PRELIMINARY SCIENTIFIC PROGRAM (Subject to changes – as of Febuary 8, 2024)

THURSDAY, MARCH 21, 2024		
08:10-10:50	**Neuroimmunology 1**	**HALL A**
Chairs:	**Bruno Gran,** UK; **Olaf Stuve,** USA	
08:10-09:00	Is Hashimoto's encephalitis/encephalopathy a valid construct in 2024?	
	Capsule: Hashimoto´s encephalopathy is a rare condition manifesting with variety of symptoms ranging from disturbances of consciousness, seizures, myoclonus to rapidly progressive cognitive decline observed in euthyroid patients with anti-thyroid antibodies. It is a steroid-responsive disorder. However, majority of described cases are from the period before tests for novel antibodies were available. Anti-thyroid antibodies in a patient with encephalopathy could be an incidental finding. Is therefore Hashimoto´s encephalitis/encephalopathy a valid construct in 2024?	
08:10-08:20	Moderator: **Uros Rot**, Slovenia Introduction and Pre-Debate Voting	
08:20-08:35	Yes: **Alasdair Coles**,UK	
08:35-08:50	No: **Divyanshu Dubey**, USA	
08:50-09:00	Discussion, Rebuttals and Post-Debate Voting	
09:00-09:50	All patients with inflammatory optic neuritis should be screened for AQP4-IgG and MOG-IgG antibodies	
	Capsule: Optic neuritis has discriminating clinical and paraclinical characteristics, different responses to treatment and prognosis in patients with MS, NMOSD or MOGAD but there is a significant clinical overlap between the entities. Immune treatment differs markedly between them. Should we therefore screen all patients with inflammatory optic neuritis for AQP4-IgG and MOG-IgG antibodies?	
09:00-09:10	Moderator: **Ruth Geraldes Nuffield,** UK Introduction and Pre-Debate Voting	
09:10-09:25	Yes: **Uros Rot**, Slovenia	
09:25-09:40	No: **Saif Huda**, UK	
09:40-09:50	Discussion, Rebuttals and Post-Debate Voting	

09:50-10:50	A prolonged (1 year) corticosteroid taper is sufficient to prevent relapses in patients with MOGAD	
	Capsule: MOGAD is a monophasic or relapsing disease associated with MOG-IgG autoantibodies, manifesting primarily as optic neuritis or as acute disseminated encephalomyelitis in children. It is controversial whether to start relapse prevention treatments following a first episode of MOGAD. Some experts have suggested that a prolonged taper of corticosteroids over 1 year is needed tol reduce the risk of relapses in MOGAD. How strong is the evidence and is this a recommendation that should be endorsed?	
09:50-10:00	Moderator: **Joab Chapman,** Israel Introduction and Pre-Debate Voting	
10:00-10:15	Yes: **Michael Levy, USA**	
10:15-10:30	No: **Friedemann Paul**, Germany	
10:30-10:50	Discussion, Rebuttals and Post-Debate Voting	
10:50-11: 20	**Coffee Break, Exhibition & ePosters Visits**	
11: 20-12:00	**Opening Ceremony**	**Plenary Hall**
11:20-11:25 **11:25-11:30** **11:30-11:35** **11:35-11:40** **11:40-12:00**	**Natan Bornstein**, Israel **Anthony Schapira**, UK **Amos Korczyn,** Israel **Richard J Davenport,** UK *Best e Posters award*	
12:00 -13:00	**Plenary Session**	**Plenary Hall**
Chairs:	**Brian G. Weinshenker**, USA; **John Hardy**, UK *CONy Excellence Recognition Award to Prof. Angela Vincent.*	
12:00-12:30	Antibody-mediated diseases: past, present and questions for the future **Angela Vincent, UK**	
12:30-13:00	The neurological manifestations of Covid-19 **Mike Zandi**, UK	
13:00-14:00	**Industry Sponsored Symposium**	**Plenary Hall**
14:00-14:50	**MTE**	
14:00-14:50	**Lunch Break, Exhibition & ePosters Visits**	
14:50-16:30	**Antibody testing**	**HALL A**
Chairs:	**Brian G. Weinshenker**, USA; **Mike Zandi**, UK	
14:50-15:40	Is long COVID an autoimmune disease?	
	Capsule: Long COVID refers to diverse symptoms, neurological and otherwise, that follow COVID-19 infection. The existence of this condition as a unique syndrome and its cause(s) remain uncertain. Is there reason to believe that long COVID is an autoimmune disease?	
14:50-15:00	Moderator: **Tom Pollak**, UK Introduction and Pre-Debate Voting	
15:00-15:15	Yes: **Michael D. Geschwind,** USA	
15:15-15:30	No : **Hans-Peter Hartung**, Germany	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	

14:50-16:30	**Antibody testing**	**HALL A**
15:40-16:30	Is it sufficient to send focused antibody testing on patients with suspected autoimmune encephalitis, or should all patients be screened with a panel of antibody tests?	
	Capsule: Autoimmune encephalitis (AE) is frequently associated with antibodies against neuronal, synaptic or glial proteins. Several clinical syndromes of AE have been reported to date. Correct diagnosis of AE depends on the disease phenotype, exclusion of alternative cause (infection, metabolic and neuropsychiatric) and identification of a specific antibody in serum and CSF. Both missed diagnosis and misdiagnosis of AE are recognised, and seronegative AE has been reported. Detection of serum antibody alone may be of uncertain significance without the clinical phenotype. Should antibody testing in suspected AE be focused on the specific syndrome or routinely carried out on an extensive panel of autoimmune and paraneoplastic antibodies?	
15:40-15:50	Moderator: **Abhijit Chaudhuri**, UK Introduction and Pre-Debate Voting	
15:50-16:05	Yes**: Eoin Flanagan, USA**	
16:05-16:20	No: **Ruth Geraldes Nuffield**, UK	
16:20-16:30	Discussion, Rebuttals and Post-Debate Voting	
16:30-16:50	**Coffee Break, Exhibition & ePosters Visits**	
16:50-18:30	**Corticosteroid treatment**	**HALL A**
Chair:	**Boleslav Lichterman** , Russia	
16:50-17:40	Diabetes mellitus-associated plexopathy is an inflammatory vasculitis and should be treated with high dose corticosteroids	
	Capsule: An infrequent but very disabling complication of diabetes mellitus is lumbosacral plexopathy (also known as diabetic amyotrophy), often initially asymmetrical accompanied by prominent pain and proximal weakness. The pathogenesis is controversial, but nerve biopsies have demonstrated evidence of microvasculitis with ischemic and inflammatory changes. Corticosteroids can be very effective in alleviating pain and many expert clinicians continue to advocate this treatment in spite of the lack of definite data	
16:50-17:00	Moderator: **Divyanshu Dubey** , USA Introduction and Pre-Debate Voting	
17:00-17:15	Yes: **Jim Dyck,** USA	
17:15-17:30	No: **Alasdair Coles**,UK	
17:30-17:40	Discussion, Rebuttals and Post-Debate Voting	
17:40-18:30	Cerebral amyloid angiopathy (CAA) may lead to inflammatory vasculopathy; patients with cerebral amyloid angiitis should receive corticosteroids on diagnosis.	
	Capsule: CAA is a vasculopathy characterised by amyloid beta (Aβ) deposition in cortical and meningeal blood vessels. Cerebrovascular Aβ deposit may provoke an inflammatory response, leading to perivascular inflammation and vasculitis. Acute, subacute, as well as chronic or progressive focal and multifocal neurological dysfunction are recognised in CAA and often attributed to the inflammatory response . Early corticosteroid therapy is considered by some to be beneficial in cerebral amyloid angiitis.	
17:40-17:50	Moderator: **Friedemann Paul**, Germany	
17:50-18:05	Yes: **Joab Chapman,** Israel	
18:05-18:20	No: **Abhijit Chaudhuri**, UK	
18:20-18:30	Discussion, Rebuttals and Post-Debate Voting	
18:30	**Networking Reception (Exhibition Area)**	

