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Central nervous system demyelinating disease protection by the human commensal Bacteroides fragilis depends on polysaccharide A expression.

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Abstract

The importance of gut commensal bacteria in maintaining immune homeostasis is increasingly understood. We recently described that alteration of the gut microflora can affect a population of Foxp3(+)T(reg) cells that regulate demyelination in experimental autoimmune encephalomyelitis (EAE), the experimental model of human multiple sclerosis. We now extend our previous observations on the role of commensal bacteria in CNS demyelination, and we demonstrate that Bacteroides fragilis producing a bacterial capsular polysaccharide Ag can protect against EAE. Recolonization with wild type B. fragilis maintained resistance to EAE, whereas reconstitution with polysaccharide A-deficient B. fragilis restored EAE susceptibility. Enhanced numbers of Foxp3(+)T(reg) cells in the cervical lymph nodes were observed after intestinal recolonization with either strain of B. fragilis. Ex vivo, CD4(+)T cells obtained from mice reconstituted with wild type B. fragilis had significantly enhanced rates of conversion into IL-10-producing Foxp3(+)T(reg) cells and offered greater protection against disease. Our results suggest an important role for commensal bacterial Ags, in particular B. fragilis expressing polysaccharide A, in protecting against CNS demyelination in EAE and perhaps human multiple sclerosis.

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