Supplementary Data

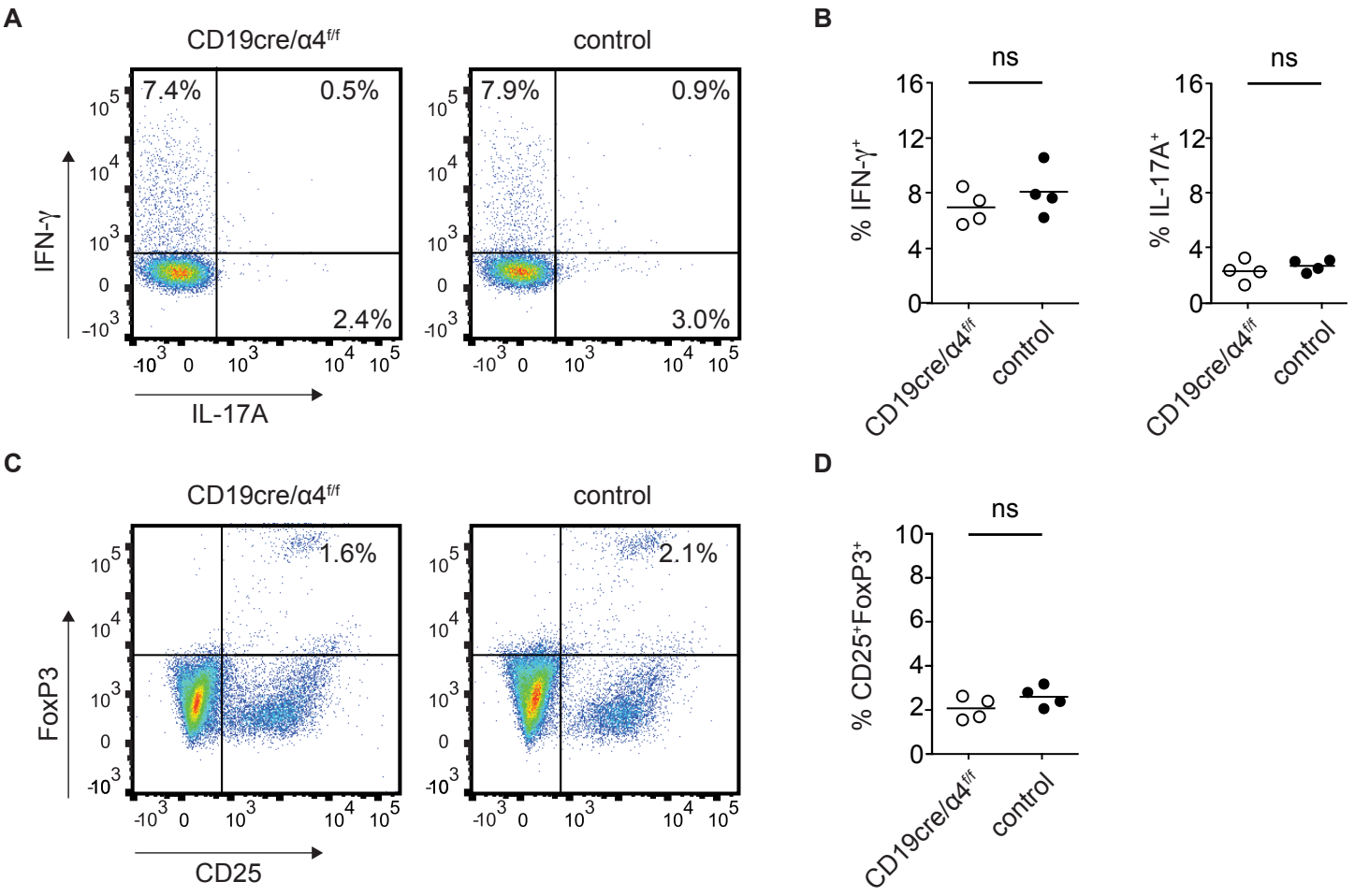

Figure e-1. Splenic proinflammatory T cell differentiation and Treg are unchanged in MOG p35-55-induced EAE in B cell VLA-4-deficient mice. CD19cre/α4$^{f/f}$ or control mice (n = 6 mice/group; pooled from 2 independent experiments) were immunized with 100 µg MOG p35-55. Spleen cells were collected 14 days after immunization. (A) Th1 (IFN-γ secreting) and Th17 (IL-17A secreting) T cell differentiation is measured by intracellular cytokine staining (ICS). Flow cytometry plots show one representative mouse per group (gated on viable CD4$^+$ T cells). (B) Frequency of Th1 (left) and Th17 (right) cells. (C) CD25$^+$FoxP3$^+$ Treg are quantified similarly and flow cytometry plots show one representative mouse per group (gated on viable CD4$^+$ T cells). (D) Frequency of Treg. All data shown in graphs are mean frequency (%) ± SEM. ns = not significant; unpaired t test with Welch's correction.

