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Research Title : Neurofascin as a novel target for autoantibody-mediated axonal injury

Neurofascin as a novel target for autoantibody-mediated axonal injury

Descriptipn : Axonal injury is considered the major cause of disability in patients with multiple sclerosis (MS), but the underlying effector mechanisms are poorly understood. Starting with a proteomics based approach we identified neurofascin (NF)-specific autoantibodies in patients with MS. These autoantibodies recognize the native form of the extracellular domains of both NF186, a neuronal protein concentrated in myelinated fibers at nodes of Ranvier, and NF155, the oligodendrocyte-specific isoform of neurofascin. Our in vitro studies with hippocampal slice cultures indicate that neurofascin antibodies inhibit axonal conduction in a complement dependent way. To evaluate whether circulating anti-neurofascin antibodies mediate a pathogenic effect in vivo we co-transferred these antibodies with myelin oligodendrocyte glycoprotein-specific encephalitogenic T cells to mimic the inflammatory pathology of MS and breach the blood-brain barrier. In this animal model antibodies to neurofascin selectively targeted nodes of Ranvier, resulting in deposition of complement, axonal injury and disease exacerbation. Together, these results identify a novel mechanism of immune mediated axonal injury that can contribute to axonal pathology in MS

Research Type : Article

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Attatchments :

File Name	Type	Description
Neurofascin.pdf	pdf	مشاهدة المقالة العلمية كاملة