THURSDAY, MARCH 21, 2024		
08:10 – 10:50	**Alzheimer's disease (AD): Biomarkers**	**HALL B**
Chairs:	**Marina Janelidze,** Georgia	
08:10-09:00	Biomarkers are useful in subjective cognitive complaints and should be tested in each patient	
	Capsule: Patients with SCC are at increased risk to develop dementia . it is important to identify who is at risk. Are there any biomarkers which van help?	
08:10-08:20	Moderator: **Tom Neylan,** USA Introduction and Pre-Debate Voting	
08:20-08:35	Yes: **Paul Edison,** UK	
08:35-08:50	No: **Zvezdan Pirtosek**, Slovenia	
08:50-09:00	Discussion, Rebuttals and Post-Debate Voting	
09:00-09:50	Are serum markers such as phospho-tau useful in diagnosing AD ?	
	Capsule: *In a chronic medical condition, early diagnosis becomes important when treatment is available that can alter its course. Regarding AD , there is hope that drugs or prevention strategies will have the capacity of slowing down the neurodegeneration. Such treatments may provide greatest benefit to early stage since higher levels of functioning, independence, and quality of life will be maintained. Blood-based biomarkers would be critical in making early diagnosis accessible in routine clinical care. This debate will focus on the central question whether AD can (and should) be diagnosed early based on biomarkers measured in blood.*	
09:00-09:10	Moderator**: Xiaoping Wang**, People's Republic of China Introduction and Pre-Debate Voting	
09:10-09:25	Yes: **Robert Perneczky**, Germany	
09:25-09:40	No: **Arfan Ikram**, The Netherlands	
09:40-09:50	Discussion, Rebuttals and Post-Debate Voting	
09:50-10:50	**Sleep, Alzheimer's and Dementia – *session in cooperation with Alzheimer's Association***	
09:50-09:55	Moderators: **Claire Sexton**, USA; **Lea Grinberg,** USA	
09:55-10:10	Sleep as risk factor - evidence and interventions: **Sharon Naismith**, Australia	
10:10-10:25	Neuropathology and neuroimaging of sleep: **Neus Falgas,** Spain	
10:25-10:40	Sleep in clinical populations: **Tom Neylan**, USA	
10:40-10:50	Panel Discussion	

10:50 -11:20	**Coffee Break, Exhibition & ePosters Visits**
11:20-12:00	**Opening ceremony (Plenary Hall)**
12:00-13:00	**Plenary Session (Plenary Hall)**
13:00-14:00	**Industry Sponsored Symposium (Plenary Hall)**
13:00-14:00	**MTE**
14:00-14:50	**Lunch Break, Exhibition & ePosters Visits**

14:50-16:30	**AD: Therapy**	**HALL B**
Chairs:	**Panteleimon Giannakopoulos**, Switzerland ; **Yvonne Freund-Levi**, Sweden	
14:50-15:40	Obstructive sleep apnea is detrimental in patients with dementia and should always be treated	
	Capsule: An overwhelming body of work suggests that obstructive sleep apnea is more prevalent in patients with dementia and may be one of the risks for development of dementia. Whilst the exact mechanics of this bidirectional relationship are not fully understood, several studies advocate that early diagnosis, and early treatment of sleep apnea in patients with dementia may improve their quality of life, and possibly also decelerate the neurodegenerative process. In this debate the major limitations and/or potential contraindications, as well as the most promising aspects of OSA-treatment approach will be discussed.	
14:50-15:00	Moderator: **Michael D. Geschwind**, USA Introduction and Pre-Debate Voting	
15:00-15:15	Yes: **Ivana Rosenzweig,** UK	
15:15-15:30	No: **Sharon Naismith**, Australia	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	
15:40-16:30	Is cognitive reserve a useful term?	
	*Capsule: The concept of reserve was established to account for the observation that a given degree of neurodegenerative pathology may result in varying severities in different individuals. There is a large amount of evidence on epidemiological risk and protective factors for neurodegenerative diseases and dementia, yet the biological mechanisms that underpin the protective effects of certain lifestyle and physiological variables remain poorly understood, limiting the development of more effective preventive and treatment strategies. Additionally, different definitions and concepts of reserve exist, which hampers the coordination of research and comparison of results across studies. Is cognitive reserve just another buzz word or is the phenomenon supported by enough scientific evidence***?***	
15:40-15:50	Moderator: **Robert Perneczky**, Germany Introduction and Pre-Debate Voting	
15:50-16:05	Yes: **Yaakov Stern** , USA	
16:05-16:20	No: **Amos Korczyn** , Israel	
16:20-16:30	Discussion, Rebuttals and Post-Debate Voting	
16:30-16:50	**Coffee Break, Exhibition & ePosters Visits**	

