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Oral Presentation

The Pathophysiological Hallmarks of MS Beyond the Blood Brain Barrier: Myelination and Neuronal Network Interactions

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Abstract

Multiple Sclerosis (MS) is a complex disease resulting from the occurrence of intermingled episodes of neuro-inflammation and degeneration. The temporal and spatial patterns in which these events occur are not well understood as well as the molecular substrates underlying it. Myelin loss and gain, as well as axonal damage are considered crucial events influencing the course of the disease but their cause/effect dependency remains unclear. Numerous recent evidence showed impaired cognitive behaviors both in MS patients and animal models, which would support a profound involvement of neurons in mediating such effects, along to the long-known MS hallmarks like locomotor deficits and slow axonal conductance, attributed mainly to myelin loss. In order to investigate the role of neurons and their functionality in the pathophysiology on MS we took advantage of different animal models of neuro-inflammation and general de- and remyelination, namely the experimental autoimmune encephalitis (EAE) and the cuprizone model, respectively. For both animals we could observe the occurrence of different cognitive impairments including loss of short and long term memory and of high functional cortical abilities. In both animals models we could associate these symptoms to an altered neuronal network excitability and therefore, we pursued pharmacological modulation in vitro and in vivo in order to verify this finding by identifying potential molecular players. Therefore, by using different novel or established compounds we tried to identify new drugable targets and new therapeutic time windows for intervention.

Keywords: Multiple Sclerosis, Animal Models, Locomotor Deficits.

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