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Identifying new Treg cells in MS patients

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Inflammation is a self-destructive process that can lead to irreversible chronic tissue destruction. The defective generation or function of $T_{regulatory}$/T_{reg} cells contributes to chronic autoimmune inflammation. We report the first identification of FoxA1 as a novel transcription factor in T-cells that upon ectopic expression conveys suppressive properties in a new T_{reg} population, hereby called FoxA1$^{+}T_{regs}$. FoxA1 bound to the *pdl1* promoter, inducing PD-L1, which was essential for FoxA1+T_{regs} to kill activated T-cells. FoxA1+Tregs had a distinct transcription profile. They express CD4, CD47and PD-L1^{hi}. IFN-β induces FoxA1+Tregs requiring IFNAR signaling; consequently *Ifnb*$^{-/-}$ and *Ifnar*$^{-/-}$ mice were defective in FoxA1+T_{regs}. Adoptive transfer of stable FoxA1+T_{regs} inhibited experimental autoimmune encephalomyelitis mediated by functional FoxA1 and PD-L1. In patients with relapsing-remitting multiple sclerosis, response to IFN-β-treatment was associated with expansion of suppressive FoxA1+T_{regs}. FoxA1 is a lineage-specification factor with a specialized role in supporting differentiation and the suppressive function of FoxA1+T_{reg} cells.

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Nogo-receptor 1 expression on B-cell populations in the central nervous system during experimental autoimmune encephalomyelitis

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Although the fact that deletion of Nogo receptor 1 (NgR1) can protect against axonal degeneration and thus progression of disease, in the animal model of multiple sclerosis (MS), experimental autoimmune encephalomyelitis (EAE), the immunological role of this receptor is unclear. To further understand the function of NgR1 in regulating immune cells, flow cytometry-based phenotypic analysis was performed on isolates from spleens, lymph nodes and spinal cords at different clinically defined stages of EAE disease. The central nervous system (CNS)-infiltrating blood cells revealed an augmented response in the B-cell population, which expressed NgR, seeing in *ngr1*$^{+/+}$ mice with the onset and progression of the disease. This population of cells could not be demonstrated within the spinal cords of EAE-induced *ngr1*$^{-/-}$ mice or during the chronic stage of disease in *ngr1*$^{+/+}$ mice. At the onset of disease onset, there was a significant increase in IgM-B-cells-expressing NgR in the spinal cord, when compared with the IgD populations. Remarkably, there was a cluster of B-cells expressing NgR present at the meninges of the spinal cords of *ngr1*$^{+/+}$ EAE-induced mice at clinical score 1.5 and these cells localised within small follicles in submeningeal regions. Furthermore, there was clustering of B-cell activating factor (BAFF) and NgR-positive immune cell infiltrates within the spinal cords of EAE-induced *ngr1*$^{+/+}$ mice at disease onset. Collectively, these data indicate that there exists the inducible expression of NgR1 in specific immune lineage cells upon the induction of EAE, as well as, a strong correlation between the expression profiles of NgR1 and BAFF on neighbouring B-cells within spinal cord follicular structures.

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