16:50-18:30	AD 3	HALL B
Chairs:	**Judith Aharon**, Israel; **Milica G. Kramberger**, Slovenia	
16:50-17:40	Antiamyloid drugs have only a very limited effect and will not be clinically useful for most patients	
	Capsule: Several large clinical trials have demonstrated potential utility of amyloid-targeting approaches in slowing the progression of AD. These treatments may change the course of the disease in some people in the early stages, giving them more time to participate in daily life. However, while promising, these treatments have also been shown to have significant side effects and high cost. In this debate the major limitations as well as the most promising aspects of amyloid-targeting approach will be discussed.	
16:50-17:00	Moderator: **John Hardy, UK** Introduction and Pre-Debate Voting	
17:00-17:15	Yes: **Dorota Religa** ,Sweden	
17:15-17:30	No: **Paul Edison,** UK	
17:30-17:40	Discussion, Rebuttals and Post-Debate Voting	
17:40-18:30	Should lecanemab use be extended beyond 18 months?	
	Capsule: There is only one phase 3 randomized trial of lecanemab and one of donanemab. Both were undertaken to support FDA marketing approval; and both have uncontrolled, long-term extended treatment options provided for participants who completed the 18 month trials and wished to continue treatment. As the clinical effects of these antibodies are small and dropouts and adverse events fairly common a question arises about whether treatment should be continued beyond the length of the trials and whether any clinical benefit might become apparent over the long-term. Only a few hundred clinical trials patients have been exposed to these antibodies beyond 18 months; and no regular clinic patient in the USA or Japan could have been exposed to lecanemab for more than 8 months. Thus this issue is ripe for debate as evidence is sparse or absent. This debate might highlight what needs to be considered for better understanding of treatment.	
17:10-17:50	Moderator: **Zvezdan Pirtosek,** Slovenia	
17:50-18:05	Yes: **Dorota Religa**, Sweden	
18:05-18:20	No: **Lon Schneider** , USA	
18:20-18:30	Discussion, Rebuttals and Post-Debate Voting	

THURSDAY, MARCH 21, 2024		
08:10 – 10:50	**Parkinson's disease (PD)**	**HALL C**
Chairs:	**Cristian Falup-Pecurariu**, Romania; **Hana Brozova**, Czech Republic	
08:10-09:00	Has the DLB vs. PD(D) distinction outlived its usefulness?	
	Capsule: The timing of dementia relative to parkinsonism is the major clinical distinction between DLB and PD. There is evidence of overlap of clinical, neuropsychological, and neuropathological features of DLB and PD, remarkably convergent neuropathologic changes at autopsy. These can be a reason to believe that PD and DLB are different phenotypic expressions of the same underlying process and therefore, maybe it is time to consider that the DLB vs. PD(D) distinction outlived its usefulness?	
08:10-08:20	Moderator: **Nestor Galvez** , USA Introduction and Pre-Debate Voting	
08:20-08:35	Yes: **Lea Grinberg** , USA	
08:35-08:50	No: **Vladimira Vuletic**, Croatia	
08:50-09:00	Discussion, Rebuttals and Post-Debate Voting	
09:00-09:50	With the limitation of dopamine-based therapy, should therapy in PD be directed in other directions?	
	Capsule: PD is not only a disease with a shortage of dopamine but is a multi-transmitter disease. Should other options than simply dopamine replacement be discussed, or does levodopa still have untapped potential?	
09:00-09:10	Moderator: **Michael Ugrumov** Russia Introduction and Pre-Debate Voting	
09:10-09:25	Yes: **Jaroslaw Slawek**, Poland	
09:25-09:40	No: **Heinz Reichmann**, Germany	
09:40-09:50	Discussion, Rebuttals and Post-Debate Voting	
09:50-10:40	Is it possible to modify the disease course in PD?	
	Capsule: As we may soon get options to diagnose PD in its prodromal phase, disease-modifying therapies may get a second chance.	
09:50-10:00	Moderator: **Avner Thaler**, Israel Introduction and Pre-Debate Voting	
10:00-10:15	Yes: **Vladimira Vuletic**, Croatia	
10:15-10:30	No: **Jaroslaw Slawek** ,Poland	
10:30-10:50	Discussion, Rebuttals and Post-Debate Voting	
10:50 -11:20	**Coffee Break, Exhibition & ePosters Visits**	
11:20-12:00	**Opening Ceremony (Plenary Hall)**	
12:00-13:00	**Plenary Session (Plenary Hall)**	
13:00-14:00	**Industry Sponsored Symposium (Plenary Hall)**	
14:00-14:50	**MTE**	

14:00-14:50	**Lunch Break, Exhibition & ePosters Visits**	
14:50-16:30	**Precision therapies in PD**	**HALL C**
Chairs:	**Gilad Yahalom,** Israel; **Weidong Le,** China	
14:50-15:40	Are we ready for precision medicine in PD?	
	Capsule: Developments in -omic technologies as well as deep phenotyping support the heterogeneity of PD risk and progression. However, this has not yet translated into "personalized medicine" outside of the research environment. Are we ready yet to tailor disease prevention and/or treatment according to different disease profiles?	
14:50-15:00	Moderator: **Yoav Ben-Shlomo**, UK Introduction and Pre-Debate Voting	
15:00-15:15	Yes: **Ray Chaudhuri,** UK	
15:15-15:30	No: **Evzen Ruzicka**, Czech Republic	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	
15:40-16:30	GBA targeted therapies are a waste of money	
	Capsule: Genetic variants of the GBA1 gene are the commonest genetic risk factor for PD. They result in a number of specific biochemical alterations including lysosomal and mitochondrial dysfunction and accumulation of alpha-synuclein. Is the GBA1 gene and its product (glucocerebrosidase) a reasonable target for therapies to modify the course of PD?	
15:40-15:50	Moderator: **Ziv Gan-Or**, Canada Introduction and Pre-Debate Voting	
15:50-16:05	Yes: **Avner Thaler**, Israel	
16:05-16:20	No : **Anthony Schapira**, UK	
16:20-16:30	Discussion, Rebuttals and Post-Debate Voting	
16:30-16:50	**Coffee Break, Exhibition & ePosters Visits**	
16:50-18:30	**PD 3**	**HALL C**
Chair:	**Yehonatan Sharabi**, Israel; **Ilana Schlesinger** , Israel	
16:50-17:40	Telemedicine is valuable for PD patients care and will become the predominant method	
	Capsule: The COVID-19 pandemia forced many of us to start applying telemedicine. It is also known for many years that the registration of movement during a whole day gives a better insight than taking the history from the patient. Thus, telemedicine may be an option for many PD patients.	
16:50-17:00	Moderator: **Yoav Ben-Shlomo**, UK Introduction and Pre-Debate Voting	
17:00-17:15	Yes: **Heinz Reichmann**, Germany	
17:15-17:30	No: **Anthony Schapira,** UK	
17:30-17:40	Discussion, Rebuttals and Post-Debate Voting	

