed. Clinical features, laboratory findings, and coexisting autoantibodies were analyzed in ELISA-positive patients.

Results: Among 32 patients diagnosed with anti-SRP IMNM using line-blot immunoassays, 28 (88%) tested positive on anti-SRP54 ELISA. The ELISA demonstrated a sensitivity of 88% and a specificity of 99%, with high test-retest reliability. All ELISA-positive patients showed proximal muscle weakness. Neck weakness, myalgia, dysphagia, or respiratory symptoms were observed in a subset of patients. Extra-muscular involvement, including interstitial lung disease and myocarditis, was uncommon. Serum creatine kinase levels were markedly elevated overall and tended to be higher in patients with coexisting anti-Ro-52 antibodies.

Conclusion: Anti-SRP54 antibody ELISA showed high diagnostic accuracy and good reproducibility in patients with anti-SRP IMNM. In routine clinical practice, this assay can serve as a reliable complementary tool to support the diagnosis of IMNM and may help increase diagnostic confidence in patients with suspected disease.

Disclosure: Nothing to disclose.

EPO-1057 | Do we really need non-steroidal immunosuppressants in generalized myasthenia gravis

treated with new targeted therapies? A pilot study

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Background and aims: Targeted biologic therapies, including complement and neonatal Fc receptor (FcRn) inhibitors, have transformed the treatment of generalized myasthenia gravis (gMG). However, the role of concomitant non-steroidal immunosuppressants (NSIST) remains uncertain. This study evaluated whether background NSIST influences clinical outcomes and corticosteroid reduction in gMG patients treated with targeted therapies.

Methods: We conducted a retrospective, observational study including adult gMG patients treated with complement inhibitors or anti-FcRn agents ($n=41$). Clinical outcomes were assessed at baseline and during follow-up using Myasthenia Gravis Activities of Daily Living (MG-ADL) and Quantitative Myasthenia Gravis (QMG) scales. Achievement of Minimal Symptom Expression (MSE), MG Foundation of America Post-Intervention Status (MGFA-PIS), and changes in corticosteroid dose were analyzed.

Results: Twenty patients received background NSIST (Group 1), while 21 did not (Group 2). Baseline MG-ADL scores and corticosteroid doses were comparable between groups (9.10 ± 4.36 vs 8.24 ± 3.38) (22.17 ± 13.81 mg vs 22.06 ± 16.43). Baseline QMG scores were slightly higher in Group 1 (14.0 ± 5.1 vs 10.9 ± 4.8 ; $p=0.049$). Both groups showed significant improvement in MG-ADL and QMG scores at all follow-up time points, without differences in clinical scale improvement comparing the two groups. Rates of MSE achievement, MGFA-PIS improvement, and corticosteroid tapering did not differ between groups.

Conclusion: Our pilot analysis indicates that concomitant use of NSIST does not appear to be associated with greater improvement in clinical outcomes, enhance corticosteroid-sparing effects or increased disease stability. Larger, prospective studies are needed to optimize treatment strategies in gMG.

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Background and aims: Understanding patients' preferences for Generalised Myasthenia Gravis (gMG) is essential for informed treatment decisions. However, limited evidence is available regarding how Chinese patients weigh safety, efficacy, onset speed, and convenience when choosing treatments. This study aimed to assess treatment preferences and willingness to pay (WTP) among Chinese patients.

Methods: This multicenter, cross-sectional survey was conducted from March to August 2025 among gMG patients across

TABLE 1 The disease characteristics and treatment of patients with gMG

Characteristic	Patients (n=909)
Duration of gMG*, months (SD)	30 (18-62-78-35)
MGFA class, n (%)	
II	478(52.3)
III	343(37.5)
IV	108(11.9)
Serological testing, n (%)	
Negative	268(29.6)
Positive	640(70.4)
Anti-AChR antibody positive	423(68.5)
Anti-MuSK antibody positive	25(2.8)
Anti-LRP4 antibody positive	30(3.3)
Anti-transient receptor potential calcium channel antibody positive	10(1.1)
MG-ADL, (min score, mean±SD)	1-9±3.25
Comorbidity, n (%)	
Yes	488(53.6)
No	421(46.4)
Type of comorbidity, n (%)	
Hypertension	228(24.2)
Diabetes	138(14.3)
Hyperlipidemia	60(6.5)
Others	21(2.3)
Thymectomy, n (%)	
Yes	36(3.9)
No	873(96.1)
Mixing	1
Treatment for gMG	
Pyridostigmine bromide	85(9.2)
Prednisone acetate	71(7.7)
Methylprednisolone	134(14.7)
Azathioprine	102(11.2)
Tacrolimus	43(4.6)
Mycophenolate mofetil	38(4.1)
Sirolimus	14(1.5)
Infliximab	21(2.3)
Flutemetamol	6(0.6)
Others	17(1.8)
Administration route of gMG treatment	
Oral	98(9.7)
Subcutaneous infusion	35(3.7)
Subcutaneous injection	24(2.5)
Others	45(4.8)
Average annual out-of-pocket costs for gMG treatment†	
<¥1000	33(3.5)
¥1000 to ¥5000	37(4.0)
¥5000 to ¥10000	103(11.2)
¥10000 to ¥50000	58(6.3)
>¥50000	26(2.8)

*Onset duration of gMG was defined as the duration from diagnosis to the first of informed consent.
†Others: These 56 patients received plasma exchange.
*AChR: acetylcholine receptor; gMG: generalized myasthenia gravis; LRP4: low-density lipoprotein receptor-related protein 4; MuSK: muscle-specific kinase; Myasthenia Gravis-Activities of Daily Living; MGFA: Myasthenia Gravis Foundation of America; MuSK: muscle-specific kinase

TABLE 2 Analysis of patients' medication preferences and willingness to pay.

Attribute (Levels)	Regression coefficient	95%CI	P value	Willingness-to-pay (per year)*
Onset time of action				
≤2 Weeks	0.157	(0.04, 0.24)	<0.0001	\$4,000
>2 Weeks	Ref	—	—	—
Administration route				
Oral	0.241	(0.16, 0.31)	<0.0001	\$7,000
Subcutaneous injection (20 min)	0.108	(0.04, 0.17)	0.0035	\$3,000
Subcutaneous infusion (2-60 min)	Ref	—	—	—
Treatment frequency				
Once daily	0.284	(0.17, 0.39)	<0.0001	\$8,000
Once weekly	0.109	(0.06, 0.15)	0.0352	\$3,000
Once biweekly	0.058	(0.03, 0.09)	0.2944	\$2,000
Once every 6-month	0.396	(0.29, 0.49)	<0.0001	\$11,000
2-3 times daily	Ref	—	—	—
Metabolic disease risk				
Low	0.477	(0.40, 0.55)	<0.0001	\$11,000
Moderate	0.366	(0.29, 0.43)	<0.0001	\$10,000
High	Ref	—	—	—
Infection risk				
Low	0.419	(0.34, 0.49)	<0.0001	\$12,000
Moderate	0.308	(0.23, 0.38)	<0.0001	\$8,000
High	Ref	—	—	—
Myelosuppression risk				
Low	0.565	(0.49, 0.63)	<0.0001	\$16,000
Moderate	0.405	(0.32, 0.48)	<0.0001	\$11,000
High	Ref	—	—	—
Liver and kidney function impairment				
Low	0.463	(0.38, 0.54)	<0.0001	\$13,000
Moderate	0.343	(0.27, 0.41)	<0.0001	\$9,000
High	Ref	—	—	—
Annual out-of-pocket costs	-0.363	(-0.39, -0.33)	<0.0001	—

*q = 10.14 in 2025.

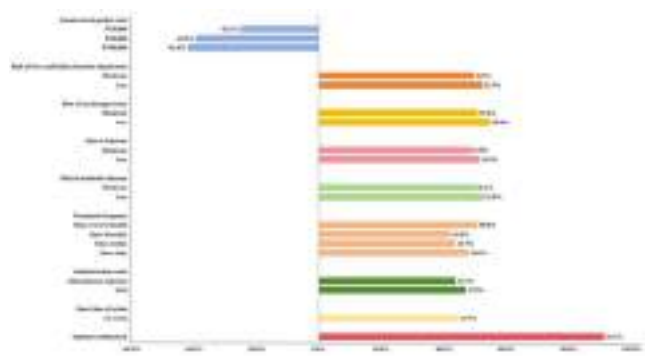


FIGURE 1 The selection probability of gMG patients.

China. A discrete choice experiment was used to quantify patient preferences by presenting hypothetical treatment scenarios.

Results: Of 909 analysed patients, treatment safety was a key determinant of patient preferences. Oral administration was most preferred, followed by subcutaneous injection. Less frequent dosing was strongly favoured: once every 6 months, and once daily, compared to 2–3 times daily. Younger patients paid more attention to low-frequency dosing, subcutaneous injection, and liver and kidney function impairment than older patients. Patients with MGFA class II were more concerned about oral administration, once-weekly dosing, infection risk, and myelosuppression. High-income patients (>¥100,000) favoured low risk of metabolic disease, infection, myelosuppression, and liver and kidney function impairment and infrequent (weekly, biweekly, or every-6-month), subcutaneous administration, and were less sensitive to cost compared to low- (< ¥50,000) and medium-income groups (¥50,000–100,000).

Conclusion: Safety, infrequent and convenient administration route (oral/subcutaneous), affordable cost, and rapid onset of action were prioritised. Patients were willing to invest financially in therapies that align with these preferences. Embedding

patient preferences in clinical decision-making and reimbursement policy can improve adherence, reduce disease burden, and enhance quality of life for individuals with gMG in China.

Disclosure: Nothing to disclose.

Neurocritical Care

EPO-1059 | **Aetiology, clinical features, and outcomes of paediatric TBI management in sub-Saharan Africa: A systematic review and meta-analysis of regional data**

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Background and aims: Despite the rising burden of paediatric traumatic brain injury (TBI) in sub-Saharan Africa, there is still fragmented evidence on its clinical profile and outcomes. This review evaluated the aetiology, clinical presentation, management, and outcomes of paediatric TBI to establish pooled and regional estimates.

Methods: Articles published between January 2005 and August 2025 on pediatric TBI and neurosurgical management were identified from PubMed, ScienceDirect, Google Scholar, AJOL, and Cochrane Library. Pooled proportions and 95% confidence intervals were calculated using random-effects meta-analyses, with I² statistic for heterogeneity.

Results: A total of 19 studies (n = 4452 patients) were included in this review. Road traffic accidents (RTA) were the primary aetiology (64%; 95% CI 57–70%), followed by falls (20%) and assaults (9%). Computed tomography scan was the dominant imaging

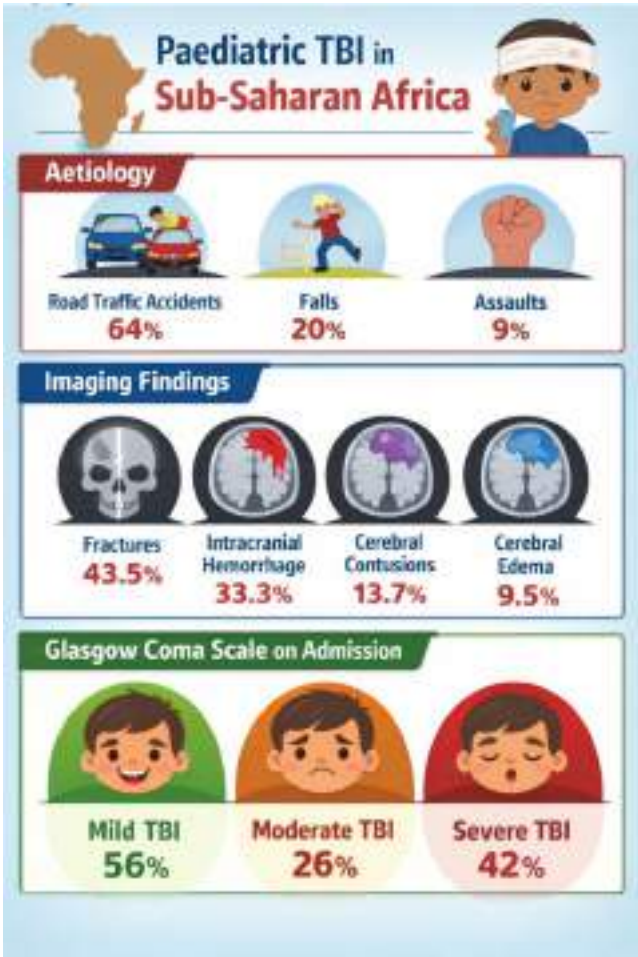


FIGURE 2 Paediatric TBI in sub-Saharan Africa.

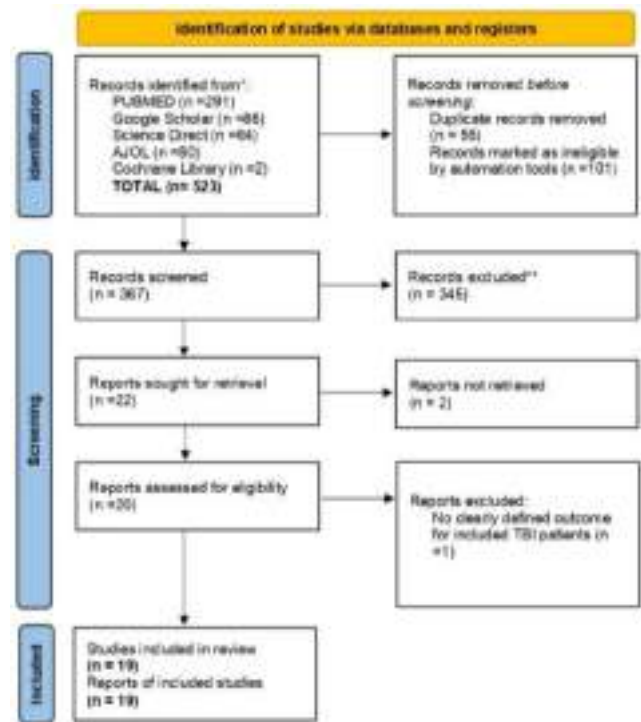


FIGURE 1 PRISMA flow diagram for included studies.

modality. Common radiological findings included fracture-related lesions (43.5%), intracranial hemorrhage (33.3%), cerebral contusions (13.70%), and cerebral edema (9.50%). On admission, the GCS (Glasgow Coma Scale) scores for mild, moderate, and severe TBI accounted for 56%, 26%, and 42% of cases, respectively. Neurosurgical intervention was reported in 15.4% of patients. The pooled favorable outcome was 81% (95% CI 67–90%), with the highest in West Africa (89%). The overall pooled mortality was 8% (95% CI 6–11%), with country-specific rates varying from 3% to 13%.

Conclusion: RTAs are the major cause of TBIs, with a significant proportion presenting with a moderate to severe GCS score at admission. Despite relatively favourable functional outcomes among survivors, mortality remains considerably high and varies across regions.

Disclosure: Nothing to disclose.

EPO-1060 | Severe chronic inflammatory demyelinating polyradiculoneuropathy relapses in the ICU: Clinical characteristics, complications and outcome

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Background and aims: Severe relapses of chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) requiring intensive care unit (ICU) admission are rare, and data on their presentation and prognosis are limited. This study aimed to describe their characteristics, management, and long-term outcomes.

Methods: We conducted a single-center retrospective study including all neurological ICU admissions for severe CIDP relapse between January 2004 and December 2024. CIDP was diagnosed according to EFNS criteria. Demographic data, indications for ICU admission, treatments, and 1-year modified Rankin Scale (mRS) scores were analyzed. Patients were stratified by the need for invasive mechanical ventilation.

Results: Fifty-two patients were included (median age 62.8 years). Main indications for ICU admission were plasma exchange for refractory disease (35%), acute respiratory failure (33%), and rapid motor worsening (33%). Twelve patients (23%) required invasive ventilation and more frequently presented with respiratory failure, bulbar dysfunction, and severe motor impairment. ICU stay was longer in ventilated patients (30 vs 7 days). Overall ICU mortality was low (3.8%). At ICU discharge,

58% of patients had significant disability (mRS 4–5). Pre-relapse mRS independently predicted 1-year functional outcome, whereas intubation did not.

Conclusion: In this long-term cohort, mortality from severe CIDP relapses was low, and invasive ventilation was not associated with worse 1-year functional outcome. Respiratory and bulbar involvement were the main predictors of intubation.

Disclosure: Nicolas Weiss declares having perceived consultant fees from Alexion and Lucane Pharma. Clemence Marois declares having perceived consultant fees from Alexion, Johnson and Jonson and UCB Timothée Lenglet declares having perceived consultant fees from LFB.

EPO-1061 | Stimulating consciousness recovery after acute brain injury with psilocybin and apomorphine

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Background and aims: What can be done to promote recovery of consciousness after brain injury in the intensive care unit (ICU)? We hypothesize that simultaneously targeting the two components of consciousness—awareness with psilocybin and arousal with apomorphine—can promote consciousness after acute brain injury. Here, we assess the safety of this combination in comatose ICU patients.

Methods: Prospective, open-label, phase 1b dose-escalation study ($n=80$). ICU coma patients receive psilocybin at doses of 1 mg (n = the first 26), 10 mg (n = next 27), or 25 mg (n = final 27), together with 2 mg apomorphine 1–2 hours after psilocybin administration (Figure 1).

Results: The first participant was recruited in February 2025, and as of January 2026, 53 (66%) participants have been enrolled, i.e., enrollment of the 1 mg and the 10 mg groups has been completed. In total, 69 serious adverse events were observed as of mid-January 2026 (Table 1). The most common events were



FIGURE 1 Distribution of the modified Rankin Scale (mRS) in the population.

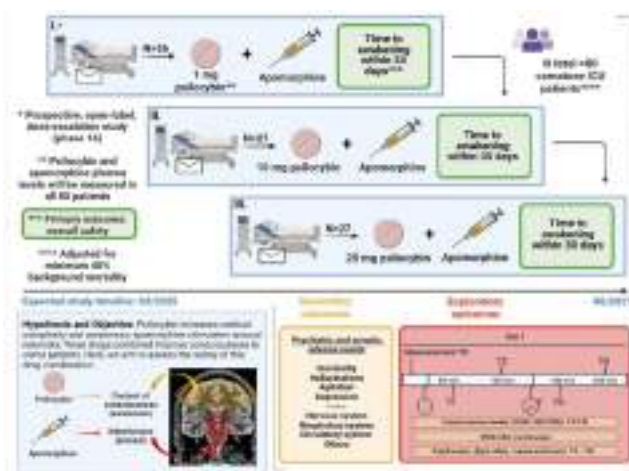


FIGURE 1 Study design.

TABLE 1 Serious adverse events.

Type of serious adverse event	N (as of 12 th of January 2026)	% of participants with event
Neurological system		
- Seizure	2	4
- Stroke	1	2
Respiratory system		
- Pneumonia	19	36
- Pneumothorax	1	2
- Pulmonary embolus	2	4
Urinary system		
- Acute kidney injury	1	2
- Urinary tract infection	13	25
Skin and soft tissue		
- Erysipelas	2	4
- Mucositis	2	4
Neuropsychiatric		
- Death Anxiety	1	2
- Delirium	4	8
Death		
- Asphyxia	1	2
- Cardiac Arrest	1	2
- Intracranial bleeding	1	2
- Withdrawal of life-sustaining therapy	13	25
Other		
- Bacteremia	1	2
- Infection of unknown origin	1	2
- Sepsis shock	1	2
Total	69	69

pneumonia ($n=19$), death after withdrawal of life-sustaining support ($n=15$), and urinary tract infection ($n=13$). We identified no serious adverse events related to the study drugs. Occasionally, apparently beneficial effects were noted in the 10 mg group.

Conclusion: No safety concerns have been identified following administration of 1 mg and 10 mg psilocybin in combination with 2 mg apomorphine so far. Pending replication of safety data in the 25 mg psilocybin group, we suggest that this combination appears safe to test in a randomized controlled trial-design to boost arousal (apomorphine) and awareness (psilocybin) in comatose ICU patients.

Disclosure: Nothing to disclose.

EPO-1062 | Evaluation of CAPE rhythm and clinical outcomes: A single-center study

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Background and aims: Cyclic Alternating Pattern of Encephalopathy (CAPE) refers to cyclical fluctuations in background electroencephalography (EEG) activity lasting 10 to 60 seconds, characterized by a spontaneous and regular alternation between two distinct patterns occurring for at least six consecutive cycles. The clinical significance of CAPE remains unclear.

In this study, we aimed to evaluate the electroencephalographic characteristics and clinical relevance of CAPE in critically ill patients.

Methods: We retrospectively reviewed EEG recordings obtained from patients hospitalized in the intensive care units of Cukurova University Hospital between 2022–2025. These patients were categorized according to age, sex, indication for intensive care admission, length of stay, clinical outcome. In addition, CAPE characteristics including the number of cycles, cycle durations, the proportions of pattern 1 and pattern 2 relative to the total CAPE duration and EEG recording time were analyzed.

Results: Among 371 patients with a total of 521 EEG recordings, CAPE rhythm was identified in 3.5% ($n=13$). The mean age was 50.8 years (range 20–79), and 9 patients (69.2%) were female. Etiology for hospitalization were CVD (23.1%), CNS infection (15.4 %), metabolic encephalopathy (23.1%), intoxication (23.1 %), intracranial malignancy and NORCE (15.4%). Mortality rate was 53.8 %. During EEG recording, 53.8% ($n=7$) of patients had a GCS score ≥ 11 . The associations between CAPE ratio, pattern 1, and pattern 2 with mortality were not statistically significant (all $p>0.05$).

Conclusion: CAPE is an increasingly recognized EEG pattern, particularly in critically ill patients. In this retrospective study, the mortality ratio was not affected according to CAPE findings.

Disclosure: Nothing to disclose.

EPO-1063 | Non-convulsive status epilepticus: Treatment adequacy matters more than treatment sequence

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Background and aims: Non-convulsive status epilepticus (NCSE) is often managed using algorithms derived from convulsive SE, despite major differences in etiological heterogeneity, diagnostic uncertainty, and patient vulnerability. Benzodiazepines (BZDs) and continuous intravenous anesthetic drugs (CIVADs) are frequently avoided due to comorbidities, respiratory risk, and benefit-risk balance. Although deviation from guideline-recommended SE treatment is associated with adverse outcomes, its clinical relevance in NCSE remains unclear.

Methods: Adult NCSE episodes were extracted from a six-year ambispective non-anoxic SE registry and classified using Salzburg criteria. Etiology was categorized according to ILAE and grouped as potentially fatal or non-potentially fatal; acute etiologies were further classified as primary or secondary CNS pathology. First-line BZD use was defined as administration before any antiseizure medication (ASM). ASMs underdosing was defined as a loading dose $<2/3$ of the recommended weight-adjusted dose. Outcomes included failure of NCSE termination, in-hospital mortality, and worsening of the modified Rankin Scale (mRS).

Results: We analyzed 189 NCSE episodes (median age 74). Failure to NCSE cessation occurred in 12.1%, in-hospital mortality in 31.7%, and mRS worsening in 63.0%. First-line BZDs were used in 50.8% of episodes and were not associated with outcomes. ASM underdosing occurred in 38.6% and was independently associated with failure to NCSE cessation (OR6.56, $p=0.002$) and

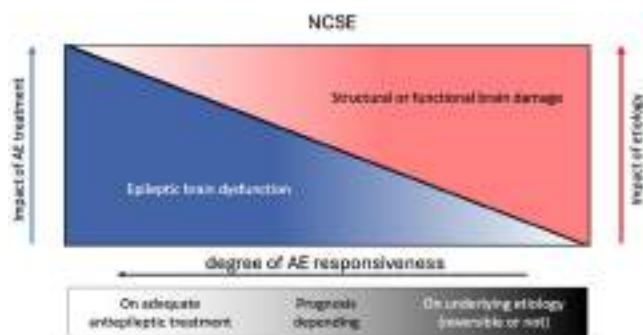


FIGURE 1 Conceptual model of NCSE showing the inverse relationship between antiepileptic treatment responsiveness and etiological impact on outcome.

in-hospital mortality (OR 4.38, $p < 0.001$). CIVADs were used in 21.7% and were not independently associated with outcomes. The negative impact of ASMs underdosing was most evident in acute primary CNS and non-potentially fatal etiologies.

Conclusion: In NCSE, adequate ASM dosing is more strongly associated with outcome than adherence to treatment sequence, supporting a personalized, etiology-driven treatment approach.

Disclosure: Nothing to disclose.

EPO-1064 | Altered level of consciousness in a tertiary emergency department: Etiologies, mortality, and predictors of outcome in a contemporary cohort

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Background and aims: Altered level of consciousness (ALC) is an emergency department (ED) presentation with diverse etiologies and substantial mortality. This study aimed to describe

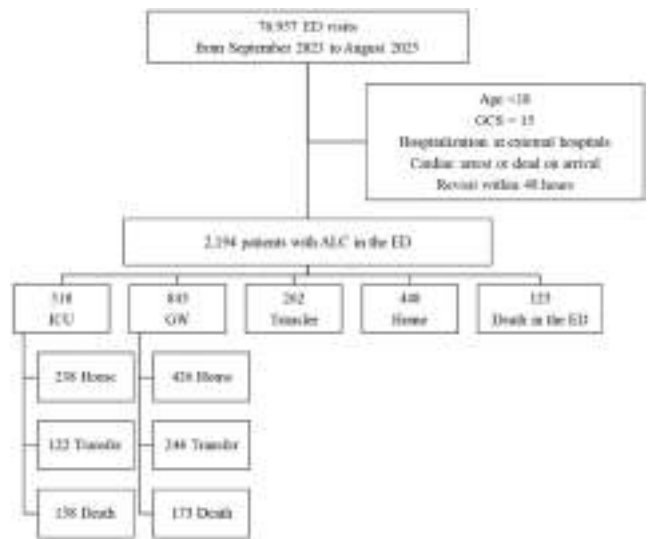


FIGURE 1 Flowchart of patients including dispositions and destinations. ALC, altered level of consciousness; ED, emergency department; GCS, Glasgow Coma Scale; GW, general ward; ICU, intensive care unit; Transfer, transferred to another hospital.

TABLE 1 Disposition and post-admission outcomes by etiology.

Etiology	N (%)	OR ± (95% CI)	Discharge from ED (%)	ICU ± (95% CI)	Discharge from ICU (%)	Transfer to (%)	Home ± (95% CI)	Death in ED ± (95% CI)
Systemic infection	545 (25.3)	1.00 ± (1.0)	Transfer 101 (18.5)	44 (8.1)	Transfer 11 (2.6)	18 (3.3)	17 (3.1)	18 (3.3)
			Stroke 101 (18.5)	44 (8.1)	Stroke 11 (2.6)	18 (3.3)	17 (3.1)	18 (3.3)
Metabolic cause	502 (22.9)	2.09 (1.18)	Transfer 101 (20.1)	10 (2.0)	Transfer 11 (2.2)	18 (3.6)	17 (3.4)	18 (3.8)
			Stroke 101 (20.1)	10 (2.0)	Stroke 11 (2.2)	18 (3.6)	17 (3.4)	18 (3.8)
Stroke	325 (14.8)	4.00 (2.04)	Transfer 101 (31.1)	10 (3.1)	Transfer 11 (3.4)	18 (5.5)	17 (5.2)	18 (5.6)
			Stroke 101 (31.1)	10 (3.1)	Stroke 11 (3.4)	18 (5.5)	17 (5.2)	18 (5.6)
Brain	250 (11.4)	1.00 ± (1.0)	Transfer 101 (40.4)	10 (4.0)	Transfer 11 (4.4)	18 (7.2)	17 (6.8)	18 (7.2)
			Stroke 101 (40.4)	10 (4.0)	Stroke 11 (4.4)	18 (7.2)	17 (6.8)	18 (7.2)
CNS	111 (5.1)	4.1 ± (2.1)	Transfer 101 (90.9)	10 (9.1)	Transfer 11 (10.0)	18 (16.2)	17 (15.3)	18 (16.2)
			Stroke 101 (90.9)	10 (9.1)	Stroke 11 (10.0)	18 (16.2)	17 (15.3)	18 (16.2)
Stroke	108 (4.9)	1.00 ± (1.0)	Transfer 101 (93.5)	10 (9.3)	Transfer 11 (10.2)	18 (16.7)	17 (15.7)	18 (16.7)
			Stroke 101 (93.5)	10 (9.3)	Stroke 11 (10.2)	18 (16.7)	17 (15.7)	18 (16.7)
Primary disorder	40 (1.8)	1.00 ± (1.0)	Transfer 101 (100.0)	10 (100.0)	Transfer 11 (100.0)	18 (100.0)	17 (100.0)	18 (100.0)
			Stroke 101 (100.0)	10 (100.0)	Stroke 11 (100.0)	18 (100.0)	17 (100.0)	18 (100.0)
CNS unknown	10 (0.5)	0.16 (0.04)	Transfer 101 (100.0)	10 (100.0)	Transfer 11 (100.0)	18 (100.0)	17 (100.0)	18 (100.0)
			Stroke 101 (100.0)	10 (100.0)	Stroke 11 (100.0)	18 (100.0)	17 (100.0)	18 (100.0)
Unintentional	111 (5.1)	1.00 ± (1.0)	Transfer 101 (90.9)	10 (9.1)	Transfer 11 (10.0)	18 (16.2)	17 (15.3)	18 (16.2)
			Stroke 101 (90.9)	10 (9.1)	Stroke 11 (10.0)	18 (16.2)	17 (15.3)	18 (16.2)

TABLE 2 Independent and severity-related factors associated with mortality.

	Adjusted OR (95% CI)	p-value
Age	1.01 (1.01–1.02)	<0.001
GCS	0.94 (0.90–0.98)	<0.001
Usage of vasopressor	19.48 (13.36–27.59)	<0.001
Stay time in ED	1.00 (1.00–1.01)	0.17
Admission to ICU	1.94 (1.52–2.49)	<0.001

the incidence, etiologic distribution, and mortality of ALC in the ED and to identify predictors of death.

Methods: This is a retrospective single-center cohort study of adults presenting with ALC to a tertiary ED between September 2023 and August 2025. ALC was defined as a Glasgow Coma Scale (GCS) score <15 or clearly abnormal orientation or behavior compared with baseline. Etiologies were categorized into ten predefined groups based on structured chart review. Logistic regression analyses were performed to identify baseline clinical predictors of overall mortality.

Results: Among 76,957 ED visits, 2,194 (2.85%) involved ALC. Systemic Infection was the most frequent etiology ($n = 516$, 23.5%), followed by Metabolic Cause ($n = 502$, 22.9%) and Stroke ($n = 325$, 14.8%). Overall mortality of ALC was 20.7%, consisting of 5.6% and 24.3% in the ED and in-hospital, respectively. The highest overall mortality occurred in Central Nervous System Infection (CNS-i) (40.9%), Stroke (31.7%), and Systemic Infection (30.6%). In the multivariable model, older age, lower initial GCS, vasopressor use, and ICU admission were independently associated with increased odds of death, whereas ED length of stay was not.

Conclusion: Mortality varied markedly across etiologic categories, with the highest risk observed for CNS-i and Stroke. These findings highlight the prognostic significance of age, initial neurologic status, and underlying etiology and may assist in risk stratification and early resource allocation.

Disclosure: Nothing to disclose.

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Background and aims: Climate change may crucially impact on various neurological diseases, which affect 40% of people globally. Socioeconomic status and green spaces could act as mitigating factors, promoting physical activity, social interaction, and psychological well-being. Objectives: (1) to evaluate the impact of extreme temperatures on emergency department visits (EDVs) among cohorts of Bologna residents with Parkinson's disease, atypical parkinsonism, epilepsy, dementia, amyotrophic lateral sclerosis, obstructive sleep apnoea, migraine, stroke; (2) to investigate the moderating role of socioeconomic status and green spaces.

Methods: Case-crossover design (Figure). Cohorts will be identified through record-linkage systems (Table 1) and compared to the adult resident population of Bologna (2015–2024). The mean daily apparent temperature represents the exposure. The primary outcome is the number of EDVs. Effect modification will be assessed for age, sex, Deprivation Index and Normalized Difference Vegetation Index.

Results: Sample sizes and data sources for the cohorts are reported in Table 1 Preliminary results on the association between

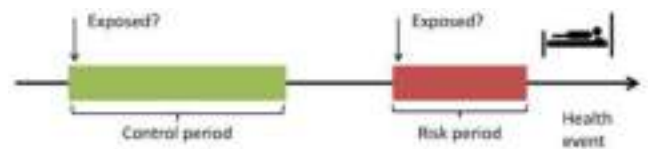


FIGURE 1 Case cross-over design: the control period is selected using a time-stratified approach, in which each case is matched to control values recorded on the same subject in different time points.

TABLE 1 Sample sizes (observed and estimated) and data sources for the cohorts of patients included in the study.

Disorder	Source	Observed/Estimated number of participants
Migraine*	CephalLink	3,200
Stroke*	Validated algorithm applied to administrative databases	9,500
Dementia*	Validated algorithm applied to administrative databases	5,000
Epilepsy**	EpiLink	400
Parkinson's disease and atypical parkinsonism**	ParkLink-Bologna	1,687 (1,311 PD and 376 APS)
Obstructive sleep apnoea syndrome**	OSASLink	380
Amyotrophic lateral sclerosis*	SEALink	80

TABLE 2 Odds ratios (OR) of emergency department visits (EDVs) per 1°C increase in mean apparent temperature, for the entire temperature range and above the 26°C threshold, with corresponding 95% confidence intervals (CI).

Univariate models	Odds ratio	95% CI	p-value
Unit increase of temperature (°C)	1.0026	(1.0015-1.0037)	<0.001
Unit increase of temperature above 26°C (°C)	1.0044	(1.0015-1.0072)	0.003

mean daily apparent temperature and EDVs in the reference population ($N=190,971$) are in Table 2 A one-unit increase in mean apparent temperature was significantly associated with increased odds of EDVs (OR 1.0026, 95%CI 1.0015–1.0037) with a stronger effect observed for each 1°C increase above the 26 °C threshold (1.0044, 1.0015–1.0072). These associations will be further compared across the specific cohorts of patients.

Conclusion: The project's findings may enhance our understanding of the role of extreme temperatures in the progression of neurological conditions and inform public health policies and preventive strategies to mitigate their impact on vulnerable populations.

Disclosure: Funded by the Fondazione del Monte di Bologna e Ravenna (grant number 2025.0163) and by IRCCS ISNB (Bando Ricerca Corrente 2025).

EPO-1066 | Endothelial activation and stress index (EASIX) as a predictor of delayed cerebral ischemia and functional outcome in subarachnoid hemorrhage

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Background and aims: Endothelial dysfunction contributes to secondary brain injury and poor outcomes following subarachnoid hemorrhage (SAH). The Endothelial Activation and Stress Index (EASIX), derived from routine laboratory parameters, has recently emerged as a marker of endothelial stress. This study aimed to evaluate the relationship between admission EASIX values and clinical outcomes in both aneurysmal and non-aneurysmal SAH.

Methods: We retrospectively analyzed 90 patients admitted to the neurointensive care unit with SAH between January 2020 and June 2025. EASIX was calculated within the first 2 h of admission as: $EASIX = (LDH \times Creatinine) / \text{Platelet count}$. Clinical variables included age, sex, aneurysm presence, Glasgow Coma Scale (GCS), Fisher grade, Hunt-Hess, and WFNS scores. Delayed cerebral ischemia (DCI) was defined according to AHA/ASA guidelines. Logistic regression was used to identify independent predictors of DCI and 3-month functional outcomes (mRS).

Results: DCI occurred in 37.7% of patients, and poor functional outcomes ($mRS \geq 3$) in 35.6%. Patients with DCI had significantly higher EASIX values (median 1.01 [IQR 0.57] vs 0.64 [IQR 0.28], $p=0.001$). Multivariable analysis identified higher EASIX (OR=26.3, $p=0.004$) and aneurysm presence (OR=7.7, $p=0.005$) as independent predictors of DCI. For functional outcome, both EASIX (OR=2.83, $p=0.039$) and age (OR=1.05, $p=0.036$) independently predicted poor prognosis.

Conclusion: EASIX independently predicts delayed cerebral ischemia and poor 3-month functional outcome in SAH, supporting its potential as a simple, readily available biomarker of endothelial stress in neurocritical care.

Disclosure: Nothing to disclose.

EPO-1067 | Precision glycemc management for diabetic neuropathy in the ICU: The HYPO-AWARE score in 96,129 admissions

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Background and aims: Diabetic neuropathy affects 50% of diabetic ICU patients, yet current glycemc protocols ignore neuropathy status despite impaired counterregulation and gastroparesis fundamentally altering glucose homeostasis. Existing hypoglycemia prediction tools achieve only modest discrimination (AUC 0.66–0.83) and exclude neuropathy-specific factors.

Methods: We analyzed 96,129 ICU admissions with 3,093,411 glucose measurements from MIMIC-IV v3.1 (2008–2022), comparing diabetic patients with neuropathy ($n=42,672$), diabetic controls ($n=37,202$), and non-diabetics ($n=16,255$). We developed the HYPO-AWARE score incorporating gastroparesis, autonomic dysfunction, hypoglycemia frequency, and glucose variability, then evaluated prediction performance using gradient boosting models with 5-fold cross-validation.

Results: Neuropathy patients exhibited paradoxically lower mean glucose (148.76 vs 156.41 mg/dL, $p<0.001$) but significantly higher coefficient of variation (26.20% vs 22.99%, $p<0.001$). Feature importance analysis revealed glucose variability dominated hypoglycemia prediction (56.6%) over mean glucose (30.9%). The HYPO-AWARE score achieved AUC 0.92 (95% CI: 0.92–0.93), substantially outperforming existing tools. Risk stratification effectively separated hypoglycemia incidence: low-risk 8.2%, moderate-risk 18.4%, high-risk 41.2% ($p<0.001$ for trend). Only 49.0% of neuropathy patients achieved adequate time-in-range ($>70\%$) compared to 81.1% of non-diabetics, representing systematic failure of universal protocols.