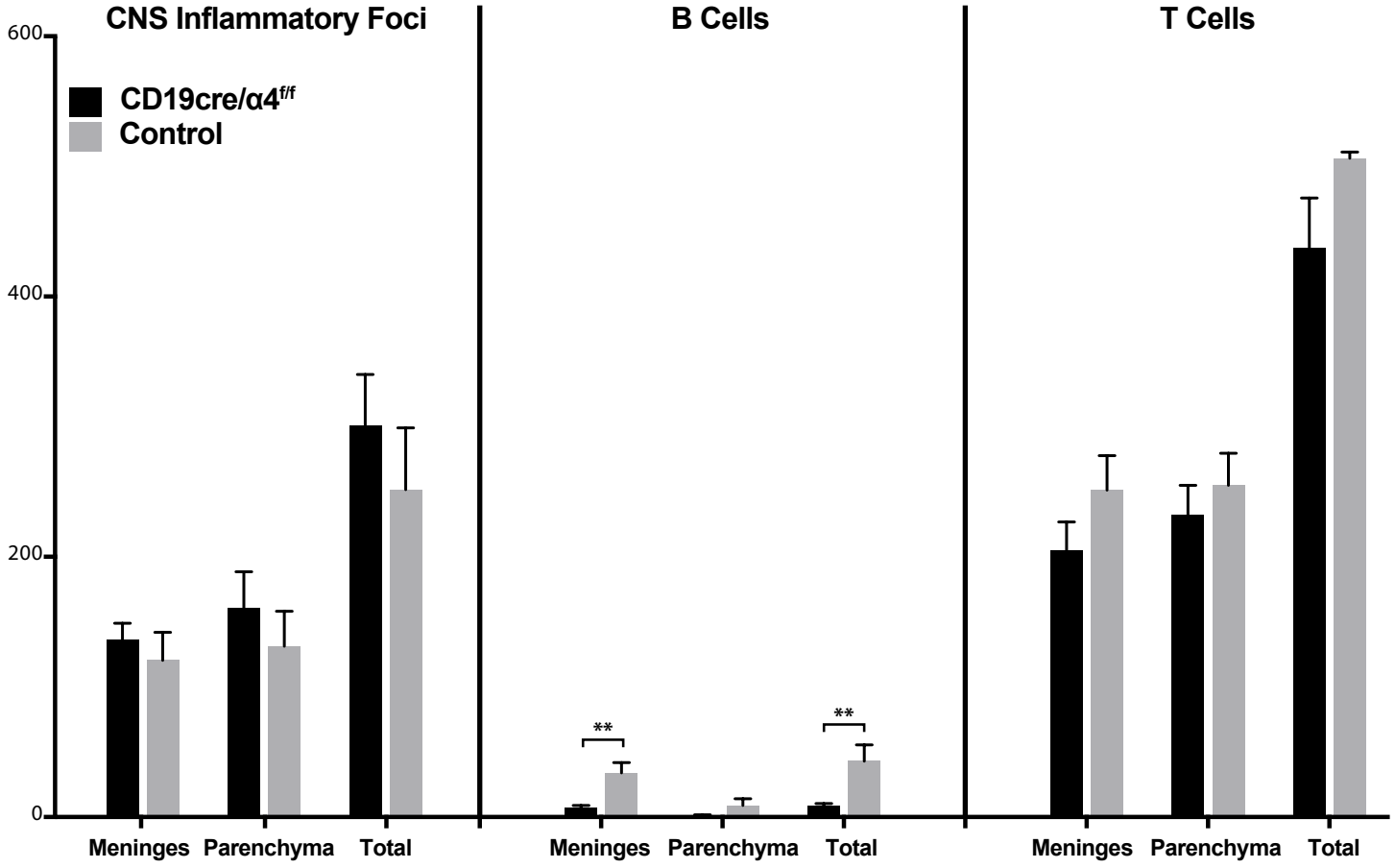

Figure e-2. B cell VLA-4 deficiency reduced CNS accumulation of B cells, but did not influence the number of inflammatory foci or accumulation of T cells. Counts of inflammatory foci were performed in paraffin sections with Luxol fast blue-H&E, (left, for inflammatory infiltrates), and immuno-histochemistry for B and T cells (middle and right). Data are from 6 CD19cre/$\alpha 4^{f/f}$ and 3 CD19cre (control) mice per group. Bar graphs show mean ± SEM. **, $p \leq 0.01$; unpaired t tests.