17:40-18:30	Should idiopathic REM-sleep behavior disorder (iRBD) patients be informed about potential PD prognosis as long as there is no disease modifying therapy (DMT) ?
	Capsule: iRBD is linked with an increased risk of PD and other alpha-synucleinopathies, but presently there is no consensus about disclosure of this risk to patients.. However, presently, there is no proven neuroprotective strategy, or DMT , to prevent the development of neurological deficits and there are only very limited data concerning counselling of iRBD patients. What are the potential ethical and clinical conundrums in prognostic counselling of iRBD?
17:10-17:50	Moderator: **Ray Chaudhuri,** UK
17:50-18:05	Yes: **Ivana Rosenzweig**, UK
18:05-18:20	No: **Danielle Wasserman Berk,** Israel
18:20-18:30	Discussion, Rebuttals and Post-Debate Voting

FRIDAY, MARCH 22, 2024		
08:30-11:00	**PARKINSON'S DISEASE (PD) 2** Advances in Diagnosis and Treatment	**HALL A**
08:30-08:45	Advances in Diagnosis and Treatment	
Chair:	Stuart Isaacson, USA	
08:45-09:30	Psychosis, Dementia, and Mortality in PD	
	Capsule:	
08:45-08:50	Moderator: TBD Introduction and Pre-Debate Voting	
08:50-09:05	Panelist: **Daniel Weintraub,** USA	
09:05-09:20	Panelist: TBD	
09:20-09:30	Discussion, Rebuttals and Post-Debate Voting	
09:30-10:15	Nonmotor symptoms should be treated when they first emerge (such as psychosis, siallorhea, cognition)	
	Capsule:	
09:30-09:35	Moderator: TBD Introduction and Pre-Debate Voting	
09:35-09:50	YES: **Rajesh Pahwa**	
09:50-10:05	NO: TBD	
10:05-10:15	Discussion, Rebuttals and Post-Debate Voting	

10:15-11:00	VMAT2 inhibitor should be firstline treatment for hyperkinetic movements in HD and TD
	Capsule:
10:15-10:20	Moderator: TBD Introduction and Pre-Debate Voting
10:20-10:35	Yes: **Daniel Kremens**, USA
10:35-10:50	No: TBD
10:50-11:00	Discussion, Rebuttals and Post-Debate Voting
11:00-11:30	**Coffee Break, Exhibition & ePosters Visits**
11:30-12:30	**Plenary Sessions (Plenary Hall)**
12:30-13:30	**Industry Sponsored Symposium (Plenary Hall)**
13:30-14:15	**MTE**
13:30-14:30	**Lunch Break, Exhibition & e-Posters Visit**

14:30-	**PD**	**HALL A**
Chairs:	**Stuart Isaacson,** USA	
14:30-15:15	Nondopaminegic mechanisms to address motor complications	
	Capsule:	
14:30-14:35	Moderator: TBD Introduction and Pre-Debate Voting	
14:35-14:50	Yes: **Sagari Betté**, USA	
14:50-15:05	No: TBD	
15:05-15:15	Discussion, Rebuttals and Post-Debate Voting	
15:15-16:00	Dopaminergic mechanisms to address motor complications (such as COMT, D1-family and D1/D2 agonists)	
	Capsule:	
15:15-15:25	Moderator: TBD Introduction and Pre-Debate Voting	
15:25-15:40	Yes: **Daniel Kremens**, USA	
15:40-15:55	No: TBD	
15:55-16:05	Discussion, Rebuttals and Post-Debate Voting	
16:05-16:50	Continuous dopaminergic stimulation (such as COMT, ER LD, and sc, infusion)	
	Capsule:	
16:05-16:10	Moderator: TBD Introduction and Pre-Debate Voting	

10:10-16:25	Yes: TBD
16:25-16:40	No: TBD
16:40-16:50	Discussion, Rebuttals and Post-Debate Voting
16:50-17:35	Optimizing subcutaneous infusions for OFF fluctuations: LD/CD vs. apomorphine
	Capsule:
16:50-16:55	Moderator: TBD Introduction and Pre-Debate Voting
16:55-17:10	Yes: **Stuart Isaacson**, USA
17:10-17:25	No: TBD
17:25-17:35	Discussion, Rebuttals and Post-Debate Voting
17:35-18:20	Wearables to inform motor complications in PD
	Capsule:
17:35-17:40	Moderator: **Rajesh Pahwa,** USA Introduction and Pre-Debate Voting
17:40-17:55	Yes: TBD
17:55-18:10	No: TBD
18:10-18:20	Discussion, Rebuttals and Post-Debate Voting

FRIDAY, MARCH 22,2024		
08:30-11:00	STROKE: Acute stroke	**HALL B**
Chair:	**Vida Demarin**, Croatia ; **Natan Bornstein**, Israel	
08:30-09:20	In people with intracerebral hemorrhage (ICH) minimally invasive neurosurgery should be routinely discussed	
	Capsule: Acute stroke due to supratentorial intracerebral hemorrhage is associated with high morbidity and mortality. Open craniotomy hematoma evacuation has not been found to have any benefit in large, randomized trials. Recently minimally invasive catheter evacuation followed by thrombolysis , with the aim of decreasing clot size and iron toxicity showed promising results .Whether minimally invasive surgery should be routinely discussed in patients with ICH is the topic of this debate.	
08:30-08:40	Moderator: **Robert Mikulik**, Czech republic Introduction and Pre-Debate Voting	
08:40-08:55	Yes: **Marina Roje Bedekovic**, Croatia	
08:55-09:10	No: **Laszlo Csiba,** Hungary	
09:10-09:20	Discussion, Rebuttals and Post-Debate Voting	
09:20-10:10	Mecanical Thrombectomy (MT) for large core infarcts is a worthwhile use of health care resources.	
	Capsule: Patients with large infarct core on baseline imaging were excluded from MT studies due to their assumed poor outcome. It is still unclear whether reperfusion therapies are safe and beneficial in this group of patients. The debate will try to answer the open questions: What is the best imaging modality to diagnose the large infarct core? Can analysis of risk-benefit balance justify MT for this group of patients? How can the risk of complications be reduced?	
09:20-09:30	Moderator: **Laszlo Csiba,** Hungary Introduction and Pre-Debate Voting	
09:30-09:45	Yes: **Ashfaq Shuaib** ,Canada	
09:45-10:00	No: TBA	
10:00-10:10	Discussion, Rebuttals and Post-Debate Voting	
10:10-11:00	Should patients with mild non-disabling stroke admitted within 4.5h be considered for thrombolysis?	
	Capsule: The role of thrombolysis in treatment of patients with AIS with low National Institutes of Health Stroke Scale (NIHSS) scores is not well understood. However, in the absence of definitive evidence, practice pattern is widely variable. Here we debate the benefit of mild thrombolysis of strokes in view of the most recent evidence.	
10:00-10:20	Moderator: **Robert Mikulik**, Czech republic	
10:20-10:35	Yes: **Ashfaq Shuaib,** Canada	
10:35-10:50	No: **Jesse Dawson**, UK	
10:50-11:00	Discussion, Rebuttals and Post-Debate Voting	
11:00-11:30	**Coffee Break, Exhibition & e-Posters Visit**	