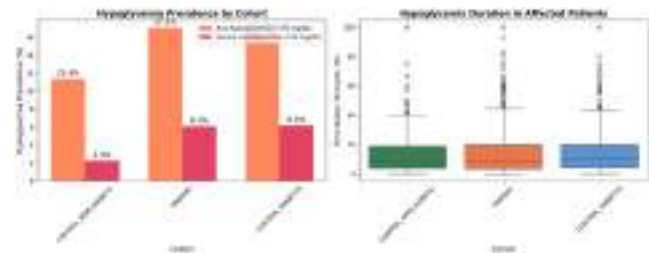


FIGURE 2

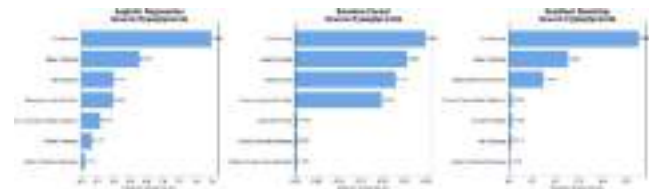


FIGURE 3

Conclusion: Diabetic neuropathy creates a distinct glycemc phenotype requiring targeted management strategies. The HYPO-AWARE score provides validated risk stratification substantially superior to existing tools. Current one-size-fits-all ICU glucose protocols fail half of neuropathy patients, demanding precision medicine approaches that prioritize variability reduction over mean glucose targets and incorporate neuropathy-specific pathophysiology.

Disclosure: Nothing to disclose.

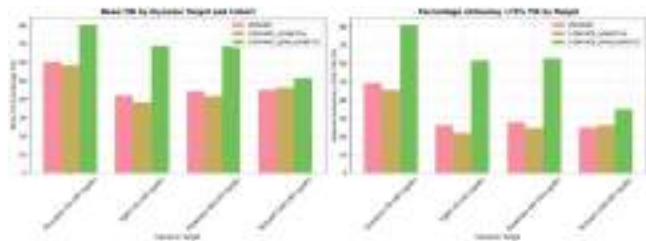


FIGURE 1

SUPPLEMENT ABSTRACTS

ePosters Virtual

Ageing and dementia

EPV-0001 | “CBI-R BADL” composite as a tool to assess functional stages in Alzheimer’s disease

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EPV-0002 | A novel NOTCH3 mutation in a patient with apathy and lacunar stroke: The importance of genotype-phenotype correlations in CADASIL

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EPV-0003 | Accelerated rise in vascular dementia being coded as a cause of death in Lithuania

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EPV-0004 | Acceptance of fixed-dose combination therapy with donepezil and memantine by patients and caregivers

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EPV-0005 | AI modelling and Alzheimer’s: How AI modelling coupled with biomarker findings improves pathophysiology discovery as well as diagnosis

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EPV-0006 | Alcohol consumption’s influence on clinical phenotype and Alzheimer’s disease biomarkers

M. Muñoz Garcia; V. Stride-Gonzalez; M. Gonzalez-Sanchez; S. Llamas-Velasco; V. Blanco-Palmero; D. Perez-Martinez; E. Morenas-Rodriguez; A. Villarejo-Galende
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EPV-0007 | Alzheimer disease co-pathology in a patient carrying pathogenic CSF1R mutation

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EPV-0008 | Analytical and clinical validation of a blood-based multiprotein biomarker panel for neurodegenerative disorders

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EPV-0009 | APOE ε4 and Alzheimer's disease: Effects on onset but not progression

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EPV-0010 | Application of the ATN framework for routine clinical diagnosis of neurodegenerative disorders in a Spanish population

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EPV-0011 | Association of microinfarcts, amyloid co-pathology, and clinical phenotype with cognitive performance in Lewy body dementia

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EPV-0012 | Attention-deficit/hyperactivity disorder as a risk factor for all-cause dementia: A systematic review and meta-analysis

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EPV-0013 | Attention-enhanced deep learning for multi-stage Alzheimer's disease detection and progression prediction: A systematic review and meta-analysis

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EPV-0014 | Attributable burden of Alzheimer's disease and other dementias due to smoking: A comparative analysis in trends across Europe (1990-2023)

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EPV-0015 | Beta-synuclein: A diagnostic and prognostic biomarker in early Alzheimer's disease

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EPV-0016 | Biomarkers of neurodegenerative diseases

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EPV-0017 | Cerebrospinal fluid LMO4 as a synaptic biomarker linked to Alzheimer's disease pathology and cognitive decline

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EPV-0018 | Challenges in assessing neuropsychiatric symptoms in dementia via telemedicine: Lessons from the “TeleCogNition” project in the Aegean

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D. Kontaxopoulou; K. Vourou; E. Smaragdaki; S. Papageorgiou
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EPV-0019 | Structural brain ageing in the Kenya

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L. Musili¹; J. Shah¹; J. Odhiambo¹; J. Miano³; S. Ponda³;
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EPV-0020 | Clinical criteria for primary age related tauopathy: Challenges in development and implementation

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EPV-0021 | Clinical, electrophysiological and neuroimaging correlates of dysphagia risk in dementia

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EPV-0022 | Cognitive reserve and confrontational naming in healthy aging

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EPV-0023 | Data-driven MRI patterns outperform volumetric measures for preclinical AD detection: Systematic review and meta-analysis

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R. Mohamed⁴; D. Atef Abouda⁵; B. Ayoub⁶; Z. Hasan⁷;
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EPV-0024 | Diagnostic performance of 3D convolutional neural networks for predicting MCI-to-AD conversion: A systematic review and meta-analysis

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EPV-0025 | Diagnostic value of CSF tTau and tTau/pTau ratio in probable Creutzfeldt–Jakob disease: A Tertiary referral center cohort study

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EPV-0026 | Early detection of dementia risk in CADASIL using integrated clinical, neuroimaging and immunogenetic markers

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EPV-0027 | Abstract withdrawn

EPV-0028 | Efficacy and Safety of phenserine and its enantiomer, buntanetap (Posiphen) in treatment of Alzheimer's disease, a systematic review and meta-analysis

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EPV-0029 | Egyptian dementia risk score: Machine learning model from EDN registry data

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EPV-0030 | Electroencephalography as a predictive marker for early diagnosis of cognitive decline

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EPV-0031 | Elevated serum sodium is linked to increased amyloid-dependent tau pathology, neurodegeneration, and cognitive impairment in Alzheimer's disease

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EPV-0032 | Emotional and cognitive engagement in Alzheimer's disease during facilitated art sessions: Observational insights from a nursing home setting

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EPV-0033 | Evaluation of prescription appropriateness in hospitalized elderly patients at a neurology institute: Cross-sectional study

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EPV-0034 | Examining time to diagnosis in a real-world memory clinic cohort

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EPV-0035 | Features of functional cognitive disorder in patients with subjective cognitive complaints and mild cognitive impairment: A prospective study

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EPV-0036 | Frontotemporal dementia associated with motor neuron disease in a progranulin mutation carrier: An exceptional phenotype beyond haploinsufficiency

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EPV-0037 | Frontotemporal dementia with C9orf72 mutation presenting with bizarre psychosis and right flail leg syndrome

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EPV-0038 | Hearing loss as a modifiable risk factor for Alzheimer's disease: Evidence from a Tunisian case-control study

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EPV-0039 | Hypersexuality and inappropriate sexual behaviors in Alzheimer's disease: A scoping review

Z. Bercini; M. Tondelli; A. Barbieri; N. Iacovino; C. Galligani; R. Maramotti; D. Ballotta; G. Vinceti; S. Cossutti; C. Carbone; A. Chiari; G. Zamboni
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EPV-0040 | Incremental value of 18F-FDG PET and other technical investigations in the diagnosis of neurodegenerative cognitive disorders

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EPV-0041 | Insulin resistance influences global cognition in patients with subjective cognitive decline and mild cognitive impairment

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EPV-0042 | IN-TeMPO: A multicenter trial of multidimensional interventions to reduce cognitive decline in older adults

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EPV-0043 | Is there a relationship between cognitive decline, surgical interventions and general anesthesia? A pilot retrospective observational study

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EPV-0044 | Long-term treatment of Alzheimer's dementia with neuronavigated mechanical Shockwaves: A new effective and safe method

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EPV-0045 | Mapping thirty years of Alzheimer's disease burden across Europe: Age-standardized disability-adjusted life year trends by region

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EPV-0046 | Neuroprotective potential of *Mansonella gagei* in a zebrafish model of Alzheimer's disease

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EPV-0047 | Neuropsychological assessment of a cohort with frontotemporal dementia

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EPV-0048 | Neuropsychological assessment of older adults: Tools and methods, with insights from the UM6P Hospitals Geriatric Center in Benguerir (Morocco)

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EPV-0049 | Newly identified mutation in exon 8 of the PSEN1 gene: Atypical presentation of Alzheimer disease in a familial early-onset dementia cluster

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EPV-0050 | NULISA synaptic biomarker signature distinguishes Alzheimer's disease from other dementias

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EPV-0051 | Oxytocin mitigates chronic sleep deprivation–Triggered apoptosis and neuroinflammation in the cerebro-cerebellar axis of aged rats

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EPV-0052 | Patterns of involvement on structural and functional neuroimaging in frontotemporal dementia

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EPV-0053 | Plasma NfL and GFAP in the preclinical stages of neurodegenerative diseases: Insights from the UK Biobank

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EPV-0054 | Prevalence study of lewy body dementia: Clinical and laboratory analysis of a cohort from Rio de Janeiro/Brazil

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EPV-0055 | Primary progressive aphasia as the initial manifestation of hereditary diffuse leukoencephalopathy with spheroids: A case report

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EPV-0056 | Quantitative relationship between Lumipulse G Measures of pTau217 in plasma and in brain

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EPV-0057 | Random forest identifies stable CSF biomarkers for AD progression: Systematic review and meta-analysis

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EPV-0058 | Real world implementation of brain health services for dementia prevention: Insights from the ICOPAD working group

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EPV-0059 | Reversible Olanzapine-induced dropped head syndrome in behavioral variant of frontotemporal dementia: A case report

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EPV-0060 | Revision of personal cases to select patients for anti-amyloid therapy

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EPV-0061 | Serum neurofilament light chain differentiates subjective cognitive decline (SCD) from non-SCD conditions in clinical practice

E. Sapiro; F. Motolese; M. Di Giesi; S. Selvaggi; G. Massa Rolandino; I. Anzuino; A. Salvati; G. Annesi; D. Norata; F. Ursini; F. Capone; V. Di Lazzaro
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EPV-0062 | Sex-specific associations between mild behavioral impairment and cognition according to APOE e4 in mild cognitive impairment

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EPV-0063 | Silent pain: Altered expression in cognitive impairment

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EPV-0064 | Six-year retrospective analysis of sporadic Creutzfeldt–Jakob disease in a tertiary academic center

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EPV-0065 | Sleep spindle density and its correlation with cognitive performance in patients with Alzheimer's disease

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EPV-0066 | Streamlining Alzheimer's disease diagnosis: Real-world validation of two-cut-off diagnostic models based on plasma p-tau/A β 42 ratios

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EPV-0067 | Survival analysis in early-onset Alzheimer's disease

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EPV-0068 | Targeting perimenopausal HPO axis dyshomeostasis: A novel strategy to mitigate women's elevated Alzheimer's disease risk

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EPV-0069 | The Protective role of cognitive reserve in the conversion from Mild cognitive impairment to Alzheimer's disease: A two-year prospective study

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EPV-0070 | Validation of plasma pTau217 versus cerebrospinal fluid biomarkers for the early diagnosis of Alzheimer's disease

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EPV-0072 | Weakly supervised CNNs overcome limited MRI annotations: Systematic review and meta-analysis

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EPV-0073 | Where do elderly patients with Alzheimer's disease die? A retrospective analysis

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EPV-0074 | YKL-40 in cerebrospinal fluid as a neuroinflammatory marker associated with Tau pathology in Alzheimer's disease

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Autonomic nervous system diseases

EPV-0075 | Adie-Holmes syndrome as the first manifestation of systemic disease: Report of two clinical cases

M. Marin; E. Campanyà; I. Saurina; C. Martinez; E. Vadillo; A. Anaya; M. Masjuan; L. Marín; D. Lopez

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EPV-0076 | AIDS-associated benign intracranial hypertension: A case report

H. Abbashar; A. Hussien; I. Hegazy; M. Alfatih; Mohamed; N. Adel; N. Abdalbagi; S. Adil
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EPV-0077 | Autonomic features, stress and cortisol response in chronic cerebral ischemia compared with healthy population

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EPV-0078 | Autonomic nervous system abnormalities in Alzheimer's disease

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EPV-0079 | Autonomic regulation via dermal vibrotactile triggering of piezoelectric channels: An emerging nonmedication approach to symptom relief

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EPV-0080 | Biological factors of perinatal anxiety

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EPV-0081 | COVID-19 aftermath: A pilot treatment protocol for autonomic dysregulation in patients with long COVID

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EPV-0082 | Evaluation of the autonomic profile in Parkinsonian syndrome

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EPV-0083 | Harlequin syndrome: Half the face, all the clues

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EPV-0084 | HPA-axis dysregulation as diagnostic marker in somatoform autonomic dysfunction

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EPV-0085 | Idiopathic Harlequin syndrome in a police force personnel: A decade of misdiagnosis

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EPV-0086 | Neuroendocrine biomarkers for differential diagnosis of somatoform autonomic dysfunction

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EPV-0087 | Prefrontal rTMS as a potential route for recovery in post-traumatic olfactory dysfunction

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EPV-0088 | Relation between body composition and functional state of autonomic nervous system

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EPV-0089 | Somatoform autonomic dysfunction in women of reproductive age with iron deficiency anemia

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EPV-0090 | Sympathetic hyperactivity in somatoform autonomic dysfunction: Salivary alpha-amylase as a diagnostic marker

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EPV-0091 | When Burnout Affects the Brain: Psychovegetative dysfunction in physicians

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Cerebrovascular diseases

EPV-0092 | A comparative study of parenchymal lesion vs. non-lesion presentations in cerebral venous thrombosis

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EPV-0093 | A new look at biomarkers of ischemic stroke

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EPV-0094 | A rare duo: Fabry disease and familial cerebral cavernomatosis

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Alto Minho, Viana do Castelo, Portugal; ³Neuroradiology
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EPV-0095 | Abdominal obesity metrics as independent predictors of stroke severity: Evidence from a population-based cohort from Bosnia and Herzegovina

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EPV-0096 | Active malignancy as an independent predictor of long-term mortality in cancer-associated stroke

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EPV-0097 | Acute ischemic cerebrovascular event following extreme breath-hold freediving

Z. Gritsopoulou; S. Stasinou; A. Tsimpiktsioglou;
M. Ioakeimidis; T. Doskas
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EPV-0098 | Acute ischemic stroke in an HIV-positive patient with syphilis: A diagnostic challenge

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EPV-0099 | Adipokines, thyroid function and low-grade inflammation in overweight and obese adults: implications for cardiometabolic and cerebrovascular risk

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EPV-0100 | Age- and sex-related differences in serum levels of vitamins B6, B9, and B12 in acute ischemic stroke

M. Asadullayev; G. Rakhimbaeva; N. Vakhabova; S. Kodirova; T. Khomidov
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EPV-0101 | Age-specific genetic phenotypes of ischemic stroke in women of Uzbek ethnicity

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EPV-0102 | Alexia without agraphia: A rare case of cortical damage without splenial involvement

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EPV-0103 | Anatomical, functional and risk factor associations of domain-specific cognitive impairment post ischemic stroke: A cross-sectional study

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EPV-0104 | Anesthetic strategy and functional outcomes after mechanical thrombectomy for acute ischemic stroke

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EPV-0105 | Anterior choroidal artery territory stroke: Insights from a short case series

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EPV-0106 | Anticoagulation in cryptogenic stroke and ESUS: A network meta-analysis

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EPV-0107 | Anticoagulation management in patients with perioperative ischemic stroke

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EPV-0108 | Antithrombotic strategies for stroke prevention in atrial fibrillation: A meta-analysis of contemporary evidence

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EPV-0109 | Artificial intelligence-based prediction of hemorrhagic stroke risk in patients with type 2 diabetes mellitus

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EPV-0110 | Assessment of health-related quality of life in patients with hemorrhagic stroke using artificial intelligence

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EPV-0111 | Association of 24-hours change of temperature and long-term disability in acute ischemic stroke

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EPV-0112 | Association of ABO blood groups with stroke etiology according to TOAST classification: A comparative study of large and small vessel atherosclerosis

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EPV-0113 | Association of admission urea to creatinine ratio to the mortality in hemorrhagic stroke: A systematic review and meta-analysis

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EPV-0114 | Association of headache with white matter lesion severity and cognitive impairment in cerebral small vessel disease

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System Diseases, I.M. Sechenov First Moscow State Medical
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EPV-0115 | Association of lipoprotein(a) with atherosclerotic burden and long-term vascular events in east Asian patients: A prospective cohort study

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EPV-0116 | Atraumatic acute subdural hematoma in moyamoya disease

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EPV-0117 | Atrial fibrillation diagnosed after ischemic stroke: Risk factors, cardiac abnormalities, and the importance of screening

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EPV-0118 | Baseline anaemia, haemoglobin levels, and functional outcomes after mechanical thrombectomy: A systematic review and meta-analysis

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EPV-0119 | Bevacizumab associated PRES

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EPV-0120 | Beyond admission scores: perioperative dynamics of mWFNS grade and outcomes in aneurysmal subarachnoid hemorrhage

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EPV-0121 | Beyond contraception: Stroke as a complication of hormonal overdose

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EPV-0122 | Beyond parenchymal hematoma: Prognostic impact of the modified graeb score in acute ICH

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EPV-0123 | Beyond reperfusion: Pre-stroke brain vulnerability as the main determinant of long-term cognitive outcome after ischemic stroke

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EPV-0124 | Beyond speed: Quality-adjusted door-to-needle time and functional outcome in acute ischemic stroke: A retrospective institutional cohort study

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EPV-0125 | Beyond the posterior pattern: A case of atypical posterior reversible encephalopathy syndrome during hemodialysis

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EPV-0126 | Beyond the third cranial nerve: Bilateral ptosis due to central caudal nucleus involvement in midbrain stroke

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EPV-0127 | Biomarkers and recurrence risk in symptomatic intracranial atherosclerotic stenosis: A single-centre retrospective study

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EPV-0128 | Brazilian adaptation of the “iHELP stroke: Improving health and lifestyle programme” and feasibility study

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EPV-0129 | Bridging therapy versus endovascular thrombectomy in acute ischemic stroke: A meta-analysis of randomized controlled trial

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EPV-0130 | Burden of undiagnosed hypertension among stroke patients in Rwanda: A five-year retrospective study

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EPV-0131 | Cancer and ischaemic stroke in a comprehensive stroke centre: A retrospective review

L. Gil Martinez; A. Sanchez Asensio; J. Cortina Garcia; J. Romero Ferro; N. Mena Garcia; M. Campos Jimenez; G. Cabañas Engeneios; R. Pastor Gonzalez; C. Matute Lozano; S. Garcia Madrona; A. de Felipe Mimbrena; A. Cruz Culebras; J. Masjuan Vallejo; R. Vera Lechuga
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EPV-0132 | Cardiac abnormalities in the early ICU course after anterior communicating artery aneurysm rupture

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EPV-0133 | Cardioembolic stroke in the era of anticoagulation therapy (AT): A two-year retrospective cohort study

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EPV-0134 | Carotid-cavernous fistula in Albania: A prospective case series on treatment modality and regression of ocular symptoms

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EPV-0135 | Case report of brainstem variant of posterior reversible encephalopathy mimicking brainstem glioma

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EPV-0136 | Cerebellar syndrome secondary to a Guillain-Mollaret triangle cavernous malformation mimicking a late-onset cerebellar ataxia

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EPV-0137 | Cerebral amyloid angiopathy related inflammation: A case report and a systematic review of the literature

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EPV-0138 | Characteristics of VA-PICA junction aneurysm rupture in polycystic kidney disease

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EPV-0139 | Clinical and cognitive outcomes after bi-thalamic stroke: Multicenter artery of percheron stroke registry

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EPV-0140 | Clinical and laboratory predictors of early hematoma expansion in spontaneous intracerebral hemorrhage: A systematic review and meta-analysis

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EPV-0141 | Clinical and prognostic relevance of lipoproteins and their receptors in acute cerebrovascular disorders

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N. Vakhabova; S. Kodirova
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EPV-0142 | Clinical and radiological profile of early and late seizures after ischemic stroke

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EPV-0143 | Clinical and safety outcomes of dexmedetomidine use during mechanical thrombectomy: A meta-analysis

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EPV-0144 | Clinical characteristics and lesion localization of organic emotional lability after ischemic stroke

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EPV-0145 | Clinical characteristics and prevalence of post-stroke emotionalism: Findings from a prospective observational study

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EPV-0146 | Clinical features of cerebrovascular pathology and cerebral hemodynamics in patients with rheumatic disease

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EPV-0147 | Clinical outcomes of mothership and drip-and-ship pathways in acute ischemic stroke: First-year experience of a comprehensive stroke center

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EPV-0148 | Clinical, imaging, and outcome spectrum of intracranial atherosclerotic disease in first-ever ischemic stroke

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EPV-0149 | Collateral perfusion delay predicts infarct growth and futile reperfusion after thrombectomy: A retrospective CT perfusion study

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EPV-0150 | Comparative effectiveness of different treatment approaches for acute ischemic stroke in patients aged 80 years and older

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EPV-0151 | Comparative efficacy and safety of DOACs versus aspirin in embolic stroke of undetermined source: A network meta-analysis

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EPV-0152 | COVID-19 vaccination and the risk of stroke and thrombotic events: An umbrella review of 35 studies

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EPV-0153 | Decompressive craniectomy after intravenous thrombolysis for malignant MCA infarction: Feasibility and outcomes in a resource-limited stroke center

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EPV-0154 | Deep medullary veins in cerebral amyloid angiopathy and hypertensive arteriopathy

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EPV-0155 | Determinants of mortality and functional outcome in spontaneous intracerebral hemorrhage: Insights from a tertiary center cohort

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EPV-0156 | Diagnostic and prognostic significance of MMP-9 levels in the hyperacute phase of hemorrhagic stroke

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EPV-0157 | Diagnostic and prognostic value of cystatin-c levels in the hyperacute phase of hemorrhagic stroke

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EPV-0158 | Diet-related stroke burden in Europe: Persistent inequities and diverging trends across subregions

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EPV-0159 | Does patient's sex influence acute ischemic stroke clinical profile and early outcome? A single-center RES-Q registry analysis

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EPV-0160 | Drug-coated balloon versus stent angioplasty in patients with intracranial atherosclerotic disease: A systematic review and meta-analysis

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EPV-0161 | Dual antiplatelet therapy after peripheral revascularisation: Implications for stroke and neurovascular risk—A systematic review & meta-analysis

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EPV-0162 | Early LADDER strategy (low-dose anticoagulation and directed dose escalation with reassessment) in patients with intra-parenchymal haemorrhage and AF

R. Ramesh; Y. Kanagavel; L. Narasimhan R; S. Shanmugam;
P. Hazeena; A. Mekala

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EPV-0163 | Effectiveness of stabilometric platform training in balance recovery in patients with vertebrobasilar insufficiency

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EPV-0164 | Efficacy and safety of PCSK9 inhibitors in acute ischemic stroke: A systematic review and meta-analysis

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EPV-0165 | Endothelial and metabolic markers across ischaemic stroke subtypes and functional outcome

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EPV-0166 | Endovascular treatment for severe symptomatic stenosis of the petrocavernous segment of the internal carotid artery

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EPV-0167 | Etiologic patterns and prognostic determinants of ischemic stroke in young adults at a secondary care hospital

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EPV-0168 | Evaluation of the Effectiveness of Safe Nutrition Program Applied to Acute Stroke Patients with Dysphagia

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EPV-0169 | Factors influencing the occurrence and development of cerebrospinal fluid-vascular distension in children

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EPV-0170 | From diplopia to recurrent stroke: A diagnostic journey towards primary CNS vasculitis

J. Aguilera Aguilera; B. Rodriguez Garcia; M. Ravelo León;

J. Rodriguez Carrillo; M. Luz Esteve; Á. Criado Antón;

L. Redondo Robles; J. Vizcaya Gaona

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EPV-0171 | From guidelines to practice: Acute care bundles in spontaneous intracerebral hemorrhage

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EPV-0172 | From hyperthyroidism to transient ischemic attack

J. Vrbica; A. Bajunović; T. Rabi Žikić; S. Simić; Ž. Živanović

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EPV-0173 | Functional outcome in intracerebral hemorrhage: Predictive factors

K. Yessine; N. Salma; J. Emna; S. Houssem; H. Anis; B. Sana

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EPV-0174 | Greek word intelligibility test for neurologically impaired dysarthric patients

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EPV-0175 | Guideline recommendations vs real-world practice: Oral anticoagulation at discharge after ischemic stroke in Armenia

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N. Nazinyan¹; M. Balasanyan¹; M. Ghambaryan¹;

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EPV-0176 | Headache with multiple cranial nerve palsies: Proximal carotid dissection or transient perivascular inflammation of the carotid syndrome?

D. Rivero-Rodríguez; A. Diezma Maritin; M. Martin martin;

R. Rodriguez Garcia; N. Muñoz Pinto; F. Marquez Peñuela;

D. Maroto Navas; J. Piernagorda Copado; C. Marsal Alonso;

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EPV-0177 | Hematological inflammatory indices in the early phase of acute ischemic stroke

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EPV-0178 | High-dose sildenafil use as a potential trigger for cerebral venous sinus thrombosis: A case report

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EPV-0179 | Higher frequency of cortical involvement in diffusion-weighted imaging-positive TIA patients

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EPV-0180 | Hormonal determinants and female sex-specific risks in cerebral venous thrombosis

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EPV-0181 | Idarucizumab for dabigatran reversal: A 10-year single-centre experience

T. Nožić Škerjanec; U. Čepin; M. Simerl Jožef; A. Trebše;

M. Zupan; S. Frol

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EPV-0182 | Identification of ITGAM and TNFSF13B as core panoptosis genes in ischemic stroke and prediction of potential therapeutic drugs

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EPV-0183 | Identifying diagnostic and treatment challenges in cerebral amyloid angiopathy: Results from a multicenter Bulgarian survey

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EPV-0184 | Imaging the skull-brain immune axis: Calvarial marrow asymmetry on MRI After thrombectomy and clinical outcomes

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EPV-0185 | Immune TTP presenting with acute dysphasia and hemiparesis: A case report

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EPV-0186 | Impact of hematoma volume and localization on clinical prognosis in intracerebral hemorrhage

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EPV-0187 | Impact of intravesical gentamicin irrigation on catheter-associated urinary tract infections in a comprehensive stroke unit

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EPV-0188 | Impact of reperfusion timing on interictal epileptiform discharges after ischemic stroke

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EPV-0189 | Impact of the socioeconomic crisis on acute ischemic stroke management and outcomes in a university hospital in Lebanon

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EPV-0190 | In lacunar stroke patients, high levels of HbA1c were associated with high burden of cerebral microbleeds and white matter hyperintensities

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EPV-0191 | In stroke patients, women are associates with more worsening of aortic atheroma compared with men

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EPV-0192 | Inflammation and immune mechanisms in Moyamoya disease: A literature review

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EPV-0193 | Influence of admission urea to creatinine ratio in ischemic stroke patients' neurological outcomes: A systematic review and meta-analysis

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EPV-0194 | Integrated lipid, cardiac, and hematologic biomarker ratios and their association with incident stroke in a high cardiovascular-risk cohort

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EPV-0195 | Integrating artificial intelligence with clinical and neuroimaging data for lacunar ischemic stroke diagnosis

M. Mirjalolkhonov; N. Vakhobova; G. Rakhimbaeva;

Y. Musaeva

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EPV-0196 | Interleukin-6 (IL-6) signalling pathway modulation in the acute and sub-acute phases of ischaemic stroke for maximising therapeutic benefits

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EPV-0197 | Interplay between arterial collateral circulation and venous hemodynamic patterns in atherosclerotic ischemic stroke

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EPV-0198 | Intracerebral haemorrhage in Malta: Epidemiology, risk factors and current management practices since March 2024

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EPV-0199 | Intracerebral haemorrhage location and intensive care unit mortality: A MIMIC-IV Cohort study

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EPV-0200 | Intravenous thrombolysis with rt-PA in patients with acute ischemic stroke: Clinical experience from two Egyptian centers

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EPV-0201 | Intravenous usage of Tenecteplase (TNK) versus Alteplase (rt-PA) before mechanical thrombectomy for Acute Ischemic Stroke: A systematic review

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EPV-0202 | Ischemic stroke due to atrial fibrillation and oral anticoagulation: A two-decade longitudinal study

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EPV-0203 | Ischemic stroke in hypereosinophilic syndrome: A case of cryptogenic stroke and multidisciplinary management

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EPV-0204 | Ischemic stroke of the left hemisphere caused by tandem occlusion treated with mechanical thrombectomy and tandem stenting

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EPV-0205 | Knowledge of cerebrovascular disease among patients aged 40–65 years in Eastern Poland

D. Jakubowicz-Lachowska; M. Bargielski; M. Bazylewicz; M. Wierciński; E. Szyłak; M. Chorazy; A. Kułakowska
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EPV-0206 | Laboratory markers of plasma coagulation, platelet function and endothelial dysfunction in patients with cerebral small vessel disease

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EPV-0207 | Late carotid in-stent thrombosis causing ischemic stroke treated with intravenous thrombolysis

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EPV-0208 | Limb-shaking transient ischemic attacks due to severe middle cerebral artery stenosis: A case report

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EPV-0209 | Linguistic disparities in stroke care and recovery among long-term care residents – A population-based cohort study

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EPV-0210 | Machine learning in stroke triage and workflow optimization for endovascular thrombectomy: A systematic review

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EPV-0211 | Management and outcomes of ischemic stroke or TIA in young patients with patent foramen ovale: A retrospective cohort study

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EPV-0212 | Mechanical thrombectomy beyond 6 hours: Outcomes in a real-world cohort

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EPV-0213 | Mechanical thrombectomy in minor ischemic stroke (NIHSS ≤5) with large- or medium-vessel occlusion: A retrospective cohort study

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EPV-0214 | Multisegmental vascular disease following fluoroquinolone use: A case of simultaneous carotid and vertebral artery dissection

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EPV-0215 | Neurological complications in children with sickle cell disease in Senegal: A retrospective study of 57 cases from January 2020 to October 2024

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EPV-0216 | Neuromodulation-Driven Neuroplasticity for Motor Recovery After Stroke: A Systematic Review of Adjunctive Rehabilitation Strategies

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EPV-0217 | Neuropsychological anxiety and opioid activity in chronic cerebral ischemia without somatovegetative syndrome

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EPV-0218 | Neurorecovery in the digital era: Smartwatches and smartphones in post-stroke care – A scoping review of validation studies and clinical trials

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EPV-0219 | On the definition and typology of cognitive deficits in stroke

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EPV-0220 | Oral anticoagulation in cerebral venous thrombosis: strategies and clinical outcomes after discharge

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EPV-0221 | Persistent gaps in the management of neurovascular diseases in Haiti: Challenges, consequences, and urgent perspectives

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EPV-0222 | Post-stroke anxiety disorders and their psychocorrection

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EPV-0223 | Potential prognostic value of blood biomarkers in clarifying the diagnosis of ischemic stroke

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EPV-0224 | Predictors of early neurological deterioration in acute minor ischemic stroke: A systematic review and meta-analysis

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EPV-0225 | Predictors of malignant brain edema after mechanical thrombectomy for ischemic stroke patients: A systematic review and meta-analysis

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EPV-0226 | Prescription of dual antiplatelet therapy beyond guidelines in acute ischemic stroke: Real-world evidence

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EPV-0227 | Primary vs rescue thrombectomy in the therapeutic management of acute ischemic stroke: A three-year comparative study

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EPV-0228 | Prioritizing components for a healthy lifestyle intervention post-stroke: A cross-country Patient and Public Involvement-based descriptive analysis

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EPV-0229 | Prognostic factors in patients with stroke undergoing endovascular treatment

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EPV-0231 | Proposed clinical practice protocol for the treatment of stroke at the hospital level

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EPV-0232 | Prourokinase in acute ischemic stroke: A review of current evidence

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EPV-0233 | Recanalization therapy of acute stroke in elderly: Two-year real-life prospective data from Armenia

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EPV-0234 | Recurrent cerebral hemorrhage as a late manifestation of 22q11.2 deletion syndrome: A case report and literature review

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EPV-0235 | Recurrent intracranial hemorrhage in a young adult revealing iatrogenic cerebral amyloid angiopathy

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EPV-0236 | Recurrent transient brachial paresis: When vascular etiology does not seem to explain everything

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EPV-0237 | Recurrent transient ischemic attacks associated with long-term Nilotinib: A case of accelerated Atherosclerosis

L. Caballero Sánchez; M. Capra; C. Gómez López de San Román; I. Bermejo Casado; A. De Luca; M. Vargas Cobos; R. Alfonso González; D. Cerdán Santacruz; A. Castrillo Sanz; A. Mendoza Rodríguez
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EPV-0238 | Repeated intravenous thrombolysis within one day for recurrent ischemic stroke

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EPV-0239 | Reperfusion therapy in stroke patients over 80 years: Risks and outcomes from a Stroke Center in the Republic of Moldova

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EPV-0240 | Results of indication for primary air transport of patients with suspected stroke based on telephone verification of FAST+ positivity

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EPV-0241 | Reversible cerebral vasoconstriction syndrome and non-aneurysmal intracerebral haemorrhage: Diagnostic role of delayed digital subtraction angiography

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EPV-0242 | Reversible cerebral vasoconstriction syndrome associated with hydrogen sulphide exposure: A rare environmental trigger

J. Aguilera Aguilera; B. Rodriguez Garcia; D. Gómez de la Torre Morales; J. Rodríguez Carrillo; M. Luz Esteve; I. Díaz Díaz; J. Vizcaya Gaona; Í. Esain González; G. Carvalho Monteiro
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EPV-0243 | Robotic assisted stroke rehabilitation: Does it improve functional recovery and patient outcomes?

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EPV-0244 | Routine coagulation parameters and severity of neurological deficit in women with ischemic stroke

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EPV-0245 | Screening for poststroke Screening for poststroke cognitive impairment (PSCI) in real practice

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EPV-0246 | Seeds from another source: Iatrogenic cerebral amyloid angiopathy

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EPV-0247 | Sex differences in acute ischaemic stroke treated with reperfusion therapy: A retrospective registry analysis

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EPV-0248 | Sex differences in acute ischemic stroke thrombolysis: Insights from a real-world registry

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EPV-0249 | Sex differences in mechanical thrombectomy timing and early outcomes in acute ischemic stroke: A single-center retrospective cohort study

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EPV-0250 | Sneezing-induced sudden headache as an initial manifestation of convexity subarachnoid hemorrhage: A case report

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EPV-0251 | Splenial ischemic stroke in a young woman with systemic inflammation and probable serotonergic toxicity

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EPV-0252 | Spontaneous triple flexion after thalamo-capsular haemorrhagic stroke

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EPV-0253 | Stress, autonomic dysfunction, and cortisol profile in chronic cerebral ischemia with and without somatovegetative syndrome

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EPV-0254 | Stroke burden and risk factors in India, 1990–2023: Age–sex stratified GBD analysis

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EPV-0255 | Stroke characteristics and outcomes in two neurology units in a Sub-Saharan African country

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EPV-0256 | Temporal trends in particulate matter-attributable stroke across Europe. Insights from the global burden of disease 2023 study (1990–2023)

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EPV-0257 | Tenecteplase versus alteplase before mechanical thrombectomy in acute ischaemic stroke: A retrospective comparative study

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EPV-0258 | Tenecteplase vs alteplase for large vessel occlusion stroke: An updated systematic review and meta-analysis

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EPV-0259 | The beta-blocker nebivolol therapy in acute ischemic stroke: Effects on nitric oxide metabolism

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EPV-0260 | The evolving epidemiology of ischemic heart disease and ischemic stroke in Europe: Observational and genetic evidence from GBD 2023

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EPV-0261 | The predictive value of the stroke oxidative-inflammatory index in hemorrhagic stroke: A retrospective cohort study

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EPV-0262 | The prevalence of cerebral amyloid angiopathy among the causes of hemorrhagic stroke

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EPV-0263 | The relationship between dehydration levels on admission, blood viscosity, and early neurological deterioration in patients with lacunar strokes

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EPV-0264 | The role of inflammatory cytokine IL-6 in cognitive impairment after acute ischemic stroke: A preliminary study

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EPV-0265 | The Role of undiagnosed obstructive sleep apnea in recurrence of transient ischemic attacks and progression of Leukoaraiosis: A pilot cohort study

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EPV-0266 | Tocilizumab for the treatment of cerebral amyloid angiopathy-related inflammation with elevated IL-6: A case report

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EPV-0267 | Trends and disparities in colon cancer and stroke-related mortality in the United States: A retrospective study

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EPV-0268 | Trends and disparities in stroke-related mortality in inflammatory bowel disease patients

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EPV-0269 | Umbrella review of endovascular thrombectomy time windows and outcomes in acute ischemic stroke

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⁶University of Buea, Cameroon; ⁷Rostov State Medical University, Rostov-on-Don, Russian Federation; ⁸AMA School of Medicine, Philippines

EPV-0270 | Using an artificial intelligence-based machine learning approach to understand the future of declining burden of stroke in the United Kingdom

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EPV-0271 | Vascular risk burden as a predictor of clinical outcomes after thrombolysis in acute ischemic stroke: Single-centre study

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EPV-0272 | Vasospasm after aneurysmal subarachnoid hemorrhage: Triggering factors and clinical outcomes

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EPV-0273 | Venous sinus stenosis in idiopathic intracranial hypertension: Intracranial compliance after best medical therapy versus venous sinus stenting

L. Souza Viana; F. Moulin de Moraes; S. Luiz De Andrade Matas; G. Sampaio Silva

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EPV-0274 | Very late-onset HTRA1-related autosomal dominant cerebral small vessel disease misdiagnosed as multiple sclerosis

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EPV-0275 | Werneck's syndrome: A case report

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EPV-0276 | White blood cells to HDL ratio as a superior predictor of ischemic stroke clinical severity

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N. Charfi; E. Smaoui; M. Damak

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Child neurology/developmental neurology

EPV-0277 | A Network meta-analysis of stem cell for cerebral Palsy: Does route of administration and stem cell type matters?

