

Supplemental Figures

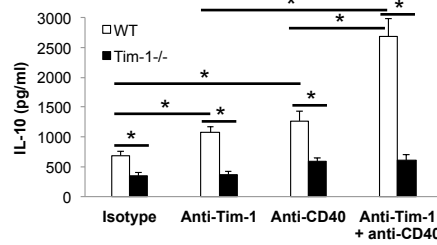


Figure S1. Tim-1 and CD40 signaling together strongly promoted B cell IL-10 production while Tim-1 defect in B cells reduced IL-10 production. Purified splenic CD19⁺ B cells from 2-3 month-old WT and Tim-1^{-/-} mice were cultured in the presence of anti-Tim-1 (clone 5F12), anti-CD40 or both. After 3 days, IL-10 production in culture supernatants was measured by CBA. * P < 0.01; n = 3.

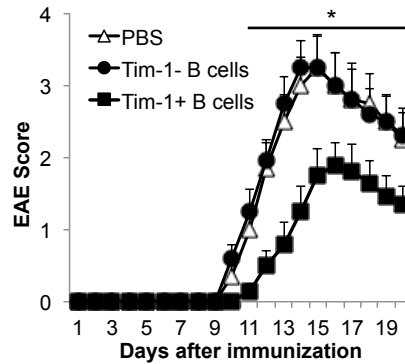


Figure S2. Transfer of Tim-1⁺ Bregs but not Tim-1⁻ B cells reduced EAE severity. Splenic Tim-1⁺ and Tim-1⁻ CD19⁺ B cells (2×10^6) purified from WT mice were transferred into WT mice. One day later, the mice were immunized with MOG35-55/CFA to induce EAE. Mice (n = 5 per group) were scored daily for clinical signs of EAE. * P < 0.05.

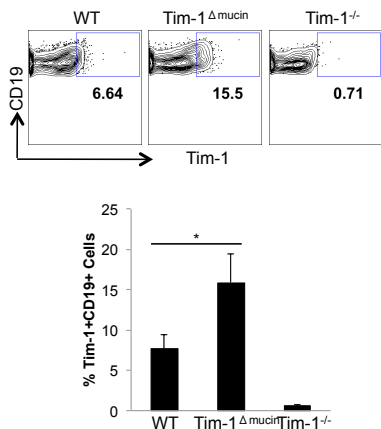


Figure S3. Tim-1^{Δmucin} mice at 12⁺ months of age showed increased Tim-1⁺ Bregs. Frequencies of Tim-1⁺ Bregs in spleens of 12⁺-month old WT, Tim-1^{-/-}, and Tim-1^{Δmucin} mice were determined by flow cytometry. Representative histograms and bar graphs with cumulative data are shown. * P < 0.01; n ≥ 3 per group.

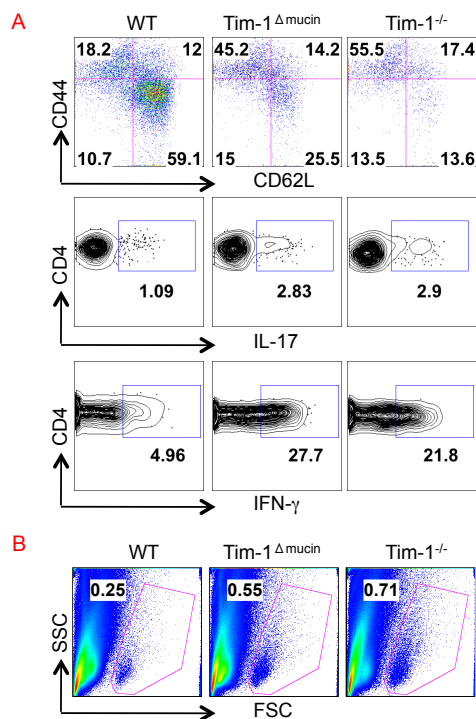


Figure S4. Tim-1^{-/-} mice, like Tim-1^{Δmucin} mice at 12⁺ months of age developed hyper-activated IFN-γ⁺ and IL-17⁺ T cells and had more mononuclear cell infiltration in livers. **A)** Splenic CD4⁺ T cell phenotypes in 12⁺-month old WT, Tim-1^{-/-}, and Tim-1^{Δmucin} mice were determined by flow cytometry. **B)** Single cell suspension of livers from mice in **A** showed increased accumulation of mononuclear cells as determined by flow cytometry. n ≥ 3 per group.