11:30-12:00	**Plenary Session**	**Plenary Hall**
Chair:	**Lea Grinberg** USA/Brazil ; **Ivana Rosenzweig**, UK	
11:30-12:00	Sleep disorders and brain health **Claudio Bassetti**, Switzerland	
12:00-12:30	The use of AI in neurology . **Sanjay Budheo**, UK	
12:30-13:30	**Industry Sponsored Symposium**	**Plenary Hall**
13:30-14:30	**MTE**	
13:30-14:30	**Lunch Break, Exhibition & e-Posters Visit**	

14:30-16:10	STROKE: Prevention and recovery	**HALL B**
Chairs:	**Christine Kremer,** Sweden; **Marina Roje Bedekovic**, Croatia	
14:30-15:20	Is there an added value to use virtual reality in rehabilitation after stroke?	
	Capsule: Within the last 10 years there is growing interest in possible applications of advanced technolgies such as virtual or augmented reality. First steps have been made to use these techniques in motor, cognitive and emotional therapies in rehabilitation after stroke. It is however controversial if this will offer additional value for patients or may even be counterproductive taking away resources which could be used better.	
14:30-14:40	Moderator: **Nirmal Surya**, India Introduction and Pre-Debate Voting	
14:40-14:55	Yes: **Volker Homberg**, Germany	
14:55-15:10	No: **Dafin Muresanu**, Romania	
15:10-15:20	Discussion, Rebuttals and Post-Debate Voting	
15:20-16:10	Should GLP-1 agonists be used to reduce stroke risk in non-diabetic obese individuals?	
	Capsule: There is evidence from studies in diabetes that GLT-1 receptor agonist treatment may reduce cardiovascular events, including stroke. A recent large study in obese people with no evidence of diabetes reveals that GLP-1 receptor agonists may also be effective in the prevention of stroke in such non-diabetic obese individuals. Should GLP-1 receptor agonists be used in obese non-diabetic individuals for stroke prevention?	
15:20-15:30	Moderator: **Vida Demarin**, Croatia Introduction and Pre-Debate Voting	
15:30-15:45	Yes: **Natan Bornstein**, Israel	
15:45-16:00	No: **Jesse Dawson,** UK	
16:00-16:10	Discussion, Rebuttals and Post-Debate Voting	
16:10-16:30	**Coffee Break, Exhibition & e-Posters Visit**	

16:30-18:10	STROKE: Prevention and recovery	**HALL B**
Chairs:		
16:30-17:20	Should we aggressively treat asymptomatic small vessels disease (SVD) with drugs?	
	Capsule: Asymptomatic SVD is perhaps the most common abnormality noted on CT and MR imaging in the elderly population. The presence of SVD increases the risk of symptomatic stroke, dementia, falls, and early mortality. Thus far no definite treatments have been shown to slow the progression of SVD. Should patients with SVD be treated aggressively with antiplatelet medications or is risk factor management sufficient to manage patients in whom SVD is present on brain imaging?	
16:30-16:40	Moderator: **Ashfaq Shuaib,** Canada Introduction and Pre-Debate Voting	
16:40-16:55	Yes: **Christine Kremer,** Sweden	
16:55-17:10	No: **Alan Cameron**,UK	
17:10-17:20	Discussion, Rebuttals and Post-Debate Voting	

FRIDAY, MARCH 22, 2024		
08:30-11:00	**HEADACHE : Migraine and cluster headache**	**HALL C**
Chair:	**VlastaVukovic Cvetkovic**, Denmark ; **Ruta Mameniskiene**, Lithuania	
08:30-09:20	Are anti-CGRP mAbs effective in prevention of cluster headache?	
	Capsule: Cluster headache is a serious medical condition that lacks disease-specific and mechanism-based treatments. There is some evidence that galcanezumab may be effective in reducing the frequency of cluster headache attacks, but the evidence is weak. Should CGRP mAbs be used in people with episodic cluster headache?	
08:30-08:40	Moderator: **Dimos Mitsikostas,** Greece Introduction and Pre-Debate Voting	
08:40-08:55	YES: **Giorgio Lambru**, UK	
08:55-09:10	NO: **Patricia Pozo-Rosich**, Spain	
09:10-09:20	Discussion, Rebuttals and Post-Debate Voting	
09:20-10:10	Premonitory symptoms in migraine and cluster headache are important for treatment	
	Capsule: Premonitory symptoms in migraine and cluster headache involve activation of the central parts of the trigeminovascular system (TVS). Whether preventive migraine treatments acting on the peripheral parts of the TVS can reduce the incidence of premonitory symptoms, not just the incidence of headache attacks, in people with migraine and/or cluster headache, remains an attractive hypothesis.	
09:20-09:30	Moderator: **Cristina Tassorelli**, Italy Introduction and Pre-Debate Voting	
09:30-09:45	YES: **Anna Andreou**, UK	
09:45-10:00	NO: **Haakan Ashina**, Denmark	
10:00-10:10	Discussion, Rebuttals and Post-Debate Voting	
10:10-11:00	Is central 5-HT1F agonism essential for ditans to be effective?	
	Capsule: 5-HT1F receptors have been identified in both peripheral and central parts of the TVS. Ditans penetrating the blood brain barrier and activating 5-HT1F receptors in both parts of the TVS, also induce adverse effects that limit their use. Is central 5-HT1F agonism essential for ditans to be effective, or is the peripheral action enough, like with all other migraine-specific and mechanism based symptomatic treatments?	
10:00-10:20	Moderator: **Dimos Mitsikostas**, Greece Introduction and Pre-Debate Voting	
10:20-10:35	Yes: **Anna Andreou**, UK	
10:35-10:50	No: **Antoinette Maassen van den Brink**, The Netherlands	
10:50-11:00	Discussion, Rebuttals and Post-Debate Voting	
11:00-11:30	**Coffee Break, Exhibition & e-Posters Visit**	

11:30-12:30	**Plenary Sessions (Plenary Hall)**
12:30-13:30	**Industry Sponsored Symposium (Plenary Hall)**
13:30-14:30	**MTE**
13:30-14:30	**Lunch Break, Exhibition & e-Posters Visit**