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EPV-0278 | A study of the clinical characteristics, etiology and predictors of severity of global developmental delay in children

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EPV-0279 | Acute peripheral facial palsy of neurological origin in the pediatric population

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EPV-0280 | Age and diagnosis determine TMS Efficacy in preschoolers with developmental disorders

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EPV-0281 | An investigation of stuttering prevalence in children in Tashkent, Uzbekistan

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EPV-0282 | Clinical and functional manifestations of diabetic cardiovascular autonomic neuropathy in children with type 1 diabetes

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EPV-0283 | Clinical and instrumental phenotyping of gait in subjects with Cri du Chat syndrome

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EPV-0284 | Clinical predictors of global developmental delay in Azerbaijani Patients with infantile epileptic spasms syndrome

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EPV-0285 | Cognitive status and non-specific school in children with attention-deficit/hyperactivity disorder

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EPV-0286 | Daily posture and movement profiles in Saudi children with cerebral palsy: An ActivPAL-based comparative study

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EPV-0287 | Development of a smartphone-based pose estimation framework for automated pediatric motor assessment

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EPV-0288 | Diagnostic accuracy of machine learning for schizophrenia spectrum disorders in pediatrics: A systematic review and meta-analysis

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EPV-0289 | Diagnostic pathway and neuro-investigations in pediatric sydenham chorea: A Tunisian cohort study

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M. Jamoussi; T. Ben Younes; I. Kraoua; H. Benrhouma
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EPV-0290 | Diagnostic strategy and genetic landscape of syndromic microcephalies

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France; ⁴Department Of Genetics, Mongi Slim Hospital, La Marsa, Tunisia

EPV-0291 | Early detection of autism spectrum disorder in children with cerebral Palsy using motor-adapted diagnostic tools

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EPV-0292 | Effectiveness of interventions for cognitive enhancement in down syndrome: A network meta-analysis

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EPV-0293 | Effectiveness of microcurrent therapy in the multidisciplinary rehabilitation of children with microcephaly

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EPV-0294 | Endovascular thrombectomy for paediatric large vessel occlusion stroke: A rapid evidence synthesis

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EPV-0295 | Experience using cenobamate in refractory epilepsy in children

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EPV-0296 | Gene-environment interactions shaping pediatric neurological phenotypes

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EPV-0297 | Health education strategies for early autism recognition in childhood: Implications for child neurology in middle-income settings

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EPV-0298 | Innovative method for the rehabilitation of motor disorders in children with cerebral Palsy

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EPV-0299 | Leigh syndrome: Consider in cases of late neurodevelopmental regression

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EPV-0300 | Lipid metabolic disorders in neonatal bilirubin encephalopathy: Novel findings from proteomic analysis in a rat model

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EPV-0301 | Metabolic changes in the brain of children and adolescents with diabetes mellitus, taking into account the presence or absence of cognitive deficit

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EPV-0302 | Neuroimaging methods in the early diagnosis of cerebral impairment in children and adolescents with type 1 diabetes mellitus

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EPV-0303 | Neurological and speech manifestations of developmental speech delay in children: Impact of residual brain damage

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EPV-0304 | Neurological and speech phenotypes of developmental speech delay in children: The role of residual brain damage

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EPV-0305 | Neurological burden in neurocutaneous syndromes: Epilepsy, cognitive impairment, and imaging patterns in children

E. Cherif; Z. Miladi; H. Klaa; A. Zioudi; M. Jamoussi; T. Ben Younes; M. Ben Hafasa; H. Benrhouma; I. Kraoua
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EPV-0306 | Neurological changes in children with congenital dysplasia of the lower limb joints

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EPV-0307 | Neurometabolic phenotype of torch-associated autism in children

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EPV-0308 | Neurophysiological and cognitive correlates of developmental language disorder in preschool children

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EPV-0309 | NK cell mediated immune alteration at the maternal fetal interface regulates the fetal neural development after influenza infection

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EPV-0310 | Non-invasive neuromodulation with repetitive transcranial magnetic stimulation for children with Tourette syndrome and chronic tic disorders

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EPV-0311 | Preliminary insights into neuroinflammatory mediators in children with autism spectrum disorder

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EPV-0312 | Prevention of static contractures in cerebral Palsy: Long-term prognostic effects of early botulinum toxin therapy

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EPV-0313 | Self-limiting epilepsies of childhood: Experience of the single referral centre in Serbia

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EPV-0314 | Trends in pediatric intracranial hemorrhage-related mortality in the United States: A retrospective analysis

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EPV-0315 | BEAM™ EEG biomarkers as early indicators of cognitive decline: Insights from a retrospective cohort with mild cognitive impairment

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EPV-0316 | Cervical dystonia: The objective evaluation of muscle tone

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EPV-0317 | Clinical and electrophysiological spectrum of monoclonal gammopathies in neuromuscular practice

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EPV-0318 | Clinical value of somatosensory and motor evoked potentials in nitrous oxide-induced myelopathy

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EPV-0319 | Cognitive disorders in patients with carbon monoxide poisoning: Risk factors and predictors for delayed neuropsychological sequelae

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EPV-0320 | Evaluation of the effectiveness of botulinum toxin type A for hemifacial spasm using the Jankovic rating scale in Sri Lanka

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EPV-0321 | Feasibility of a portable, low-cost multichannel EEG system using commercially available components

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EPV-0322 | From focal targets to network phenotypes in deep brain stimulation and the circuit reframing of OCD

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EPV-0323 | Neuroinflammatory effects of repetitive transcranial magnetic stimulation and their clinical relevance in depressive disorders

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EPV-0324 | Progressive ataxia at the metabolic-neurodegenerative crossroads: Central diabetes insipidus as a clue to juvenile metachromatic leukodystrophy

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EPV-0325 | Referral pattern, waiting time and outcome distribution in EMG: A retrospective descriptive study

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EPV-0326 | Self-resolving, isolated Anti-GD3-positive atypical Guillain-Barré syndrome: Case report and literature review

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EPV-0327 | Spindle coma associated with acute carbamazepine intoxication in a paediatric patient

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I. Lopera Rodríguez¹; J. Ciriero Macías¹; L. Garcia Granados¹;

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EPV-0328 | The electroencephalography response to the olfactory stimulus: Comparing automated and individualized approaches

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EPV-0329 | Unveiling neuroradiological and neurophysiological features in a novel proteolipid protein 1 (PLP1) related disorder

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M. Pengo¹; V. Yahya¹; A. De Grado⁴; E. Vola¹; M. Gastaldi³;

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IRCCS Istituto Neurologico “Carlo Besta”, Milan, Italy

EPV-0330 | “Neuroendocrine cognitive dysregulation syndrome” developmental structure in thyroid disorders

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EPV-0331 | Apraxia severity independently predicts functional decline across neurodegenerative movement and cognitive disorders

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EPV-0332 | Are there cognitive dysfunction in myasthenia gravis? An Italian cohort study

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EPV-0333 | Associations between sleep neurophysiology and cognitive performance in rem sleep behavior disorder: Evidence from a wearable PSG device

F. Bolengo¹; L. Dell'Acqua¹; E. Cini¹; A. Della Valle¹; F. Casoni¹; P. Proserpio¹; D. Levendowski²; A. Galbiati¹; L. Ferini-Strambi¹

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EPV-0334 | Central gain, peripheral restraint: A cholinergic strategy for Parkinson disease dementia

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EPV-0335 | Chronic anti-NMDAR encephalitis: An unusual course

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EPV-0336 | Cognitive function in middle-aged adults and its differential associations with sedentary behaviour type: Findings from the 1970 British birth cohort

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EPV-0337 | Comorbid psychoemotional disturbances with apathy in Parkinson's disease

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EPV-0338 | Corneal transplantation-associated probable iatrogenic Creutzfeldt–Jakob disease with cerebellar presentation and myopathic EMG findings

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EPV-0339 | Creutzfeldt–Jakob disease in north Africa: Insights from a Tunisian case series

L. Bouafia; R. Zouari; D. Ben Mohamed; Z. Said; A. Rachdi; F. Nabli; S. Ben Sassi

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EPV-0340 | Diagnostic accuracy and clinical utility of imaging and CSF biomarkers in sporadic CJD: A systematic review and meta-analysis

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EPV-0341 | Diagnostic Performance of the Number Connection Test for Minimal Hepatic Encephalopathy

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EPV-0342 | Early cognitive impairment in CADASIL: The value of SDMT, MMSE and MRI findings

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EPV-0343 | Efficacy and safety of perioperative intranasal insulin in preventing postoperative delirium: A systematic review and meta-analysis of RCTs

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Medicine, Irbid, Jordan; ⁶Kasr-alainy Faculty of Medicine, Cairo University, Cairo, Egypt

EPV-0344 | Emotional disorders and quality of life in chronic lymphocytic leukemia

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EPV-0345 | From in- to outpatient: day-hospital lumbar puncture for shunt prediction in NPH

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EPV-0346 | Hidden neuropsychological profiles in amyotrophic lateral sclerosis beyond standard cognitive screening

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EPV-0347 | HIV-associated neurocognitive disorder. A case report

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EPV-0348 | Immediate effects of motor imagery on muscle strength in healthy young adults: A within-subject study

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EPV-0349 | Insulin resistance, subclinical brachiocephalic atherosclerosis, and cognitive performance in young adults

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EPV-0350 | Ketogenic dietary intervention in early cognitive decline: A pilot study in mild cognitive impairment and subjective cognitive decline

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EPV-0351 | Mindfulness-based intervention in idiopathic generalised epilepsy: results of a randomised controlled trial

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EPV-0352 | Neurobehavioral trajectory of eating patterns and body image after bariatric surgery: A 9-month prospective study

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EPV-0353 | Neurocognitive fatigue and burnout phenotypes among physicians in Uzbekistan: Cross-sectional MBI and MFI-20 analysis

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EPV-0354 | Night-shift load and circadian disruption as drivers of neurocognitive fatigue and burnout in physicians: Cross-sectional data from Uzbekistan

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EPV-0355 | Obstructive sleep apnea in adults with down syndrome: prevalence and screening limitations

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EPV-0356 | Preserved cerebral hemodynamics across gender during an activation task

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EPV-0357 | Quality of life in patients with severe acquired brain injury in long-term care facilities: a multi-centre cross-sectional study

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EPV-0358 | Recurrent traumatic direct carotid-cavernous fistula: Case report and literature review

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EPV-0359 | Specificity of cognitive disorder structure in patients with advanced post-traumatic epilepsy

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EPV-0360 | The cerebellar stroke, beyond motor manifestations

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EPV-0361 | The impact of androgen deficiency on neuropsychological status and quality of life in men

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EPV-0362 | The impact of migraine on cognitive processing and central auditory processing: A study evaluating patients' history

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EPV-0363 | Toward earlier detection: A delphi Study on non-vital-sign early behavioural & cognitive indicators of postoperative delirium in surgical inpatients

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EPV-0364 | Value of ptau217 in the differential diagnosis of Alzheimer's disease in a clinical cohort

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EPV-0365 | When biomarkers conflict with the clinic: Positive RT-QuIC in the absence of clinical Creutzfeldt-Jakob disease – A case report

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EPV-0366 | When cognition leaves a digital footprint: Evidence on passive smartphone-based biomarkers

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Coma and chronic disorders of consciousness

EPV-0367 | Conscious but confused – What's in a name?

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EPV-0368 | Diagnostic advances for patients with disorders of consciousness: The ultrasound assessment

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EPV-0369 | EEG-derived network parameters for the fine grading and economic bridging of cognition-motor dissociation in DoC

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EPV-0370 | Exploring cardiac interoceptive self-related processes in disorders of consciousness

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EPV-0371 | Family perspectives on prolonged disorders of consciousness after intensive neurorehabilitation: A nationwide qualitative study

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R. Koopmans; W. van Erp
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EPV-0372 | Higher incidence and prevalence of coma in the USA than in the UK

M. Hassani; S. Stückler; M. Amiri; M. Othman; D. Kondziella
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EPV-0373 | Specialized long-term care for severe acquired brain injury in the netherlands: Organizing nationwide networks

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EPV-0374 | TMS-EEG as a unique tool to probe cortical dysconnectivity after severe TBI

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EPV-0375 | Value of bedside diagnostic tools for predicting outcome in acute disorders of consciousness

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Education in neurology

EPV-0376 | Anterior insula and dACC as regulatory hubs: Network convergence and brain-behavior links

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EPV-0377 | Assessing two methods of medical education on prolonged seizures and Rapid and Early Seizure Termination (REST)

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EPV-0378 | Assessment of nutritional knowledge in patients with multiple sclerosis in Opole and Silesian Province, Poland

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EPV-0379 | Awareness and perception of neurology as a career choice among medical students in a low-resource setting

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EPV-0380 | Bridging knowledge and practice: Neurology trainees' views on functional neurological disorders

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EPV-0381 | Bridging the gap: Assessing stroke knowledge disparities among future healthcare professionals in Sudan

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EPV-0382 | Cinemeducation in neurology: Teaching empathy and bioethics through cinema and narrative ethics

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EPV-0383 | Computer-aided localization in neurology: Integrating structured digital assessment with machine learning for automated diagnostic support

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EPV-0384 | Determinants of poor adherence to antihypertensive medication among primary hemorrhagic stroke patients in the north Indian population

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EPV-0385 | Education on biosimilars and JC virus testing in neurology: Knowledge gains through online CME

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EPV-0386 | Education on myasthenia gravis diagnosis results in practice changes and identifies diagnostic barriers

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EPV-0387 | Emotional knowledge integration through boundary-spanning search in multiple sclerosis

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EPV-0388 | Epidemiology and educational implications of neurophobia in Germany, Austria and Switzerland: A trination study

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EPV-0389 | From data to decision: The librarian–neurologist alliance in neurology education

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EPV-0390 | How ReCognition Health (RCH) has fast tracked patients to receive amyloid targeted treatment

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EPV-0391 | How ReCognition Health (RCH) has fast tracked patients to receive Amyloid Targeted Treatment (ATT)

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EPV-0392 | Improvements in implementing the latest practical strategies for myasthenia gravis after continuing medical education

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EPV-0393 | Improving competence in addressing under- and over-treatment in epilepsy through medical education

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EPV-0394 | Improving knowledge, competence and confidence through curriculum-based continuing medical education in focal epilepsy

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EPV-0395 | Knowledge and attitudes towards Parkinson's disease among the Sudanese population: A nationwide cross-sectional study

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EPV-0396 | Medical education on shared decision-making in epilepsy significantly improves knowledge of key topics to discuss with patients

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EPV-0397 | Medication reconciliation in hospitalized neurology patients: A cross-sectional study

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EPV-0398 | Meeting of the minds 2025: A student-led model for advancing neuroeducation through immersive and inclusive learning

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EPV-0399 | Neuroimmunology matters: Accelerating cumulative learning and improving outcomes through centralised education hubs

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EPV-0400 | Online postgraduate neuronutrition program for neurologists: An interdisciplinary educational model

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EPV-0401 | Patient and public willingness to accept disease-modifying therapies for Alzheimer's and Parkinson's: Drivers and barriers

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EPV-0402 | Right patient, right pathway: Consultant-led neurology referral management outcomes from a UK tertiary centre

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Environmental Neurology/Climate Change

EPV-0403 | Climate change as a disruptor of acute neurology: A surge and squeeze threat to time critical care

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EPV-0404 | Climatic predisposing factors for posterior circulation aneurysm rupture

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EPV-0405 | Clinical and neuropsychological characteristics of chronic cerebral ischemia in patients living in the Aral environmental crisis region

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EPV-0406 | Environmental factors in multiple sclerosis focusing climate change

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EPV-0407 | Increasing incidence of Lyme disease in the Republic of Moldova: National data and potential neurological implications in climate change context

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EPV-0408 | Neuroplasticity in spaceflight: Adaptive and maladaptive brain changes in microgravity

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EPV-0409 | Rising temperatures, rising risks: Heatwave effects on Parkinson's disease

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Epilepsy

EPV-0410 | “The Silent Rust of the Brain”: A case of delayed diagnosis of idiopathic superficial siderosis of the CNS

K. Duve; S. Shkrobot; R. Nasalyk
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EPV-0411 | “Seizure-like phenomena (SLP)” in the context of propofol sedation after recovered cardiac arrest: A case report

I. Ruiz Salcedo; V. Carmona Bravo; A. Torres Moral; F. Sánchez Fernández; C. Conde Velasco; A. Rincón Valencia; J. Montaner Villalonga; S. Pérez Sánchez
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EPV-0412 | A multimodal automated seizure diagnosis model for seizure detection and type classification using video-electroencephalogram data

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EPV-0413 | A rare complication of radiotherapy: SMART syndrome

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EPV-0414 | A review of the burden of status epilepticus, approaches to its treatment, and any gaps in the Asia-Oceanic region

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EPV-0415 | A systematic review of health economic evaluations of treatments for patients with drug-resistant epilepsy

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EPV-0416 | A systematic review of human status epilepticus in organophosphate poisoning: A real-world stage 1 plus model?

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EPV-0417 | ABCC2 polymorphisms and antiseizure medication resistance: A systematic review and meta-analysis with power assessment

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EPV-0418 | Age and sex disparities in healthcare utilization and costs among epilepsy patients: A population-based study in Korea

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EPV-0419 | Applications of machine learning in predicting valproic acid pharmacokinetics, safety and treatment response in epilepsy

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EPV-0420 | Beyond Drug Resistance: Rethinking Early Surgery in Lesional Focal Epilepsy

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EPV-0421 | Brain arteriovenous malformations and epilepsy – A retrospective analysis from a tertiary centre

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EPV-0422 | Clinical and neurophysiological diagnosis and surgical treatment of post-traumatic drug-resistant temporal lobe epilepsy

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EPV-0423 | Clinical characteristics and outcomes of patients with refractory/super refractory status epilepticus: A retrospective analysis

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EPV-0424 | Clinical characteristics, etiology, and outcomes of motor Epilepsia Partialis continua: A 12-patient cohort study

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EPV-0425 | Clinical correlates of depressive symptoms and the treatment gap of depression among adults with epilepsy

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EPV-0426 | Clinical outcomes of adult-initiated cenobamate in developmental and epileptic encephalopathy versus other drug-resistant epilepsies

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EPV-0427 | Clinical, EEG and CSF predictors of outcome in status epilepticus: A retrospective study

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EPV-0428 | Comparative analysis of wearable machine learning-based and EEG-based seizure detection methods

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EPV-0429 | Comparative performance of the Montreal cognitive assessment and the mini-mental state examination for cognitive screening in patients with epilepsy

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EPV-0430 | Comparison of prognostic score performance in refractory status epilepticus treated with continuous intravenous anesthetic drugs at 90 days

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EPV-0431 | Correlation of EEG findings outcome in Juvenile Myoclonic Epilepsy (JME) – A multi-centric retrospective study

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EPV-0432 | Deep brain stimulation in drug-resistant epilepsy: Our experience in clinical practice

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EPV-0433 | Detection of anxiety disorders in tension-type headache and evaluation of the effectiveness of cognitive behavioral therapy

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EPV-0434 | Detection of anxiety-phobic disorders in epilepsy and evaluation of the effectiveness of medical-psychological intervention

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S. Yusuphodjayeva; G. Ernayeva

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EPV-0435 | Dynamics of epilepsy in patients with supratentorial brain ventricular system tumours depending on surgical approach

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EPV-0436 | Early cenobamate as a third-line option in drug-resistant focal epilepsy: A paradigm shift?

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EPV-0437 | Early interventions for children with epileptic encephalopathies: a systematic review

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EPV-0438 | EEG-VL: Integrating visual features with large language models for automated seizure detection

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EPV-0439 | Effects of seizure frequency, EEG and MRI findings on quality of life and psychological burden in epilepsy

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EPV-0440 | Efficacy and safety of adjunctive cenobamate in patients with intellectual disability: A real world experience

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EPV-0441 | Efficacy and safety of omega-3 fatty acid supplementation on seizure outcomes in epilepsy: A systematic review and meta-analysis

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EPV-0442 | Epilepsy-on-a-chip: In vitro platforms for seizure mechanisms and drug discovery

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EPV-0443 | Eslicarbazepine acetate exposure in pregnant women with epilepsy: From clinical development to 16 years post-marketing experience

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EPV-0444 | Facial emotion recognition in focal epilepsy: Localization is not the main factor

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EPV-0445 | Ferroptosis as a key mechanism in the pathogenesis of symptomatic epilepsy

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EPV-0446 | Focal epilepsy with variable foci. Relevance of genetics in epilepsy

G. Torres Sánchez; H. El Mouhajir Mohamed; M. González Campos; A. Tapia Barrantes; J. Mederos Hernández;

G. Velamazán Delgado

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EPV-0447 | Focal myoclonic status epilepticus without coma presenting as perioral and cervical myoclonias in an elderly patient

L. Dominguez-Jimenez; M. Garcia-Ruiz; E. Lopez-Valdes;

A. Lafuente-Almaden; P. Abizanda-Saro; M. Alvarez-Gutierrez; J. Melgar; M. Bonnin-Bravo; P. Mayo-Rodriguez

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EPV-0448 | From benign infantile epilepsy to paroxysmal kinesigenic dyskinesia: The clinical spectrum of PRRT2-related disorder

C. Hurtado Alcázar; R. Calle Calle; C. Santillana Ávila; Á. Morales Lahoz; I. Del Pino Díaz

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EPV-0449 | Genetic generalized epilepsy: phenotypic characterization and prognostic factors

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EPV-0450 | Gray matter heterotopia in adults: Clinical and radiologic features across morphologic subtypes

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EPV-0451 | Identification of risk factors and evaluation of clinical and electrophysiological features in epilepsy-related psychosis

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EPV-0452 | Inflammatory-micronutrient phenotype in epilepsy: Associations with EEG abnormalities, and seizure burden in an outpatient cohort

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EPV-0453 | Inherited instability of sleep architecture creating a light-NREM seizure window in juvenile myoclonic epilepsy

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EPV-0454 | Lamotrigine monotherapy as an effective treatment option in drug-resistant epilepsy associated with tuberous sclerosis complex

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EPV-0455 | Late-onset Rasmussen encephalitis, should early hemispherectomy be considered?

B. Jové; M. Olivera; O. Brengaret; N. Bennouna; M. Ruiz-Vives; J. Tutusaus; E. Conde-Blanco; M. Centeno; M. Carreño
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EPV-0456 | Lennox–Gastaut syndromes secondary to chromosome 15 rearrangement: description of a case series and response to cannabidiol treatment

M. Callejo¹; A. Marinas¹; P. Ceballos²; I. Garamendi¹; I. Escalza³; A. Moreno⁴; M. Horvarth⁵; M. Martínez¹; Á. López¹; N. Marcos¹; F. Angoso¹; L. Pastor¹; J. Mallada¹; A. Rodriguez-Antigüedad¹

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EPV-0457 | Levetiracetam for new onset epilepsy in older adults: A 24 week prospective cohort study

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EPV-0458 | Levetiracetam for seizure prophylaxis post-neurosurgery: Prescription patterns and ADR monitoring

R. Baxi; P. Marathe; S. Kamat; S. Mishra; J. Jha; T. Nadkarni; D. Muzumdar
Seth GS Medical College & KEM Hospital, Mumbai

EPV-0459 | Machine learning-based prediction of post-stroke epilepsy

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EPV-0460 | Multifocal epileptiform activity as an independent predictor of mortality in hospitalized patients

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EPV-0461 | Multimodal AI decision support for epilepsy surgery under functional uncertainty

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EPV-0462 | Next-generation sequencing: application for Kazakhstani patients with epilepsy

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EPV-0463 | NGS-based identification of genetic variants potentially influencing AED response in pediatric epilepsy

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EPV-0464 | Optimization of treatment strategy in DEE-SWAS: Role of early hormonal therapy

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EPV-0465 | Post-encephalitic epilepsy after autoimmune encephalitis

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EPV-0466 | Preoperative and postoperative evaluation of cognitive functions in patients undergoing temporal and extratemporal epilepsy surgery

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EPV-0467 | Prevalence of depression in patients with epilepsy and evaluation of the effectiveness of medical-psychological interventions

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EPV-0468 | Psychiatric adverse-event monitoring in epilepsy: Lesson from a systematic literature review of eslicarbazepine acetate, lacosamide, and levetiracetam

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EPV-0469 | Pyridoxine for preventing and treating levetiracetam-induced behavioural and neuropsychiatric adverse effects in patients with epilepsy

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EPV-0470 | Quality of life, self stigma, and associated factors in patients with epilepsy in Colombo, Sri Lanka

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EPV-0471 | Rasmussen encephalitis: clinical spectrum, complementary investigations, and therapeutic strategies from a case series

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EPV-0472 | Real-life experience of switching from branded to generic eslicarbazepine acetate

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EPV-0473 | Refractory status epilepticus in posterior reversible encephalopathy syndrome resolved with perampanel after failure of barbiturate coma

A. Herrera Ortega; M. Muñoz Pasadas; J. Vaamonde Gamio; P. Gómez Ramírez; A. Sánchez Gómez; M. El Harmochi Daoud; V. Ros Escolar; M. Carrillo Carrillo

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EPV-0474 | Retrospective prescription based analysis of levetiracetam usage in various types of epilepsies in Indian outpatient's settings

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EPV-0475 | Routine EEG versus 24-hour EEG in the discontinuation of anti-seizure medications in epileptic patients with long-term seizure freedom

J. Almada Silva; S. Bernardo; P. Lopes das Neves; A. Martins; J. Peres

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EPV-0476 | Seizure control in mesiotemporal epilepsy after 6 months of treatment by means of focal cortical stimulation

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EPV-0477 | Seizure frequency as a determinant of cognitive impairment severity in epilepsy

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EPV-0478 | Seizure worsening as a sentinel sign of penicillamine-induced aplastic anemia in wilson disease

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EPV-0479 | Seizures reported under stroke code: What is truly stroke?

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EPV-0480 | Abstract withdrawn

EPV-0481 | Sex-specific differences in presentation, management and outcomes in status epilepticus: A retrospective cohort study

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EPV-0482 | Siamese-ES: A multi-task learning framework leveraging sleep features for interictal epileptiform discharge detection

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EPV-0483 | Status epilepticus: Critical review and experience. First step towards an effective protocol

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A. Sanchez Rodriguez; M. Sanchez-Suárez López
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EPV-0484 | The double twenty: Unmasking a rare chromosomal epilepsy in a young adult

B. Chelaru; S. Petrescu; C. Gheti; M. Martoiu; A. Daneasa;
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EPV-0485 | The epidemiology, cultural perceptions, and management of epilepsy in roma communities: A systematic review

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EPV-0486 | The role of stress sensitivity in the genetics of juvenile myoclonic epilepsy

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EPV-0487 | Circadian distribution of seizures and their impact on sleep structure in adults with epilepsy

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EPV-0488 | Transforming care for people with tuberous sclerosis complex: A collaborative, nurse-led evolution toward a national multidisciplinary model

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Neurology, Trinity College Dublin, Ireland; ⁵TSC Ireland,
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EPV-0489 | Transition of care from paediatric to adult epilepsy services in malta: A clinical review

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EPV-0490 | Use of cenobamate in a patient with super-refractory status epilepticus

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EPV-0491 | Vagus nerve stimulation for RSE/SRSE: Systematic review of survival and seizure freedom outcomes

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EPV-0492 | Velopharyngeal myoclonic status as a manifestation of cortical stroke

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EPV-0493 | Where do young individuals with epilepsy die? A study of mortality locations in patients under 18

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Ethics in neurology

EPV-0494 | Decision-making capacity and safeguards in voluntary assisted dying for neurodegenerative disorders

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EPV-0495 | Epistemic authority in adaptive DBS: When neural metrics compete with lived experience

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EPV-0496 | Ethical and Governance Challenges in Neurotechnology: Implications for Clinical Neurology

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EPV-0497 | Presymptomatic disease-modifying therapies in neurodegenerative disorders: Clinical promise and ethical challenges

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EPV-0498 | The ethics of chiropractic cervical spinal manipulation

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EPV-0499 | “Interictal photophobia as a potential risk factor for migraine chronification and treatment resistance: A prospective study proposal”

M. Alkhawaja; M. Fragoso; J. Paz; P. Comes; G. Salazar
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EPV-0500 | A Comparison of the efficacy, tolerability and adverse effects of two Anti-CGRP (Erenumab and Galcanezumab) in migraine prevention

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EPV-0501 | Absenteeism caused by migraine among Norwegian hospital nurses

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EPV-0502 | Achieving higher standards for migraine prevention in a series of 639 patients with chronic migraine treated with OnabotulinumtoxinA

C. Sánchez-Rodríguez; E. Varas-Martín; C. Montero Grande; P. Jiménez Caballero; Á. Sierra-Mencía; A. Recio-García; P. Caballero-Lillo; M. Peñas-Martínez; A. Gonzalez-Martinez; Á. Guerrero-Peral
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EPV-0503 | Alterations in interoception among patients with medication-overuse headache

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EPV-0504 | Atogepant for migraine prevention, including patients previously treated with anti-CGRP therapies: A real-world cohort

R. Nunes Rato; A. Neves; C. Sousa; R. Dias; M. Pinto; M. Pinto; A. Costa
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EPV-0505 | Beyond headache freedom: Crystal-clear and unclear days to assess residual interictal burden in migraine treated with CGRP monoclonal antibodies

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EPV-0506 | Beyond headache frequency: Sex-specific clinical and lifestyle signatures of migraine chronicity

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EPV-0507 | Beyond pain: impact of atogepant on migraine-related symptoms

C. Sousa; A. Neves; R. Rato; R. Dias; M. Pinto; M. Pinto; A. Costa
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EPV-0508 | Breath holding cerebrovascular responses in migraine: longitudinal fNIRS comparison of CGRP mAb and oral preventives

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EPV-0509 | Cerebrospinal fluid biomarkers in idiopathic intracranial hypertension: Targeted proteomics analysis

B. Arora; A. Pandit; S. Shree; P. Tangri; R. Singh; M. Nath
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EPV-0510 | CGRP level in patients with vestibular migraine and the therapeutic effect of onabotulinumtoxinA

B. Holmuratova; N. Rashidova; K. Khalimova
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EPV-0511 | Changes in muscle tone after electrotherapy in patients with chronic migraine and cervicalgia

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EPV-0512 | Characterisation of patients with new daily persistent headache treated at a tertiary care hospital

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EPV-0513 | Clinical characteristics and outcomes of patients with idiopathic intracranial hypertension treated by transvenous venous sinus stenting

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EPV-0514 | Clinical outcomes of rimegepant in acute and prophylactic migraine therapy in a real-world setting

N. Öksüz¹; E. Ekizoğlu²; N. Tepe³; R. Karacı⁴; T. Cerrahoğlu Şirin⁵; A. Atalar⁶; E. Acıman Demirel⁷; B. Polat⁸; P. Yalınay Dikmen⁹; D. Vurallı¹⁰; H. Genç¹¹; F. Domaç⁴; E. Ilgaz Aydınlar⁹; E. Kocasoy Orhan²; A. Özge¹

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EPV-0515 | Clinical outcomes of topiramate and onabotulinumtoxinA in chronic migraine prophylaxis

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EPV-0516 | Cluster headache and sleep: A questionnaire-based cross-sectional study on the bidirectional relationship between attacks and sleep disturbances

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EPV-0517 | Cluster-BIO: Cross-sectional study of plasma biomarkers (CGRP, PACAP-38, and VIP) in patients with cluster headache

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EPV-0518 | COACT study: CGRPmAbs + OnabotulinumtoxinA assessment of chronic migraine treatments study

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EPV-0519 | Cognitive complaints in migraine: response to preventive treatment with atogepant

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EPV-0520 | Cognitive flexibility, resilience and executive control in chronic migraine with and without medication overuse

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EPV-0521 | Comparative efficacy and safety of magnesium sulfate versus metoclopramide for treating acute migraines: A systematic review and meta-analysis

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EPV-0522 | Comparative efficacy and safety of metoclopramide versus sumatriptan in acute management of migraine: A systematic review and meta-analysis

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EPV-0523 | Comparative efficacy and safety of propranolol versus flunarizine for migraine prophylaxis: A systematic review and meta-analysis

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EPV-0524 | Comparing neurologist and neurologist-customised semantic search engine management plans for chronic migraine: A prospective pilot study from Kenya

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EPV-0525 | Data of efficacy and safety of Atogepant in the preventive treatment of migraine in a cohort of highly treatment-refractory migraine patients

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EPV-0526 | Diagnostic yield of non-contrast cranial CT in chronic tension-type headache: A retrospective cohort

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EPV-0527 | Distinct clinical features in adult and pediatric HaNDL syndrome: A literature-based analysis

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EPV-0528 | Effectiveness and safety in patients receiving atogepant

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EPV-0529 | Effectiveness of pulsed radiofrequency for migraine: A systematic review and meta-analysis

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EPV-0530 | Effects of astrocytic metabolic dysfunction and glucose stress on gene expression during cortical spreading depression

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EPV-0531 | Efficacy and safety of Eptinezumab 100 mg and 300 mg for migraine prevention: A systematic review and meta-analysis of randomized trials

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EPV-0532 | Efficacy and safety of fentanyl for prophylaxis against postdural puncture headache in cesarean section: A meta-analysis

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EPV-0533 | Efficacy and safety of Gabapentin versus placebo against postdural puncture headache: A systematic review and meta-analysis

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EPV-0534 | Efficacy and safety of magnesium in preventing post-dural puncture headache after cesarean delivery: A systematic review

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EPV-0535 | Efficacy and safety of ondansetron versus conservative treatment in post-dural puncture headache prophylaxis in cesarean section: A meta-analysis

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EPV-0536 | Efficacy and safety of Rimegepant 75 mg for migraine prevention: A meta-analysis of randomized controlled trials

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EPV-0537 | Efficacy and safety of valproate versus topiramate for pediatric migraine prophylaxis: A systematic review

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EPV-0538 | Efficacy of coenzyme Q10 for migraine: a systematic review, meta-analysis, and trial sequential analysis

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EPV-0539 | Efficacy of Rimegepant 75 mg for acute migraine treatment: A systematic review and meta-analysis of randomized controlled trials

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EPV-0540 | Eptinezumab for difficult to treat migraine: Who responds and who need more

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EPV-0541 | Frequency of migraine phenotype in individuals at risk for MEFV variations: A neuroinflammatory reflection of familial mediterranean fever

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EPV-0542 | Global prevalence of medication-overuse headache: A systematic review and meta-analysis

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EPV-0543 | Headache features among military personnel hospitalized following combat deployment

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EPV-0544 | Headache in chiari malformation type I: Clinical phenotypes and diagnostic pitfalls

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EPV-0545 | High standards of migraine control in patients treated with anti-CGRP monoclonal antibodies

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EPV-0546 | How does war affect the next generation of doctors? Cognitive flexibility and headache burden among sudanese medical students

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EPV-0547 | Immunogenetic drivers of migraine: Evidence for microglial inflammasome and IL-1 β involvement

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EPV-0548 | Impact of ATGepant on interictal burden and crystal CLEAR days in difficult-to-treat migraine: The ATO-CLEAR study

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EPV-0549 | Impact of premonitory symptoms on quality of life in patients with migraine without aura

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EPV-0550 | Impact of sex on clinical presentation and diagnostic delay in cluster headache

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EPV-0551 | Implementation and impact of medication reviews on patients eligible for eptinezumab treatment in severe migraine in a pain clinic

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EPV-0552 | Interpretation of increased resistive index in the middle meningeal artery during migraine attacks

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EPV-0553 | Intracranial hypertension due to a giant Pacchionian granulation: A case report with complete endovascular resolution

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EPV-0554 | Less is more: Low-dose divalproex for recurrent dialysis headache

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EPV-0555 | Machine learning for differential diagnosis of migraine: A systematic review

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EPV-0556 | Machine learning in headache: A systematic review of diagnostic and classification approaches

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EPV-0557 | Medication misuse of CGRP-targeted therapies without adverse effects in chronic migraine

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EPV-0558 | Migraine and pregnancy: Can we do better?

