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	

14:30-16:10	**Multiple sclerosis 2**	**HALL C**
Chair:	**Homa Ebrahimi,** Iran	
14:30-15:20	Epstein-Barr virus (EBV) is a driver of ongoing MS disease activity	
	Capsule: 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	
16:30- 18:10	**MS : Diagnosis and pathogenesis**	**HALL C**
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	

16:30- 18:10	**MS : Diagnosis and pathogenesis**	**HALL C**
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**	