14:30-16:10	**HEADACHE : Ditans and Gepants**	**HALL C**
Chairs:	**Marjan Zaletel,** Slovenia ; **Tomas Nezadal,** Czech Republic	
14:30-15:20	Addition of a gepant for the acute care of migraine attacks is safe and effective in patients on anti-CGRP mAbs	
	Capsule: The consideration of using gepants for aborting migraine attacks remains a topic of debate when managing patients who are concurrently prescribed anti-CGRP mAbs as preventive therapy. Moreover, considering the safety aspects of combining two drugs that target CGRP signaling, it's important to discuss adding a gepant for immediate migraine relief in patients on anti-CGRP mAbs preventive treatment	
14:30-14:40	Moderator: **Messoud Ashina**, Denmark Introduction and Pre-Debate Voting	
14:40-14:55	Yes:**Cristina Tassorelli**, Italy	
14:55-15:10	No: **Gisela Terwindt**, The Netherlands	
15:10-15:20	Discussion, Rebuttals and Post-Debate Voting	
15:20-16:10	Are gepants and ditans efficacious and safe for people with vascular risk factors?	
	Capsule: Preclinical studies showed that CGRP is a potent vasoactive neuropeptide, yet activation of 5-HT1F receptors does not constrict coronary or cerebral arteries. Is there evidence supporting the use of treatments targeting either the CGRP neuropeptide, e.g., the gepants, or the 5-HT1F receptor, e.g., the ditans, in people with migraine and vascular risk factors?	
15:20-15:30	Moderator: **Messoud Ashina**, Denmark Introduction and Pre-Debate Voting	
15:30-15:45	Yes: **Jose Miguel Lainez**, Italy	
15:45-16:00	No: **Gisela Terwindt**, The Netherlands	
16:00-16:10	Discussion, Rebuttals and Post-Debate Voting	
16:10-16:30	**Coffee Break, Exhibition & e-Posters Visit**	
16:30-18:10	**Medication overuse headache and artificial intelligence**	**HALL C**
Chair:	**Licia Grazzi,** Italy	
16:30-17:20	Is Artificial Intelligence (AI) ready for inclusion in headache management?	
	Capsule: AI systems have gotten our attention as they have emerged in all aspects of the working and education worlds. In medicine, AI was initially promoted as a way to improve the quality of diagnosis and treatment planning, as well as a way to deliver care more efficiently. But most would agree it has not lived up to its promise. Recently however, with increased power of operating systems and new designs for analyzing data, this may be about to change and it may revolutionize medicine including the care of patients with headache disorders. It the time now?	

16:30-16:40	Moderator: **Alan Rapoport**, USA Introduction and Pre-Debate Voting
16:40-16:55	Yes: **Robert Cowan**, USA
16:55-17:10	No: **Morris Levin**, USA
17:10-17:20	Discussion, Rebuttals and Post-Debate Voting
17:20-18:10	Is it necessary to detoxify most patients with Medication Overuse Headache (MOH) in order to achieve success?
	Capsule: Ever since the concept for MOH was promoted, a key tenet was that patients with this syndrome could not improve until the overused medication was removed. Only then, would preventive medications be effective in reversing the chronification of the primary headache disorder. Recent data calls this dogma into question, but there is still disagreement about the best way(s) to help patients.
17:20-17:30	Moderator: **Cristina Tassorelli**, Italy Introduction and Pre-Debate Voting
17:30-17:45	Yes: **Morris Levin**, USA
17:45-18:00	No: **Alan Rapoport**, USA
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting

SATURDAY,MARCH 23,2024		
08:20-11:00	**Amyotrophic lateral sclerosis/Motorneuron disease** (ALS/MND)	**HALL A**
Chair:		
08:20-09:10	Human cell models are better than mouse models in therapy development for ALS/MND	
	Capsule: Given the limitations of currently available mouse models of ALS/MND and the lack of a mouse model for sporadic disease, human cellular models have key advantages in the evaluation of potential neuroprotective therapies.	
08:20-08:30	Moderator: **Amir Dori,** Israel Introduction and Pre-Debate Voting	
08:30-08:45	Yes : **Pam Shaw,** UK	
08:45-09:00	No: **Linda Greensmith** ,UK	
09:00-09:10	Discussion, Rebuttals and Post-Debate Voting	
09:10-10:00	ALS/MND treatments have to demonstrate efficacy on both prolonging survival and slowing disease progression	
	Capsule: In the absence of improved function, prolonged survival by a few months may not necessarily indicate attenuation of disease progression. Therefore, evidence for slowed disease progression is required	
09:10-09:20	Moderator: **Amir Dori, Israel** Introduction and Pre-Debate Voting	
09:20-09:35	Yes: **Albert Ludolph**, Germany	
09:35-09:50	No: **Osman Sinanovic**, Bosnia and Herzegovina	
09:50-10:00	Discussion, Rebuttals and Post-Debate Voting	

10:00-11:00	The euphoria on personalized ALS/MND treatment with Antisense oligonucleotides (ASO) is premature
10:00-10:10	Capsule: Treatment with ASOs for specific genetic subtypes of ALS/MND has shown some promising results, but euphoria is premature given the rarity of these subtypes and the failure of some ASO approaches.
10:10-10:25	Moderator: **Osman Sinanovic**, Bosnia and Herzegovina Introduction and Pre-Debate Voting
10:25-10:40	Yes: **Pam Shaw,**UK
10:40-11:00	No: **Giancarlo Logroscino**, Italy
11:00-11:30	**Coffee Break, Exhibition & e-Posters Visit**
11:30-12:30	**Plenary Session** **Plenary Hall**
Chairs:	**George Chakhava**, Georgia; **Odelia Elkana,** Israel
11:30-12:00	Aphantasia – when all is dark in the mind's eye **Adam Zeman**, UK
12:00-12:30	The impossible pricing of anti-amyloid medication. A global challenge. **Paola Barbarino, UK**
12:30-13:30	**Industry Sponsored Symposium (Plenary Hall)** **Plenary Hall**
13:30-14:30	**MTE**
13:30-14:30	**Lunch Break, Exhibition & e-Posters Visit**
14:30- 16:10	Amyotrophic lateral sclerosis/ motorneuron disease (ALS/MND) 2 **HALL A**
Chair:	**Ervin Jancic,** Croatia
14:30-15:20	Tracheostomy ventilation in ALS/MND should be offered to all patients
	Capsule: Neuromuscular respiratory failure is the cause of death in most patients with ALS/MND. Invasive tracheostomy ventilation adds a burden of care while non-invasive respiratory support is readily available.
14:30-14:40	Moderator: **David Oliver ,** UK Introduction and Pre-Debate Voting
14:40-14:55	Yes: **Giancarlo Logroscino**, Italy
14:55-15:10	No: **Albert Ludolph**, Germany
15:10-15:20	Discussion, Rebuttals and Post-Debate Voting
15:20-16:10	gMG patients with highly active disease should be treated with innovative treatments earlier
	Capsule :
15:20-15:30	Moderator: TBA
15:30-15:45	Yes: **Celia Oreja-Guevara** , Spain
15:45-16:00	No: TBA
16:00-16:10	Discussion, Rebuttals and Post-Debate Voting
16:10-16:30	**Coffee Break, Exhibition & e-Posters Visit**

