

Supplementary Figure 1. RELMa promotes DSS-induced colitis and Th17 cell responses. A. *Retnla* real-time PCR analysis of cDNA from naïve and day 4 DSS-treated colonic tissue, measured as fold induction over naïve. B. Immunofluorescent staining of PFA-fixed colonic tissue from naïve or day 7 DSS-treated mice reveals DSS-induced recruitment of RELMa+ cells to the lamina propria. RELMa, red; DAPI, blue. Bar, 50 µm. C. DSS-induced weight loss in WT or RELMa-/- mice. D. Pathology score of day 7 DSS-treated WT or RELMa-/- mice. E. PAS/Alcian blue staining of colonic tissue sections from day 7 DSS-treated WT or RELMa-/- mice. Bar, 50 µm. F-G. Mesenteric lymph node cells from day 7 DSStreated WT or RELMa-/- mice were stimulated with α CD3/ α CD28 for 3 days followed by ELISA of supernatants for IL-17A and IFN- γ (F) and flow cytometry analysis of CD4+ T cells (G). H. Real-time PCR analysis of cDNA from day 4 DSS-treated colonic tissue of WT and RELMa-/- mice. ***P*<0.01, **P*<0.05. Data are representative of 2 experiments with 4-5 mice per group.

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Supplementary Figure 2. Leukocyte frequencies and MHCII expression are equivalent in macrophages and dendritic cells in the colons of WT and RELMα^{-/-} **mice.** Single leukocyte preparations from colonic tissue day 10 *Citrobacter*-infected (A-C) or naïve (D-E) mice were analyzed by flow cytometry. A. Gating strategy for macrophages (mac, Ly6G⁻F4/80⁺CD11c⁻) and dendritic cells (DC, Ly6G⁻CD11c⁺F4/80⁻). B. Gating strategy for monocytes (mono, Ly6G⁻CD11c⁻F480⁻CD11b⁺) was validated by surface expression of Ly6C. C. Frequency of leukocyte populations in the colons of WT or -/- mice. D-E. MHCII expression in macrophages (D) and dendritic cells (E) was measured as mean fluorescent intensity (MFI). ns, not significant.



Supplementary Figure 3. RELM α promotes *Citrobacter* infection-induced intestinal inflammation through selectively promoting IL-17A expression. 1. *Citrobacter* infection induces RELM α expression by intestinal epithelial cells, macrophages and eosinophils. 2. RELM α promotes intestinal inflammation by stimulating IL-17A production through 3. activation of macrophages to produce IL-23.