



**PRELIMINARY SCIENTIFIC PROGRAM** (Subject to changes – as of February 13, 2024)

THURSDAY, MARCH 21, 2024		
08:10-10:50	<b>Neuroimmunology 1</b>	<b>HALL A</b>
Chairs:	<b>Bruno Gran</b> , UK; <b>Olaf Stuve</b> , USA	
08:10-09:00	Is Hashimoto's encephalitis/encephalopathy a valid construct in 2024?	
	<i>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?</i>	
08:10-08:20	Moderator: <b>Uros Rot</b> , Slovenia Introduction and Pre-Debate Voting	
08:20-08:35	Yes: <b>Alasdair Coles</b> , UK	
08:35-08:50	No: <b>Divyanshu Dubey</b> , 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	
	<i>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?</i>	
09:00-09:10	Moderator: <b>Ruth Gerald Nuffield</b> , UK Introduction and Pre-Debate Voting	
09:10-09:25	Yes: <b>Uros Rot</b> , Slovenia	
09:25-09:40	No: <b>Saif Huda</b> , 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	
	<i>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 to reduce the risk of relapses in MOGAD. How strong is the evidence and is this a recommendation that should be endorsed?</i>	
09:50-10:00	Moderator: <b>Joab Chapman</b> , Israel Introduction and Pre-Debate Voting	
10:00-10:15	Yes: <b>Michael Levy</b> , USA	
10:15-10:30	No: <b>Friedemann Paul</b> , Germany	
10:30-10:50	Discussion, Rebuttals and Post-Debate Voting	
<b>15:00-16:40</b>	<b>Antibody testing</b>	<b>HALL A</b>
Chairs:	<b>Brian G. Weinshenker</b> , USA; <b>Mike Zandi</b> , UK	
<b>15:00-15:50</b>	Is long COVID an autoimmune disease?	
	<i>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?</i>	
15:00-15:10	Moderator: <b>Thomas Pollak</b> , UK Introduction and Pre-Debate Voting	
15:10-15:25	Yes: <b>Michael D. Geschwind</b> , USA	
15:25-15:40	No : <b>Hans-Peter Hartung</b> , Germany	
15:40-15:50	Discussion, Rebuttals and Post-Debate Voting	
<b>15:50-16:40</b>	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?	
	<i>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?</i>	
15:50-16:00	Moderator: <b>Abhijit Chaudhuri</b> , UK Introduction and Pre-Debate Voting	
16:00-16:15	Yes: <b>Eoin Flanagan</b> , USA	
16:15-16:30	No: <b>Ruth Geraldine Nuffield</b> , UK	
16:30-16:40	Discussion, Rebuttals and Post-Debate Voting	

<b>17:00-18:40</b>	<b>Corticosteroid treatment</b>	<b>HALL A</b>
Chair:	<b><u>Boleslav Lichterman</u></b>	
<b>17:00-17:50</b>	Diabetes mellitus-associated plexopathy is an inflammatory vasculitis and should be treated with high dose corticosteroids	
	<i>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</i>	
17:00-17:10	Moderator: <b><u>Divyanshu Dubey</u></b> , USA Introduction and Pre-Debate Voting	
17:10-17:25	Yes: <b><u>Jim Dyck</u></b> , USA	
17:25-17:40	No: <b><u>Alasdair Coles</u></b> ,UK	
17:40-17:50	Discussion, Rebuttals and Post-Debate Voting	
<b>17:50-18:40</b>	Cerebral amyloid angiopathy (CAA) may lead to inflammatory vasculopathy; patients with cerebral amyloid angiitis should receive corticosteroids on diagnosis.	
	<i>Capsule: CAA is a vasculopathy characterised by amyloid beta (A<math>\beta</math>) deposition in cortical and meningeal blood vessels. Cerebrovascular A<math>\beta</math> 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.</i>	
17:50-18:00	Moderator: <b><u>Friedemann Paul</u></b> , Germany	
18:00-18:15	Yes: <b><u>Joab Chapman</u></b> , Israel	
18:15-18:30	No: <b><u>Abhijit Chaudhuri</u></b> , UK	
18:30-18:40	Discussion, Rebuttals and Post-Debate Voting	
	<b>Networking Reception (Exhibition Area)</b>	