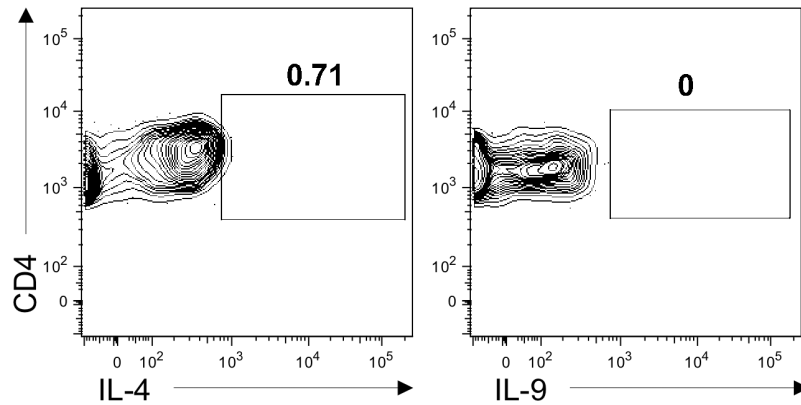
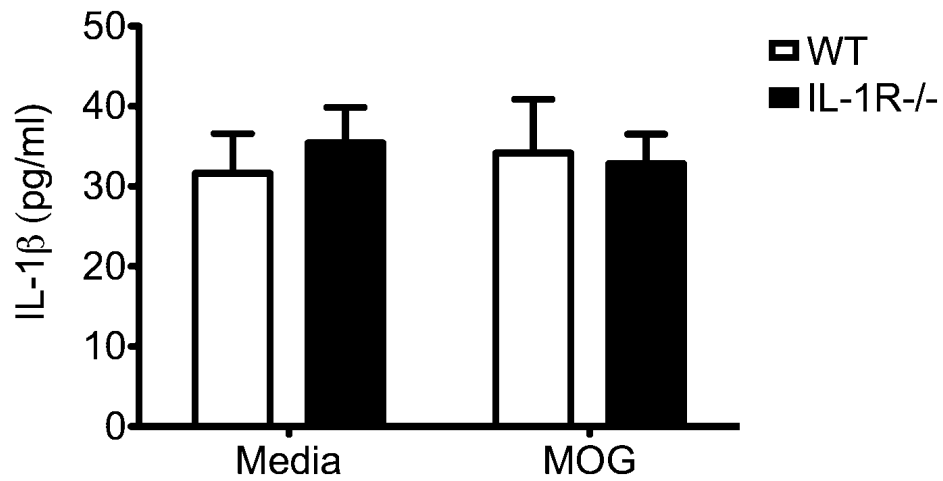


Supplemental Figure 1. Anti-CD3 stimulation induces low but detectable levels of IL-1 β . Splenocytes and lymph node cells were harvested from wild-type (WT) or IL-1R^{-/-} mice. Cells were stimulated with anti-CD3 in the presence of IL-1 β , IL-23, IL-1 β and IL-23, IL-12, or IL-12 and IL-1 β . As a control, dLN cells from IL-1 β ^{-/-} mice that were immunized with MOG/CFA were collected on day 10 and restimulated with MOG peptide. Supernatants were collected at 72 hrs for analysis of IL-1 β production by ELISA. Data are representative of four independent experiments with 2-3 mice per group. Data represent mean \pm SEM.



Supplemental Figure 2. Pathogenic CD4⁺GM-CSF⁺IL-17⁺IFN- γ ⁻ T cells are not T_H2 or T_H9 cells. WT mice were immunized with MOG/CFA. On day 33, splenocytes were stimulated with PMA/ionomycin for 5 hrs followed by intracellular staining for GM-CSF, IL-17, IFN- γ , IL-4, and IL-9. CD4⁺ T cells that produce GM-CSF were gated and then this population was expanded to evaluate the expression of IL-17 and IFN- γ . The levels of IL-4 (left panel) and IL-9 (right panel) was assessed in the CD4⁺GM-CSF⁺IL-17⁺IFN- γ ⁻ T cell population. Data are representative of eight mice.



Supplemental Figure 3. No difference in IL-1 β levels in the absence of IL-1R during EAE. WT and IL-1R^{-/-} mice were immunized with MOG/CFA. (A) On day 10, splenocytes were stimulated for 48 hrs with MOG peptide and IL-1 β was measured by ELISA. Data are representative of four independent experiments with at least 4 mice per group.