16:30-18:10	**Neurodegenerative diseases** **HALL A**
Chair:	**Johannes Attems,** UK
16:30-17:20	Will anti-tau drugs be useful in fighting tau-driven diseases?
	Capsule: Age/ageing is a major predisposing factor for the development of AD and PD yet its role in enhancing neuronal loss is seldom incorporated into concepts of these diseases at either the laboratory or clinical level. The relevance of ageing in the pathological processes in AD and PD remains poorly understood and poorly studiedbut it may hold the key to effective prevention and treatment of the most common neurodegenerative illnesses. Should more effort be put in to understanding the relevance of the ageing process to the onset of neurodegenerative disease or is it a bystander that merely increases risk but does not affect whether pathological change occurs or not?
16:30-16:40	Moderator: **Bogdan Popescu**, Romania Introduction and Pre-Debate Voting
16:40-16:55	Yes: **Lea Grinberg,** USA
16:55-17:10	No : **Jesse Cedarbaum** , USA
17:10-17:20	Discussion, Rebuttals and Post-Debate Voting
17:20-18:10	Pathological changes in microglia were linked to pathology in different neurodegenerative diseases. Can we target microglia to develop a therapeutic approach?
	Capsule: Activation of microglia is considered a pathological hallmark in many neurodegenerative diseases. While microglia are essential for normal brain activity, impairment in their activity may exacerbate neuroinflammation and result in neuronal death. Should microglia be a target for therapeutic intervention to ameliorate disease pathology in neurodegenerative diseases?
17:20-17:30	Moderator: **Jesse Cedarbaum**, USA Introduction and Pre-Debate Voting
17:30-17:45	Yes: **Dan Frenkel,** Israel
17:45-18:00	No: **Bogdan Popescu,** Romania
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting

SATURDAY, MARCH 23, 2024		
08:20-11:00	**Epilepsy: Drug treatment**	**HALL B**
Chair:	**Natasa Pejanovic Skobic** , Bosnia and Herzegovina	
08:20-09:10	Should we routinely utilize therapeutic drug monitoring when prescribing drug therapy for people with epilepsy?	
	Capsule: It is debated whether monitoring of serum levels improves care for people with epilepsy, and the literature contains conflicting recommendations. What should we do?	
08:20-08:30	Moderator**: Michael Sperling,** USA Introduction and Pre-Debate Voting	
08:30-08:45	Yes: **William Theodore**, USA	
08:45-09:00	No: **Elinor Ben-Menachem**, Sweden	
09:00-09:10	Discussion, Rebuttals and Post-Debate Voting	
09:10-10:00	Should women who wish to become pregnant be prescribed supplemental folic acid? Rationale: A recent study suggests that cancer risk is increased in children born to women who took folic acid during pregnancy. Is this study sufficient to alter clinical practice?	
	Capsule: A recent study suggests that cancer risk is increased in children born to women who took folic acid during pregnancy. Is this study sufficient to alter clinical practice?	
09:10-09:20	Moderator: **Martin Holtkamp**, Germany Introduction and Pre-Debate Voting	
09:20-09:35	Yes: **Jacqueline Frenc**h,USA	
09:35-09:50	No: **Alla Guekht**, Russia	
09:50-10:00	Discussion, Rebuttals and Post-Debate Voting	
10:00-11:00	Should polytherapy be used early, after one drug has failed to control seizures?	
	Capsule: Is rational polytherapy a sensible practice after failure of one antiseizure medication, given the low probability of response to another monotherapy? Is there evidence that polytherapy is superior to monotherapy?	
10:00-10:10	Moderator: **Zeljka Petelin-Gadze,** Croatia Introduction and Pre-Debate Voting	
10:10-10:25	Yes:**Manjari Tripathi**, India	
10:25-10:40	No: **Elinor Ben-Menachem**, Sweden	
10:40-10:50	Discussion, Rebuttals and Post-Debate Voting	
11:00-11:30	**Coffee Break, Exhibition & e-Posters Visit**	
11:30-12:30	**Plenary Sessions (Plenary Hall)**	
12:30-13:30	**Industry Sponsored Symposium (Plenary Hall)**	
13:30-14:30	**MTE**	
13:30-14:30	**Lunch Break, Exhibition & e-Posters Visit**	

14:30-16:10	**The unusual cases**	**HALL B**
Chair:	**Nandan Yardi** , India	
14:30-15:20	Mix of interesting cases related to diagnosis and management challenges in epilepsy, discussing advanced drug therapy and a modes of administration. Case Studies**: Michael Sperling** ,USA	
15:20-16:10	Should we prefer one method of neurostimulation over others?	
	Capsule: Vagus nerve stimulation, deep brain stimulation, responsive neurostimulation, and extracranial stimulation are used to treat people with uncontrolled seizures. Is there evidence that some method is superior or inferior to others?	
15:20-15:30	Moderator: **Alla Guekht**, Russia Introduction and Pre-Debate Voting	
15:30-15:45	Yes: **Martin Holtkamp**, Germany	
15:45-16:00	No: **Ivan Rektor,** Czech republic	
16:00-16:10	Discussion, Rebuttals and Post-Debate Voting	
16:10-16:30	**Coffee Break, Exhibition & e-Posters Visit**	
16:30-18:10	**Epilepsy 3**	**HALL B**
Chairs:	**Man Mohan Mehndiratta**, India; **Andreja Bujan Kovac**, Croatia	
16:30-17:20	Should laser interstitial thermal ablation therapy (LITT) be preferred over open surgery, either anterior temporal lobectomy or amygdalohippocampectomy, for mesial temporal sclerosis?	
	Capsule: Thermal ablation has been used for over 11 years and offers clear advantages with regard to perioperative pain, morbidity, and period of disability. Are these advantages sufficient to recommend this procedure as the first preferred therapy for people with seizures due to mesial temporal sclerosis?	
16:30-16:40	Moderator: **Lilach Goldstein**, Israel Introduction and Pre-Debate Voting	
16:40-16:55	Yes: **Michael Sperling**, USA	
16:55-17:10	No: **Matthias Koepp**, UK	
17:10-17:20	Discussion, Rebuttals and Post-Debate Voting	

