Special Issue

The Immune System as a Target for Therapy of Progressive Multiple Sclerosis (MS) in Patients and Animal Models

Message from the Guest Editor

In progressive MS (PMS), the relatively intact blood–brain barrier generates compartmentalized CNS immune inflammation, decorated by ectopic lymphoid structures and characterized by the accumulation of lymphocytes, macrophages and microglia. These cells release reactive oxygen and nitrogen species and pro-inflammatory cytokines/chemokines, leading to demyelination, axonal lesions and mitochondrial damage. While relatively many disease-modifying therapies have shown efficacy in ameliorating relapsing-remitting MS (RRMS), the majority of these have been ineffective for PMS (e.g., copaxone, azathioprine), and the number of approved medications for PMS is very limited. The differential therapeutic effect between RRMS and PMS is possibly related to qualitative or quantitative differences in inflammatory phenotypes, e.g., distinct cell subpopulations, cytokine profiles and inflammation sites. Analyzing these inflammatory phenotypes as well as other differences may help to understand why beneficial medications are documented in RRMS, but not in PMS, what characteristics underline the few approved PMS medications and how PMS drugs should be designed.

Guest Editor

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Deadline for manuscript submissions

closed (15 March 2024)

International Journal of Molecular Sciences

an Open Access Journal by MDPI

Impact Factor 4.9
CiteScore 9.0
Indexed in PubMed

mdpi.com/si/120918

International Journal of Molecular Sciences
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