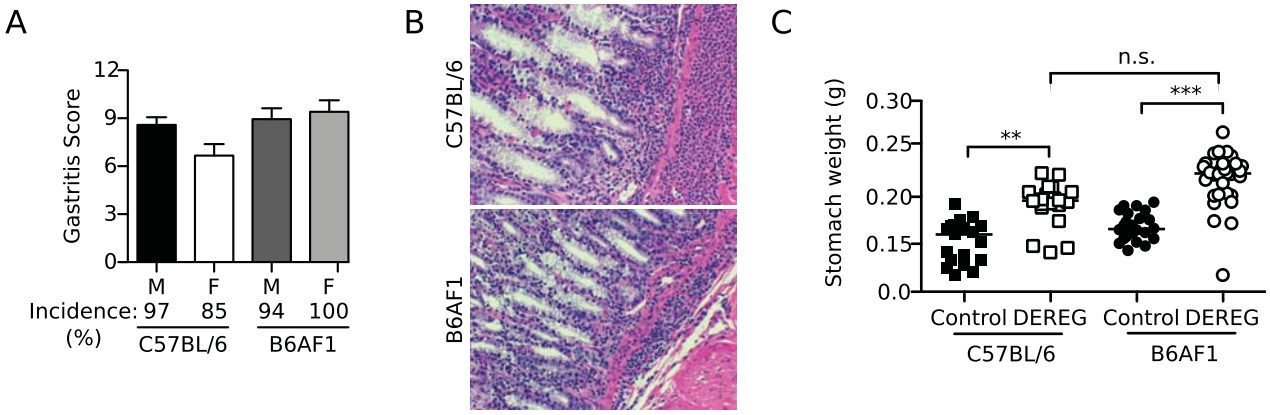
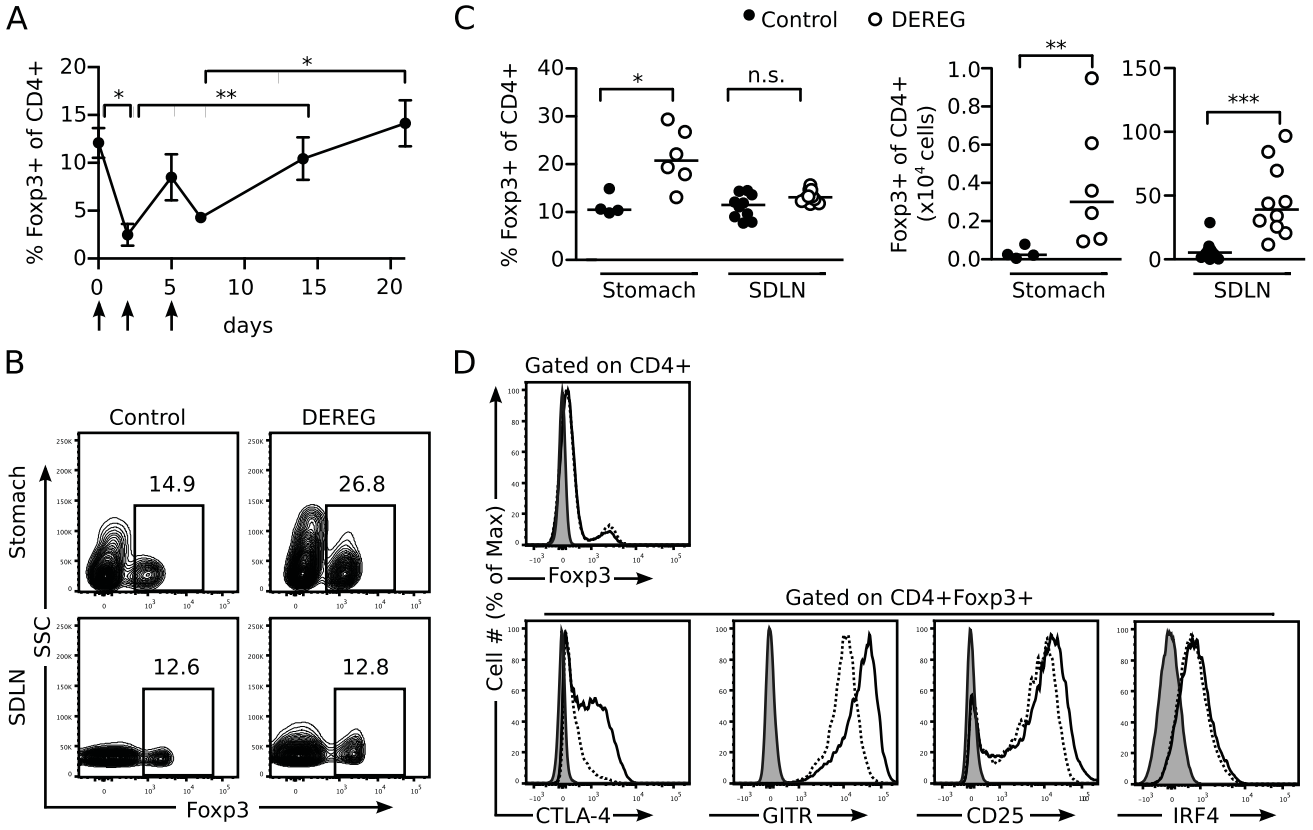
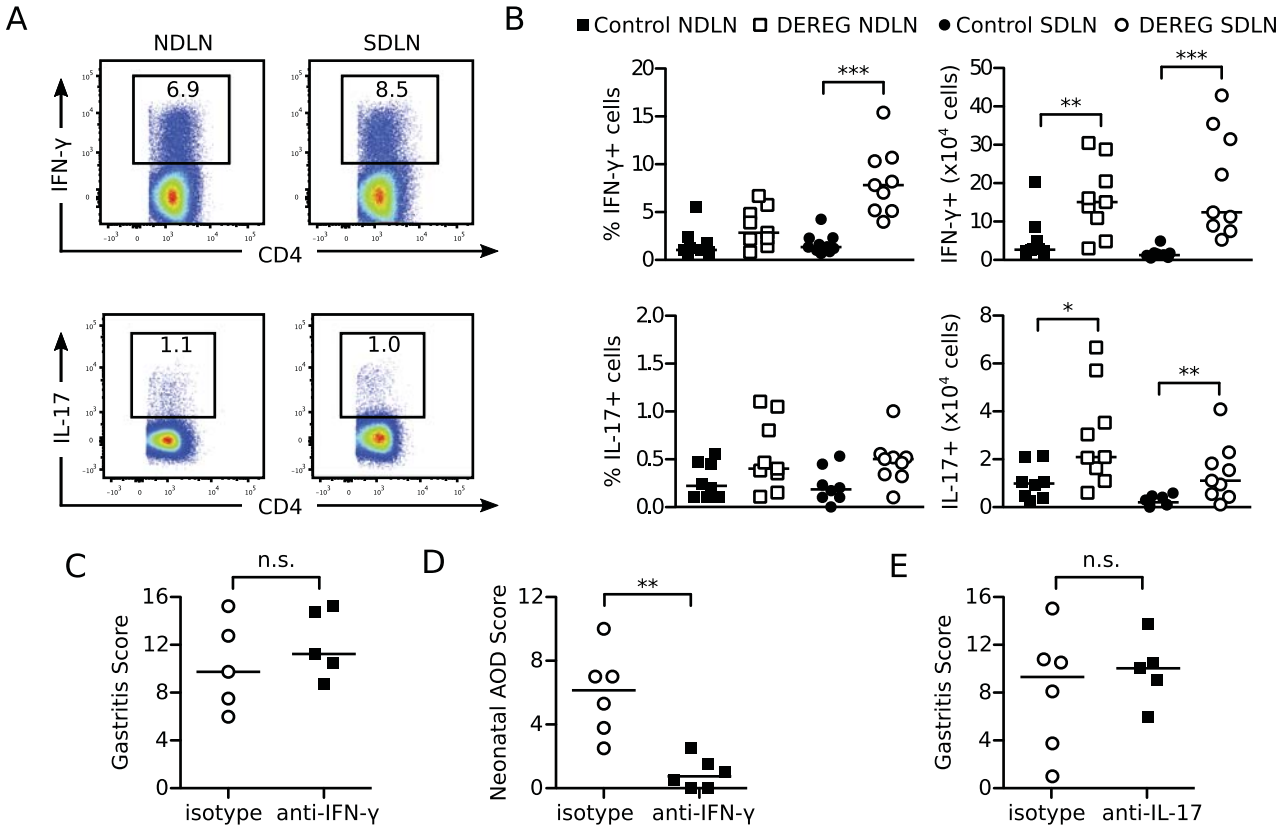


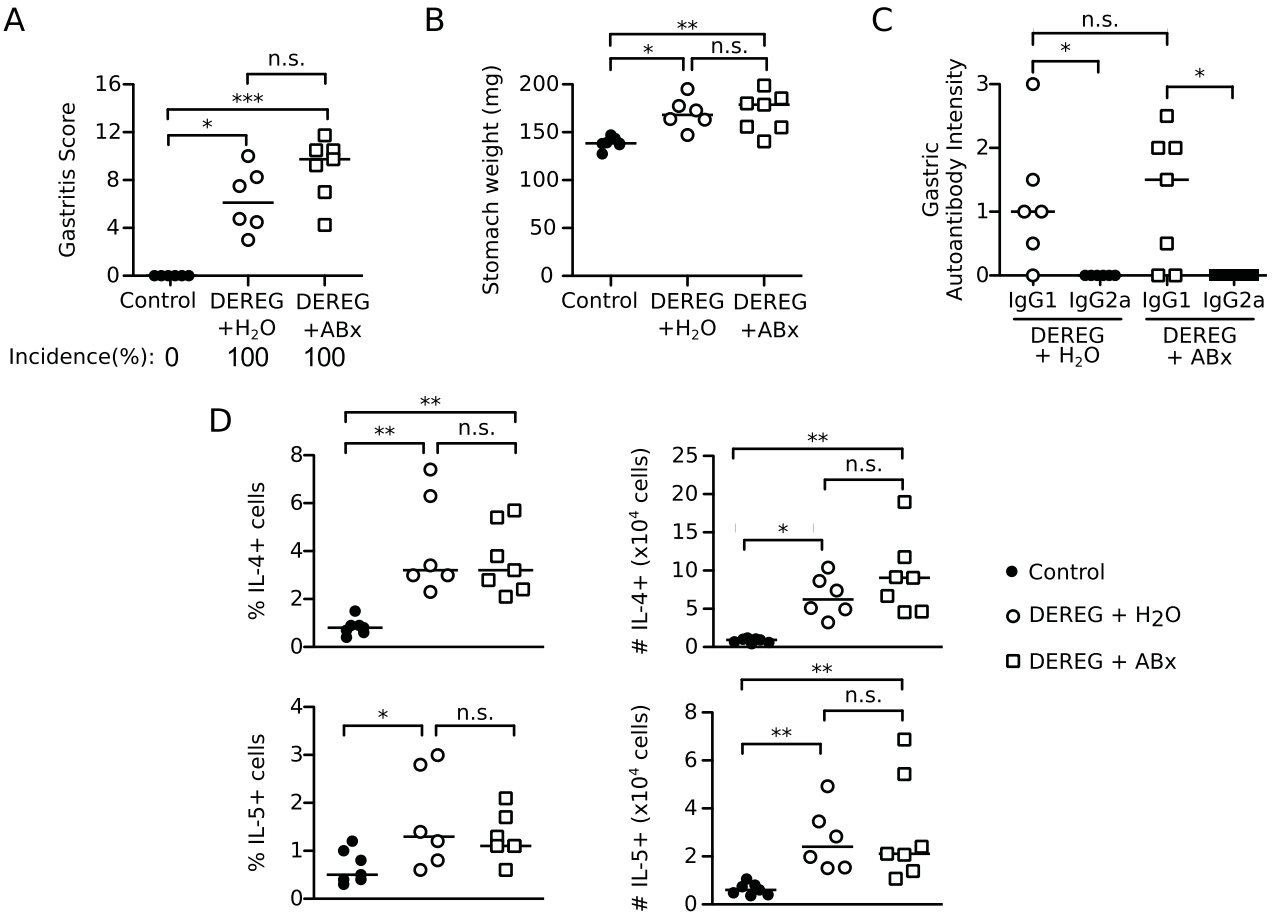
Supplemental Figure 1: AIG incidence and severity are comparable between C57BL/6 and B6AF1 mice and between male and female mice. (A) Gastritis severity and incidence at 3 weeks in male (M) and female (F) C57BL/6 and B6AF1 DEREG mice. (B) Representative stomach histopathology (H&E, x200) at 3 weeks. (C) Comparison of stomach weights between control and Treg cell-depleted C57BL/6 and B6AF1-DEREG mice. Data in (A) display the mean and SEM. Data were pooled from 3-11 independent experiments per group. Each symbol in (C) represents an individual mouse. $p>0.05$ for all comparisons in (A), $^{**}p<0.01$ and $^{***}p<0.001$; Kruskal-Wallis test with Dunns posttest. Data in subsequent experiments were pooled from both mouse strains and genders.