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EPV-0559 | Migraine diagnosis across Europe: EMHA migraine in women survey results, H2 2025

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EPV-0560 | Migraine in low-income community: Prevalence, care practice and access barriers – a cross-sectional study

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EPV-0561 | Migraine with aura in female patients with psoriasis: A systematic review

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EPV-0562 | Migrainous infarction masked by Patent Foramen Ovale: Diagnostic challenge and preventive management

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EPV-0563 | OnabotulinumtoxinA for occipital neuralgia: results from a series of 15 patients

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EPV-0564 | OnabotulinumtoxinA in patients with painful trigeminal neuropathy. Experience in a headache unit

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EPV-0565 | Outcomes of blind epidural blood patching in intracranial hypotension

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EPV-0566 | Peripheral blood electrolyte profiles in patients with migraine with and without aura

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EPV-0567 | Prevalence and characteristics of headaches in adolescents – is problem being underestimated? Subgroups analysis of the HY-LIFE study

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EPV-0568 | Prevalence and clinical features of cough headache: A systematic review with meta-analysis

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EPV-0569 | Prophylactic efficacy and safety of sphenopalatine ganglion block versus greater occipital nerve block against headache disorders: A meta-analysis

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EPV-0570 | Real-world effects of onabotulinumtoxinA and anti-CGRP monoclonal antibodies on disability and clinical phenotype in chronic migraine

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EPV-0571 | Real-world efficacy and tolerability of atogepant for migraine prevention after 6 months of treatment: a double center experience

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EPV-0572 | Real-world long-term effectiveness and retention of CGRP-targeted therapies: A 4-year retrospective study in a Thai tertiary headache clinic

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EPV-0573 | Remote electrical neuromodulation (Ren) with nerivio device. Our experience in 4 cases of new daily persistent headache (Ndph)

J. Tutusaus; N. Bennouna; B. Jové; M. Ruiz; D. Asín; V. Obach-Irimia; R. Armand; A. Grajea; M. Marco; A. Sosa; T. Topczewski; S. Fernández; N. Fabregat; V. Obach
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EPV-0574 | Retrospective evaluation of patients with idiopathic intracranial hypertension: A single-center experience

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EPV-0575 | rTMS in treating patients with chronic migraine and depression: A real-life exploratory analysis

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EPV-0576 | Serum and CSF neurofilament light chain and glial fibrillary acidic protein levels in idiopathic intracranial hypertension

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EPV-0577 | Sex differences in headache unit referral in patients with chronic migraine

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EPV-0578 | Short-term effectiveness of new anti-CGRP therapies in chronic and episodic migraine

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EPV-0579 | Sleep disorders in medication-overuse headache: prevalence, associated factors, and reversibility after treatment

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EPV-0580 | Spontaneous Intracranial Hypotension (SIH): heterogeneity in clinical, radiological, and therapeutic responses—A case series

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EPV-0581 | Spontaneous intracranial hypotension mimicking status migrainosus in a woman with a known history of classic migraine

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EPV-0583 | Study of blood CGRP level patterns in migraine subtypes and other headaches in 1 tertiary healthcare setup of north India

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EPV-0584 | Targeted gut-focused dietary intervention in migraine patients with gastrointestinal comorbidity: A real-world observational study

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EPV-0585 | Targeted intermittent atogepant for breakthrough attacks in partial responders to fremanezumab: A real-world case series

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EPV-0586 | Tension-type headache and new postural headache: Clues to low cerebrospinal fluid pressure

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EPV-0587 | The impact of Erenumab and Galcanezumab upon quality of life among migraine patients in Kuwait

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EPV-0588 | The Nordic Chronic Migraine Trial of CGRP monoclonal antibody and onabotulinumtoxin A dual therapy—A randomized placebo-controlled double-blind trial

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EPV-0589 | The relationship between computer vision syndrome, technostress, and headache: A multicenter cross-sectional study

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EPV-0590 | Three decades of tension-type Headache burden in Europe: Regional variation in age-standardized disability-adjusted life years

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EPV-0591 | Tryptophan used as a dietary supplement: A new option for migraine treatment?

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EPV-0592 | Use of corticosteroid-containing greater occipital nerve injections in the perinatal period

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Higher cortical functions

EPV-0593 | Baseline kynurenine pathway metabolites and post-stroke cognitive performance: a prospective cohort study

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EPV-0594 | Functional MRI-informed EEG identification of active cortical regions in a silent naming task

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EPV-0595 | Functional neuroplasticity in response to a giant left-sided arachnoid cyst: An fMRI and DTI analysis of language preservation

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EPV-0596 | Method for assessing stereognosis to evaluate the morphofunctional organization of tertiary cortical areas in preschool children

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EPV-0597 | Mixed reality-based functional screening for hemispatial neglect in acute right parietal stroke

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EPV-0598 | Oculomotor apraxia as a window into visuomotor network stroke

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EPV-0599 | Survey on the actual conditions of traumatic brain injury resulting from bicycle traffic accidents in Japan

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EPV-0600 | Telemedicine-based assessment of limb apraxia in neurodegenerative diseases: Feasibility and diagnostic implications

E. Smaragdaki; I. Stamelos; E. Angelopoulou; K. Vourou; D. Kontaxopoulou; J. Papatrifiantayllou; L. Stefanis; V. Konstantinidis; S. Papageorgiou
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History of neurology

EPV-0601 | A concept of autoimmune etiology of ALS- a historical evolution and current state

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EPV-0602 | Historical perspectives on epilepsy care in rural areas in the Philippines

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EPV-0603 | Merging culture-bound hyperstartle syndromes into a single nosological entity: What survived from Tourette's original description of tic disease?

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EPV-0604 | Neuro-FakeNews: Major historical errors in neurology

J. Sosa Luis; D. Machado Sampedro; L. Portela Martínez; M. Lozano López; R. Boto Martínez; G. Lafuente Gómez
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EPV-0605 | The awards of Hungarian neurology in the last 90 years

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EPV-0606 | A diagnostic paradox: Central nervous system tuberculosis in the setting of parvovirus B19 infection

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S. Martínez-García; G. Sánchez-Rincón
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EPV-0607 | A treatable mimic of dementia: Neurosyphilis with progressive speech apraxia and brain atrophy—Two cases and literature review

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EPV-0608 | A wicked malady tale-acute dystonia and residual hemidystonia following viral infection in high-risk acute lymphoblastic leukaemia pediatric patient

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EPV-0609 | Aseptic meningitis revisited: The meninges strike back

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EPV-0610 | Association of level of csf sugar, protein and cells with mortality amongst cases with bacterial and tubercular meningitis

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EPV-0611 | Clinical and neurophysiological features of post-COVID and Diabetic Polyneuropathy

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EPV-0612 | Clinical cases of bacterial meningoencephalitis during pregnancy

M. Vasilieva; A. Bondarciuc; A. Bejenari; E. Zota; D. Belitei;
G. Oglinda-Catirau; S. Groppa
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EPV-0613 | Clinical spectrum and cerebrospinal fluid predictors of adverse outcomes in Central Nervous System Tuberculosis – A retrospective cohort study

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EPV-0614 | Diagnostic challenges in multifocal brain lesions: A case of HIV-related toxoplasmic encephalitis

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EPV-0615 | Disseminated neurocysticercosis: Clinical manifestations, diagnostic challenges, and therapeutic outcomes

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EPV-0616 | Elsberg syndrome presenting as acute lumbosacral radiculitis: two case reports

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EPV-0617 | Epstein-barr virus-related neurological disease: Two cases illustrating heterogeneous clinical presentations

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EPV-0618 | Facial diplegia as the initial manifestation of HIV infection: A case report

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EPV-0619 | Froin syndrome as a diagnostic marker of tuberculous cauda equina arachnoiditis in inflammatory polyradiculopathy

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EPV-0620 | Fulminant *Listeria monocytogenes* meningoencephalitis in an immunocompromised patient: A case report

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EPV-0621 | Herpes simplex encephalitis presenting as super – refractory status epilepticus with retrograde amnesia sequelae in an immunocompetent adult

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EPV-0622 | Intramedullary abscess in a non-surgical candidate: Is medical management a viable option?

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EPV-0623 | Isolated oculomotor nerve palsy as an early neurological manifestation of HIV infection

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EPV-0624 | Keeping the great imitator in mind: A case of syphilitic encephalitis

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EPV-0625 | Management of *Nocardia farcinica* brain abscess with subdural empyema and septic sinus thrombosis in an elderly immunocompetent patient

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EPV-0626 | Meningoencephalitis caused by *streptococcus salivarius* in a 63-year-old patient with no significant medical history

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EPV-0627 | mRNA cytokine profiling in tuberculous meningitis associated infarctions

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EPV-0628 | Multiple cerebral space-occupying lesions: From presumed metastases to the diagnosis of cerebral tuberculosis

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EPV-0629 | Neuroaspergillosis mimicking acute polyradiculoneuritis

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EPV-0630 | Atypical neurobrucellosis: Lymphocytic meningoencephalitis with a solitary cystic brain lesion

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EPV-0631 | Neuromelioidosis – A life threatening masquerader

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EPV-0632 | Neurosyphilis presenting with rapidly progressive dementia and status epilepticus: A case report

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EPV-0633 | Non-convulsive status epilepticus-misdiagnosis in the Creutzfeldt-Jakob disease. Clinical cases

M. Vasilieva; G. Oglinda-Catirau; C. Munteanu; M. Junbei; S. Groppa
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EPV-0634 | Painful ophthalmoplegia and diplopia: Beyond Tolosa-Hunt syndrome

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EPV-0635 | PD-L1 immune checkpoint as a potential target for PML immunotherapy: Insights from 2 cases

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EPV-0636 | Post-infectious encephalitis associated with coxiella burnetii: A case with aphasia and focal seizures

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EPV-0637 | Post-infectious upper limb monoradiculopathy following dengue infection: A case report

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EPV-0638 | Post-rituximab progressive multifocal leukoencephalopathy treated with pembrolizumab

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EPV-0639 | Post-SARS-CoV-2 acute disseminated encephalomyelitis in a 4-year-old child: A case report

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EPV-0640 | Progressive multifocal leukoencephalopathy in a patient with pancytopenia and pulmonary tuberculosis: A case report

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EPV-0641 | Progressive primary aphasia with asymmetric temporal lobe atrophy as an initial presentation of neurosyphilis: A case report

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EPV-0642 | Pseudobulbar palsy due to Herpes Simplex 1 meningoencephalitis with bilateral opercular involvement

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EPV-0643 | Rapidly progressive dementia as a diagnostic challenge: A case of probable Creutzfeldt–Jakob disease

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EPV-0644 | Recurrent multifocal infarcts in a heart transplant patient with disseminated phaeohyphomycosis

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EPV-0645 | Survival after primary amebic meningoencephalitis: A rare case of naegleria fowleri infection in a diabetic adult patient

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EPV-0646 | The burden of persisting COVID-19 associated loss of smell on quality of life, health and mood: Baseline data from the SMELL-trial

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EPV-0647 | The unseen threat: A two-month cluster of probable sporadic CJD in Eastern India

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EPV-0648 | Tuberculous meningitis with negative microbiological studies: a diagnostic challenge

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EPV-0649 | Varicella zoster virus associated meningoradiculitis in an elderly patient: A case report

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EPV-0650 | Vasculopathy in central nervous system tuberculosis: A retrospective cohort study from Sri Lanka

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EPV-0651 | When Bell's palsy is not idiopathic: A diagnostic pitfall leading to Ramsay Hunt syndrome with brain encephalitis

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EPV-0652 | When shingles strikes the brain: A case of VZV vasculopathy with ophthalmoplegia and stroke

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EPV-0653 | When SSPE begins with blindness: A rare presentation with recurrent visual loss and hemimyoclonus

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Motor neurone diseases

EPV-0654 | Care-related regret and procrastination as determinants of burnout among healthcare professionals managing spinal muscular atrophy

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EPV-0655 | Clustering and personalized prognostic prediction in patients with amyotrophic lateral sclerosis

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EPV-0656 | Contribution of nucleus accumbens and amygdala subnuclei to the emergence of positive and negative behavioural symptoms in ALS

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EPV-0657 | Does genetic background influence serum creatine phosphokinase levels in amyotrophic lateral sclerosis?

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EPV-0658 | Dropped head syndrome identifies a malignant phenotype in FUS-associated ALS

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EPV-0659 | Early and mild cognitive-behavioral changes independently predict caregiver burden in ALS: a longitudinal study using the MBNI classification

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EPV-0660 | Home-based tDCS in amyotrophic lateral sclerosis: Effects on clinical progression and neurodegeneration biomarkers

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EPV-0661 | Immune aging connects chronic gastritis and Parkinson's disease through a gastric-brain network

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EPV-0662 | Incidence and prevalence of amyotrophic lateral sclerosis in Curitiba, Brazil

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EPV-0663 | Long-term respiratory and clinical evaluation in adult SMA type 2 patients treated with risdiplam: A 48-month observational study

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EPV-0664 | Motor neuron diseases in latvia: A nationwide epidemiology study

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EPV-0665 | Our experience with intrathecal tofersen treatment in patients with SOD1-associated ALS

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EPV-0666 | Real-world treatment with risdiplam in adults with Spinal Muscular Atrophy (SMA): A single center experience

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EPV-0667 | Results of the molecular genetic study of amyotrophic lateral sclerosis (ALS) in the Republic of Belarus

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EPV-0668 | Risdiplam in adult patients with spinal muscular atrophy: 24-month follow-up

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EPV-0669 | Rising disability from motor neuron disease across Europe: Mapping long-term trends in the regional burden

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EPV-0670 | Safety of memantine in adults with amyotrophic lateral sclerosis: A systematic review

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EPV-0671 | Sensory nerve involvement in motor neuron disease

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EPV-0672 | Systematic review of fluid biomarkers in Parkinson's disease for tracking progression and early diagnosis

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EPV-0673 | TDP-43 deficiency-induced triglyceride catabolism disorder in ALS: Lipid droplets accumulation, insufficient energy supply and neuronal degeneration

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EPV-0674 | Tracking the individualized longitudinal tempo of ALS through clinical and inflammatory trajectories

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EPV-0675 | Unraveling the mystery of alien hand syndrome: When your hand has a mind of its own. Abstract

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EPV-0676 | Wearable sensors detect postural abnormalities in spinal muscular atrophy

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Movement disorders

EPV-0677 | A case of neurodevelopmental disorder mitochondrial with abnormal movements and lactic acidosis with or without seizures (nemmlas)

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EPV-0678 | A case of PLA2G6-associated neurodegeneration

A. Gamboa Berastegui; M. Cortes Rubiales; A. Rodriguez Valer; S. Cajaraville Vicente; C. Sifre Peña; A. Santesteban Nedderhoff; A. Rodriguez Lopez; A. Barquin Toca; A. Pinedo Brochado
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EPV-0679 | A novel homozygous PLA2G6 mutation in a patient with young-adult-onset dystonia-parkinsonism

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EPV-0680 | A pilot, cross-sectional study on sleep quality and anxiety between Parkinson's disease (PD) patients and their caregivers

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EPV-0681 | A preventable clinical cascade: Levodopa-induced polyneuropathy as a contributor to traumatic cervical myelopathy in Parkinson's disease

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EPV-0682 | Accelerating real-world evidence generation in Parkinson's disease using the AccessPD registry: Case studies from digital sub-studies in the UK and US

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EPV-0683 | Access to essential medicines for Parkinson's disease in armenia: A health system gap analysis

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EPV-0684 | Access to Parkinson's disease treatments in the Middle East: A multi-national survey of availability, affordability, and utilization patterns

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EPV-0685 | Adaptive deep brain stimulation in Parkinson's disease: Clinical outcomes, biomarkers and translational challenges—A focused literature review

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EPV-0686 | Adult-onset idiopathic tic disorder: a rare clinical entity

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EPV-0687 | Adult-onset vanishing white matter disease presenting with dopa-responsive dystonia

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EPV-0688 | Advocacy, care coordination, education & support services: ACCESS HD novel national Huntington's disease community outreach care pathway in Ireland

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EPV-0689 | Alpha-synuclein in neuron-derived extracellular vesicles in Parkinson's disease

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EPV-0690 | Amantadine as first-line treatment in early Huntington's disease: A one-year observational study

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EPV-0691 | Amyloid status as a moderator of the association between physical activity and progression of Parkinson's disease

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EPV-0692 | Archimedes' spiral-based analysis using machine learning model for tremor's classification

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EPV-0693 | Assessment of psychosis in Parkinson disease (PD)

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EPV-0694 | Association of Restless Leg Syndrome (RLS) with Parkinson's disease, migraine, multiple sclerosis: A systematic review and meta-analysis

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EPV-0695 | Association of sleep hygiene practice with disturbed sleep in early-stage Parkinson's Disease

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EPV-0696 | Atherogenic lipid indices and disease severity in restless legs syndrome: A cross-sectional case-control study

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EPV-0697 | Atypical tremor associated with genes involved in metal metabolism and neurovascular signaling

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EPV-0698 | Baseline clinical characteristics of Parkinson's disease at first presentation to a National Tertiary Referral Center in the Republic of Moldova

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EPV-0699 | Beyond motor control: Neuropsychiatric benefits of botulinum toxin type A in Parkinson's disease: A systematic review and meta-analysis

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EPV-0700 | Beyond motor symptoms in dystonia: Non-motor symptoms and retinal OCT features

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EPV-0701 | Beyond motor symptoms in Multiple System Atrophy (MSA): Our experience in northern tenerife

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EPV-0702 | Caregiver burden in people with Parkinson's disease treated with deep brain stimulation: How does it evolve over time?

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EPV-0703 | CARN1 regulates neurohomeostasis in Parkinson's disease via STUB1-mediated ubiquitination and carnosine axis

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EPV-0704 | Changes in tremor, anxiety and depression after MR-guided focused ultrasound for essential tremor: A prospective study and exploration of predictors

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EPV-0705 | Characteristics of dystonia in Niemann-Pick disease type C

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EPV-0706 | Characterizing the bradykinesia complex in GBA-associated Parkinson's disease: A series of three cases

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EPV-0707 | Clinical and genetic characteristics of ANO3-associated movement disorders in a large Chinese cohort

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EPV-0708 | Clinical characterization of the response to Fampridine in SCA27B: Challenges and opportunities

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EPV-0709 | Clinical drug-drug interaction profile of BIA 28-6156: Results from three phase I studies

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EPV-0710 | Clinical effectiveness and safety of unilateral and staged-bilateral MR guided focused ultrasound for essential tremor: Systematic literature reviews

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EPV-0711 | Clinical outcomes after dual-target MR-guided focused ultrasound in Parkinson's disease: A single-center experience

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EPV-0712 | Clinical phenotype and early axial involvement in very late-onset Parkinson's disease: A real-world cohort study

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EPV-0713 | Clinical profile and burden of non-motor symptoms in Parkinson's disease: A cross-sectional study

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EPV-0714 | Clinical, radiological, and genetic characterization of neurodegeneration with Brain Iron Accumulation (NBIA): A case series

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EPV-0715 | Cognitive decline and motor severity across progressive supranuclear palsy phenotypes

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EPV-0716 | Cognitive impairment in people with idiopathic dystonia in Bulgaria – A prospective cohort study

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EPV-0717 | Cognitive profile of LRRK2-G2019S Parkinson's disease in a Tunisian cohort

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EPV-0718 | Complications of levodopa–carbidopa intestinal gel therapy: Polyneuropathy, cacosmia, and peritonitis

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EPV-0719 | Continuous Subcutaneous Levodopa Infusion in Advanced Parkinson's Disease patients with Subthalamic Nucleus Deep Brain Stimulation

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EPV-0720 | Correlation between tnf- α levels and psychoemotional disorders in Parkinson's disease

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EPV-0721 | Could abdominal hernia be an early clue for Parkinson's disease?

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EPV-0722 | Deep brain stimulation for medically refractory tremor in multiple sclerosis: A single case and review of the literature

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EPV-0723 | Depression and anxiety in Parkinson's disease: A comparison according to motor phenotypes

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EPV-0724 | Depression and quality of life in patients with Parkinson's disease

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EPV-0725 | Development of a standardized glossary to improve multidisciplinary and patient-centered communication in Progressive Supranuclear Palsy (PSP)

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EPV-0726 | Diagnostic revision in Parkinson's disease within five years after diagnosis

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EPV-0727 | Diagnostic value of MRI Nigrosome-1 visualization vs DaTSCAN SPECT in Parkinson's disease

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EPV-0728 | Differentiating functional dystonia from organic dystonia – A tertiary care centre experience

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EPV-0729 | Digital biomarkers for detecting wearing-off in Parkinson's disease: A pilot study of the remepy mobile platform

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EPV-0730 | Digital narrative medicine in Parkinson's disease: Patient and caregiver stories to inform clinical care

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EPV-0731 | Domain-specific associations between pain and non-motor symptoms in Parkinson's disease

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EPV-0732 | Dopamine dysregulation syndrome induced by dopaminergic therapy in Parkinson's disease: A case report

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EPV-0733 | Dropped head syndrome after radiotherapy for Hodgkin's lymphoma: Cervical muscles as a potential target for botulinum toxin

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EPV-0734 | Dysregulated EEG theta activity suggests impaired synaptic build-up in levodopa-induced dyskinesia in Parkinson's disease

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EPV-0735 | Early real-world experience with foslevodopa/foscarbidopa infusion in a UK neurology centre

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EPV-0736 | Early-onset mixed phenotype in a patient with Huntington's Disease and SCA2: A clinico-pathological case description

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EPV-0737 | Effect of continuous subcutaneous infusion of Foslevodopa/Foscarbidopa on motor fluctuations, dyskinesia, and sleep; subjective vs. PKG watch scores

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EPV-0738 | Effects of Transcranial Pulse Stimulation (TPS) intervention in patients with Parkinson's disease

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EPV-0739 | Efficacy and safety of cannabinoid-based therapies in Parkinson's disease: A systematic review and meta-analysis of randomized controlled trials

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EPV-0740 | Efficacy and safety of opicapone in early Parkinson's disease: A prospective observational study

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EPV-0741 | Efficacy of Citicoline as an add-on therapy in Parkinson's disease: The CITIPARK TRIAL trial

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EPV-0742 | Efficacy of Foslevodopa/Foscarbidopa infusion on gait disturbances and non-motor symptoms in advanced Parkinson's disease

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EPV-0743 | Enteric alpha-synuclein pathology in early-onset Parkinson's disease and an incidental case in spinocerebellar ataxia type 6

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EPV-0744 | Epidemiology and genetic landscape of hyperkinetic movement disorders in a Belgian tertiary care centre

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EPV-0745 | Factors of health-related quality of life in elderly patients with Parkinson's disease

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EPV-0746 | Failed and inconclusive clinical trials in Parkinson's disease: A systematic review

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EPV-0747 | Fatigue as a key independent predictor of quality of life in Parkinson's disease

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EPV-0748 | Flow reduction and local tolerability after opicapone use with continuous foslevodopa/foscarbidopa infusion

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EPV-0749 | Foslevodopa-foscarbidopa subcutaneous administration as bridging therapy in deep brain stimulation withdrawal syndrome

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EPV-0750 | From waiting list to treatment: A "Traffic Light" triage algorithm for Foslevodopa/Foscarbidopa initiation in advanced Parkinson's disease

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EPV-0751 | Functional movement disorders – A one-year neuropsychiatric model to prevent chronicity

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EPV-0752 | Gait Instability in Parkinson's disease with motor fluctuations: Composite gait score analysis in clinical and real-world settings

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EPV-0753 | Genotype–phenotype–biomarker integration in Parkinson's disease and parkinson-plus syndromes: A prognostic matrix approach

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EPV-0754 | Glioblastoma following cell therapy in a patient with Parkinson's disease: A cautionary tale

M. Martinez Seijas; A. Lopez Prado; A. Lagüela Alonso; V. Anciones Martin; M. Callejo Seguela; N. Marcos Fernandez; L. Pastor Martin; J. Mallada Abellan; F. Angoso Berrocal; B. Tijero Merino; J. Gomez Esteban; M. Ruiz Lopez
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EPV-0755 | Gut dysbiosis and inflammation in Parkinson's disease: An integrated microbiome–biomarker assessment

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EPV-0756 | Gut microbiota dysbiosis and alpha-synuclein pathogenesis in Parkinson's disease: Exploring the gut-brain axis and fecal microbiota transplantation

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EPV-0757 | “Hot Cross Bun Sign” revealing multiple system atrophy: A case report

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EPV-0758 | If you look, you find: a novel CSF1R gene variant as a possible cause of parkinsonism

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EPV-0759 | Imaging characteristics of ANO10-related Ataxia

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EPV-0760 | Immunosuppression and Parkinson's disease risk: Unveiling the link – A systematic review of the literature and meta-analysis

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EPV-0761 | iMOVA: A scalable multimodal prototype for objective movement disorder assessment

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EPV-0762 | Impact of a 3-month ketogenic diet on sleep quality, daytime sleepiness, and body composition in patients with Parkinson's disease

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EPV-0763 | Improving useability and safety for Parkinson's patients – Use of the ENFit extension tube for delivery of LECIG infusion

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EPV-0764 | Impulse control disorders in Parkinson's disease: associations with dopaminergic burden and dosing patterns

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EPV-0765 | Inflammatory processes and the impact of MAO-B inhibitors in early-stage idiopathic Parkinson's disease

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EPV-0766 | Interlocking pentagons task and visuospatial deficit in parkinsonian syndrome: A quantitative approach

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EPV-0767 | International multicentre survey of driving and road traffic accidents in Parkinson's disease: The Drive-PD study

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EPV-0768 | Investigating spontaneous smiling in Parkinson's disease through the Whistle-Smile reflex

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EPV-0769 | Is there a relationship between sleep disturbances and bradykinesia features in essential tremor?

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EPV-0770 | Ketotic hyperglycemia-related generalized chorea: A rare and atypical presentation

M. Vargas Cobos; A. De Luca; R. Alfonso González; M. Capra; C. Gómez López de San Román; L. Caballero Sánchez; I. Bermejo Casado; D. Cerdán Santacruz; A. Castrillo Sanz; A. Mendoza Rodríguez
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EPV-0771 | Levodopa-sparing drugs persistence in advanced Parkinson's disease: a retrospective cohort study on device-aided therapies

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EPV-0772 | Local vascular mimics of asymmetrical limb tremor: A two-case report

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EPV-0773 | Longitudinal assessment of cognitive function in essential tremor

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EPV-0774 | Longitudinal changes and clinical utility of the neurological pupillary index in de novo Parkinson's disease

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EPV-0775 | Long-term 4-aminopyridine efficacy in SCA27B: Up to 18-month case series

Á. López Prado; M. Martínez Seijas; M. Callejo Seguela; N. Marcos Fernández; A. Lagüela Alonso; V. Anciones Martín; A. Rodríguez-Antigüedad Zarrantz; I. Rouco Axpe
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EPV-0776 | Longterm efficacy and safety of levodopa/entacapone/carbidopa intestinal gel in patients with advanced Parkinson's disease in Slovakia

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EPV-0777 | Lowering the occlusion pressure detection setting of the CRONO® LECIG pump can have a positive impact on device useability and safety

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EPV-0778 | Magnetic resonance imaging-based analysis of morphometric changes in grey matter in patients with Parkinson's disease and depression

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EPV-0779 | Abstract withdrawn

EPV-0780 | Mirror paresis of the contralateral limb following botulinum toxin injection

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EPV-0781 | Mortality trends in Stroke and paralytic syndrome among older adults in the United States: A 24-year retrospective analysis

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EPV-0782 | Motor phenotype of familial Parkinson's disease: A Tunisian cohort study

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EPV-0783 | Multimodal mixed-reality assessment for automated prediction of Movement Disorder Society clinical ratings

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EPV-0784 | Neuroinflammatory and axonal biomarkers as decision-support tools in Parkinson's disease and atypical Parkinsonism

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EPV-0785 | Neuropsychiatric outcomes after STN-DBS versus stereotactic lesioning in Parkinson's disease

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EPV-0786 | nOH ratio and risk of falls in patients with Parkinson disease

N. Centno Pique; J. Camacho Velasquez; P. Bermell Campos;

M. Bea Sintes; V. Adell Ortega; E. Franquet Gomez

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EPV-0787 | Non-motor subtypes in Parkinson's disease and atypical parkinsonian syndromes: A cluster-based longitudinal study from the PROATYP cohort

M. Makrygianni¹; D. Potamianou¹; T. Pipikos¹; L. Florentin²; A. Sarafi¹; E. Papadogeorgaki¹; C. Mourmouris¹; M. Xilouri³; M. Stamelou¹

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EPV-0788 | Non-motor symptoms in Parkinson's disease are under-recognised in routine care: Evidence from an Uzbek cohort

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EPV-0789 | Nothing alike: A PSP-like case

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EPV-0790 | OPAL-PD: A longitudinal observational study assessing health-related quality of life after opicapone initiation in Parkinson's disease

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EPV-0791 | Optimising DBS for dystonia using local field potentials: LFP-DYT protocol and anticipated early cohort data

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EPV-0792 | Orofacial drinking tremor: A case series and literature review

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EPV-0793 | Outcome of subthalamic stimulation in tremor-dominant Parkinson's disease

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EPV-0794 | Overlapping etiologies in hpca-related dystonic syndrome

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EPV-0795 | Pain in Parkinson's disease

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EPV-0796 | Painful Legs and Moving Toes Syndrome: When treatable factors make the difference

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EPV-0797 | Pallidal bursting correlates with dystonia severity

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EPV-0798 | Paradoxical tremor amplification and impaired arithmetic performance under cognitive load in functional tremor

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EPV-0799 | Parkinson's Disease (PD) and predictors of Driving Cessation: A 7-year longitudinal prospective study

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EPV-0800 | Patient adherence to botulinum toxin therapy in the context of dystonia epidemiology (real-world data from western Moscow)

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EPV-0801 | Patient satisfaction with genetic counseling in GBA1-associated Parkinson's disease

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EPV-0802 | Patient voices driving innovation in drug delivery: How user insights guide improvements to the Crono® LECIG pump

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EPV-0803 | Peripheral lipid and inflammatory markers in Parkinson's disease: Clinical associations of ApoA1 and CRP

A. Mishra; S. Rout; A. Verma; R. Shukla
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EPV-0804 | PGC-1α small molecule activator attenuates dopaminergic degeneration in Parkinson's disease via regulating mitochondrial homeostasis

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EPV-0805 | Population characteristics and motor fluctuation detection in early Parkinson's disease using a wearable sensor

J. Rodriguez Carrillo; M. Ravelo Leon; D. Gomez de la Torre; I. Diaz Diaz; M. Luz Esteve; L. Abad Reguera; A. Hernandez Gonzalez; I. Redondo Robles; G. Carvalho Monteiro; J. Aguilera Aguilera
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EPV-0806 | Posterior tibial nerve stimulation in the management of urinary symptoms in idiopathic Parkinson's disease: The National Hospital of Pikine experience

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EPV-0807 | Precision disease stratification in Parkinson's disease: Fairness-aware imputation and longitudinal subtype discovery

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EPV-0808 | Predicting MSA and PSP based on clinical and neuropsychological scores by employing machine learning

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EPV-0809 | Predictive model of one-year motor outcomes after bilateral STN deep brain stimulation in Parkinson's disease: An internally validated study

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EPV-0810 | Prevalence and clinical features of Jaw Tremor in Parkinson's disease

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EPV-0811 | Prevalence of burning mouth syndrome in Parkinson's disease: A systematic review and meta-analysis

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EPV-0812 | Problematic internet use in Parkinson's disease patients: Risk factors and independent predictors

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EPV-0813 | Prodromal symptoms in multiple system atrophy and Parkinson's disease: A comparative study

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EPV-0814 | Progressive supranuclear palsy mimics-a cohort with redeemable features

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EPV-0815 | Proteomics – Potential biomarker of INPH shunting outcome

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EPV-0816 | Quantitative analysis of reflexive and voluntary saccades in Parkinson disease and multiple system atrophy

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EPV-0817 | SAFIN-DREAM: Real-world effectiveness of safinamide on non-motor symptoms as first add-on therapy to levodopa

F. Salazar Hernández¹; M. Ruiz Perelló¹; B. Gómez Gozávez¹; D. López Segura¹; A. Savolainen¹; M. Lorente¹; A. Sánchez Pedrós¹; B. Palazón Cabanes²; M. Gómez Pacheco¹; D. Vidal Mena¹; J. Bermejillo Barrera¹; A. Báidez Guerrero¹; M. Cerdán Sánchez¹
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EPV-0818 | Satisfaction with and functional impact of subcutaneous Foslevodopa/Foscarbidopa treatment – User perspective

M. Palermo; M. Saianda Duarte; V. Fonseca; J. Neiva Correia; T. Lampreia; J. Vale; R. Simões
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EPV-0819 | SCA3 mitochondrial phenotype is rescued by an antisense oligonucleotide targeting expanded CAG repeats in Ataxin-3 pre-mRNA

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EPV-0820 | Sebum and cerumen analysis for the identification of biomarkers in Parkinson's disease

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EPV-0821 | Sequential use of device-aided therapies in advanced Parkinson's disease: An individualized strategy

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EPV-0822 | Sex differences in the severity of non-motor symptoms in Parkinson's disease: A systematic review and meta-analysis

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EPV-0823 | Simulation of Foslevodopa/Foscarbidopa extra doses to achieve levodopa steady-state exposures following nighttime infusion rate reduction

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EPV-0824 | Skin biomarkers for neurodegenerative disorders

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EPV-0825 | Sleep as a physiological window into cervical dystonia: REM suppression and multidomain PSG abnormalities

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EPV-0826 | Sleep quality in patients – A cross-sectional audit of patients with and without Deep brain stimulation

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EPV-0827 | Sleep quality in patients with Huntington's disease

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EPV-0828 | SMaRT-PD: Developing a scalable and explainable clinical decision support system for Parkinson's disease management

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EPV-0829 | Smartwatch-based assessment of tremor and dyskinesia in Parkinson's disease in the Uzbek population

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EPV-0830 | Spectrum of genetically confirmed ataxias in the Latvian population: A single-center experience

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EPV-0831 | Stroke in Friedreich ataxia: Prevalence and cardioembolic risk beyond CHA2DS2-VA

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EPV-0832 | Subcutaneous levodopa in advanced Parkinson's disease: Is it feasible beyond tertiary centers and 'ideal' patients?

