Supporting Information

B cell-derived transforming growth factor-β1 expression limits the induction phase of autoimmune neuroinflammation

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Short title: B cell-derived TGF-β1 production in CNS autoimmunity

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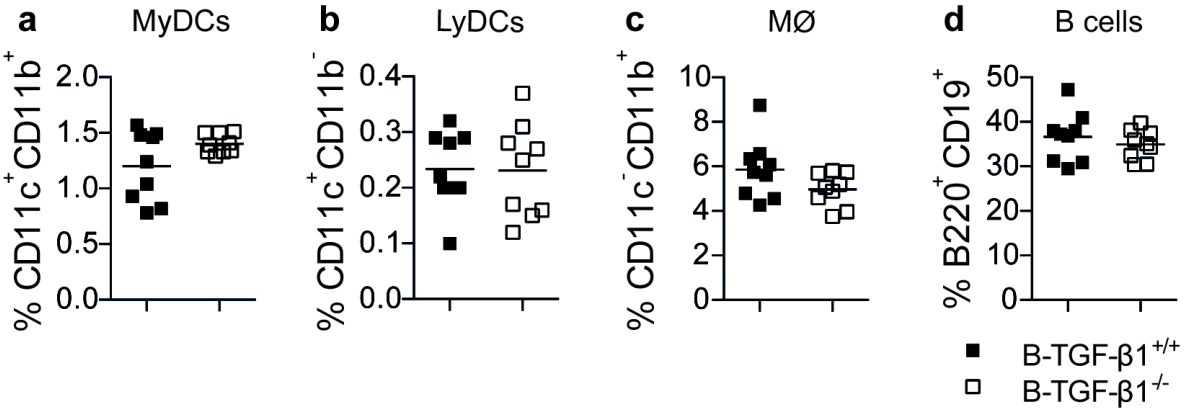
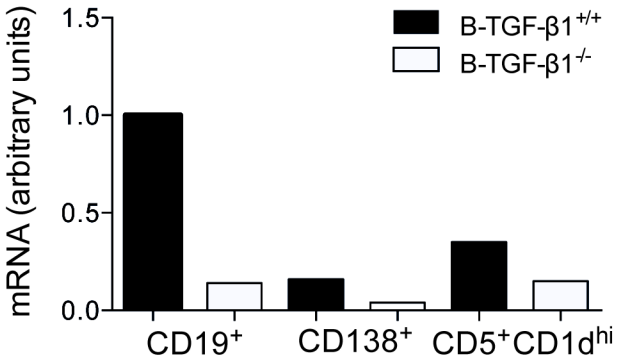
Supplementary Figure S1



Supplementary Figure S1: Selective B cell TGF-β1−deficiency does not regulate the frequencies of APCs in naïve mice. APC subpopulations were characterized by flow cytometry from B–TGF-$\beta1^{+/+}$ (black) and B–TGF-$\beta1^{-/-}$ (open) naïve mice. Frequency of (**a**) myeloid ($CD11c^{+}CD11b^{+}$) DCs, (**b**) lymphoid ($CD11c^{+}CD11b^{-}$) DCs, (**c**) monocytes/macrophages ($CD11c^{-}CD11b^{+}$), (**d**) and B cells ($B220^{+}CD19^{+}$). Data are a composite of two independent experiments with similar results.

Supplementary Figure 2



Supplementary Figure S2: Quantification of TGF-β1 expression by $CD19^{+}CD5^{-}$ $CD1d^{lo}$ B cells, $CD138^{+}$ plasma cells and $CD19^{+}CD5^{+}CD1d^{hi}$ B cells from B–TGF-$\beta1^{+/+}$ and B–TGF-$\beta1^{-/-}$ EAE mice. Levels of TGF-β1 mRNA in $CD19^{+}CD5^{-}$ $CD1d^{lo}$ B cells, $CD138^{+}$ plasma cells, and $CD19^{+}CD5^{+}CD1d^{hi}$ B cells (n=1) sorted from spleen of rmMOG-immunized B–TGF-$\beta1^{+/+}$ (black) and B–TGF-$\beta1^{-/-}$ (open) mice were assessed by quantitative real-time PCR. Data are from one experiment representative of two independent experiments with similar results.