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Poster Presentation

The Role of Kv7-Channels in the Pathophysiology of Multiple Sclerosis

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Abstract

Multiple sclerosis is an autoimmune CNS-disease characterized by inflammatory neurodegenerative events occurring with de- and remyelination. Recent evidence show that demyelinated neurons are less excitable than myelinated ones while at early stages of remyelination these neurons seem to be hyperexcitable. The latter is a transitory condition that, very likely, leads to impaired neuronal network functioning also at late remyelination stages. The mechanism underlying these conditions are not clear but recent studies indicate the Kv7 channels as potential molecular players as they were shown to be altered following demyelination and, involved in regulating neuronal excitability. Therefore, we focus on assessing their role in MS pathophysiology, both in influencing the immune-system or the neuronal functionality. Using the experimental autoimmune encephalitis model (EAE) in C57Bl/6 mice, we can show that a prophylactic treatment with the specific KV7-channel opener Retigabine improves the course of the disease. We observe that Retigabine does not change the proliferation of stimulated splenocyte in culture. By quantifying the cytokines contained in their supernatant, we see no differences in which type of immune cells are proliferating. This indicates that Retigabine does not have a direct effect on the immune system. Furthermore, we investigate the neuronal contribution to the disease by using the cuprizone mouse model of general de- and remyelination. Following myelin loss, a pavlovian conditioning paradigm shows altered cognition in vivo and altered excitability in vitro. Pharmacological modulation performed during the spontaneous remyelination following cuprizone-induced myelin loss will target neurons and try to achieve neuronal protection. Taken together, our results add some interesting information about the involvement of Kv7 channel in MS pathophysiology paving the way for further studies

Keywords: Autoimmune, Multiple Sclerosis, Inflammatory

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