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M. Vargas; A. De Luca; R. Alfonso González; M. Capra; I. Bermejo Casado; L. Caballero Sánchez; J. Berrio; G. Suárez Fernández; A. Castrillo Sanz; A. Mendoza Rodríguez
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EPV-0833 | Sweet spot for pallidal deep brain stimulation in the dystonias

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EPV-0834 | Systematic review of deep brain stimulation effects on non-motor symptoms in Parkinson's disease: STN versus GPi

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EPV-0835 | Thalamic lesion tremor: Comparing a typical and an atypical case

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EPV-0836 | The association of GSTT1 deletion, HindIII C>G PAI-1, and rs11808092 polymorphisms linked to Parkinson's disease vulnerability in Egyptian cohort

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EPV-0837 | The burden of Friedreich ataxia in Brazil: Results of an online survey

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EPV-0838 | The Burden of Friedreich Ataxia in Europe: Results of an online survey

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EPV-0839 | The Burden of Friedreich Ataxia in Italy: Results of an online survey

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EPV-0840 | The clinical and etiological spectrum of Hemiballismus: A case series

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EPV-0841 | The compelling case of Parkinson's disease presenting as pseudo-orthostatic tremor: Valuable insights from the neurophysiology lab

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EPV-0842 | The identification of a single axis to Archimedes spiral drawing and its correlates in terms of tremor's type and severity

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EPV-0843 | The impact of Subthalamic Deep Brain Stimulation on socio-occupational functioning in Parkinson's disease

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EPV-0844 | The influence of anxiety and depression over the sleep disturbances in Parkinson's disease

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EPV-0845 | The non-motor symptom burden as an independent predictor of disease severity in Parkinson's disease: A cross-sectional study from a Moldovan center

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EPV-0846 | The relationship between swallowing function, trunk control, and hand dexterity in patients with Parkinson's disease

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EPV-0847 | The shape of sequence effect in Parkinson's disease

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EPV-0848 | Tips and strategies for subthalamic nucleus aDBS programming in Parkinson's disease

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EPV-0849 | Twelve-year longitudinal assessment of cognitive and motor function in Parkinson's disease versus healthy controls

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EPV-0850 | Ultrasound-guided botulinum toxin injections for upper limb tremor: A portuguese single-center case series

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EPV-0851 | Unilateral spinal-generated pseudo-dystonia due to chronic cervical cord compression: A case report

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EPV-0852 | Unmet needs in movement disorders: Similarities and differences in European neurologists perspectives on essential tremor and Parkinson's disease

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EPV-0853 | Unsupervised phenotyping of beta bursts in Percept RC recordings: A machine learning approach to non-stationary Parkinson's disease biomarkers

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EPV-0854 | Use of “Kneu Health” app for Parkinson's phenotype stratification to improve patients' selection for advanced therapies

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EPV-0855 | Validation of a smart shoe system for remote gait monitoring in people with Parkinson's disease

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EPV-0856 | Vascular risk factors as modifiers of Parkinson's disease progression

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EPV-0857 | Vertebral artery dissection associated with tourette syndrome: Case report

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EPV-0858 | VPS16-associated generalized dystonia misdiagnosed as wilson disease: A diagnostic pitfall

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EPV-0859 | When infection imitates degenerative movement disorders: A diagnostic trap in late neuroborreliosis

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MS and related disorders

EPV-0860 | Correlations between coroid plex volume, EPVS and MRI markers of MS pathology

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G. Corsini¹; M. Aiello²; A. De Rosa³; D. Ranucci¹; V. Nicoletta¹;
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EPV-0861 | “Shrimp Sign”: A peculiar imaging marker with life-threatening impact

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EPV-0862 | A single-centre retrospective review of autologous hematopoietic stem cell transplantation for multiple sclerosis in Singapore

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EPV-0863 | Access to immunomodulatory treatment for patients with multiple sclerosis in the Republic of Moldova: A mixed-methods study

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EPV-0864 | AI-driven MRI measures for predicting NEDA failure in multiple sclerosis

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EPV-0865 | Amantadine for multiple sclerosis-related fatigue: A systematic review and meta-analysis

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EPV-0866 | Analytical comparability of SIMOA and NULISA platforms for CSF GFAP and NFL in treatment-naïve multiple sclerosis

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EPV-0867 | Anti-CD20 treatment induced neutropenia: A retrospective case series

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EPV-0868 | Anticipatory anxiety before follow-up visits in early-stage relapsing-remitting multiple sclerosis: Prevalence, predictors, and clinical implications

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EPV-0869 | Applicability of NfL and GFAP as prognostic biomarkers in multiple sclerosis: A prospective study

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EPV-0870 | Assessing the effects of induced fatigability on cognition and balance performance in Multiple Sclerosis: A multicenter cross-sectional study

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EPV-0871 | Assessment of pain and quality of life in multiple sclerosis, neuromyelitis optica spectrum diseases, and MOG antibody associated disease

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EPV-0872 | Assessment of tolerability of relapse treatment in people with Multiple Sclerosis using oral or intravenous methylprednisolone

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EPV-0873 | Association of psychological resilience, and quality of life with brain and lesion volumes in patients with early diagnosis of multiple sclerosis

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EPV-0874 | Associations between visual evoked potentials and MRI lesion load in relapsing-remitting multiple sclerosis

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EPV-0875 | Autologous haematopoietic stem cell transplantation affects leptomeningeal enhancement in multiple sclerosis: A longitudinal study

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EPV-0876 | Autologous hematopoietic stem cell transplantation versus alemtuzumab in multiple sclerosis: A systematic review and meta-analysis

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EPV-0877 | Blood–cerebrospinal fluid barrier permeability and disability outcomes in multiple sclerosis: A real-world cohort study

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EPV-0878 | Cerebellar lesions and epileptic seizures as unusual manifestations of AQP4-IgG-Positive NMOSD

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EPV-0879 | Challenges and treatment gap of multiple sclerosis in Sudan: A cross-sectional study

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EPV-0880 | Characterizing the natalizumab wearing-off phenomenon: Real-world evidence from a clinically stable multiple sclerosis population

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EPV-0881 | ChatMS study – Awareness and use of Large Language Models among people with multiple sclerosis: a large Italian survey

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EPV-0882 | Chronic pain in multiple sclerosis: Mechanisms, clinical characteristics and treatment strategies

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EPV-0883 | Cladribine as an effective and safe alternative in the treatment of relapsing–remitting multiple sclerosis

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EPV-0884 | Clinical course and multimodal imaging findings in MOG-antibody-associated optic neuritis

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EPV-0885 | Clinical phenotype–based evaluation of serum kappa free light chains in relapsing: Remitting multiple sclerosis

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EPV-0886 | Clinical significance of neurotrophic and inflammatory CSF biomarkers in multiple sclerosis and neuromyelitis optica spectrum disorder

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EPV-0887 | CNS demyelination following adalimumab treatment for hidradenitis suppurativa: A single-center experience

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EPV-0888 | Cognition in multiple sclerosis: Determined by age and education rather than sleep quality or insomnia

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EPV-0889 | Comparative viral antibody signatures under anti-CD20 therapy in MS: Ocrelizumab vs ofatumumab

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EPV-0890 | Comparison of clinical and demographic characteristics of on-treatment MS patients between two MS center in Denmark and Romania

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EPV-0891 | Comparison of fatigue and restless legs syndrome severity across NMOSD, MOGAD, and multiple sclerosis

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EPV-0892 | Comparison of STRATIFY JCV and IMMUNOWELL JCV assays for anti-JCV antibody detection: A multicenter study across Tuscan neurology centers

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EPV-0893 | Contribution of thalamus-cortex network and cortical habituation mechanisms in multiple sclerosis

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EPV-0894 | Cryoneurolysis of the tibial nerve as a treatment for spasticity in patients with multiple sclerosis

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EPV-0895 | De-escalation of high efficacy disease-modifying therapies in multiple sclerosis: A systemic review and meta-analysis assessing relapse rate

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EPV-0896 | Determinants of resilience in Tunisian patients with multiple sclerosis

M. Becha; A. Souissi; N. Msadek; A. Gharbi; Y. Abida; A. Atrous; I. Kacem; A. Gargouri; R. Gouider
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EPV-0897 | Development of pulmonary sarcoidosis in a patient with multiple sclerosis on sphingosine-1-phosphate modulator. A case report

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EPV-0898 | Differentially expressed miRNAs in Multiple Sclerosis patients stratified for NEDA status

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EPV-0899 | Do obesity related hormones play a role in multiple sclerosis?

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EPV-0900 | Early functional, cognitive, and neuropsychiatric outcomes in newly diagnosed multiple sclerosis: A two-year longitudinal study

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EPV-0901 | Effect of ofatumumab on brain atrophy and its association with clinical outcomes in relapsing-remitting multiple sclerosis

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EPV-0902 | Effectiveness and safety of ofatumumab treatment in RRMS patients – One site RWE experience study

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EPV-0903 | Effectiveness of a dance-based intervention on motor outcomes in young adults with multiple sclerosis: Results of the ESPRIMO study

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EPV-0904 | Effects of fingolimod exposure on birth weight and prematurity in Multiple Sclerosis

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EPV-0905 | Effects of oral disease-modifying therapies on lymphocyte counts in relapsing-remitting multiple sclerosis: A real-world experience

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EPV-0906 | Efficacy, safety, and neurofilaments evaluation in secondary progressive multiple sclerosis patients treated with siponimod

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EPV-0907 | EPVS in mid brain in multiple sclerosis correlation with cerebellar and brainstem atrophy and clinical disability

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EPV-0908 | Erythrocyte morphological alterations in multiple sclerosis assessed by white-light diffraction phase microscopy

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EPV-0909 | Dissociating cognitive and physical fatigue in multiple sclerosis: A differential roles of psychophysiological vs. neurological-disease related factors

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EPV-0910 | Evaluation of undetectable K-Fle index in suspected multiple sclerosis

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EPV-0911 | Exploring the effect of cerebrospinal fluid cytokine levels on MRI biomarkers of inflammation and neurodegeneration in multiple sclerosis

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EPV-0912 | Fatigue and depression in multiple sclerosis: A bidirectional relationship

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EPV-0913 | Fatigue and disability drive sexual dysfunction in multiple sclerosis: a cross-sectional study in Spain using the MSISQ-15

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EPV-0914 | Fatigue severity in multiple sclerosis: preliminary results of a cross-sectional study

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EPV-0915 | Fatigue, depression and anxiety as determinants of physical and mental quality of life in MS types

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EPV-0916 | Feasibility and neurological safety of a ketogenic dietary intervention in relapsing-remitting multiple sclerosis: A pilot study

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EPV-0917 | Features of cognitive and neuropsychological disorders in multiple sclerosis

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EPV-0918 | Frailty in elderly patients with Multiple Sclerosis (MS)

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EPV-0919 | From biomarker to a new clinical perspective: Kappa free light chain index and disability progression in multiple sclerosis

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EPV-0920 | Functional outcomes after immune reconstitution therapy: A multidimensional evaluation of multiple sclerosis patients treated with cladribine

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EPV-0921 | Gastric diffuse large B-cell lymphoma in a multiple sclerosis patient undergoing treatment with natalizumab: A case-report and literature review

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EPV-0922 | Gastrointestinal symptoms and health-related quality of life in relapsing-remitting multiple sclerosis: A GSRS and SF-36 based study

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EPV-0923 | Hemifacial spasm as the initial manifestation of multiple sclerosis: a diagnostic pitfall

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EPV-0924 | Hidden disabilities in multiple sclerosis: Incontinence and sexual dysfunction

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EPV-0925 | How will climate change impact severity and incidence of multiple sclerosis?

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EPV-0926 | Hyper-reflecting foci in multiple sclerosis: Association with clinical, optical coherence tomography, and laboratory markers

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EPV-0927 | Immunobiological and gender-specific features of multiple sclerosis in patients of Uzbek ethnicity

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EPV-0928 | Immunoglobulin profiles in multiple sclerosis: A real-world comparison of ofatumumab and ocrelizumab

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EPV-0929 | Immunological and hormonal shifts in pregnant women with multiple sclerosis and their impact on pregnancy outcomes

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EPV-0930 | Impact of Aquaporin-4 serostatus on disease activity and disability in NMOSD: A five-year comparative study

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EPV-0931 | Increasing age is associated with lesser decreases in neurofilament levels under disease-modifying treatments for MS

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EPV-0932 | Investigation of functional capacity, reaction time, and cognitive functions in individuals with multiple sclerosis

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EPV-0933 | Is it safe to stop? A systematic review and meta-analysis of discontinuation versus continuation of disease-modifying therapies in multiple sclerosis

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EPV-0934 | Kappa free light chains – A quantitative alternative to oligoclonal bands in multiple sclerosis

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EPV-0935 | Kappa-free light chain in predicting long term MS disability progression

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EPV-0936 | Ketogenic diet and fatigue in relapsing-remitting multiple sclerosis: exploring a neuro-metabolic signal in a pilot study

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EPV-0937 | Life-threatening hepatitis requiring liver transplantation in a patient with multiple sclerosis treated with ocrelizumab

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EPV-0938 | Linking corpus callosum diffusion tensor imaging biomarkers to MSFC and cognitive–motor dual-task costs in RRMS

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EPV-0939 | Management of multiple sclerosis-related fatigue: An umbrella review of systematic reviews with meta-analyses

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EPV-0940 | Metabolomic alterations in central and peripheral nervous system demyelinating diseases

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EPV-0941 | Migraine and tension-type headache in multiple sclerosis: A two-year, prospective, longitudinal, controlled study

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EPV-0942 | Motivations and treatment shifts in older multiple sclerosis patients: Understanding treatment changes after age 50

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EPV-0943 | Motor–cognitive phenotypes in early multiple sclerosis: Insights from a multidimensional assessment

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EPV-0944 | Multiparametric assessment of MS at diagnosis: is kappa free light chain index associated with clinical, radiological, and retinal findings?

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EPV-0945 | Multiple sclerosis phenotype transition patterns

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EPV-0946 | Multiple sclerosis relapse presenting with broken heart syndrome – A case report

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EPV-0947 | Multiple sclerosis-related uveitis and its prognostic value

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EPV-0948 | Nabiximols improves sleep architecture in multiple sclerosis: A prospective polysomnographic study

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EPV-0949 | Neurofilament light as a biomarker for stratifying treatment intensity in newly diagnosed multiple sclerosis

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EPV-0950 | Objective assessment of fatigue in relapsing–remitting multiple sclerosis

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EPV-0951 | Ocrelizumab effect on progression: real-world data in large cohort of multiple sclerosis patients

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EPV-0952 | OCT measures at multiple sclerosis onset and early therapeutic management: A retrospective, real-world study

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EPV-0953 | Ofatumumab efficacy, safety, adherence, and retention in Cypriot Multiple Sclerosis patients

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EPV-0954 | Participation and perceived benefits of physical activity in multiple sclerosis over a decade

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EPV-0955 | Parvovirus B19 infection associated with ocrelizumab in a patient with multiple sclerosis: A case report

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EPV-0956 | Passive smartphone usage data as a digital biomarker for cognitive impairment in multiple sclerosis

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EPV-0957 | Patient-reported outcomes in Serbian ocrelizumab-treated relapsing multiple sclerosis patients with advanced disability: The MuSicalE Interim Analysis

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EPV-0958 | Patient-reported wearing-off symptoms in Ocrelizumab-treated multiple sclerosis and their impact on quality of life

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EPV-0959 | Physical disability predicts multidomain cognitive dysfunction in multiple sclerosis

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EPV-0960 | Plasma biomarkers as early indicators of cognitive and motor impairment in Multiple Sclerosis: A prospective observational study

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EPV-0961 | Pre-diagnostic fatigue and fatigue burden at diagnosis in multiple sclerosis

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EPV-0962 | Predictors of treatment failure following immune reconstitution therapies in relapsing multiple sclerosis: A real-world cohort

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EPV-0963 | Predominant verbal fluency deficit in multiple sclerosis

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EPV-0964 | Pregnancy and clinical outcomes in women with multiple sclerosis: data from the Republic of Moldova

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EPV-0965 | Preserving professional autonomy in multiple sclerosis: A multimodal management of lower urinary tract dysfunction

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EPV-0966 | Prevalence and treatment efficacy of trigeminal neuralgia in patients with multiple sclerosis

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EPV-0967 | Prevalence of Epstein–Barr virus infection in inflammatory diseases of the central nervous system

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EPV-0968 | Psychiatric disorders preceding the diagnosis of multiple sclerosis: A case series

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EPV-0969 | Quality of reporting randomized controlled trials in exercise approaches for multiple sclerosis: A CONSORT-based assessment (up to 2025)

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EPV-0970 | Radiologically isolated syndrome: New challenges for prognosis and management

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EPV-0971 | Real-world treatment satisfaction in multiple sclerosis: Digital patient reported outcomes comparison between intravenous and subcutaneous anti-CD20

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EPV-0972 | Real-world use of Ublituximab: Patient profile in clinical practice

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EPV-0973 | Retrospective analysis of lymphocyte dynamics in multiple sclerosis patients treated with fingolimod

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EPV-0974 | Revisiting corpus callosum index data after 20years is it sensitive enough to predict evolution?

G. Medeiros Andrade Figueira; S. Paula Vallegas Soares; S. Raquel Custodio; C. Viviane Tavares Carvalho; V. Alexandra Seide Cardoso; M. Nunes Ferreirinha Leite de Castro; F. Tome do Nascimento; F. Faria Andrade Figueira

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EPV-0975 | Rituximab as a single therapeutic strategy in coexisting takayasu arteritis and multiple sclerosis: A case report

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EPV-0976 | Screening chronic fatigue in MS with MFIS: Clinical correlates in a real-world cohort

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EPV-0977 | Sepsis mortality in multiple sclerosis: Trends and disparities using CDC WONDER

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EPV-0978 | Serum calprotectin as a biomarker of inflammatory activity and treatment response in relapsing multiple sclerosis

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EPV-0979 | Serum glial fibrillary acidic protein and neurofilament light chain as biomarkers of progression and cognitive decline in multiple sclerosis

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EPV-0980 | Serum neurofilament light chains and serum glial fibrillary acidic protein in relation to cognitive performance in multiple sclerosis

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EPV-0981 | Sexual dysfunction and related factors in multiple sclerosis patients

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EPV-0982 | Silent rebound: Tumefactive demyelinating lesion following the discontinuation of teriflunomide

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EPV-0983 | Sleep disorders and quality of life in multiple sclerosis

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EPV-0984 | SmartBioMS: Smartphone application for investigating speech as a passive biomarker to track progression in Multiple Sclerosis

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EPV-0985 | Social cognition and cognitive impairment across RRMS and SPMS

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EPV-0986 | Social cognition: A significant modifier of Quality of Life in MS

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EPV-0987 | Statistical properties of tract density for MS, NMOSD and MOGAD

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EPV-0988 | Statistical properties of tract density in corpus callosum for MS, NMOSD and MOGAD patients

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EPV-0989 | Supportive interventions to reduce caregiver burden among family caregivers of Multiple Sclerosis (MS) patients: A systematic review and meta-analysis

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EPV-0990 | The evolving burden of multiple sclerosis and migraine in G20 countries: A geospatial and socioeconomic analysis from 1990 to 2023

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EPV-0991 | The rising role of cyberchondria and ehealth literacy in multiple sclerosis: Psychological reflections of online health information seeking

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EPV-0992 | Theory of mind and empathy deficits are associated with verbal and executive dysfunction in relapsing–remitting multiple sclerosis

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EPV-0993 | Therapeutic adherence in multiple sclerosis: The role of patient self-management behaviors

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EPV-0994 | Time to disability milestones in NMOSD and MOGAD: A study of a Tunisian cohort

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EPV-0995 | Timing of high-dose corticosteroids and motor recovery after immune-mediated myelitis: a Girona neuroimmunology cohort

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EPV-0996 | Transverse myelitis potentially linked to undifferentiated connective tissue disease: Clinical clues from a single case

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EPV-0997 | Tuberculous longitudinally extensive transverse myelitis in an elderly patient: A diagnostic and therapeutic challenge

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EPV-0998 | Tumefactive multiple sclerosis: A single-centre experience

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EPV-0999 | Understanding real-world engagement with a patient monitoring app: Insights from the Remote Multiple Sclerosis Care in Antwerp (ReMSCA) study

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EPV-1000 | Unplanned pregnancy during ponesimod therapy in RRMS: Clinical course and outcome

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EPV-1001 | Using AI chatbots as a source of information for patients with multiple sclerosis

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EPV-1002 | Video-oculography markers of subclinical brainstem involvement in multiple sclerosis

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K. Grammatchikova; A. Abramova; D. Ganin; A. Broutian;
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EPV-1003 | Viral antibody profiles in multiple sclerosis and healthy controls: a cross-sectional study

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EPV-1004 | When treating skin uncovers the brain: A case of multiple sclerosis in a patient on dupilumab for atopic dermatitis

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Muscle and neuromuscular junction disorder

EPV-1005 | A phase 3 study to evaluate the efficacy and safety of Telitacicept in patients with generalized myasthenia gravis: Trial design update

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EPV-1006 | Association of complement deposition with disease severity and target treatment in dysferlinopathy

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EPV-1007 | CAPTIVATE trial design: A pivotal phase 3 study of claseprubart in chronic inflammatory demyelinating polyneuropathy

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EPV-1008 | Cardiovascular burden among patients with myasthenia gravis in France: A retrospective study using the french national insurance database

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EPV-1009 | Clinical characteristics, outcomes and comparison of conventional and newer therapies in myasthenia gravis

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EPV-1010 | Clinical, serological and therapeutic characteristics of immune-mediated necrotizing myopathy: A case series

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EPV-1011 | Combined central and peripheral demyelination: Incidental findings or a single disease entity?

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EPV-1012 | Comparative efficacy of one cycle of efgartigimod versus standard of care in generalized myasthenia gravis

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EPV-1013 | Content validity of the Myasthenia Gravis Impairment Index (MGII) patient-reported ocular score in patients with ocular myasthenia gravis

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EPV-1014 | Diagnostic value of muscle ultrasound as screening tool for congenital myopathies

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EPV-1015 | Early cerebrospinal fluid cytokine changes after nusinersen loading phase in adult spinal muscular atrophy

B. Gębka- Kępińska; D. Gębka; J. Majchrzak; M. Cwynar; J. Jurkiewicz; B. Adamczyk; N. Morawiec; M. Lis; N. Niedziela; J. Bączyk; M. Adamczyk-Sowa
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EPV-1016 | Eculizumab enables rapid remission and successful embryo transfer in refractory generalized myasthenia gravis: A case report

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EPV-1017 | Effect of nipocalimab on sustained myasthenia gravis control during infections: Post-hoc analysis of the vivacity-MG3 study

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EPV-1018 | Effectiveness and tolerability of rozanolixizumab in generalized myasthenia gravis: A single-center retrospective analysis of 18 cases

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EPV-1019 | Efficacy and safety of eculizumab versus intravenous immunoglobulin in the acute exacerbation of myasthenia gravis

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EPV-1020 | Abstract withdrawn

EPV-1021 | Efficacy and safety of efgartigimod versus intravenous immunoglobulin in moderate to severe generalized myasthenia gravis

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EPV-1022 | Efficacy of nipocalimab in adult patients with none-to-mild ocular manifestations of generalized myasthenia gravis in Phase 3 VIVACITY-MG3

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EPV-1023 | Emergency use of eculizumab in impending and manifest myasthenic crisis: A retrospective case series

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EPV-1024 | Evaluation of decrement patterns in distal and proximal muscles using repetitive nerve stimulation in myasthenia gravis

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EPV-1025 | Experiences with Myotonic Dystrophy Type 1 (DM1) and treatment with Del-desiran: Interviews with participants in MARINA-OLE™ and their caregivers

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EPV-1026 | Generalized but not ocular weakness predicts quality of life in myasthenia gravis: A factor-analytic and regression study

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EPV-1027 | Head drop in myasthenia gravis – How does it influence patients' quality of life and acceptable symptoms state (PASS)?

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EPV-1028 | Hypokalaemic periodic paralysis: Don't miss thyrotoxicosis

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EPV-1029 | Impact of efgartigimod treatment on patients with generalized myasthenia gravis with distinct antibody profiles in a single-center study

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EPV-1030 | Impact of high-dose methylprednisolone pulse therapy for ocular myasthenia gravis in a single-center study

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EPV-1031 | Impact of published single-fiber EMG reference values on diagnostic classification in myasthenia gravis

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EPV-1032 | Indirect comparison of nipocalimab safety versus FcRn blockers, C5 complement inhibitors, rituximab and IVIg in generalized myasthenia gravis

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EPV-1033 | Late-onset multiple Acyl-CoA dehydrogenase deficiency: Clinical features, diagnostic challenges, and the role of oxidative stress in pathophysiology

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EPV-1034 | Long-term clinical and functional outcomes in 5q spinal muscular atrophy of nusinersen-treated patients (AME-NUSILONG)

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EPV-1035 | Long-term user assessment of ME&MGTM, an app tracking generalized Myasthenia Gravis manifestations

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EPV-1036 | ME&MGTM digital biomarkers to monitor generalized Myasthenia Gravis symptoms: DOMYA, a validation study

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EPV-1037 | Metabolic myopathy due to carnitine palmitoyltransferase II (CPT II) deficiency: A case report

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EPV-1038 | Mild clinical presentation of anti-SRP immune-mediated necrotizing myopathy: A case report

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EPV-1039 | Muscle anoctaminopathies: Clinical and genetic spectrum in a case series

N. Marcos; A. Jauregi; A. González; M. Callejo; A. Lagüela; V. Anciones; Á. López; M. Martínez; L. Pastor; J. Mellada; F. Angoso; A. Rodríguez-Antigüedad
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EPV-1040 | Myasthenia gravis and pregnancy: A retrospective analysis from a large tertiary care centre in Russia

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EPV-1041 | Neurophysiological assessment of the hypoglossal nerve in oculopharyngeal muscular dystrophy

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EPV-1042 | Ocular-onset myasthenia gravis in a real-world Italian referral center: Clinical characteristics and risk of generalization

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EPV-1043 | Passive and active digital biomarkers for comprehensive monitoring of generalized myasthenia Gravis: Design of the HARMONY 360 study

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EPV-1044 | Persistent muscle damage after statin discontinuation: A retrospective series of 78 patients submitted to muscle biopsy

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EPV-1045 | Phenotype, serum biomarkers, and quantitative muscle MRI in patients with mitochondrial myopathy due to single large-scale mtDNA deletions

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EPV-1046 | Portuguese Myasthenia gravis epidemiological and clinical study: Preliminary data

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EPV-1047 | Possible impact of very late onset disease on clinical presentation and myasthenic crisis in myasthenia gravis

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EPV-1048 | Potential mechanistic advantages of aC1s targeting in myasthenia gravis: Upstream versus downstream complement blockade

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EPV-1049 | Predictors of conversion of myasthenic crisis from impending myasthenic crisis

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EPV-1050 | Pregnancy enhanced tracking with neonatal and infant assessment (PETUNIA): Design of a safety study for nipocalimab in generalized myasthenia gravis

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EPV-1051 | Quality of life outcomes in triple-seronegative and MuSK antibody-positive myasthenia gravis patients: Evidence from MyRealWorld-MG

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EPV-1052 | Real word data on alglucosidase – Cipaglucosidase/miglustat switching in LOPD: an Italian multiCenter experience

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EPV-1053 | Real-world analysis of healthcare resource utilization, costs, and work loss in patients in the united states initiating therapy for myasthenia gravis

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EPV-1054 | Real-world evidence on the timing of immunosuppressive therapy and post-thymectomy outcomes in myasthenia gravis: A multicenter retrospective study

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EPV-1055 | Relationship between peripheral nervous system damage and fatigue in immune checkpoint inhibitors

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EPV-1056 | Rethinking myasthenia gravis care through brain health lens: Systematic review on socio-economic burden, patient pathways, digital tools adoption

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EPV-1057 | Retrospective analysis of the association between extrathymic neoplasms and Myasthenia Gravis in a patient cohort

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EPV-1058 | Semi-quantitative analyses of muscle magnetic resonance imaging for pattern recognition in early idiopathic inflammatory myopathies

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EPV-1059 | Short-term comparison of efgartigimod vs. eculizumab in patients with myasthenia gravis: a dual-center real-world study

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EPV-1060 | Sleep disturbances in pediatric duchenne muscular dystrophy: Insights from the sleep disturbance scale for children

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EPV-1061 | Sleep quality, depression, and functional status in adult patients with spinal muscular atrophy

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EPV-1062 | Spectrum of orthopedic complications in children with autosomal recessive limb-girdle muscular dystrophy: A tunisian cohort study

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EPV-1063 | Successful treatment of Glycogenosis Type V (McArdle's disease) with high-dose vitamin B6

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EPV-1064 | Sustained clinical benefit with limited retreatment: Real-world experience with efgartigimod inachr-positive generalized myasthenia gravis

G. D'Alvano; F. D'Anna; D. Marigliano; F. Capuozzo; R. Sansone; V. Todisco; A. Tessitore; A. Biseco
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EPV-1065 | Switching from alglucosidase alfa to avalglucosidase: Real-world data from a single-center experience

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EPV-1066 | The role of the QMG score in determining the therapeutic approach in patients with myasthenia gravis – a prospective clinical study

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EPV-1067 | The role of vital capacity (VC) in myasthenia gravis (MG) patients without clinical signs (CS) of respiratory dysfunction (RD)

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EPV-1068 | The story behind a foot drop: When EMG requires a second reading

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EPV-1069 | The true chameleon: A single center experience on Congenital Myasthenic Syndromes

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EPV-1070 | Treatment with anti-FcRn and C5-inhibitors in drug-resistant gMG-real world data

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EPV-1071 | Variable phenotypic expression associated with a recurrent ANO5 variant: A clinical, MRI and muscle biopsy case series

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EPV-1072 | Wearable-based activity markers in Myasthenia Gravis

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EPV-1073 | Where do patients with myasthenia gravis die? A retrospective study

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Neurocritical care

EPV-1074 | A study of serum fibrinogen level in acute ischemic stroke patients in critical care unit

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EPV-1075 | Beyond seizure detection: ICU based cEEG-guided management of NORSE targeting Ictal-Interictal Continuum and BIRDs – A case series

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EPV-1076 | Cerebral edema in ICU patients: Etiological spectrum and mortality predictors

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EPV-1077 | Cerebral EEG activity response to mannitol in acute ischemic malignant stroke

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EPV-1078 | Comparative outcomes of toxic versus metabolic encephalopathy in critically ill adults: A MIMIC-IV analysis

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EPV-1079 | Delayed anoxic leukoencephalopathy: When recovery isn't the end

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EPV-1080 | Determinants of mortality and functional outcomes in neurological intensive care unit patients

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EPV-1081 | Evaluation of nasogastric tube placement via X-ray in neurological intensive care patients

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EPV-1082 | Improving diagnostic accuracy and rehabilitation outcomes in transient ischemic attack and ischemic stroke using the NIRSIT device

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EPV-1083 | Intrathecal dexamethasone for FIRES: Therapeutic experience and review of current evidence

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EPV-1084 | Non-convulsive status epilepticus in the neurological intensive care unit: Electro-clinical outcome predictors

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EPV-1085 | Procalcitonin and complications in non-aneurysmal subarachnoid hemorrhage: A single-centre cohort study

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EPV-1086 | Prognostic value of routinely assessed cerebrospinal fluid parameters in spontaneous subarachnoid hemorrhage

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EPV-1087 | Validation of entrustable professional activities assessment for neurocritical care advanced practice providers via simulation

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Neuroepidemiology

EPV-1088 | Analysis of cerebrovascular disease incidence and seasonal trends: A 10-year nationwide study in Georgia (2014–2023)

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EPV-1089 | Association of occupational factors and environmental tobacco smoke with risk of Alzheimer's disease

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EPV-1090 | Case-mix, referral drivers, and admissions in a rapid-access neurology clinic: A 12-month evaluation from a middle-income setting

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EPV-1091 | Clinical and etiological predictors of stroke severity in young adults with ischemic stroke

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EPV-1092 | Comparative epidemiology of optic neuritis before and after the COVID-19 pandemic: A comprehensive literature review

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EPV-1093 | Diagnostic value of nerve growth factor in cognitive impairment in generalized epilepsy

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EPV-1094 | Estimating the prevalence of mild behavioral impairment in a population-based greek cohort: Findings from the HELIAD study

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EPV-1095 | Impact of war-related stress on multiple sclerosis relapse in iranian patients: Lessons from a recent war in the Middle East

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EPV-1096 | Incidence, prevalence, and disability associated with neurological disorders in Jordan between 1990 and 2023: An analysis based on the GBD Study 2023

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EPV-1097 | Lifestyle and comorbidities for cerebrovascular risk in adults 40–65, Eastern Poland

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EPV-1098 | Long-term trends in idiopathic epilepsy in India: Evidence from the global burden of disease study, 1990–2023

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EPV-1099 | Paternal perinatal depression in Pakistan: A mixed-methods systematic review

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EPV-1100 | Phenotype distribution and differential disease burden in multiple sclerosis: Real-world data from the apolo study

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EPV-1101 | Post-COVID changes in thyroid cancer incidence: Possible neuroendocrine and ACE2-mediated mechanisms

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EPV-1102 | Serum S100B protein levels in neurodegenerative diseases

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EPV-1103 | Sex differences in ischemic stroke among young adults: A North African cohort study

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EPV-1104 | Sex-specific trends in multiple sclerosis burden in India, 1990–2023: A GBD 2023 analysis

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EPV-1105 | Sociodemographic and clinical profile of an outpatient population with functional neurological disorder over a decade

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EPV-1106 | Superficial siderosis of the central nervous system: An underdiagnosed cause of hearing loss and progressive ataxia

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EPV-1107 | The impact of psychological stress on epileptic seizure frequency during wartime: A study following the “Swords of Iron” War in October 2023

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EPV-1108 | A 62-year-old woman with hyperorality and apathy drug resistant, A new variant of CHMP2B mutation

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EPV-1109 | A novel mutation in the RNF216 gene associated with gordon holmes syndrome: Two Turkish sibling cases

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EPV-1110 | A recurrent pathogenic MTM1 variant manifesting as hereditary spastic paraplegia (HSP) expands the phenotypic spectrum of X-linked myotubular myopathy

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EPV-1111 | Accelerated brain aging and anatomical substrates in spinocerebellar ataxia type 3

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EPV-1112 | Adult-onset congenital myasthenic syndrome: Genetic variability and clinical phenotype

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EPV-1113 | Application of genetic tests in clinical practice: challenges in dealing with atypical phenotypes in neurodegenerative diseases

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EPV-1114 | Association study between polymorphisms of the PTH gene and the morphology of the Sella Turcica

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EPV-1115 | Atypical presentation of spinocerebellar Ataxia 34 associated with a Novel Elov14 mutation

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EPV-1116 | Autosomal dominant cerebellar ataxia in siblings with a shared PDYN frameshift variant

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EPV-1117 | Beyond ALS genes: Identification of causative variants associated with neurodegenerative disorders in Greek ALS patients

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EPV-1118 | CADASIL atypical motor presentation

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EPV-1119 | Challenges in diagnosing pediatric neurodegeneration with brain iron accumulation: A case report

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EPV-1120 | Clinical and genetic characterization of a Portuguese cohort with familial multiple cavernous malformations

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EPV-1121 | Decoding Parkinson's genetics: Causal mutations, risk factors, and clinical phenotypes

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EPV-1122 | Defining a rare mtDNA phenotype: MELAS-like presentation and status epilepticus linked to MT-TT m.15933G>A

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EPV-1123 | Determination of genotype-phenotype characteristics in 194 cases with hereditary ataxia

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EPV-1124 | Early-onset parkinsonism associated with intermediate CAG/CAA expansion in the TBP gene

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EPV-1125 | Epileptic and developmental encephalopathy associated with wdr45 mutation

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EPV-1126 | Evaluation of inflammatory and neurotrophic gene expression profiles in the epileptogenesis process

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EPV-1127 | Exploring the clinical spectrum of LRSAM1-related disorder: A Greek case series

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EPV-1128 | Fabry's disease: Rare symptom in a rare disease

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EPV-1129 | Fragile X-associated tremor/ataxia syndrome (FXTAS) with mosaicism in genetic testing and different clinical course in two female siblings

E. Zeis; P. Kalligerou; A. Daskalaki; C. Koros; V. Konstantinidis; N. Skarmas; S. Papageorgiou
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EPV-1130 | Genetic evaluation of hereditary ataxia patients in Türkiye using targeted next-generation sequencing: The impact of consanguinity

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EPV-1131 | Genetic risk factors in neurodegenerative parkinsonism

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EPV-1132 | Genetic spectrum and phenotypic heterogeneity in Parkinson's disease patients undergoing whole-exome sequencing in a tertiary referral center

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EPV-1133 | Hemiplegic migraine with epileptic seizures and reversible cortical abnormalities in a male adolescent: A case report

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EPV-1134 | Hypercapnic respiratory failure in familial cavernous cavernoma: Neurogenetic diagnostic criteria and significance of lesion progression – A rare case

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EPV-1135 | Identification of genetic modifiers in adult Moyamoya disease using whole-exome sequencing

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EPV-1136 | Inflammation-related genetic variants and copy number changes in Alzheimer's disease in a Saudi cohort

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EPV-1137 | Late presentation of megalencephalic leukoencephalopathy with subcortical cysts (MLC) in a female patient with ataxia, dystonia and seizure

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EPV-1138 | Machine learning applications in Huntington's disease genomics: A systematic review

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EPV-1139 | Measuring functional abilities and assistance level in niemann-pick disease type C, GM2 gangliosidosis, and ataxia

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EPV-1140 | Mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes: A case series

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A. Rachdi; Z. Saied; F. Nebli; S. Ben Sassi

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EPV-1141 | Molecular diagnosis of trinucleotide repeat expansion disorders in Singapore (1996–2024)

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EPV-1142 | Neuromuscular manifestations in patients with lipodystrophy

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EPV-1143 | Peak expiratory flow causally affects brain neuronal activity: A Mendelian randomization study

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EPV-1144 | Persistent sleep disturbance in youth: A clue to Wilson disease

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EPV-1145 | Progressive external ophthalmoplegia due to a POLG gene mutation: A case report

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EPV-1146 | Progressive spastic paraparesis misdiagnosed as cerebral palsy: a case of ARSACS

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EPV-1147 | Pseudodemyelinating MRI changes in neurofibromatosis type 1: Clinical significance of UBO/FASI

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EPV-1148 | Rare case of neuromuscular oculauditory syndrome: Genetic challenges

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EPV-1149 | RORA-Related IDDECA with cerebellar atrophy: Marked clinical response to valproate in an adult patient

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EPV-1150 | Transcranial alternating current stimulation for treatment of multiple system atrophy cerebellar type: A randomized controlled trial

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EPV-1151 | VPS16-dystonia linked pathogenic variant in Parkinson's disease: Expanding the phenotype?

