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Influence of Type I IFN signalling on anti-MOG-mediated demyelination.

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Background

Multiple Sclerosis (MS) is an inflammatory demyelinating disease of the central nervous system (CNS). Although considered a T cell-mediated disease, many MS lesions show deposition of antibodies and activated complement (C). Myelin oligodendrocyte glycoprotein (MOG) is a candidate autoantigen in MS. Interferon beta (IFN-β**),** a Type I IFN, is used to treat MS. Type I IFN signaling regulates leukocyte infiltration and demyelination and severity of experimental autoimmune encephalomyelitis (EAE), an MS-like disease in mice.

Aim

To investigate the influence of Type I IFN signaling on anti-MOG antibody-mediated pathology in EAE.

Methods

EAE was induced by immunization of C57BL/6 and IFNAR1-KO mice with MOGp35-55. Purified IgG2a anti-MOG Mab and mouse C were stereotactically injected into the corpus callosum (CC) as soon as mice showed symptoms. Demyelination (loss of luxol fast blue staining) was quantitated using ImageJ.

Results and Conclusions

Anti-MOG Mab induced C-dependent demyelination in CC of C57BL/6 mice, that was significantly reduced in IFNAR1-KO mice. Severity and time of onset of EAE were similar between C57BL/6 and IFNAR1-KO mice, and EAE did not induce CC demyelination in either strain. By contrast, anti-MOG Mab + C-induced demyelination in CC of mice with EAE was significantly increased in IFNAR1-KO compared to C57BL/6 mice.

Conclusion: The effect of Type I IFN signaling on antibody-mediated demyelinating pathology is influenced by the CNS inflammatory environment.

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