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Mechanisms of T-B cell cooperation important for MOG antibody mediated demyelination

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Article:

Antibodies to MOG have a demyelination phenotype and affect oligodendrocyte cytoskeleton

Dale RC, Tantsis EM, Merheb V, et al. Antibodies to MOG have a demyelination phenotype and affect oligodendrocyte cytoskeleton. *Neurol Neuroimmunol Neuroinflamm*. 2014 May 22; 1(1):e12. doi: 10.1212/NXI.0000000000000012

Letter in Response to Above Article:

Mechanisms of T-B cell cooperation important for MOG antibody mediated demyelination

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The article by Dale et al. is significant, reporting the presence of demyelinating antibodies to MOG in pediatric relapsing ON. [1] The role of complement activating antibodies to MOG is suggested in pathogenesis of adult AQP4- ON. [2] In vitro experiments have indicated that MOG antibodies isolated from pediatric ON patients perturb organization of oligodendrocyte microtubule cytoskeleton. [1]

Similarly, pronounced demyelination, damage of axonal cytoskeleton, and oligodendrocytes were observed in experimental autoimmune encephalomyelitis (EAE) induced by immunization with MOG35-55, in (B6 x SJL)F1 (H-2b/s) mice. These mice developed relapsing-remitting disease, with prominent inflammation by CD4+ T cells followed by B220+ B cells, scattered in small perivascular infiltrates throughout spinal cord. Production of a cytokine IL-16 by infiltrating CD4+ and B220+ T cells in CNS of relapsing EAE mice, has been demonstrated. [3]

IL-16 is a CD4+ T cell specific chemotactic cytokine. This cytokine regulates fundamental biological properties of CD4+ T cells, including T - B cell cooperation. [4] Cooperation between T and B cells in regulation of autoimmune responses to MOG35-55 is critical as demonstrated by double transgenic EAE mouse models. [5] Studies in appropriately selected animal models, which resemble immunopathology and histopathology of anti-MOG+, AQP4- ON are needed.

1. Dale RC, Tantsis EM, Merheb V, et al. Antibodies to MOG have a demyelination phenotype and affect oligodendrocyte cytoskeleton. *Neurol Neuroimmunol Neuroinflamm* 2014;1:e12.

2. Mader S, Gredler V, Schanda K, et al. Complement activating antibodies to myelin oligodendrocyte glycoprotein in neuromyelitis optica and related disorders. *J Neuroinflammation* 2011;8:184.

3. Skundric DS, Dai R, Zakarian VL, et al. Anti-IL-16 therapy reduces CD4+ T-cell infiltration and improves paralysis and histopathology of relapsing EAE. *J Neurosci Res* 2005;79:680-693.

4. Kaser A, Dunzendorfer S, Offner FA, et al. B lymphocyte-derived IL-16 attracts dendritic cells and Th Cells. *J Immunol* 2000;165:2474 -2480.

5. Bettelli E, Baeten D, Jager A, et al. Myelin oligodendrocyte glycoprotein-specific T and B cells cooperate to induce a Devic-like disease in mice. *J Clin Invest* 2006;116:2393-2402.

For disclosures, please contact the editorial office at nnjournal@neurology.org.

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