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EPV-1152 | AI in healthcare: The potential of ChatGPT as an assisting tool in diagnosing stroke

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EPV-1153 | Beyond the classical triad: Cerebral and spinal cord involvement in Kearns-Sayre syndrome

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EPV-1154 | Changes in brain morphometry in post COVID-19 patients: An exploratory study

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EPV-1155 | CHANTER syndrome: An emerging diagnosis in toxic encephalopathy

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EPV-1156 | Characterizing the cellular distribution of iron in Parkinson's disease from MRI data

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EPV-1157 | Clinical and radiological features of autoimmune encephalitis: A monocentric study from Tunisia

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EPV-1158 | Clinical utility of DaTscan in detecting neurodegenerative causes of Parkinsonism

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EPV-1159 | Correlation between FDG and amyloid-beta PET uptake in patients with neurodegenerative diseases

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EPV-1160 | Deep learning-based synthesis of [11C]PiB MicroPET images from [18F]FDF in an Alzheimer's disease animal model

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EPV-1161 | Diencephalo-mesencephalic Junction Dysplasia Type B: Diagnostic challenges and clinico-imaging correlations

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EPV-1162 | Differentiating neurovascular conflict in classical trigeminal neuralgia: A 15-year study on the use of multispiral computed tomographic angiography

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EPV-1163 | Distinct cerebral perfusion patterns in drug-induced parkinsonism versus Parkinson's disease: A dual-phase 18F-FP-CIT PET analysis

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EPV-1164 | Enlarged perivascular spaces alongside white matter hyperintensities predict poor MoCA outcomes among elderly individuals

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EPV-1165 | Evaluation of the correlation of neuromelanin loss in the Substantia Nigra on MRI

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EPV-1166 | Fahr disease diagnosed in an elderly patient: A puzzling clinical case

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EPV-1167 | FDG-PET in Creutzfeldt-Jakob disease presenting as corticobasal syndrome

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EPV-1168 | Hypertrophic olivary degeneration secondary to venous infarction: a previously unreported presentation

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Gutiérrez; L. Domínguez Jiménez; Á. Lafuente Almadén;

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EPV-1169 | Hypomagnesemia-associated cerebellar syndrome: A reversible cause of acute ataxia

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EPV-1170 | Improving diagnostic sensitivity for temporal lobe epilepsy by using deep learning-based MRI reconstruction

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EPV-1171 | Inflammatory cerebral amyloid angiopathy: Report of two clinical cases

M. Ruiz Perelló; F. Salazar Hernandez; B. Gómez González;

D. Lopez Segura; A. Maija Savolainenhg; M. Lorente;

A. Sancho; A. Gomez Esteban; D. Vidal Mena; M. Lopez Lopez;

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EPV-1172 | Intermittent diplopia revealing an aneurysmal persistent trigeminal artery

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EPV-1173 | Intravenous thrombolysis in wake-up and unknown-onset ischemic stroke selected by CT-based imaging: A systematic review and meta-analysis

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EPV-1174 | Intravenous thrombolysis in wake-up and unknown-onset ischemic stroke selected by MRI-based imaging: A systematic review and meta-analysis

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EPV-1175 | Longitudinal neuroimaging evidence of cognitive changes in essential tremor

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EPV-1176 | Neuroimaging spectrum of central nervous system tuberculosis – A retrospective cohort study

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EPV-1177 | Not everything is as it seems: Pseudo–midbrain atrophy on neuroimaging

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EPV-1178 | Persistent trigeminal artery causing transient oculomotor nerve palsy: A neuroimaging case

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EPV-1179 | Preserved global topology with frontoparietal control–centered cross-network coupling alterations after carbon monoxide poisoning

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EPV-1180 | pyogenic cerebellar abscesses leading to ataxia and intractable hiccups

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EPV-1181 | Radscale score and clinical response to tap-test in patients with normal pressure hydrocephalus

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EPV-1182 | Reversible splenial lesion syndrome after fentanyl exposure: Expanding the etiological spectrum

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EPV-1183 | Safety and feasibility of non-contrast follow-up magnetic resonance imaging in clinically stable multiple sclerosis patients

J. García Granado¹; E. Santana Suárez²; M. Pérez García³; J. de la Nuez González¹; M. Pérez Vieitez¹; M. Quintana García²; V. Serrano Romero-De Ávila²; Á. Hernández Pérez²; M. Genescà Gómez²; A. Peñate Medina²; R. García García²; V. González Pérez²; S. Dorta Gámez²; L. Ramos Gil²; F. Cabrera Naranjo¹; A. González Hernández¹

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EPV-1184 | Sensory-onset multiple sclerosis with posterior column lesions: Multivariate predictors of long-term disability

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EPV-1185 | Simulation-assisted trans-interpeduncular approach for resection of a midline mesencephalic cavernous malformation

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EPV-1186 | Subclavian steal due to fibromuscular dysplasia: A rare neurovascular challenge

D. Nicula; A. Pavel; A. Chesnoiu; L. Marinescu; S. Stefan; A. Ribigan; F. Antochi; C. Tiu
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EPV-1187 | Subtentorial Subdural Empyema: A case report

I. Bermejo Casado; M. Vargas Cobos; A. De Luca; R. Alfonso González; M. Capra; C. Gómez López de San Román; L. Caballero Sánchez; D. Cerdán Santacruz; A. Castrillo Sanz; A. Mendoza Rodriguez
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EPV-1188 | The bloomy rind sign: The first case of ovarian cancer-associated leptomeningeal carcinomatosis with bloomy rind sign

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EPV-1189 | The Medusa's spasm

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EPV-1190 | The use of dual-energy computed tomography in ischemic stroke patients after endovascular thrombectomy

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EPV-1191 | Two cases of diplopia caused by arteriovenous fistulas and a short literature review

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EPV-1192 | When brain calcifications meet cancer: Glioblastoma in Fahr's disease

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Neuroimmunology

EPV-1193 | A qualitative study of factors affecting multiple sclerosis management

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EPV-1194 | A real-life experience with eculizumab in neuromyelitis optica spectrum disorders patients

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EPV-1195 | A triple challenge in anti-GAD65 encephalitis: Diagnostic mimicry, therapeutic refractoriness, and the herpes simplex virus conundrum

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EPV-1196 | Acute encephalopathy with leptomenigeal-predominant amyloid- β -related angiitis: A diagnostic blind spot in clinicoradiologic criteria

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EPV-1197 | Acute presentation of anti-IgLON5 encephalitis with response to immunotherapy

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EPV-1198 | Anti-cyclic citrullinated peptide in cerebrospinal fluid as a diagnostic marker of aseptic meningitis in rheumatoid arthritis: Two case reports

M. El Harmochi Daoud; M. Muñoz Pasadas; P. Gómez Ramírez¹; A. Sánchez Gómez; A. Herrera Ortega; M. Carrillo Carrillo; V. Ros Escolar; A. Hernández González; M. Usero Ruiz; M. Corrales Arroyo; J. Vaamonde Gamo
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EPV-1199 | Anti-IgLON5 encephalitis: A diagnostic challenge

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EPV-1200 | Anti-SOX1 autoantibody positivity in an 11-year-old child after COVID-19: A case report

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EPV-1201 | Apathy is a frequent symptom but does not correlate with sNfL and sGFAP in multiple sclerosis

F. Massaro¹; T. Zacccone²; C. Tambussi³; A. Mingione¹; C. Monzani¹; K. Piscopo⁴; A. Corona¹; S. Mesiti¹; M. Guidetti³; E. Vegni⁴; S. Marcegaglia³; T. Bocci⁵; S. Oliveri⁵; A. Priori⁵; F. Martinelli Boneschi⁶

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EPV-1202 | Association between EDSS and brain metrics during peri-pregnancy in women with multiple sclerosis

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EPV-1203 | Association between surgical waiting time and prognosis after thymectomy in non-thymomatous MG patients: A multicenter real-world study

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EPV-1204 | Attention-deficit/hyperactivity disorder and subsequent risk of multiple sclerosis: A population-based retrospective case-control study

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EPV-1205 | Autoimmune brainstem encephalitis: A cohort study and topic modelling using artificial intelligence based BERTopic model on PubMed abstracts

M. Jin¹; J. Kerstens¹; R. van Steenhoven¹; E. Erdag Turgeon¹; M. Nagtzaam¹; M. van Duijn¹; S. Veenbergen²; J. de Vries¹; M. Titulaer¹

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EPV-1206 | Autoimmune limbic encephalitis additional onset features

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EPV-1207 | Beyond IgG1: Predominance of IgG2 and IgG3 subclasses in low-positive MOG-IgG profiles and its diagnostic implications

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EPV-1208 | Beyond neurology – The multidisciplinary journey of patients with multiple sclerosis: Portrait of a Portuguese cohort

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EPV-1209 | Beyond Stiff Person Syndrome: A case of Progressive Encephalomyelitis with Rigidity and Myoclonus (PERM) with status epilepticus and stroke mimicry

E. Garcés Becerril; D. Ginarte Milanés; Á. Antón Conejos; S. García Rubio; E. Bellosta Diago; R. Caldu Agud
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EPV-1210 | Cerebrospinal fluid spectrum in Weston-Hurst acute hemorrhagic leukoencephalitis: A four-case series

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EPV-1211 | Characterization of sleep disturbances in patients with demyelinating diseases: A comparative study of MOGAD, NMO, and multiple sclerosis

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EPV-1212 | Clinical and immunological spectrum of Central Nervous System Demyelinating Diseases in Sri Lanka: A retrospective observational study

A. Wanniarachchi; P. Wijethunga; P. Ruwanpathirana; B. Senanayake
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EPV-1213 | Clinical and radiological response to Pembrolizumab in a newly diagnosed HIV-associated progressive multifocal leukoencephalopathy: A case report

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EPV-1214 | Clinical characteristics, treatment and outcomes in chronic meningitis in neurosarcoidosis: A retrospective cohort study

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EPV-1215 | Clinical outcome of AQP4-IgG seropositive neuromyelitis optica spectrum disorders in Hong Kong Chinese

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EPV-1216 | Clinical profile of patients with myasthenia gravis in China: A nationwide prospective cohort study

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EPV-1217 | Clinical recognition pathways in autoimmune GFAP astrocytopathy: Insights from three cases

S. Yamamoto; G. Watanabe; G. Akao; A. Ishikawa; A. Tamagake; K. Tsukita; Y. Suzuki
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EPV-1218 | Clinical spectrum and serological features of neuromyelitis optica spectrum disorder in a Sri Lankan cohort

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EPV-1219 | Cognitive impairment in rheumatoid arthritis: Association with disease activity and inflammation

M. Abzalova; M. Yakubova; N. Abdurakhmanova
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EPV-1220 | Comparability of serum neurofilament light chain measurements by SIMOA and ECLIA in stroke and multiple sclerosis patients

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EPV-1221 | Comparison of patients with familial and sporadic multiple sclerosis – selected risk factors of the disease – Neurological Clinic in Zabrze, Poland

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EPV-1222 | Comparison of symptom frequency and heritability in patients with familial and sporadic multiple sclerosis. Neurological Clinic in Zabrze, Poland

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EPV-1223 | CSF oligoclonal bands are not a useful biomarker for predicting early reactivation after discontinuation of first-line multiple sclerosis therapies

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EPV-1224 | Eculizumab for refractory thymoma-associated myasthenia gravis with dual impending myasthenic crisis and cholinergic crisis: A new hope?

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EPV-1225 | Efficacy and safety of eculizumab in refractory spontaneous 3M syndrome associated with malignancy

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EPV-1226 | Efficacy and safety of ozanimod in multiple sclerosis: A systematic review and meta-analysis

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EPV-1227 | Features of humoral immunity in children and adolescents with migraine

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EPV-1228 | From childhood to adulthood: Phenotypic differentiation and clinical evolution in Myelin oligodendrocyte glycoprotein antibody-associated disease

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EPV-1229 | GFAP astrocytopathy associated with Epstein–Barr virus infection: A case report

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EPV-1230 | Glutamate decarboxylase antibody-associated neurological disorders: Clinical features, immunotherapy, and short-term outcomes in a russian cohort

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EPV-1231 | Immune-related adverse events due to immune checkpoint inhibitors: A single-centre case series

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EPV-1232 | Impact of prior infection and vaccination on immune-mediated neurological syndromes: A tertiary hospital experience

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S. Arés Ballesteros; S. Sánchez Velasco; L. Lacruz Ballester
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EPV-1233 | Inborn errors of immunity as a novel etiology for neuroinfectious and neuroinflammatory disorders

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EPV-1234 | Increased vulnerability to autoimmune CNS disorders after post-COVID herpes zoster: A global real-world analysis

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EPV-1235 | Kappa and Lambda Index as diagnostic and prognostic biomarkers in autoimmune neurological disorders

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M. Matta²; S. Rolla¹; A. Sala³; A. Di Sapio³; M. Clerico¹
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EPV-1236 | Kappa Free light chain index in CSF: A biomarker beyond multiple sclerosis

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EPV-1237 | Longitudinal [¹⁸F]FDG-PET changes map onto distinct cholinergic/dopaminergic and serotonergic territories in autoimmune encephalitis

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EPV-1238 | Longitudinal study on apathy and social cognition in early relapsing-remitting multiple sclerosis

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EPV-1239 | Long-term clinical and functional burden of autoimmune encephalitis: A targeted literature review

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EPV-1240 | Mapping of neuromyelitis optica spectrum disorders in Belgium: Interim results

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EPV-1241 | Mapping the clinical trajectory of MOGAD

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EPV-1242 | Mitochondrial-immune overlap in leber hereditary optic neuropathy: A case report and lessons learned

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EPV-1243 | Modulation of AMPA-receptors through combination of medium-chain triglycerides and perampanel to control autoimmune status epilepticus: Case report

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EPV-1244 | Multiple sclerosis and autoimmune comorbidities

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EPV-1245 | Natalizumab: a drug to avoid in multiple sclerosis patients with atypical lesions? – A case report

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EPV-1246 | Neurological chronic toxicity induced by immune checkpoint inhibitors: A case of encephalitis, optic neuropathy and polyneuropathy

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EPV-1247 | Neurological complications of immune checkpoint inhibitors therapy in patients with malignant melanoma

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EPV-1248 | Neurological manifestations in rheumatoid arthritis: prevalence and inflammatory correlates

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EPV-1249 | Neurosarcoidosis mimicking a high-grade brain tumor: A diagnostic challenge

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EPV-1250 | Ofatumumab in refractory anti-GABABR encephalitis with active infection

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EPV-1251 | Optimization of treatment of autonomic disorders and vegetative dysfunction in patients with rheumatoid arthritis

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A. Amriddinov

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EPV-1252 | Outcomes within 5 years following different immunosuppressive treatment protocols in cerebral amyloid angiopathy-related inflammation: A case series

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EPV-1253 | Overlap between anti-NMDAR encephalitis and multiple sclerosis: A rare neuroimmunological overlap

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EPV-1254 | Overlap syndrome of myositis, myocarditis, and myasthenia gravis secondary to immune checkpoint inhibitors (ICI): A tertiary-center case series

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EPV-1255 | Pediatric MOGAD atypical fulminant or insidious presentations: monophasic or relapsing conditions?

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EPV-1256 | Posterior cortical atrophy caused by antiphospholipid syndrome Identified by the “railroad track” sign on brain PET

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EPV-1257 | Pre-diagnostic disease trajectories preceding central nervous system inflammation: A UK Biobank Study

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EPV-1258 | Prognostic significance of thyroid autoantibodies in MOGAD and NMOSD: A biomarker for disease activity

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EPV-1259 | Progressive Hemifacial Atrophy (Parry-Romberg Syndrome): Long-term clinical course with traumatic onset and late stabilization

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EPV-1260 | Real-life study of multiple sclerosis patients under treatment with cladribine

A. Costa; S. Gomes; S. Moreira; J. Ferreira Pinto; A. Santos; F. Sousa; J. Cerqueira
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EPV-1261 | Recurrent aseptic meningitis associated with nivolumab: A rare immune-related neurological adverse event

L. Caballero Sánchez; M. Vargas Cobos; A. De Luca; R. Alfonso González; M. Capra; C. Gómez López de San Román; I. Bermejo Casado; D. Cerdán Santacruz; A. Castrillo Sanz; A. Mendoza Rodríguez
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EPV-1262 | Reduction of immune cell subsets and autoantibodies by cladribine in generalised myasthenia gravis peripheral blood mononuclear cell-based assays

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EPV-1263 | Role of anti-HBs antibody titers in HBV reactivation risk stratification in pOBI patients with multiple sclerosis treated with anti-CD20 therapy

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EPV-1264 | Safety of anti-CD20 monoclonal antibodies in multiple sclerosis: A network meta-analysis of infection-specific outcomes

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EPV-1265 | Seizure semiology and EEG patterns across subtypes of Autoimmune Epilepsy: Insights for early diagnosis

O. Sakli; R. Zouari; Z. Saied; A. Rachdi; D. Ben Mohamed; F. Nabli; L. Bouafia; S. Ben Sassi
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EPV-1266 | Steroids or blindness: When optic neuritis does not evolve as expected

M. Luz Esteve¹; D. Gómez de la Torre Morales¹; M. Ravelo León¹; J. Aguilera Aguilera¹; J. Rodríguez Carrillo¹; I. Díaz Díaz¹; A. Hernández González¹; L. Abad Reguera¹; B. Rodríguez García¹; P. Serafim Reis²; M. Polo Martín¹
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EPV-1267 | Susac's silent storm: how high clinical suspicion leads to a masquerading diagnosis. Case report

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EPV-1268 | Targeting ICOS/ICOS-L signaling ameliorates disease in an autoimmune mouse model of neuromyelitis optica spectrum disorders

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EPV-1269 | The Gut-Brain-Immune axis in neurodegeneration: Diet as a molecular modulator

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EPV-1270 | The prevalence and nature of neurocognitive deficits in MS patients – Implications for routine inpatient assessment

M. Lis; M. Schneider; J. Molenda; M. Machej; R. Szymaniak; M. Huber; P. Maj; J. Jurkiewicz; M. Adamczyk-Sowa
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EPV-1271 | Timing matters: Study of the impact of chrononutrition on multiple sclerosis-related fatigue

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EPV-1272 | Transcriptomic analysis of B cells treated with evobrutinib in a mouse model of neuromyelitis optica spectrum disorders

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EPV-1273 | Trial in progress – Impact of continuous assessment of generalized Myasthenia Gravis using a mobile application: myMGverse APP observational study

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EPV-1274 | Two cases of cerebral amyloid angiopathy-related inflammation following immune checkpoint inhibitor treatment

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EPV-1275 | Ublituximab for relapsing forms of MS: Interim results on real-world treatment satisfaction, quality of life and tolerability from the BRILL study

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EPV-1276 | Unilateral mydriasis and severe ataxia: Anti-Hu paraneoplastic encephalomyelitis as a herald of small-cell lung cancer

M. González Campos; G. Torres Sánchez; H. El Mouhahjir Mohamed; A. Ortega Ruiz; A. Tapia Barrantes; J. Mederos Neurology, Juan Ramón Jiménez Hospital, Huelva, Spain

EPV-1277 | Very late-onset AQP4-positive neuromyelitis optica associated with autoimmune hemolytic anemia and thrombocytopenia: a case report

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EPV-1278 | Vestibular tests and virtual reality-based evaluation of otolith organs in MS: Associations with brainstem lesions and serum GFAP levels

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EPV-1279 | Viral seroprevalence and risk of reactivation in MS patients treated with ocrelizumab: A real-world cohort study

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EPV-1280 | Vitaccess Real MG Registry: baseline characteristics and patient-reported outcomes scores from an international myasthenia gravis patient registry

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EPV-1281 | When immunotherapy unlocks the door to autoimmunity: A case report of MOGAD triggered by Pembrolizumab in a patient with Gray Zone Lymphoma

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EPV-1282 | When miller fisher strikes twice: A rare case of recurrent miller fisher syndrome

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EPV-1283 | When the eyes dance and the gait fails: A case of anti-GFAP associated opsoclonus-myoclonus-ataxia

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Neuroinformatics

EPV-1284 | Artificial intelligence in neurology: Preliminary results of a nationwide survey among Spanish neurologists

A. De Luca; M. Vargas Cobos; R. algonzo González; C. Gómez López de San Román; L. Caballero Sánchez; I. Bermejo Casado; D. Cerdán Santacruz; J. Eguizabal Aguado; A. Castrillo Sanz; A. Mendoza Rodriguez; M. Capra

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EPV-1285 | Beyond craniotomy: Endovascular approach to brain-computer interface

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EPV-1286 | Clinical characteristics and outcomes of acute stroke patients based on RES-Q data in Uzbekistan

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EPV-1287 | Data sharing in neurology: An analysis of the data availability statements in the European Journal of Neurology 2024–2025

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EPV-1288 | Leveraging single-cell and spatial omics for brain tumor insights to improve therapeutic strategies

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EPV-1289 | Lipid induced energetic lability in serotonin and dopamine transporter assembly could be resisted by truncation isoforms

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EPV-1290 | Network-based identification of strategic research opportunities in neurologic disorders: A bibliometric and graph analytics approach

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EPV-1291 | Research frontiers and gaps across 3,343 neurological diseases: A systematic bibliometric analysis of the PubMed database

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EPV-1292 | Reverse-QTY engineering converts soluble glutamate transporter isoforms into high-affinity inhibitors

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EPV-1293 | Validation of brain fog scale in Farsi

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Neurological manifestation of systemic diseases

EPV-1294 | Acute bilateral visual loss as the initial manifestation of Wernicke encephalopathy: A rare case report

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EPV-1295 | Acute confusional syndrome as the initial presentation of hemophagocytic syndrome secondary to high-grade lymphoma

B. Quintana López; A. O. Sanchez; D. Alonso Vallin; V. García Gracia; C. Castaño Ron; P. Diaz Aguirre; M. Cabezas Arias
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EPV-1296 | an unusual cause of axonal polyradiculoneuropathy: Metastatic infiltration from nasopharyngeal carcinoma

D. Cerdán Santacruz; M. Vargas Cobos; A. De Luca; R. Alfonso González; M. Capra; C. Gómez López de San Román; I. Bermejo Casado; L. Caballero Sánchez; A. Castrillo Sanz; G. Suárez Fernández; A. Mendoza Rodríguez
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EPV-1297 | Anti-Hu mediated small-fiber neuropathy: A rare manifestation of small-cell neuroendocrine prostate cancer

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EPV-1298 | Anti-PL-7 (Anti-Threonyl-tRNA Synthetase) positive myopathies: Two distinct clinical cases

A. Başel Pala; K. Işık; Ö. Çakır Nartsoy; G. Taşçatan; E. Acıman Demirel; M. Açıkgöz; U. Çelebi; H. Atasoy
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EPV-1299 | Anti-seizure and anti-epileptic effects of angiotensin receptor blockers in patients after acute stroke in observational studies

Z. Dr. Kiss; B. Fülöp; Z. Hermann; B. Jassey; A. Koszta; K. Kovács-Kusza; K. Magyari-Zuberecz; E. Pordány-Bagoly; K. Radó; D. Dani; G. Szegleti; V. Varga; Z. Szupera
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EPV-1300 | Anxiety and depression in overt and subclinical hypothyroidism: Single-centre observational study

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EPV-1301 | Atypical neurological presentation of autoimmune polyglandular syndrome type 2: A case report

N. Singh i Kaur; L. Rosés Hernansáez; G. Rumaldo Gutti; C. Guerrero Castaño
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EPV-1302 | Bedtime vs morning antihypertensive dosing: cardiovascular and neurovascular outcomes — A systematic review & trial sequential analysis

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EPV-1303 | Blind to the diagnosis: Marchiafava-Bignami disease

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EPV-1304 | Central nervous system demyelination in primary Sjögren's syndrome: Clinical, imaging and antibody findings in seven patients

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EPV-1305 | Cognitive and neurological manifestations in rheumatoid arthritis: Clinical and inflammatory correlates

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EPV-1306 | Cognitive impairment as an initial manifestation of diffuse large B-cell lymphoma: A case report

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EPV-1307 | Fluctuating encephalopathy and vomiting in adolescence as a presentation of late onset ornithine transcarbamylase deficiency

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EPV-1308 | From lung to brain: Invasive aspergillosis by *Aspergillus nidulans* in lung transplant recipient and stroke mimic case

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EPV-1309 | Hypertrophic pachymeningitis associated with Polyarteritis Nodosa: An unusual cause of headache and visual disorders

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EPV-1310 | Intracerebral hemorrhage as the initial presentation of severe undiagnosed immune thrombocytopenic purpura

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EPV-1311 | Invisible myelopathy: Lupus myelitis overlapping with anti-AQP4 NMOSD

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EPV-1312 | Lithium: From stabilizer to disruptor

R. Alfonso González; M. Vargas Cobos; A. C. De Luca; M. Capra; C. Gómez López de San Román; L. Caballero Sánchez; I. Bermejo Casado; J. Eguizábal Aguado; D. Cerdán Santacruz; A. Castrillo Sanz; A. Mendoza Rodríguez
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EPV-1313 | MELAS presenting as Anton syndrome: Beyond diagnostic challenges

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EPV-1314 | Misdiagnosed schizoaffective disorder revealing Fahr's syndrome secondary to hypoparathyroidism: A case report

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EPV-1315 | Misfortunes never come singly – Neurological involvement in hematologic malignancy

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EPV-1316 | Neuropsychological burden, stigma and social participation in autoimmune vitiligo: A cross-sectional study

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EPV-1317 | New-onset generalized seizure as the initial manifestation of Neuropsychiatric SLE (NPSLE)

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EPV-1318 | Numb chin syndrome: A silent warning sign

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EPV-1319 | Oligoclonal bands in diabetic lumbosacral radiculoplexus neuropathy: An unusual presentation of Bruns–Garland syndrome

M. Alvarez-Gutierrez; P. Mayo-Rodríguez; A. Lafuente-Almaden; J. Melgar-Jara; L. Dominguez-Jimenez; M. Bonnin-Bravo; E. Lopez-Valdes; M. Garcia-Ruiz
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EPV-1320 | Rapidly progressive cognitive decline as an elderly onset of neurological symptoms in an 80-year-old woman with MELAS

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EPV-1321 | Recurrent Wernicke-Korsakoff syndrome of non-alcoholic etiology: A case report

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L. García Granados; L. Fernández Espigares; J. Román Rueda;
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EPV-1322 | Rising mortality trends related to type 2 diabetes mellitus and cardiac arrest among american adults: A 21-year national analysis

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EPV-1323 | Role of bowel wash in recovery from hepatic encephalopathy

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EPV-1324 | Spectrum of non-psychotic neuropsychiatric syndromes in women with alimentary obesity: A clinical study

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EPV-1325 | The neuroprotective effect of dual GIP/GLP1-RA agonists: A scoping review of preclinical and clinical evidence

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EPV-1326 | Thirty-year trends in late and late latent syphilis in the United States: National, sex-specific, and regional patterns

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EPV-1327 | Transverse myelitis revealing primary Sjögren's syndrome in elderly patients: A case series

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EPV-1328 | Treosulfan vs Busulfan for HSCT: A meta-analysis of key outcomes

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EPV-1329 | When water becomes neurotoxic: Acute hyponatremia causing seizure in chronic polydipsia

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EPV-1330 | Indian classical music and the brain: Neurological insights into rhythm, emotion, and cognition

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Neurology and arts

EPV-1331 | Neurology on canvas

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EPV-1332 | What happens if it's not dementia?

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Neuro-oncology

EPV-1333 | A reversible mimic of dementia with Lewy bodies: Anti-LGI1 encephalitis

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EPV-1334 | An integrated framework for epileptogenicity, diagnostic evaluation and clinical management of pediatric low-grade epilepsy-associated tumors

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EPV-1335 | Assessment of cognitive functions in glial brain tumors

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EPV-1336 | Autoimmune GFAP astrocytopathy encephalomyelitis and ovarian teratoma: A case report and etiological differential diagnosis

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J. Rodríguez Quinchanequa; A. Baz Cárdenas; V. Torres do Rego
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EPV-1337 | Bloomy rind sign as a diagnostic clue in IDH-wild-type brainstem glioma

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EPV-1338 | Bridging the gap: Evaluating FLASH radiotherapy in the context of glioblastoma

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EPV-1339 | Calcifications in extra-axial metastases from prostate adenocarcinoma: A case report

A. De Luca; M. Vargas Cobos; R. Alfonso González; M. Capra;
C. Gómez López de San Román; L. Caballero Sánchez;
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EPV-1340 | CAR-T-Related myelopathy after anti-BCMA therapy: The first reported case in plasma cell leukemia

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EPV-1341 | Cavernous sinus squamous cell carcinoma masquerading as sinusitis: a diagnostic challenge

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EPV-1342 | Cerebral venous thrombosis associated with immune checkpoint inhibitor therapy in malignant melanoma: A case report

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EPV-1343 | Demyelination of the central nervous system CNS associated with cancer immunotherapy

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EPV-1344 | Diagnosis of brain tumors in pediatric emergency department-single center experience

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EPV-1345 | Effects of memantine on temozolomide-induced apoptotic signaling in glioma cells

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EPV-1346 | Inter-perforator microdebulking: A vascular-based approach to clinoidal meningioma resection

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EPV-1347 | Intravascular lymphoma mimicking a longitudinally extensive transverse myelitis: A case series and review of the literature

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EPV-1348 | Isolated inflammatory leptomeningitis after CAR-T therapy: ICANS or TIAN? An atypical case report

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EPV-1349 | Longitudinally extensive spinal cord lesion mimicking autoimmune myelitis: A case of primary spinal glioblastoma

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EPV-1350 | Mild Encephalitis/Encephalopathy with a Reversible Splenic Lesion (MERS) in acute myeloid leukemia during pregnancy: A case report

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EPV-1351 | Myeloradiculitis associated with cancer: A diagnostic challenge

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EPV-1352 | Myositis-myocarditis syndrome secondary to Immune Checkpoint Inhibitor drugs and experience with Abatacept-Ruxolitinib in a tertiary hospital

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EPV-1353 | Neurological complications in hematological malignancies: A retrospective national cohort analysis

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EPV-1354 | Outcome prediction in skull-base neurosurgery: A preliminary analysis

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EPV-1355 | Paraneoplastic sensory ganglionopathy unveiling bronchogenic carcinoma in a heavy smoker

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EPV-1356 | Person in the Barrel Syndrome following immune checkpoint inhibitor therapy: Bilateral brachial plexopathy as a form of peripheral neurotoxicity

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EPV-1357 | Preoperative functional, sociodemographic, and psychological factors as predictors of postoperative outcomes in brain tumor surgery

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EPV-1358 | Presentation and types of childhood cancer at the Muhimbili National Hospital (MNH), 2023 retrospective observational study

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EPV-1359 | Prophylactic antiepileptics in glioma: Do they prevent first seizures? A systematic review

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EPV-1360 | Rapidly progressive cerebellar syndrome secondary to primary peritoneal carcinoma with positive CV-2/CRMP5 antibodies: A novel association

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EPV-1361 | Real-world distribution, diagnostic delay, and management of primary brain tumors in a tertiary neurology center: Insights from a patient-level cohort

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EPV-1362 | Survival, response, and safety outcomes of oncolytic viral therapy in pediatric brain tumors: A meta-analysis

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EPV-1363 | Sustained visual recovery with the MEKi Trametinib in an adult patient with sporadic optic pathway glioma harboring a rare BRAF mutation

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EPV-1364 | The growing burden of central nervous system cancers across central asian states

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EPV-1365 | The role of genetic mutations in determining immunotherapy response in meningioma patients: A prospective study with MRI monitoring

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EPV-1366 | To assess the prevalence of anxiety and depressive disorders in patients with breast cancer, as well as the possibilities for their early detection

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EPV-1367 | To evaluate the effectiveness of cognitive-behavioral therapy in reducing anxiety and depressive symptoms in patients with breast cancer

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EPV-1368 | Unexpected but not unmanageable: A rare CNS manifestation of Chronic Lymphocytic Leukemia

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EPV-1369 | Unexpectedly high incidence and clinical presentation patterns of glioblastoma: A population-based study from a defined health area

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EPV-1370 | Altered sensory integration in vestibular migraine: Effects of visual input, cervical proprioception and mastoid vibration on postural responses

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EPV-1371 | A disease in the differential diagnosis of orbitopathy: IgG4-related disease

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EPV-1372 | A study to evaluate the risk of osteoporosis and osteopenia in patients with benign paroxysmal positional vertigo

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EPV-1373 | Analysis of catch-up saccades during video head impulse testing in vestibular migraine

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EPV-1374 | Anti-IL-6 receptor antibody ameliorates impaired visual function in AQP4 peptide-immunized mice

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EPV-1375 | Artificial intelligence-based assessment of optical coherence tomography in optic nerve disorders: A comparative study of large language models

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EPV-1376 | Atypical visual involvement in Miller-Fisher syndrome: Case report

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EPV-1377 | Audio and vestibular symptoms in Post Traumatic Stress Disorder

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EPV-1378 | Bilateral abducens nerve palsy following Bexsero (meningococcal group B vaccine) administration – Case report

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EPV-1379 | Central retinal artery occlusion – The tip of the iceberg of cerebrovascular disease

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EPV-1380 | CGRP-based treatment approaches for vestibular migraine: Assessing therapeutic outcomes

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EPV-1381 | Clinic-ophthalmological characteristics of migraine with aura

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EPV-1382 | Diagnostic value of bedside eye movement examination in movement disorders

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EPV-1383 | Does lumbar puncture cause changes in magnetic resonance imaging findings in patients diagnosed with pseudotumour cerebri?

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EPV-1384 | Dorsal midbrain syndrome due to complicated posterior reversible encephalopathy syndrome

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EPV-1385 | Euthyroid thyroid eye disease with negative thyroid antibodies

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EPV-1386 | Evaluation of ocular higher-order aberrations in patients with migraine: A comparative study

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EPV-1387 | Features of vestibular disorders in patients with sensorineural hearing loss

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EPV-1388 | Global burden of hearing loss in people with otitis media: A systematic review and meta-analysis

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EPV-1389 | Identification of blood-based inflammatory biomarkers for vestibular neuritis using a proximity extension assay

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EPV-1390 | Intellectual disability in episodic ataxia type 2

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EPV-1391 | Is there any prognostic factor for benign intracranial hypertension: Clinical, MRI, and OCT findings

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EPV-1392 | Marked improvement of infantile nystagmus with memantine: A video-documented case

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EPV-1393 | Motorist's vestibular disorientation syndrome: Clinical profile of 24 patients with proposed diagnostic criteria

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EPV-1394 | Multimodal assessment of visual pathway involvement in multiple sclerosis without optic neuritis: correlating OCT metrics with neurological parameters

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EPV-1395 | Ophthalmological predictors of idiopathic intracranial hypertension in patients with chronic migraine

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EPV-1396 | Orbital myositis as a rare manifestation of varicella zoster virus reactivation: A case report

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EPV-1397 | Possibilities for improving postural control in a virtual reality environment in patients with multiple sclerosis

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EPV-1398 | Posterior Ischemic Optic Neuropathy After Herpes Zoster Ophthalmicus

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EPV-1399 | Predictors of long-term disability in multiple sclerosis following optic neuritis

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EPV-1400 | Quality of life in visual snow syndrome

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EPV-1401 | Quantitative analysis of smooth pursuit and saccadic eye movements in multiple sclerosis

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EPV-1402 | Safety and efficacy of high-dose tradipitant for motion sickness: A systematic review and meta-analysis of randomised controlled trials

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EPV-1403 | The relationship between binaural auditory integration at the brainstem level and cognition in Parkinson's disease

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EPV-1404 | Two cases of micropsia after an ischemic stroke

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EPV-1405 | Validation study of oculometric biomarkers in parkinson's disease

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EPV-1406 | Velocity-dependent horizontal optokinetic nystagmus dysfunction in Multiple Sclerosis: A possible oculomotor distinctive feature

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EPV-1407 | Vestibular Function Tests in patients with Spinocerebellar Ataxia Type 3 (SCA3) and Cerebellar Ataxia Neuropathy Vestibular Areflexia (CANVAS)

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EPV-1408 | Visualization aspects of endolymphatic hydrops in patients with recurrent cochleovestibular disorders

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Neuropathies

EPV-1409 | A rare mimicker of CIDP: A case of POEMS syndrome presenting with progressive polyneuropathy

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EPV-1410 | Acute demyelinating polyneuropathy associated with diabetic amyotrophy: a complex clinical presentation in an elderly patient

B. Quintana López; D. Alonso Vallin; A. O. Sanchez; V. García Gracia; P. Díaz Aguirre; C. Castaño Ron; J. González Fernández
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EPV-1411 | Associations between short-chain fatty acid profiles and sensory diabetic polyneuropathy in patients with type 2 diabetes and NAFLD: A pilot study

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EPV-1412 | Burden of illness for patients with multifocal motor neuropathy: A quantitative survey of individuals from the United States

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EPV-1413 | Clinical and electrophysiological correlates of Guillain-Barré syndrome in a tertiary care cohort

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EPV-1414 | Clinical characteristics and determinants of cognitive decline in patients with Parkinson's disease

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EPV-1415 | Clinical spectrum and treatment outcomes in Anti-MAG neuropathy: A real-world case series

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EPV-1416 | Cognitive dysfunction and peripheral neuropathy in a conflict-affected diabetic population: A cross-sectional study from Sudan

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EPV-1417 | Differentiating multifocal motor neuropathy and multifocal variant of CIDP: A multiparametric diagnostic approach

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EPV-1418 | Eosinophilic granulomatosis with polyangiitis presenting as acute polyneuropathy mimicking Guillain-Barre syndrome

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EPV-1419 | Evolution of genetic testing in hereditary neuropathies: Experience of a centre

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EPV-1420 | Genetic and neurophysiological insights into PIGG-associated disorders

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EPV-1421 | Guillain-Barré syndrome in complex clinical settings — Diagnostic and therapeutic importance of nerve conduction studies

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EPV-1422 | Interactions of cerebrospinal fluid protein level in chronic inflammatory demyelinating neuropathy types

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EPV-1423 | Intravenous immunoglobulin to subcutaneous efgartigimod PH20 transition in CIDP: A phase 4 study in progress

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EPV-1424 | Late-onset, autosomal dominant, axonal, sensorimotor neuropathy due to the new variant c.1A_G in MPZ

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EPV-1425 | Mass balance and metabolism of BIA 28-6156, an allosteric activator of beta-glucocerebrosidase, in humans

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EPV-1426 | Misdiagnosis of chronic inflammatory demyelinating polyneuropathy: Frequency, causes, and mimics

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EPV-1427 | Morvan's syndrome: A systematic review

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EPV-1428 | Multiple cranial neuropathies as the presenting form of a parotid neoplasm

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EPV-1429 | Neurological disturbances according to chronic inflammatory demyelinating neuropathy types

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EPV-1430 | Prognostic factors for unfavorable long-term outcomes in chronic inflammatory demyelinating polyneuropathy

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EPV-1431 | Relapsing focal chronic inflammatory demyelinating polyneuropathy – An uncommon presentation

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EPV-1432 | Riliprubart Phase 2 trial results: Efficacy analysis using immune neuropathy-no evidence of disease activity framework

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EPV-1433 | Sensory ganglionopathy: The value of integrated clinical and neurophysiological criteria

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EPV-1434 | Sensory neuronopathies: Etiological review in our cohort

M. Callejo; A. Jauregui; A. Gonzalez; M. Martinez; A. Lopez; N. Marcos; F. Angoso; L. Pastor; J. Mallada; A. Rodriguez-Antigüedad

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EPV-1435 | Severe sensorimotor axonal polyneuropathy following enfortumab vedotin: Report of two cases from Türkiye

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EPV-1436 | Small fiber neuropathy and autonomic dysfunction in Sjogren's syndrome

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EPV-1437 | Structural peripheral nerve changes in systemic lupus erythematosus: A high-resolution ultrasound study

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EPV-1438 | Surgical versus conservative treatment of patients with ulnar neuropathy at the elbow: A randomized clinical trial

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Neurorehabilitation

EPV-1439 | A community-based dyadic music therapy social prescribing program for people with dementia: A 12-week feasibility study in Taiwan

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EPV-1440 | A goal-directed framework for the dopaminergic treatment of apathy and hemispatial neglect following brain injury

