PD-1 deficiency protects experimental colitis via alteration of gut microbiota

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Supplementary Figure 1. Enhanced production of IL-12 is not responsible for the resistance to colitis in PD-1−/− mice. (A) Colon lamina propria mononuclear cells (LPMCs) from DSS-untreated (DSS negative) or DSS-treated (DSS positive) mice for 9 d were isolated and stimulated in vitro with CpG DNA for 24 h. Each cytokine production in the culture supernatant was measured by ELISA. The bar graph shows amount (mean ± SEM) of indicated cytokines (pg/ml). Data are pooled from three different experiments (n = 4 - 7 in each group). *p < 0.05, **p < 0.01 vs. WT controls by Student’s t-test. Representative data (mean ± SEM) from two independent experiments are shown. (B) At 1 d before and 2 d after DSS administration, 200 µg of αIL-12/p40 mAb was intravenously injected to PD-1−/− mice. Same volume of PBS was injected as a control. Data are representative of two independent experiments with similar results (n = 5 per each group).
Supplementary Figure 2. Cytokine production in colon lamina propria after DSS administration. Colon lamina propria mononuclear cells (LPMCs) from DSS-untreated (DSS negative) or DSS-treated (DSS positive) mice for 9 d were isolated and stimulated in vitro with CpG DNA for 24 h. Each cytokine production in the culture supernatant was measured by ELISA. The bar graph shows amount (mean ± SEM) of indicated cytokines (pg/ml). Data are pooled from three different experiments (n = 4 - 7 in each group). *p < 0.05, **p < 0.01 vs. WT controls by Student’s t-test. Representative data (mean ± SEM) from two independent experiments are shown.