17:20-18:10	Should intracranial EEG be largely restricted to patients with non-lesional epilepsy unless there is discordance between the lesion and the scalp EEG?
	Capsule: Some would argue that intracranial EEG is vastly overused without adequate evidence that it improves outcome in patients with obvious epileptogenic lesions. Is there evidence to support its use or is excision of a lesion with adequate margin sufficient to achieve a good post-surgical outcome?
17:20-17:30	Moderator: **Matthias Koepp**, UK Introduction and Pre-Debate Voting
17:30-17:45	Yes: **William Theodore**, USA
17:45-18:00	No: **Ivan Rektor,** Czech republic
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting

SATURDAY, MARCH 23, 2024		
08:20-11:00	**Multiple sclerosis: drug therapy**	**HALL C**
Chair:	**Jacek Losy,** Poland**; Dimitrios Karussis**, Israel	
08:20-09:10	Radiologically isolated syndrome (RIS): Disease modifying therapies (DMT) should only be started when symptoms have occurred	
	Capsule: RIS is considered a possible early indicator of MS or "pre-MS", but since no clinical symptoms have occurred, it is unknown how long one can have RIS without actually ever developing MS. With studies showing that more than half of RIS patients still remain symptom-free for years+, there may not be a clear indication for starting therapy	
08:20-08:30	Moderator: **Alicia Kalinowska**, Poland Introduction and Pre-Debate Voting	
08:30-08:45	Yes: **Agne Straukiene**, UK	
08:45-09:00	No: **Daniel Salo Reich,** USA	
09:00-09:10	Discussion, Rebuttals and Post-Debate Voting	
09:10-10:00	DMT should be discontinued after prolonged stability	
	Capsule: There is a sense that in some patients, MS "burns out" and patients no longer have relapses or new MRI lesions. Since all DMT are directed at reducing MS activity (relapse and MRI), it is perhaps reasonable to stop the therapy after a period of time when no MS activity has been perceived.	
09:10-09:20	Moderator: **Konrad Rejdak**, Poland Introduction and Pre-Debate Voting	
09:20-09:35	Yes: **Per Soelberg Soerensen,** Denmark	
09:35-09:50	No: **Klaus Schmierer**, UK	
09:50-10:00	Discussion, Rebuttals and Post-Debate Voting	

10:00-11:00	The central vein sign should be used as a diagnostic criterion
	Capsule: The central vein sign (CVS) adds an "in vivo histology" marker to the pattern approach of the McDonald criteria, largely based on lesion distribution in space and time. Through its apparent specificity for MS lesions, the CVS may enable a more accurate diagnosis, departing from the McDonald-proviso of "no better explanation". However, is the evidence sufficiently strong, and MRI-technology available widely enough, to include the CVS in the standard set of criteria?
10:00-10:10	Moderator: **Konrad Rejdak**, Poland Introduction and Pre-Debate Voting
10:10-11:25	Yes: **Declan Chard,** UK
10:25-10:45	No : **Daniel Salo Reich,** USA
10:45-11:00	Discussion, Rebuttals and Post-Debate Voting
11:00-11:30	**Coffee Break, Exhibition & e-Posters Visit**
11:30-12:30	**Plenary Session (Plenary Hall)**
12:30-13:30	**Industry Sponsored Symposium (Plenary Hall)**
13:30-14:30	**MTE**
13:30-14:30	**Lunch Break, Exhibition & e-Posters Visit**

14:30-16:10	**Multiple sclerosis 2**	**HALL C**
Chairs:	**Homa Ebrahimi,** Iran	
14:30-15:20	Epstein-Barr virus (EBV) is a driver of ongoing MS disease activity	
	C*apsule: EBV is a confirmed risk factor for the development of MS. However, does EBV re-activation play an important role even after the disease has been triggered? Recent evidence suggest that MS patients display aberrant EBV gene expression and regulation of both viral and cellular genes in B cells and dysregulated EBV latency. Antiviral treatments can ameliorate EBV replication, viral loads, lytic gene expression, and EBV-mediated inflammation suggesting that dysregulation of EBV latency drives MS activity. But is the evidence robust enough, and what are the counter arguments?*	
14:30-14:40	Moderator: **Floriana De Angelis**, UK Introduction and Pre-Debate Voting	
14:40-14:55	Yes: **Francesca Aloisi** , Italy	
14:55-15:10	No : **Gavin Giovannoni**, UK	
15:10-15:20	Discussion, Rebuttals and Post-Debate Voting	
15:20-16:10	Multiple Sclerosis is progressive from onset	
	Capsule: The classical vision of MS characterized in more than 90% of cases by two sequential phases, an initial relapsing remitting course followed by a secondary progressive course is now challenged by the recent observation that a progression independent from relapse activity (PIRA) can be seen very early in the disease. So is MS always progressive from the beginning?	
15:20-15:30	Moderator: **Per Soelberg Sorensen,** Denmark Introduction and Pre-Debate Voting	

15:30-15:45	Yes: **Floriana De Angelis**, UK
15:45-16:00	No: **Giancarlo Comi** , Italy
16:00-16:10	Discussion, Rebuttals and Post-Debate Voting
16:10-16:30	**Coffee Break, Exhibition & e-Posters Visit**
16:30- 18:10	**MS : Diagnosis and pathogenesis**
Chair:	**Ruth Itzhaki,** UK
16:30-17:20	Microglia are the main drivers of progressive MS
	Capsule: Microglia are known for their involvement in the immune response within the central nervous system, and their activation may contribute to inflammation observed in MS. Activated microglia can release neurotoxic substances, potentially damaging surrounding neurons and exacerbating the progression of MS. On the other hand, MS is a complex and multifactorial disease with contributions from various immune cells, genetics, and environmental factors. In addition, microglia are a heterogeneous cell population and their functions can vary based on their activation state and include also neuroprotective effects. Focusing solely on microglia may overlook other crucial elements of MS pathogenesis and might oversimplify the complex nature of the disease. Are microglia indeed the main drivers of progressive MS?
16:30-16:40	Moderator: **Ron Milo**, Israel Introduction and Pre-Debate Voting
16:40-16:55	Yes: **Alicja Kalinowska,** Poland
16:55-17:10	No: **Sharmilee Gnanapavan,** UK
17:10-17:20	Discussion, Rebuttals and Post-Debate Voting
17:20-18:10	***Enhancing recovery in MS – in cooperation with European Charcot Foundation (ECF)***
Chairman:	**Giancarlo Comi**, Italy Strategies to promote remyelination: **Hans Peter Hartung**, Germany Advances in neurorehabilitation: **Peter Feys**, Belgium Role of neuromodulation in enhancing recovery: **Letizia Leocani**, Italy
18:10	**Closing ceremony (Plenary Hall)**