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EPV-1441 | Association between cerebral venous hemodynamic status and three-month functional recovery in atherosclerotic ischemic stroke

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EPV-1442 | Barriers to rehabilitation in patients with mild neurological deficits after elective supratentorial neurosurgery

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EPV-1443 | Challenges during the rehabilitation for a patient with late-onset Anti-Jo 1 positive Antisynthetase syndrome – Case report

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EPV-1444 | Clinical application of machine learning empowered brain computer interfaces in amyotrophic lateral sclerosis – A systematic review and meta-analysis

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EPV-1445 | Clinical autonomic screening in neurorehabilitation: Associations between objective orthostatic findings and patient-reported symptoms

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EPV-1446 | Combined land-based and aquatic physiotherapy in Parkinson's disease and rheumatoid arthritis: A case study

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EPV-1447 | Devices and outcomes in lower-limb exoskeleton rehab after stroke and spinal cord injury (SCI): evidence map and trial landscape

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EPV-1448 | Effectiveness and perspectives on the application of traditional Korean and Western medicine in the treatment of lumbosacral dorsopathy

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EPV-1449 | Effectiveness of psychomotor therapy in post-stroke rehabilitation in the south aral sea region

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EPV-1450 | Effectiveness of screening tools for assessing depressive risk in patients with ischemic stroke

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EPV-1451 | Effectiveness of telemedicine-based cognitive rehabilitation in patients with chronic cerebral ischemia stages I–II

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EPV-1452 | Effectiveness of vocational rehabilitation on return to work after acquired brain injury: A systematic review and meta-analysis

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EPV-1453 | Effects of a home-based calligraphic exercise program on manual dexterity in Parkinson's disease: A randomized controlled exploratory trial

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EPV-1454 | Efficacy of High-Intensity Laser Therapy (HILT) on hemiplegic shoulder pain in post-stroke patients: A systematic review and meta-analysis

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EPV-1455 | Electrophoresis and magnetophoresis in treatment of neuronal retinal changes after non-ischemic retinal vein occlusion

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EPV-1456 | Exoskeleton-assisted hand training after stroke: Motor outcomes and patient-reported experience in an ongoing randomized trial

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EPV-1457 | Feasibility of an online fatigue self-management programme for people with Parkinson's disease: A pilot RCT

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EPV-1458 | Immersive virtual reality for post-stroke upper limb rehabilitation: A systematic review and meta-analysis with trial sequential analysis

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EPV-1459 | Intermittent Theta Burst Stimulation (iTBS) priming for post-stroke upper limb motor recovery: A systematic review and meta-analysis

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EPV-1460 | Length of stay after stroke in hospitals and rehabilitation centers: A systematic review and meta-analysis from Arab Countries and Saudi Arabia

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EPV-1461 | Mesenchymal stem cells in the complex treatment of diffuse and focal brain lesions

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EPV-1462 | Perineuronal net-mediated regulation of calcium homeostasis and oxidative stress in post-stroke neuroplasticity

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EPV-1463 | Post-stroke home-based rehabilitation: Functional impact and patient satisfaction

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EPV-1464 | ReFresh: A feasibility randomised trial of an online fatigue-management programme for people with Parkinson's disease

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EPV-1465 | Regression of pain and paresthesia symptoms in patients with trigeminal neuralgia following TENS therapy

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EPV-1466 | Rehabilitation of patients after stroke through multifactorial analysis

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EPV-1467 | Technology readiness in neurological chronic patients in Denmark

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EPV-1468 | Telehealth intervention using the HEARTS Technical Package and an activity monitor to increase physical activity post-stroke: a feasibility study

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EPV-1469 | The effect of repeated use of BCI-controlled hand exoskeleton in a patient with post-stroke severe paresis

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EPV-1470 | The effectiveness of transcranial magnetic stimulation in reducing neurological complications in children with hydrocephalus

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EPV-1471 | The experience of using psychomotor techniques in rehabilitation in psychiatry

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EPV-1472 | Treatment of post-COVID symptoms with auricular vagus nerve stimulation

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EPV-1473 | Use of an artificial intelligence-based clinical assistant in cognitive rehabilitation of post-stroke patients

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EPV-1474 | Virtual reality rehabilitation in patients with total knee replacement: A study from Rural Iran

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EPV-1475 | Vitamin D supplementation on cognitive and functional outcomes in patients with traumatic brain injury: A systematic review and meta-analysis

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Neurosonology

EPV-1476 | Correlation between echo intensity and abnormal spontaneous activity in cubital tunnel syndrome: A preliminary study

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EPV-1477 | Cranial ultrasonography and circulatory markers of inflammation as predictors for atherosclerosis in patients with depression

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EPV-1478 | Impact of clinical characteristics and nomogram model in adult patients with ependymoma: A population-based study

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EPV-1479 | Pediatric stroke in arid regions shows dehydration-related severity, with hemiparesis, seizures, and ischemic stroke predominating

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EPV-1480 | Pilot study: Differences on distance between median nerve and ulnar artery in various wrist motions

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Neurotoxicology/occupational neurology

EPV-1481 | Comparative evaluation of neurological adverse effects of typical and atypical antipsychotics in patients with schizophrenia

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EPV-1482 | Environmental neurotoxicology in marginalized communities: A systematic review focusing on lead exposure among Roma children

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EPV-1483 | Marchiafava-Bignami Disease: An Uncommon Corpus Callosum Disorder

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EPV-1484 | Neurological adverse events of antidepressant medications: A pharmacovigilance study

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EPV-1485 | Neurological effects of nitrous oxide: A case of combined spinal cord degeneration secondary to recreational use

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EPV-1486 | Subacute chorea following pesticide exposure: A case report

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EPV-1487 | When mycophenolate strikes: Unveiling the neurotoxic danger of immunosuppressive therapy

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Neurotraumatology

EPV-1488 | A consolidation gate linking ketamines signaling to durable epigenetic remodeling in depression

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EPV-1489 | Assessing the diagnostic requisites in paediatric Traumatic Brain Injury (TBI) using Artificial Intelligence (AI) – A meta-analysis

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EPV-1490 | Mesenchymal stem cells in the treatment of traumatic brain injury

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EPV-1491 | Severe traumatic brain injury in Brazil: 15years of outcomes and regional disparities from DATASUS

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EPV-1492 | Smartphone quantitative pupillometry for traumatic brain injury: Systematic review of diagnostic performance and feasibility

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EPV-1493 | The bone metabolism disorders in young men with epilepsy

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EPV-1494 | Use of artificial intelligence in the prediction of traumatic brain injury outcomes in paediatric population: A systematic review and meta-analysis

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Pain

EPV-1495 | Acute myofascial facial pain management in dentistry patients

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EPV-1496 | Additional dopaminergic replacement for nociceptive pain in Parkinson’s disease: A randomized double-blind placebo-controlled trial

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EPV-1497 | Association of allostatic load and diet quality with depression and pain comorbidity: NHANES 2009-2010

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EPV-1498 | Botulinum toxin A in treatment of resistant neuropathic pain syndrome: Case series

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EPV-1499 | Can repurposed ibudilast mitigate fibromyalgia pain?

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EPV-1500 | Cannabinoids in chronic pain: Clinical outcomes, adverse effects and legal challenges

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EPV-1501 | Clinical effectiveness of gabapentin in trigeminal neuralgia

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EPV-1502 | Complex use of manual and training therapy with audiovisual vibrotactile stimulation in Zero-G for low back pain treatment in athletes

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EPV-1503 | Effectiveness and safety of oral herbal medicine for postherpetic neuralgia: A systematic review and meta-analysis

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EPV-1504 | Efficacy and safety of adjunctive 10-Hz rTMS in central post stroke pain: A randomized double-blind placebo-controlled trial

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EPV-1505 | Examining how non-pharmacological interventions that affect sleep quality impact pain-related neurotransmitters in patients with fibromyalgia

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EPV-1506 | fMRI markers of pain catastrophizing and placebo effects in patients with chronic neuropathic pain

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EPV-1507 | Hemiplegic shoulder pain

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EPV-1508 | Optimization of therapy in patients with trigeminal neuralgia

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EPV-1509 | Pain in children with Duchenne muscular dystrophy: An underrecognized symptom

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EPV-1510 | Pain management in the neurology clinics: Level of knowledge and assessment of the needs of patients suffering from pain among the EAN members

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EPV-1511 | Paroxysmal extreme pain disorder: A winding road to a definitive diagnosis

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EPV-1512 | Perception of pain and its management in people with multiple sclerosis

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EPV-1513 | Real-world outcomes of intranasal esketamine in treatment-resistant major depression with chronic pain

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EPV-1514 | TENS in chronic pain management: An 11-year nationwide analysis using the French national health data system

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EPV-1515 | The impact of barometric pressure changes on migraine attacks severity, frequency, and duration: A systematic review of the literature

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EPV-1516 | Trigeminal autonomic cephalalgias: A single-centre retrospective real-world study from the Erebouni pain clinic

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Palliative care

EPV-1517 | A National neuropalliative care training programme: Experience from the Spanish society of neurology

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Peripheral nerve disorders

EPV-1518 | A single-center cohort description of patients diagnosed with leprosy in a tertiary hospital in Catalonia

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EPV-1519 | Acute onset chronic inflammatory demyelinating polyneuropathy mimicking Guillain Barré syndrome: Clinical and electrophysiological insights

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EPV-1520 | Atypical sensorimotor polyradiculoneuropathy: Have you considered POEMS syndrome?

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EPV-1521 | Case report: A diagnostic Odyssey leading to neurosyphilis

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EPV-1522 | Characterization of neuropathies associated with IgM monoclonal gammopathy without anti-MAG antibodies

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EPV-1523 | Chronic inflammatory demyelinating polyneuropathy and thymoma

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EPV-1524 | Chronic inflammatory demyelinating polyneuropathy: Clinical spectrum and treatment outcomes in a Tunisian cohort

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EPV-1525 | Clinical, electrophysiological profile and therapeutic outcome in acute, subacute and chronic axonal polyradiculoneuropathy in a tertiary hospital

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EPV-1526 | Cognitive dysfunction and peripheral neuropathy among diabetic patients: Clinical features and contributing factors in conflict-affected region

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EPV-1527 | Comparative analysis of laser doppler flowmetry parameters in patients with severe and mild diabetic angiopolineuropathy

U. Mukarramov; S. Rustambekova; S. Kuranbayeva; G. Raximbaeva; S. Kalandarova
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EPV-1528 | Comparison of Asbury criteria versus Brighton collaboration criteria in early diagnosis of patients with Guillain-Barre syndrome

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EPV-1529 | De novo POLR3B mutation results in demyelinating polyneuropathy and spastic paraparesis with dental abnormalities

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EPV-1530 | Diagnostic accuracy of ultrasound for carpal tunnel syndrome taking nerve conduction studies as gold standard: A meta-analysis in Asian populations

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EPV-1531 | Efficacy and safety of rTMS in peripheral neuropathic pain: A meta-analysis of randomized sham-controlled trials

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EPV-1532 | Insights on neuralgic amyotrophy pathophysiology from an acute case

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EPV-1533 | Juvenile myasthenia gravis: Clinical features, management, and outcomes

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EPV-1534 | Linezolid-associated sensory axonal neuropathy: Longitudinal clinical observations with neurophysiological correlation

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EPV-1535 | Nodopathy with autoantibodies against contactin-1 and contactin-associated protein-1 complex associated with clear cell renal carcinoma

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EPV-1536 | Polineuropathy revealing rare TTR Ile127Val amyloidosis

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EPV-1537 | Prevalence of Guillain-Barré syndrome subtypes in Pakistan: A meta-analysis

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EPV-1538 | Prognostic factors in Guillain–Barré syndrome: A retrospective cohort study in a tertiary referral centre

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EPV-1539 | Risk factors for pregnancy-related carpal tunnel syndrome

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EPV-1540 | Role of upper extremity mixed nerve conduction studies in the electrophysiological diagnosis of chronic inflammatory demyelinating polyneuropathy

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EPV-1541 | Seasonal variation and clinical severity of Guillain–Barré syndrome: A five-year single-center experience

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EPV-1542 | Serum neurofilament light chain as a biomarker in immune-mediated and non-inflammatory polyneuropathies

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EPV-1543 | Abstract withdrawn

EPV-1544 | Unilateral peripheral facial paralysis in Guillain-Barre syndrome: A single-center study

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EPV-1545 | Unmasking bilateral Parsonage–Turner syndrome: The critical role of neuroimaging

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Sleep-wake disorders

EPV-1546 | “Dark Ain’t That Dark”: A qualitative study exploring biopsychosocial barriers and facilitators in the everyday lives of patients with RLS

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EPV-1547 | “We are family”: Experiences of social support, focusing on facilitators and barriers, among family members of patients with RLS

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EPV-1548 | Assessment of the impact of psycho-emotional disorders on the sleep onset process and determination of the effectiveness of relaxation psychotherapy

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EPV-1549 | Association between neck myoclonus and spontaneous cervical artery dissection: A case-control study

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EPV-1550 | Clinical structure of chronic insomnia in neurotic disorders and its psychocorrection

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EPV-1551 | Efficacy and safety of transcranial alternating current stimulation in treating Chinese patients with primary insomnia: A meta-Analysis

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EPV-1552 | Evaluation of body mass index and HDL/Triglyceride ratio in patients diagnosed with OSAS: A retrospective cohort study

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EPV-1553 | Focusing on minor parasomnia events: Is delta sleep combined with open eyes enough to diagnose disorders of arousal?

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EPV-1554 | Impact of circadian rhythm correction on treatment-refractory migraine in night-shift workers: A wearable-based pilot study

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EPV-1555 | Informational needs among patients with restless legs syndrome (RLS): Psychometric testing of the informational needs of RLS inventory

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EPV-1556 | Insomnia among hospitalized inpatients: Old issues, new challenges

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EPV-1557 | Neurobehavioral and autonomic correlates of sleepiness severity in central disorders of hypersomnolence

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EPV-1558 | Predictive value of the easy sleep apnea predictor in asymptomatic driver's license applicants with increased body mass index

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EPV-1559 | Sleep characteristics and associated factors in healthcare workers: Findings from a nationwide population-based study

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EPV-1560 | Sleep in post-traumatic epilepsy

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EPV-1561 | Sleep quality in a series of people with multiple sclerosis

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EPV-1562 | Somatic symptoms and sleep quality in Ukrainian medical students during wartime stress

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EPV-1563 | The effect of hand-wrist pain consistent with carpal tunnel syndrome on sleep quality among employees at Pamukkale

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EPV-1564 | The relationship between ALPS-index and motor and domain-specific cognitive functions in de novo Parkinson's disease

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Spinal cord and root disorders

EPV-1565 | Acute myeloradiculitis unveiling hepatitis B virus infection: Case report

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EPV-1566 | Beyond the usual: Spondylodiscitis as a hidden cause of L5 radiculopathy

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EPV-1567 | Chronic inflammatory sensory polyradiculopathy: A case report

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EPV-1568 | Early presentations of multiple sclerosis: Two adult cases

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EPV-1569 | International standards to document autonomic function after spinal cord injury: Third edition

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EPV-1570 | Learning from the two spinal cord infarct cases missed in initial diagnoses

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EPV-1571 | Neurogenic Bladder in Children with Myelomeningocele

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EPV-1572 | Spinal dural arteriovenous fistula: A crucial differential diagnosis in myelopathy

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EPV-1573 | Uncommon case of methotrexate-induced myelopathy

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EPV-1574 | Use of neural implants and brain-computer interfaces in patients with spinal cord paralysis

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EPV-1575 | When a benign lesion becomes aggressive: Vertebral hemangioma

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ABSTRACTS

Late Breaking Abstracts

Monday, June 29 2026

Late-breaking Oral Session

LB_01 | Central disorders of hypersomnolence: linking CSF hypocretin-1 to symptom severity

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Background and aims: To identify non-cataplexy symptoms indicating hypocretin-1 deficiency, and to investigate the association between CDH symptom presentation and varying CSF hypocretin-1 levels.

Methods: We included 302 CDH individuals from the European Narcolepsy Network database. Hypocretin-1 levels were categorized as ≤ 40 , 41–110, 111–200, and > 200 pg/mL. Machine learning (stochastic gradient boosting) was used to explore non-cataplexy predictors of hypocretin-1 deficiency. Logistic and quantile regression models examined associations between absolute hypocretin-1 levels and demographic, subjective symptom, polysomnography, and multiple sleep latency test (MSLT) measures, adjusted for age, sex, and body mass index. Cataplexy features were analyzed among individuals with cataplexy and hypocretin-1 levels ≤ 200 pg/mL.

Results: Non-cataplexy variables predicted degrees of hypocretin-1 deficiency with moderate certainty. In all individuals, lower hypocretin-1 levels were associated with shorter sleep latency and rapid eye movement sleep latency on both polysomnography and MSLT, more MSLT sleep-onset rapid eye movement periods, higher Epworth Sleepiness Scale scores, increased periodic limb movement index, and more frequent disturbed nocturnal sleep. Among individuals with cataplexy and hypocretin-1 levels ≤ 200 pg/mL, lower levels were associated with earlier excessive daytime sleepiness onset, younger age at diagnosis, greater diagnostic certainty of hypnagogic hallucinations and sleep paralysis, and more frequent and complete cataplexy attacks.

Conclusion: Lower hypocretin-1 levels were associated with more severe symptoms, both in the overall CDH population and in individuals with cataplexy and low hypocretin. Even within the deficient range, hypocretin-1 variation contributed to individual differences in symptom presence and severity.

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Takeda and Jazz; received a speaker fee from Pharmanovia; and is a member of a Safety Review Committee for Centessa. Rolf Fronczek did advisory work for and received research support from Takeda and Jazz; received a speaker fee from Pharmanovia; and is a member of a Safety Review Committee for Centessa. Geert Mayer received consulting fees from Takeda; received honoraria for a lecture from Löwenstein Medical; and received support for attending meetings and/or travel from Bioprojet. Markku Partinen reports no competing interests. Sebastiaan Overeem received consulting fees paid to his institution from Bioprojet, Takeda, Jazz Pharmaceuticals, and UCB Pharma. Joan Santamaria reports no competing interests. Karel Šonka participated in Alkermes-sponsored clinical trials. Claudio L. Bassetti received research support from the Swiss National Science Foundation and the University of Bern (IRC Grant); received consulting fees from Takeda and Idorsia; and received honoraria for educational events from Boehringer Ingelheim and Takeda. Julia van der Meer reports no competing interests. Rafael del Río-Villegas received consultancy fees from Alkermes, Bioprojet, and Takeda; and travel funds from Bioprojet, Jazz, and Takeda. He is a member of the International Board for Alkermes and Takeda. Christian Veauthier serves as Chair of the Berufsverband Deutscher Schlafmediziner (BDS e.V.) and as Secretary of the Deutsche Gesellschaft für Schlafforschung und Schlafmedizin (DGSM e.V.). Silvia Miano reports no competing interests. Rosa Peraita-Adrados reports no competing interests. Ulf Kallweit received consulting fees from Takeda and Bioprojet; received honoraria for educational lectures from Bioprojet and Pharmanovia; served on advisory boards for Takeda and Bioprojet; and serves as a Board Member of the European Academy of Neurology (EAN) and Vice-Speaker of the Committee on Hypersomnias of the German Sleep Society (DGSM), both unpaid roles. Eva Feketeova reports no competing interests. Jitka Bušková reports no competing interests. Claire E.H.M. Donjacour reports no competing interests. Ramin Khatami received consulting fees from Takeda and Idorsia; received honoraria for lectures from Idorsia and Takeda; and served on advisory boards or data safety monitoring boards for Takeda and Idorsia. Gert Jan Lammers received consulting fees from Takeda, Bioprojet, Alkermes, Jazz, and Daiichi Sankyo; received honoraria for a lecture from Bioprojet; and received support for attending meetings and/or travel from Takeda and Bioprojet.

LB_02 | Apathy marks brain network and genetic vulnerability to cognitive decline in Parkinson's disease

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Background and aims: Motivational deficits such as apathy and impulse-control behaviours (ICBs) are common in Parkinson's disease (PD), with increasing evidence suggesting their relevance to cognitive function. However, the mechanisms by which they influence cognitive trajectories remain under investigation.

Methods: Using the Parkinson's Progression Markers Initiative (PPMI), we examined apathy and ICBs in relation to longitudinal cognition, resting-state functional connectivity, structural imaging, and genetic susceptibility in early PD.

Results: In 1502 participants at baseline, apathy-containing phenotypes were comparatively stable over time, whereas isolated ICB was more transient, consistent with a stronger medication-linked component. Across up to 15 years of follow-up, higher baseline apathy robustly predicted steeper decline in both latent global cognition and Montreal Cognitive Assessment performance, independent of demographic, motor, depressive, and treatment covariates; ICB-related effects on decline were weak and not robust. In resting-state functional MRI ($n=310$), the Network-Based Statistic identified an apathy-related somato-motor–dorsal-attention connectivity signature independent of disease severity and dopaminergic medication, whereas any ICB-related connectivity attenuated after levodopa-equivalent dose adjustment. Lower connectivity within this subnetwork predicted steeper cognitive decline, mediated the apathy–decline association in an age-dependent manner, and tracked smaller hippocampal volume and faster subsequent atrophy. Apathy-related cognitive decline was selectively amplified in GBA-associated PD, with apathetic GBA carriers declining approximately five-fold faster than non-apathetic sporadic cases.

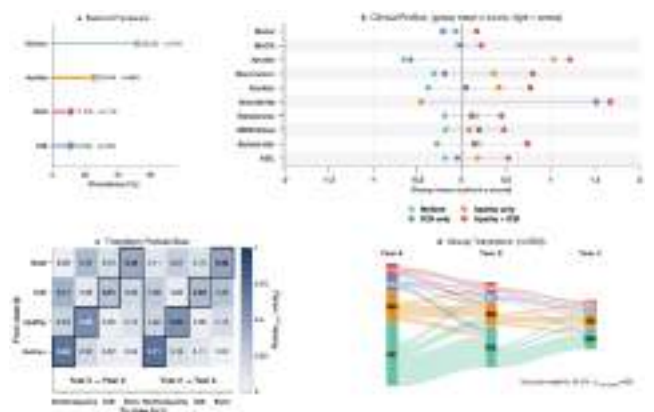


FIGURE 1 Apathy-ICB co-occurrence and longitudinal stability in early Parkinson's disease.

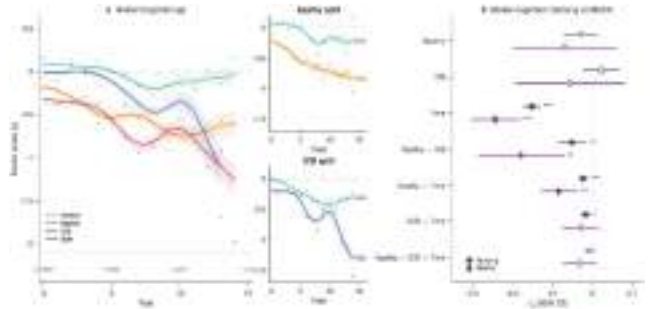


FIGURE 2 Apathy preferentially predicts faster global cognitive decline in early Parkinson's disease.

Conclusion: pathy, rather than ICB severity, identifies a stable and clinically meaningful motivational marker of adverse cognitive trajectory in early PD, linked to a specific connectivity phenotype and amplified by GBA status.

Disclosure: None.

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Background and aims: Hypomyelinating leukodystrophies (HLDs) are traditionally defined radiologically by persistent hypomyelination on brain MRI. However, many rare HLD-associated genes remain represented only by isolated reports or very small case series, limiting understanding of their full clinical, radiological, and biological spectrum.

Methods: We performed retrospective multicentre deep phenotyping and neuroradiological review of individuals with pathogenic variants in several rare HLD-associated genes, including FAM177A1, AIMP1, AIMP2, HSPD1, EPRS1, and NKX6.2.

Results: Cross-cohort analysis identified reproducible clinical subgroups, including classic Pelizaeus–Merzbacher disease-like HLD, movement-predominant HLD, epileptic HLD, peripheral neuropathy-associated HLD, and syndromic/systemic HLD phenotypes. Notably, approximately one-third of individuals with EPRS1-related disease lacked hypomyelination despite significant neurological involvement. Prominent movement disorders, particularly dystonia and tremor, were frequent in HSPD1- and NKX6.2-related disease. Seizures, often severe or refractory and associated with developmental impairment, were core features in AIMP1- and AIMP2-related disease. Progressive macrocephaly emerged as a consistent feature in individuals with FAM177A1 deficiency.

Conclusion: These findings suggest that MRI-defined HLD classifications do not fully capture the clinical and biological heterogeneity of these disorders. An integrated framework combining clinical subgrouping with imaging and molecular diagnosis may better reflect disease mechanisms, improve diagnostic interpretation, and guide future research.

Disclosure: Nothing to disclose.

LB_04 | Early immune activation in the preclinical phases of immune-mediated neurological diseases

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Background and aims: The temporal relationship between disease-specific autoantibodies and biomarkers of CNS injury before diagnosis of multiple sclerosis (MS), myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), and aquaporin-4-IgG-positive neuromyelitis optica spectrum disorder (NMOSD) remains unknown. The objective was to elucidate the preclinical trajectory of EBNA-1-specific, AQP4 as well as MOG antibodies and their association with CNS injury to improve understanding of early pathobiology and biomarker-based risk stratification.

Methods: We performed a multicenter, retrospective, longitudinal case-control study with pre-diagnostic plasma samples from 362 individuals who later developed MS, 145 who later developed MOGAD, and 60 who later developed NMOSD. Plasma IgG levels directed against CNS antigens, MOG, and AQP4, as well as plasma concentrations of neurofilament light chain (pNfL), were quantified, and temporal relationships between immune activation and neuroaxonal injury and clinical disease onset were modeled using linear mixed-effects models and survival analyses.

Results: In MS, EBNA-1-specific and CNS-cross-reactive IgG were elevated up to 77.8 months before diagnosis and preceded first increases in pNfL by mean intervals of 44.9 months, respectively. In NMOSD, AQP4-IgG seroconversion occurred a mean of 32.5 months before diagnosis and preceded elevations in pNfL by 40.4 months, respectively. In contrast, in MOGAD, elevations in pNfL preceded MOG-IgG seroconversion by 11.2 months, respectively.

Conclusion: In MS and NMOSD, disease-specific humoral autoimmunity precedes detectable blood biomarkers of CNS injury, whereas in MOGAD, neuroaxonal and astroglial injury becomes evident before circulating MOG-IgG. These distinct temporal patterns suggest different early pathophysiological mechanisms and may inform early diagnosis and intervention strategies.

Disclosure: The study was funded by the Center for Virology (Medical University of Vienna, Vienna), the Department of Neurology (Medical University of Vienna, Vienna), the Austrian Science Fund (FWF): SYNABS, I6565-B, the Austrian Research Promotion Agency (FFG, project number FO999920011) and the Austrian Society of Neurology, Austria.

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Background and aims: CASPR2-associated disease (CASPR2-ad) is a rare autoimmune neurological disorder characterized by recurrent clinical events and frequent long-term disability. We aimed to assess the effect of baseline features and treatments on disease course and neurological outcomes.

Methods: All patients testing positive for anti-CASPR2 antibodies at the French Reference Centre for Autoimmune Encephalitis (2011–2025) were retrospectively included. Neurological outcomes, mortality, and clinical event rate were analyzed using generalized linear mixed models, stratified Cox models, and Andersen-Gill counting-process models, with treatments as time-varying covariates.

Results: Among 171 patients (89% male; median age 66 years; median follow-up, 3 years [IQR 1.3–4.8]), the predominant phenotypes were limbic encephalitis (73%), Morvan syndrome (12%), and Isaacs syndrome (9%). Mortality differed markedly across syndromes ($p < 0.001$; Fig. 1A), with thymoma as the only independent predictor (HR 4.59 [1.26–16.73], $p = 0.021$; Fig. 1B). In two-year survivors, higher baseline mRS and female sex were independently associated with worse neurological outcome (OR 0.19 [0.09–0.39], $p < 0.001$ and OR 0.12 [0.02–0.71], $p = 0.02$) while Morvan syndrome was associated with higher odds of favorable outcomes (OR 12.06 [1.24–117.35], $p = 0.032$; AUC 0.868; Fig. 2). Active rituximab therapy was independently associated with a 56% reduction in clinical event rate (HR 0.44 [0.25–0.77], $p = 0.004$; Fig. 3), with a stronger effect in limbic encephalitis patients (HR 0.35 [0.18–0.66], $p = 0.001$).

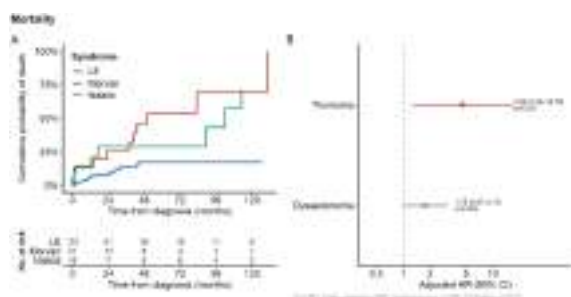


FIGURE 1 Mortality.

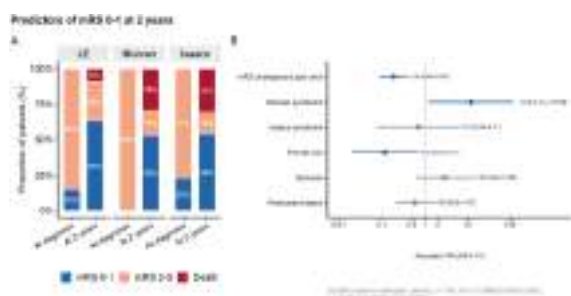


FIGURE 2 Neurological outcomes at 2 years.

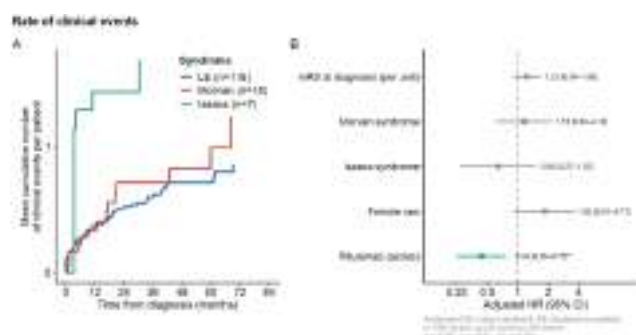


FIGURE 3 Rate of clinical events (patients with ≥ 6 months follow-up).

Conclusion: Baseline severity and thymoma are the main determinants of neurological outcome and mortality, respectively. Rituximab markedly reduces clinical event rate, supporting its use to control CASPR2-ad activity. These findings are limited by the retrospective design and potential confounding by indication.

Disclosure: This study has been developed within the BETPSY project (ANR-18-RHUS-0012).

LB_06 | Clemastine as remyelinating treatment in patients with multiple sclerosis and internuclear ophthalmoplegia

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Background and aims: Remyelination failure contributes to disability and disease progression in multiple sclerosis (MS). Clemastine has shown promise for therapeutic remyelination in optic neuropathy, but its efficacy in other neural pathways remains uncertain. A key area to explore is the brainstem where lesions cause internuclear ophthalmoplegia (INO) in 25% of people with MS, which can transiently improve with fampridine. We investigated whether clemastine promotes brainstem remyelination and evaluated fampridine response as a remyelination predictor.

Methods: In this RCT, 47 people with MS and INO received 4 mg clemastine or placebo twice daily for 6 months. Horizontal saccades conjugacy, a measure of brainstem myelination, was quantified by infrared oculography at baseline, 3, 6, 12, 24 and 36 months. The primary outcome measure was the Versional

Figure 1 consists of three panels, A, B, and C, each showing a graph of Horizontal eye position (degrees) on the y-axis (ranging from 0 to 16) versus Time (ms) on the x-axis (ranging from 0 to 600). The legend indicates that the red line represents the Left eye and the blue line represents the Right eye. Panel A shows the Left eye (red) rising to a steady state of approximately 14 degrees, while the Right eye (blue) rises more rapidly to a peak of about 15.5 degrees at 100 ms before settling at the same level. Panel B shows both eyes rising to a steady state of approximately 14 degrees, with the Right eye (blue) rising slightly faster than the Left eye (red). Panel C shows both eyes rising to a steady state of approximately 14 degrees, with the Right eye (blue) rising very rapidly to the steady state within about 50 ms.

Results: The trial ended on 15-Aug-2025. Positive fampridine response was observed in 23 subjects (49%), with a balanced distribution between clemastine ($n=12$) and placebo ($n=11$) groups (Table 1). Fampridine response was associated with VDI improvement over time ($p=0.004$). Full treatment efficacy data will be presented at the EAN conference.

Table 1. Baseline characteristics		
	Gemtane N = 24	Placebo N = 23
Demographics		
Age, years	51 ± 10	51 ± 8
Gender, female	8 (33%)	16 (70%)
Disease duration from onset (years)	19 (5, 33)	16 (12, 24)
MS (subtype)		
RRMS	13 (54%)	10 (43%)
SPMS	9 (38%)	9 (39%)
PPMS	2 (8.3%)	7 (30%)
DMT use	16 (67%)	18 (78%)
DMT type		
High efficacy DMT	11 (46%)	10 (43%)
Moderate efficacy DMT	5 (21%)	5 (22%)
Low efficacy DMT	0 (0%)	1 (4.3%)
EDSS	4.3 (3.8, 5.5)	4.4 (4.0, 4.8)
EDSMT	45 (34, 55)	45 (36, 49)
LCJA (months)	26 ± 11	21 ± 16
HCJA (months)	59 (45, 80)	57 (40, 78)
gRISQ, (cm)	88 ± 13	90 ± 13
HCFLPL (cm)	88 ± 13	78 ± 17
Oculoplethysmography		
INO		
Bilateral INO	16 (67%)	13 (57%)
Unilateral INO	4 (17%)	5 (22%)
Bilateral PNO	8 (33%)	5 (22%)
Painful responders	12 (50%)	11 (48%)
Baseline VDI-4-UC	3.88 (3.32, 4.78)	3.87 (3.20, 4.78)

Mean (SD) or (%). Median (Q1, Q3)

Abbreviations: DMT = Disease Modifying Therapy; EDSS = Expanded Disability Status Scale; HCJA = High-Contrast Letter Acuity; INO = Intraocular Oculoplethysmography; LCJA = Low-Contrast Letter Acuity; RRMS = Relapsing-Remitting MS; SPMS = Secondary Progressive MS; PPMS = Primary Progressive MS; SGMT = Symbol Gist Modulator Test; VDI-4-UC = Visual Disability Index of Area Under the second Curve; INOOR, = Intraocular Oculoplethysmography (all inner Retiniform Layer); gRISQ = Retiniform Reflexive Gaze Scale

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Late-breaking ePoster Session

F. Nuti¹, M. Valletta¹, D. Vetrano¹, E. Laukka¹, G. Kalpouzos¹,
M. Canevelli¹, C. Fredolini³, G. Bruno², G. Grande¹

Background and aims: Growth differentiation factor 15 (GDF-15) is a circulating cytokine reflecting biological aging linked to cognitive decline. Evidence on brain structural correlates is limited. We investigated the associations of blood GDF-15 with brain volumes and cerebrovascular burden in community-dwelling older adults free from dementia.

Methods: We included 400 community-dwelling participants from the Swedish National Study on Aging and Care in Kungsholmen (SNAC-K), free from major baseline cerebrovascular and neurological diseases. GDF-15 was measured at baseline and analyzed in tertiles. Brain MRI was performed at baseline and after 3 and/or 6 years. Linear mixed models were adjusted for age, sex, education, and chronic comorbidities; secondary analyses further accounted for blood p-tau217.

Fluorometer	Overall N = 362	T1 N = 154	T2 N = 153	T3 N = 155	p-value
Demographic					
Age (years)					<0.01
40-49	112 (31.2%)	60 (39.0%)	59 (38.6%)	34 (21.9%)	
50-59	101 (28.2%)	60 (39.0%)	55 (35.9%)	37 (23.8%)	
70-79	101 (28.2%)	53 (34.4%)	51 (33.3%)	28 (17.9%)	
≥80	48 (13.4%)	11 (7.1%)	89 (58.2%)	74 (47.4%)	
Sex (male)	112 (31.2%)	60 (39.0%)	60 (39.2%)	60 (38.9%)	0.98
Ethnicity, unknown	162 (44.8%)	97 (62.9%)	112 (73.2%)	44 (27.9%)	<0.01
Hispanic/Latino, non-Hispanic/Latino	28 (7.7%)	26 (16.9%)	26 (16.9%)	26 (16.8%)	0.002
Health domain					
Chronic kidney disease	36 (9.9%)	11 (7.1%)	12 (7.8%)	12 (7.7%)	<0.01
Heart failure	61 (16.9%)	33 (21.4%)	34 (22.2%)	34 (21.9%)	<0.01
Stroke	18 (5.0%)	11 (7.1%)	11 (7.2%)	16 (10.3%)	0.01
Cardiovascular MRI measures					
Prevalence of left atrial enlargement	166 (45.8%)	107 (69.5%)	109 (71.2%)	109 (70.3%)	<0.001
Volume (mL)	31.0 (4.5)	31.0 (4.5)	31.0 (4.5)	31.0 (4.5)	0.98
Myocardial mass (g)	5.5 (0.45)	5.5 (0.45)	5.5 (0.45)	5.5 (0.45)	0.98
Left atrial volume (mL)	150 (39.5)	140 (91.5%)	139 (91.5%)	135 (87.1%)	<0.001
Left atrial volume index (mL/m ²)	3.7 (0.5)	3.7 (0.5)	3.7 (0.5)	3.7 (0.5)	0.98

Results: Mean age was 70 (SD 9) years; 58% women. Higher GDF-15 tertiles, adjusted for multiple confounders, showed an association with faster atrophy of total brain tissue (T3 vs T1: β -year = -0.026, 95% CI -0.041, -0.010), hippocampus

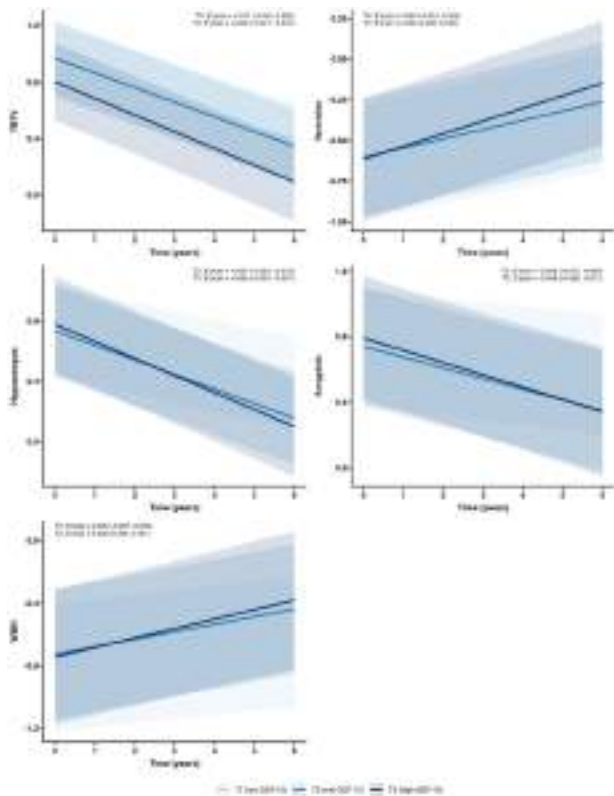


FIGURE 1 Annual rate of change in volumes of total brain tissue (TBT), lateral ventricles, hippocampus, amygdala, and white matter hyperintensities (WMH) accumulation over time, by baseline tertiles levels of GDF-15.