Supplemental Figure 2: The stomach- and draining lymph node distribution and suppressive capacity of normal and rebounded Treg cells is comparable. (A) Kinetics of changes in the percent of Foxp3+ Treg cells among total CD4+ T cells in pooled lymph nodes after DT treatment (indicated by arrows). (B) Representative flow cytometry contour plots depicting the frequency of Foxp3+ Treg cells in the stomach and SDLN of control or Treg cell-depleted mice at 3 weeks (numbers indicate percentages of cells in the gates). (C) Quantitation of B as percentage (left) and absolute number (right). (D) Expression levels of Foxp3, CTLA-4, GITR, CD25, and IRF4 on Treg cells from the SDLN of control and Treg cell-depleted mice at 3 weeks. A representative histogram showing the MFI of Foxp3 among live CD4+ cells (top) and of CTLA-4, GITR, CD25, and IRF4 among live CD4+ Foxp3+ cells (bottom) are shown. The gray shaded peaks are isotype controls; dotted and continuous lines are control and Treg cell-depleted DEREG mice, respectively. Data were pooled from 6 (B, C) and 3 (A, D) independent experiments. Each symbol represents an individual mouse. *$p<0.05$, **$p<0.01$; Kruskal-Wallis with Dunns posttest (A); Mann-Whitney t tests (C).



Supplemental Figure 3: Th1 and Th17 responses do not contribute to AIG development in Treg cell-depleted mice. (A) Representative flow cytometry dot plots of IFN-γ and IL-17 producing CD4+ T cells in the NDLN and SDLN of control and Treg cell-depleted DEREG mice at 3 weeks. Cells were stimulated *ex vivo* with PMA and ionomycin before flow cytometry analysis (numbers indicate percentages of cells in the gates). (B) Quantitation of A. (C) Gastritis scores at 3 weeks of Treg cell-depleted DEREG mice with continuous IFN-γ antibody or isotype IgG treatment. (D) Neonatal autoimmune ovarian disease (nAOD) scores at 2 weeks after disease induction in mice with continuous IFN-γ antibody or isotype IgG treatment. (E) Gastritis scores at 3 weeks of Treg cell-depleted DEREG mice with continuous IL-17 antibody or isotype IgG treatment. Data were pooled from 3 (A, B, C, E) or 4 (D) independent experiments. Each symbol represents an individual mouse. *p<0.05, **p<0.01; Mann-Whitney t tests (B right, C, D, E); Kruskal-Wallis test with Dunns posttests (B left).



Supplemental Figure 4: Intestinal microbiome alteration by oral antibiotic treatment did not affect the development of Th2-dominant AIG in Treg cell-depleted DEREG mice. (A) Gastritis severity and prevalence, (B) stomach weights, and (C) serum IgG1 and IgG2a gastric autoantibody intensities by immunofluorescence staining at 3 weeks in DT-treated control and DEREG mice treated with water or antibiotics (ABx). (D) Quantification of flow cytometry analysis of IL-4 (top) and IL-5 (bottom) producing CD4+ T cells in the SDLN of control and Treg cell-depleted DEREG mice treated with water or ABx (cells were stimulated *ex vivo* by PMA and ionomycin before flow cytometry analysis; left: percentage; right: absolute number). Data were pooled from 2 independent experiments. Each symbol represents an individual mouse. *p<0.05, **p<0.01, ***p<0.001; Kruskal-Wallis tests with Dunns posttests.