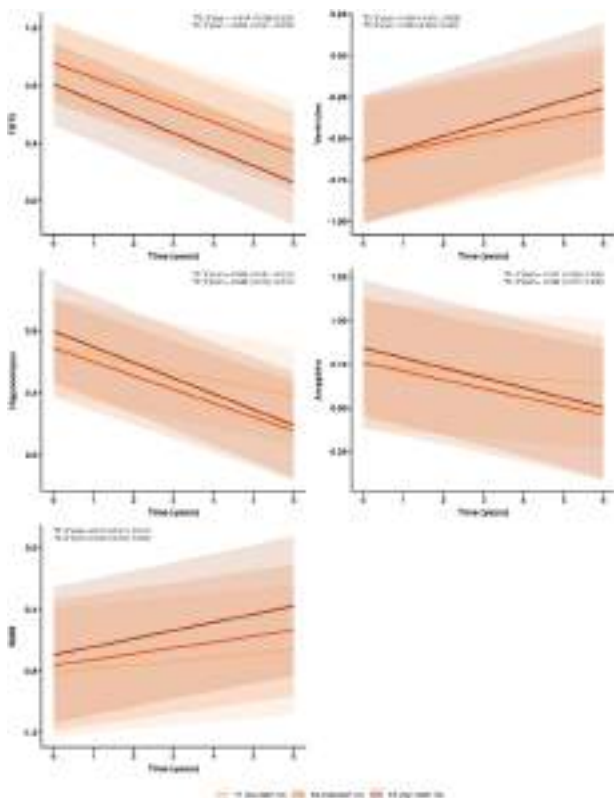


FIGURE 2 Annual rate of change in volumes of total brain tissue (TBT), lateral ventricles, hippocampus, amygdala, and white matter hyperintensities (WMH) accumulation over time, by baseline tertiles levels of GDF-15 adjusted for baseline ptau217 levels.

(β -year = -0.054 , 95% CI -0.081 , -0.027), and amygdala (β -year = -0.048 , 95% CI -0.085 , -0.011), and with faster enlargement of the ventricles (β -year = $+0.026$, 95% CI 0.006 , 0.045) and accumulation of white matter hyperintensities (WMH) (β -year = $+0.032$, 95% CI 0.003 , 0.061) over time. Findings were robust to comorbidity adjustment and APOE $\epsilon 4$; associations with total brain tissue, hippocampal, and ventricular trajectories remained significant after adjustment for blood p-tau217.

Conclusion: Higher GDF-15 levels were associated with faster atrophy of total brain tissue, hippocampus, and amygdala, and with ventricular enlargement and WMH over 6 years, supporting GDF-15 as a biomarker of accelerated age-related brain atrophy.

Disclosure: Nothing to disclose.

LB_08 | Real-world clinical outcomes with lecanemab in APOE $\epsilon 4$ heterozygotes and non-carriers: findings from the LEADER study

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Background and aims: Real-world data describing lecanemab clinical and safety outcomes by apolipoprotein E (APOE $\epsilon 4$) status, including amyloid-related imaging abnormalities (ARIA) incidence, remain limited. This interim analysis from LEADER characterizes clinical outcomes with lecanemab in APOE $\epsilon 4$ heterozygotes and non-carriers (APOE $\epsilon 4$ homozygotes were excluded to align with the ex-US label).

Methods: LEADER is a multicenter, retrospective, real-world study conducted across 15 geographically diverse U.S. neurology practices. Sites abstracted deidentified medical chart data for patients who received ≥ 7 biweekly intravenous lecanemab infusions. This descriptive analysis assessed clinician-reported

Results: Of 309 patients in LEADER, 256 were included in this analysis (excluding APOE $\epsilon 4$ homozygotes): 155 (50.2%) were heterozygous and 101 (32.7%) were noncarriers. ARIA—edema/effusion (ARIA-E) occurred in 12 (7.7%) heterozygotes and 7 (6.9%) noncarriers. No intracerebral hemorrhages (> 1 cm) occurred. Mean treatment duration at last follow-up was 471.3 and 437.6 days, respectively. Over the observation period, 85.2% of heterozygotes were stable or improved (80.0% stable; 5.2% improved) and 87.1% of noncarriers were stable or improved (81.2% stable; 5.9% improved).

[illegible]

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LB_09 | Bocunebart (anti-PACAP monoclonal antibody Lu AG09222) in adults with migraine and prior preventive treatment failures: Phase 2 program outcomes

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Methods: The phase 2 program comprised the proof-of-concept phase 2a HOPE trial (NCT05133323) and the adaptive phase 2b PROCEED trial (NCT06323928), evaluating dose and route of administration. The PROCEED primary endpoint was change from baseline in monthly migraine days (MMDs) over Weeks

1–12. Efficacy in participants with chronic migraine (CM) was evaluated in pooled post-hoc analyses across all doses and parts of PROCEED+HOPE.

Results: For PROCEED (intravenous part), the primary endpoint was met; a statistically significant reduction in MMDs was observed with bocunebart dose A versus placebo over Weeks 1–12 (−4.24 [standard error 0.63] vs −2.86 [0.53]; $p=0.0178$ [2-sided]). Over Weeks 9–12, in the pooled CM subgroup (PROCEED+HOPE), pooled bocunebart doses demonstrated greater improvements from baseline versus placebo in MMDs (−5.94, [0.42] vs −3.63 [0.53]; $p<0.001$), monthly headache days (−6.90, [0.42] vs −4.32 [0.54]; $p<0.001$) and MMDs with acute medication use (−5.21, [0.39] vs −3.53 [0.50]; $p<0.05$). Bocunebart was generally well tolerated; no new safety signals were identified.

Conclusion: Positive phase 2 findings support PACAP inhibition as a novel approach in migraine prevention. Overall, bocunebart reduced migraine burden in a severely impacted population with high unmet need. These findings support bocunebart's continued development as a potential first-in-class anti-PACAP mAb.

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LB_10 | ROSSINI 12-month interim results: Safety and effectiveness of 24-hour Foslevodopa/Foscarbidopa in Parkinson's disease

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Background and aims: Foslevodopa/foscarbidopa (LDp/CDp) is a nonsurgical 24-hour/day continuous subcutaneous infusion for patients with advanced Parkinson's disease (aPD) whose motor fluctuations are uncontrolled on oral medications. Twelve-month safety and effectiveness data from routine clinical practice were captured.

Methods: ROSSINI (NCT06107426) is an ongoing 3-year multi-country, prospective, observational study of adults with aPD who are LDp/CDp-naïve (cohort A) or transitioning from LDp/CDp

TABLE 1 Baseline characteristics and overview of adverse events through 12 months of LDp/CDp treatment in the second interim analysis population (SAS).

Baseline characteristic	n	Cohort A (n = 205)
Age, years, mean (SD)	72.1	68.1 (8.1)
< 65 years, %	66	32.8
≥ 65 years, %	125	67.2
Sex, male, %	122	65.7
Country, %		
Austria	13	6.0
Canada	7	3.5
Denmark	17	8.5
Germany	85	46.5
Israel	33	18.4
Romania	12	6.5
Spain	33	17.4
Sweden	15	7.5
United States	2	1.0
PD duration, years, mean (SD)	19.0	12.1 (8.7)
< 10 years, %	19	9.7
≥ 10 years, %	125	65.3
Time since start of motor fluctuations, years, mean (SD)	18.7	11.7 (8.3)
Previous use of device-aided treatment, %	8	4.0
MMSE score, mean (SD)	19.9	27.4 (3.2)
Hoehn and Yahr disease stage score, mean (SD)	1.9	2.0 (0.8)
OFF time hours (modified MDS-UPDRS-IV, item 4.1), mean (SD)	11.3	4.8 (2.4)
Cykloset time hours (modified MDS-UPDRS-IV, item 4.1), mean (SD)	1.8	1.2 (0.5)
MDS-UPDRS-IV total score, mean (SD)	14.1	17.9 (9.3)
PDQ-39 summary index score, mean (SD)	1.9	38.5 (18.5)
AE overview, n (%)		
Any AE	148	74.1
Severe AE	48	23.9
Serious AE	57	28.4
AE leading to withdrawal of study treatment	23	11.4
Death	8	3.0
AEs occurring in ≥ 1% of patients		
Halucination	23	11.4
Abuse	15	7.5
Skin issues	13	6.6
Fall	12	6.0
On and off phenomenon	12	6.0
Injection site infection	10	5.0
Nausea	10	5.0
Skin infection	10	5.0
Skin reaction	7	3.5
Abdominal issues	7	3.5
Celulitis	7	3.5
Infection site cellulitis	6	3.0
Infection site erythema	6	3.0
Urinary tract infection	5	2.0
Serious AEs occurring in ≥ 1% of patients		
On and off phenomenon	7	3.5
Halucination	5	2.5
Abdominal wall issues	3	1.5
Abuse	3	1.5
Skin infection	3	1.5
*From the SAS. †This list includes events occurring after the 12-month visit and within the data cutoff. ‡One death was due to pneumonia; one to lymphoma; pleural effusion; one to drug-induced (metoprolol) severe Type A/B/C gastritis; (cyclosporin), aspiration pneumonia; two with unknown causes; and one to unknown cause. All deaths were considered unrelated to study treatment and/or occurred after the 12-month visit. Abbreviations: AE, adverse event; FAS, full analysis set; LDp/CDp, foslevodopa/foscarbidopa; MDS-UPDRS-IV, Movement Disorder Society-United Parkinson's Disease Rating Scale Part II or IV; MMSE, Mini-Mental State Examination; PD, Parkinson's disease; PDQ-39, 39-item Parkinson's Disease Questionnaire; SAS, safety analysis set.		

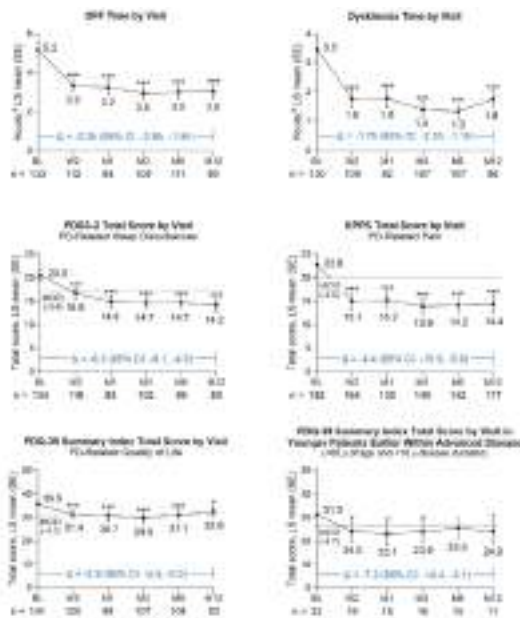


Figure 1. LS means by visit for OFF time, dyskinesia time, PDSS-2, KPPS, and PDQ-39 following LDp/CDp treatment in the second interim analysis population (FAS, N = 189). * $P < .05$, ** $P < .01$, *** $P < .001$ for LS mean difference from baseline to time point. Dashed lines indicate thresholds for meeting defined MCID from baseline. Values represent LS mean change from baseline to M12, estimated from a mixed model for repeated measures that includes visit and country as fixed effects, along with age group, baseline OFF time, disease duration, and time since motor fluctuation (primary). *As assessed by the modified MDS-UPDRS-IV, item 4.3. *As assessed by the modified MDS-UPDRS-IV, item 4.1. Abbreviations: BL, baseline; FAS, full analysis set; GDS-PD, Gastrointestinal dysfunction in PD scale; H, hour; KPPS, King's Parkinson's Disease Pain Scale; LDp/CDp, levodopa/carbidopa; LS, least squares; M, month; MDS-UPDRS-IV, Movement Disorder Society Unified Parkinson's Disease Rating Scale Part IV; MCID, minimal clinically important difference; PDSS-2, Parkinson's Disease Sleep Scale-2; PDQ-39, 39-item Parkinson's Disease Questionnaire; SE, standard error; W, week.

TABLE 2 LS mean by visit and change from baseline to month 12 for additional effectiveness outcomes among patients on LDp/CDp treatment in the second interim analysis population (FAS, N 0 189).

Outcome score, n, LS mean (SE)	Baseline	Week 2	Month 1	Month 3	Month 6	Month 12	CFB to Month 12, (95% CI)
GDS-PD, PD-related GI dysfunction	125 19.8 (3.6)	111 18.0 (3.8)	85 17.2 (3.9)	96 16.3 (3.9)**	94 16.6 (3.9)**	76 16.7 (3.5)*	-3.1 (-5.6, -0.6)
MDS-UPDRS-II, mCS	131 17.8 (3.5)	118 15.4 (3.9)**	76 16.4 (3.8)	96 15.8 (3.7)**	97 16.2 (3.5)**	76 16.3 (3.4)*	-1.6 (-2.9, 0.2)
MDS-UPDRS-III, Motor symptoms	179 31.7 (3.8)	159 29.8 (3.9)**	121 30.1 (3.9)**	139 31.7 (3.9)**	128 31.0 (3.9)**	92 30.8 (3.7)**	-5.8 (-8.0, -3.6)
PDQ-39, Freedom of gal	121 10.7 (3.0)	116 6.4 (3.6)**	86 8.7 (3.8)**	96 7.5 (3.8)**	96 8.5 (3.8)**	76 8.8 (3.8)**	-1.9 (-3.0, -0.7)
ESS, Sleep-related symptoms	133 8.4 (3.7)	115 7.1 (3.7)	86 7.7 (3.9)	102 7.2 (3.7)**	98 7.3 (3.7)**	60 7.9 (3.8)	-0.4 (-1.7, 1.1)
BDI, Depressive symptoms	132 13.8 (3.5)	122 11.8 (3.5)**	86 11.2 (3.4)**	106 10.7 (3.5)**	101 12.1 (3.5)**	81 12.3 (3.4)*	-0.8 (-2.0, 0.1)
MCSI, Cognitive symptoms	81 8.8 (3.0)	55 8.4 (3.0)	48 8.1 (3.0)	40 8.0 (3.0)	48 8.9 (3.0)	41 8.4 (3.1)	0.0 (-1.7, 2.0)

* $P < .05$, ** $P < .01$, *** $P < .001$ for LS mean difference from baseline to time point. CFB values represent LS mean change from baseline to M12, estimated from a mixed model for repeated measures that includes visit and country as fixed effects. Abbreviations: BDI, Beck Depression Inventory; CFB, change from baseline; ESS, Epworth Sleepiness Scale; FAS, full analysis set; PDQ-39, Freedom of Gal Questionnaire; LDp/CDp, levodopa/carbidopa; GDS-PD, Gastrointestinal Dysfunction Scale in Parkinson's disease; LS, least squares; MCSI, Modified Caregiver Strain Index; mCS, motor symptoms of daily living; MDS-UPDRS-II, III, or IV, Movement Disorder Society Unified Parkinson's Disease Rating Scale Part II, III, or IV; PD, Parkinson's disease; PDQ-39, 39-item Parkinson's Disease Questionnaire.

open-label extension studies (NCT04379050/NCT04750226, cohort B). Second interim results for cohort A patients (N=202) enrolled ≥ 12 months (M) before 16December2025 are presented. Adjusted mixed-effects models for repeated measurements (continuous outcomes) were utilized.

Results: At baseline, patients had a mean (SD) age of 68.1 y (9.1 y), PD duration of 12.1 y (5.7 y), and OFF time of 4.8 h (2.6 h, Table 1). From initial prescription to M12, mean daily LD equivalent dose during infusion (1414.7 mg–1471.9 mg) and LDp/CDp base infusion rates (58.9 mg/h LD–67.1 mg/h LD) moderately increased; 21/201 (10.4%) patients received LDp/CDp as a monotherapy until final visit/M12. Patients on LDp/CDp showed statistically significant sustained reductions in motor fluctuations and nonmotor symptoms by M12, including in King's Parkinson's Disease Pain Scale scores (–8.4; Figure 1, Table 2). Patients < 65 y of age and < 10 y PD duration ($n = 22$) showed numerically larger reductions in 39-item Parkinson's Disease Questionnaire scores than the overall population (Figure 1). For safety, 149/201 (74.1%) patients reported ≥ 1 adverse event, mostly nonserious and mild-moderate, with hallucinations and infusion site events most frequent (Table 1).

Conclusion: ROSSINI interim results demonstrate sustained reductions in PD symptoms following 12M of LDp/CDp treatment with an acceptable safety profile.

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Teva, UCB, LB, SP, SC, MG, PK, MO, JCP, MBS: AbbVie, JA: Abbott, AbbVie, AC Immune, Allergan (now AbbVie), Annovis, Aptinyx, AstraZeneca, Atara, Athira, Biogen, Biovie, Boston Scientific, Celgene, Cerevance, Cerevel, Denali, EIP, Eli Lilly, Impax, Inhibikase, IRL Therapeutics, Medtronic, Merz, Neuraly, Neurocrine, Neuoderm, Novartis, PD Gene/PSG, Praxis, Revance, Roche/Genentech, Sage, Sanofi/Genzyme, Scion Neurostem, Takeda, Teva, Theravance, Triplet/HSG, UCB, US World Meds.

LB_11 | Efficacy and safety of Efgartigimod PH20 SC in adults with ocular myasthenia gravis: ADAPT OCULUS interim results

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Background and aims: There is an unmet need for approved and effective treatments for patients with ocular myasthenia gravis (oMG). Subcutaneous (SC) efgartigimod PH20 is a human immunoglobulin G1 (IgG1) antibody Fc fragment coformulated with recombinant human hyaluronidase PH20 that selectively reduces IgG levels by blocking neonatal Fc receptor-mediated IgG recycling. Retrospective data analyses supporting the approval of efgartigimod for treatment of generalized MG indicated improvements in ocular symptoms. Here, we present interim results from the Phase 3 ADAPT OCULUS trial (NCT06558279) evaluating the efficacy and safety of efgartigimod PH20 SC in adults with oMG.

Methods: Adults with oMG (MGFA Class I) and Myasthenia Gravis Impairment Index (patient-reported outcome; MGII [PRO]) ocular score ≥ 6 were randomized 1:1 to receive 4 once-weekly efgartigimod PH20 SC 1000 mg or placebo injections via prefilled syringe, with 4 weeks of follow-up. Participants could continue to Part B, a ≤ 2 -year open-label period.

Results: The primary endpoint was met with statistically significant ($p = 0.012$) improvement from baseline in MGII (PRO) ocular score at Week 4 with efgartigimod compared with placebo. Least squares mean change from baseline to Week 4 in MGII

(PRO) ocular score was -4.04 points with efgartigimod versus -1.99 for placebo. A reduction in all ocular symptoms was reported, and no new safety concerns were identified.

Conclusion: ADAPT OCULUS is the first registrational trial designed specifically to evaluate a targeted therapy for patients with oMG. These results support the efficacy and safety of efgartigimod PH20 SC treatment across multiple cycles in participants with oMG.

Disclosure: VCJ, CZ, and JX have nothing to disclose. OL, RHJ, FG, and FMV are employees of argenx. CB-T, AM, JFHJ, AU, SWR, KGC, CA, LR, RÁ-V, and SHW have reported financial/nonfinancial relationships with argenx at the time of submission

LB_12 | Neuronal apoptosis redirects disease-associated microglial states in presymptomatic Alzheimer's iPSC models

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Background and aims: This study investigated how microglia from presymptomatic AD patients respond to an AD-like neuronal environment using patient-derived induced pluripotent stem cell (iPSC) models.

Methods: Twenty iPSC lines from presymptomatic AD participants from the Deep and Frequent Phenotyping study were differentiated into microglia. iPSC-microglia were exposed to non-apoptotic (NAN) or apoptotic neurons (AN) and analysed by scRNA-sequencing. Following quality control, 14 lines (11 APOE3/E4, 3 APOE4/E4) were retained. Data were preprocessed using Panpipes and annotated using a published microglia atlas. Clustering and batch correction were performed with Monocle3, while trajectory inference and pseudotime-associated gene analysis were performed using Slingshot and tradeSeq. Differential expression and gene ontology enrichment analyses were conducted using Seurat and ClusterProfiler.

Results: Trajectory analysis identified two trajectories leading from cycling to disease-associated microglia (DAM). Trajectory towards GPNMB-high CXCR4+ TREM2+ Lipox-DAM via TREM2+ IFN responsive microglia was preferred in NAN environment. Alternatively, when subjected to AN, trajectory towards LPL-high TREM2+ DAM via MITO-high DAM was more prominent. Comparing microglia subtype ratios between NAN and AN environments showed a decrease in TREM2+ IFN responsive microglia (14.2% in NAN to 1.0% in AN) and an increase in MITO-high DAM (7.2% in NAN to 15.3% in AN). Compared with TREM2+ IFN-responsive microglia, MITO-high DAM showed enrichment of endocytosis-related pathways and reduced immune-response signatures.

Conclusion: Our findings suggest that neuronal apoptosis redirects presymptomatic AD microglia toward a metabolically active, endocytosis-associated DAM state while reducing IFN-responsive immune programs. These results raise the possibility that AD-related neuronal damage may impair protective microglial immune responses early in disease pathogenesis.

Disclosure: Nothing to disclose.

LB_13 | How did Pyridostigmine gain its reputation with POTS? A systematic review and head-to-head intervention meta-analysis

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Background and aims: Pharmacotherapy of postural orthostatic tachycardia syndrome (POTS) commonly includes beta-blockers, ivabradine, midodrine, fludrocortisone and pyridostigmine. However, the use of pyridostigmine in POTS is still largely based on limited evidence.

To perform a systematic review and head-to-head intervention meta-analysis of studies evaluating the efficacy of pyridostigmine in reduction of heart rate in standardized autonomic function testing in comparison to beta-blockers, ivabradine and midodrine.

Methods: We searched PubMed, Embase, CINAHL as well as Cochrane Library for results up to May 25th, 2026, and evaluated full text articles for 38 of 443 search results. Six articles met all selection criteria. The respective heart rate change results (before and after intervention) of these 6 articles were pooled together and analyzed in a head-to-head manner (Pyridostigmine vs. beta-blockers and Pyridostigmine vs. ivabradine). All analyses were carried out using Review Manager 5.4.

Results: In the 6 articles, pyridostigmine showed no superiority in comparison to beta-blockers or ivabradine. The standardized mean difference (SMD) was -10.38 (95% confidence interval (CI) -47.90 to 27.14) for pyridostigmine vs. beta-blockers, and -3.63 (CI -56.54 to 49.27) for pyridostigmine vs. ivabradine.

Conclusion: Based on the currently available literature, pyridostigmine should not be considered a first-line therapy in patients with POTS. Our head-to-head meta-analysis and systematic review showed that the available literature is highly heterogeneous and published studies reported diverse primary endpoints. Our findings highlight the need for unanimous and standardized reporting of results (including symptomatologic questionnaires) to accurately evaluate the success of POTS pharmacological interventions.

Disclosure: Nothing to disclose.

LB_14 | AI-Augmented EEG interpretation: Unlocking timely diagnosis for new-onset epilepsy patients

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Background and aims: Approximately five million individuals are diagnosed with epilepsy each year, while many others present with uncertain first-seizure events. Early diagnosis is essential because timely treatment improves outcomes, yet delays are common due to limited access to electroencephalography (EEG) expertise. Interictal epileptiform discharges (IEDs), the only routinely used EEG biomarker, are highly specific but have low sensitivity. This study aimed to develop and validate an artificial intelligence (AI)-based diagnostic classifier using resting-state

EEG and routinely available clinical variables to improve early differentiation between epileptic and non-epileptic first-seizure presentations.

Methods: A transformer-based model was trained on approximately 10-minute resting-state EEG recordings from a real-world cohort. Patients were classified as having epilepsy or non-epileptic events after a standardized 2-year follow-up. Model inputs included EEG features, expert-marked IEDs, and clinical variables (age, sex, lesion status). A subject-level 5-fold cross-validation framework was used, with performance assessed using the area under the receiver operating characteristic curve (AUROC), sensitivity, specificity, and accuracy.

Results: The cohort included 1248 patients (621 epilepsy; 627 non-epilepsy). EEG alone yielded an AUROC of 0.72, decreasing to 0.67 in patients without IEDs or epileptogenic lesions. Adding clinical variables improved performance to 0.79. A fusion model combining EEG, IEDs, and clinical data achieved the highest performance (AUROC = 0.82; accuracy = 0.74; specificity = 0.86; sensitivity = 0.61).

Conclusion: AI-derived resting-state EEG in combination with routinely available clinical variables provides diagnostic value beyond conventional interpretation and may reduce diagnostic uncertainty and treatment delays after a first seizure.

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LB_15 | Long-term safety and tolerability of del-desiran in myotonic dystrophy type 1: Final results from MARINA-OLE™

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Background and aims: Myotonic dystrophy type 1 (DM1) is a rare, progressive neuromuscular disorder caused by DMPK mutations, leading to toxic mRNA accumulation, muscle dysfunction, and reduced life expectancy. Delpacibart etedesiran (del-desiran; AOC 1001) is an investigational antibody oligonucleotide conjugate comprised of a monoclonal antibody targeting transferrin receptor 1 for muscle delivery and an siRNA that induces degradation of DMPK mRNA to treat the underlying cause of DM1.

Methods: MARINA-OLE™ was a phase 2, open-label extension study assessing long-term safety and tolerability of del-desiran in adults with DM1, with follow-up extending to 37 months. Participants received del-desiran at either 2 or 4 mg/kg every 13 weeks and later transitioned to 4 mg/kg every 8 weeks, consistent with the phase 3 trial regimen. All participants completed approximately 1 year on this regimen.

Results: 37 participants received del-desiran, 101.3 patient-years of cumulative exposure with 506 infusions. Most treatment-emergent adverse events (TEAEs) were mild/moderate. No severe or serious TEAEs were treatment-related, and none led to treatment discontinuation or death. Laboratory values, including hematology parameters, were stable over time.

Conclusion: Del-desiran showed favorable long-term safety and tolerability in adults with DM1. These data represent the longest safety results for a DMPK-targeted therapy and highlight potential of del-desiran to drive a new treatment paradigm for DM1.

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JH served on Clinical Advisory Committees for Dyne Therapeutics and Vertex Pharmaceuticals; provided disease specific information to Design Therapeutics; as a Q&A panel member for Avidity Biosciences, A Novartis Company, at the MDA Conference; and as the overall Principal Investigator for the PepGen Freedom DM1 Study.

SHS has participated in Advisory Boards for Amicus Therapeutics, Inc., Fulcrum Therapeutics, PTC Therapeutics, Inc., and Reata; is a Principal Investigator for clinical trials by Avidity Biosciences, A Novartis Company, Biogen, Inc., Biohaven Pharmaceuticals, Inc., Fulcrum Therapeutics, Harmony Biosciences, PTC Therapeutics, Inc., Reata, Reneo, and Retrotope; and has discussed forthcoming trials with Solid Biosciences.

PS has a Research Contract Grant through UCLA to perform del-desiran clinical trials with Avidity Biosciences, A Novartis Company.

JS has served as a Consultant on trial design (Armatus Bio and Epic Bio), Advisory Boards (Avidity Biosciences, A Novartis Company, Dyne, F. Hoffmann-La Roche, Fulcrum Therapeutics, and Vertex Pharmaceuticals), and for Kate Therapeutics; received funding for clinical studies from Avidity Biosciences, A Novartis Company, Dyne, F. Hoffman-La Roche, FSHD Canada, and NINDS; received a grant for Muscular Dystrophy Clinical Research Network from MDA; and receives options as part of a non-employee compensation plan from Dyne.

MF received research support from Alnylam, Argenx, Catalyst Pharmaceuticals, Fulcrum Therapeutics, Momenta Pharmaceuticals, Inc., and UCB, Inc.; has been provided consulting fees by Argenx, Johnson & Johnson, and UCB Inc. for participation in Advisory Boards and by UCB Inc. for serving as a faculty member for speakers bureau training; and has served as Principal Investigator in clinical trials sponsored by Avidity Biosciences, A Novartis Company.

DQ receives clinical research support from Alnylam, Bridge Bio, Momenta, and Avidity; serves on a data safety monitoring board for Argenx; and has received payment from Astra Zeneca for a speaking engagement.

SV, AD, BM, MF, and EJA are employees of Avidity Biosciences, A Novartis Company.

LB_16 | Safety and efficacy of SR750 in patients with trigeminal neuralgia: A double-blind, randomized, placebo-controlled, phase 2 study

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Background and aims: Trigeminal neuralgia (TN) is a severely disabling disorder featuring sudden, recurrent, electric shock-like facial pain in the trigeminal nerve distribution. SR750, a first-in-class alpha5-GABAA receptors negative allosteric modulator (NAM) developed for neuropathic pain, has the potential to improve the condition of TN. This study assessed the efficacy and safety of SR750 in Chinese patients with TN.

Methods: A phase 2 randomized, double-blind and placebo-controlled study (NCT06571448) was initiated in July 2024. Patients diagnosed with primary TN were recruited and randomized into three arms at 1:1:1 ratio to receive SR750 50 mg, 150 mg or placebo twice a day (BID) for 6 weeks. The primary endpoint was change from baseline at week 6 in the weekly average of the daily average pain score (DAPS) of paroxysms using pain intensity numerical rating scale.

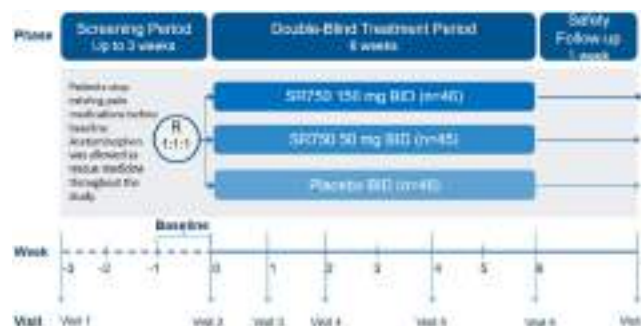


FIGURE 1 Study design.

Results: A total of 138 patients were enrolled, with 45 patients treated with SR750 50 mg BID, 47 treated with SR750 150 mg BID, and 46 treated with placebo. For the primary endpoint, the least square mean (LSM) change from baseline were -0.89 , -1.83 , and -1.97 in the placebo, SR750 50 mg BID and SR750 150 mg BID groups, respectively. Both SR750 treatment groups were superior to placebo, with between-group LSM differences of -0.94 (90% CI -1.66 , -0.23 , $p=0.031$) and -1.08 (90% CI -1.78 , -0.37 , $p=0.012$), respectively.

Conclusion: Overall, SR750 low dose and high dose demonstrated statistically and clinically significant efficacy versus placebo, with a favorable safety and tolerability profile, supporting its further investigation in late-stage trials.

Disclosure: Funded by SIMR. B. Fan reports no conflicts of interest. All other authors are employees of SIMR, including S. Li, the CEO, who participated in study oversight and approval. The funder was involved in study design, data analysis and manuscript preparation.

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Background and aims: Upper limb injuries and amputations in military personnel are associated with PTSD, anxiety, depression, and cognitive impairment. While standard rehabilitation is effective, the additional effects of robotic systems require further study.

Methods: Sixty military personnel with upper limb amputations were enrolled. Group 1 ($n=30$) received standard rehabilitation, while Group 2 ($n=30$) additionally underwent robotic therapy (DIEGO®). Assessments at baseline, 14, 30, and 120 days included PCL-5, GAD-7, PHQ-9, and MoCA. Data were analyzed using repeated-measures ANOVA, Cohen's d , and η^2 .

Results: At baseline, both groups had high psychoemotional distress and moderate cognitive impairment without significant differences: PCL-5 58.2 ± 6.1 vs. 57.9 ± 5.8 ($p=0.84$), GAD-7 15.3 ± 3.4 vs. 15.1 ± 3.2 ($p=0.79$), PHQ-9 17.8 ± 4.0 vs. 18.0 ± 3.8 ($p=0.88$), MoCA 23.1 ± 2.3 vs. 23.3 ± 2.5 ($p=0.67$).

PCL-5 scores improved: day 14 51.6 ± 5.7 vs. 45.9 ± 5.4 ($p=0.04$), day 30 43.8 ± 5.4 vs. 35.2 ± 4.9 ($p=0.003$), day 120 34.5 ± 5.2 vs. 26.1 ± 4.8 ($p=0.002$; $d=1.68$; $\eta^2=0.31$).

GAD-7 differences were significant only at day 120: 9.2 ± 2.6 vs. 6.3 ± 2.1 ($p=0.004$; $d=1.22$; $\eta^2=0.24$).

PHQ-9 improvement: day 14 15.0 ± 3.6 vs. 13.6 ± 3.3 ($p=0.06$), day 30 12.8 ± 3.3 vs. 9.6 ± 2.9 ($p=0.01$), day 120 10.4 ± 3.1 vs. 7.2 ± 2.5 ($p=0.006$; $d=1.13$; $\eta^2=0.21$).

MoCA: day 14 23.8 ± 2.2 vs. 25.1 ± 2.0 ($p=0.02$), day 30 24.4 ± 2.1 vs. 26.3 ± 1.9 ($p=0.01$), day 120 25.0 ± 2.1 vs. 27.4 ± 1.8 ($p=0.001$; $d=1.25$; $\eta^2=0.28$).

Conclusion: Robotic therapy (DIEGO®) added to standard rehabilitation leads to faster and greater improvement in psychoemotional and cognitive functions, supporting its integration into military rehabilitation.

Supported by the National Research Foundation of Ukraine (Project No. 2025.07/0021).

Disclosure: Nothing to disclose.

LB_18 | GRAND CANYON pivotal cohort of Sevasseten in adults with Becker muscular dystrophy: Rationale, study design and baseline characteristics

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Background and aims: There are no currently approved BMD therapies. Sevasseten, an investigational skeletal myosin inhibitor, is designed to protect muscle against contraction-induced damage in Becker muscular dystrophy (BMD). GRAND CANYON (NCT05291091), a double-blind, placebo-controlled cohort, evaluates sevasseten efficacy and safety.

Methods: Ambulatory BMD adults were randomized and stratified by NSAA and rise from floor (RFF). Primary endpoint is NSAA change from baseline at 18 months; secondary endpoints are TFTs, stride velocity 95th percentile (SV95C), and MRI fat fraction.

Results: 175 participants enrolled (mean age (SD): 33 (9) years; baseline NSAA: 19.6 (7.6); 58% with NSAA <22). 4-Stair Climb (4SC), 10-meter walk run (10MWR), and 100-m walk run (100MWR) tests were 6.3 (6.1), 8.2 (6.5), and 83.1 (33.8) seconds, respectively. RFF was 5.4 (3.0) seconds. SV95C was 1.2 (0.24) meters/second. Upper limb function was largely maintained, with 15% having entry level PUL 2.0 <6. In the initial CGI-S, 51% of participants had moderately or markedly severe disease and 48% reported moderate or severe disease per PGI-S. Pre-treatment NSAA strongly correlated with 4SC ($r=0.90$), 10MWR ($r=0.89$), 100MWR ($r=0.82$), RFF ($r=0.87$), and SV95C ($r=0.83$), all $p \leq 0.001$.

Conclusion: GRAND CANYON is the largest BMD interventional trial; results could support approval as the first targeted BMD therapy. Pre-treatment NSAA correlation analysis provides initial insight into potential alternatives to assessing functional status.

Disclosure: The GRAND CANYON trial is sponsored by Edgewise Therapeutics. Craig McDonald, Eugenio Mercuri, Kristl Claey's, Michela Guglieri, Erik H. Niks, Katherine Mathews, Anne M. Connolly, Luca Bello, Barry Byrne, Aravindhan Veerapandian are consultants for Edgewise. Jim MacDougall, Roxana Donisa Dreghici, and Joanne Donovan are employees and shareholders of Edgewise Therapeutics. All non-Edgewise authors are investigators in the GRAND CANYON trial.

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