

Fig. S1. Signaling through BMPR1 α alters T cell lineage commitment. (A) qRT-PCR analysis of *BMPR2* transcripts in CD4⁺ T cell subsets. Data are presented as means ± SD pooled from three independent experiments. **p < 0.01 and ***p < 0.001, as determined by Student's t-test. (B) PCR analysis of cytokine and transcription factor expression in sorted, naive CD4⁺ T cells stimulated for 4 days in Th cell polarizing conditions; either in the absence or presence of BMP2/4/7. Results representative of three independent stimulations.



Fig. S2. Cytokine production in WT and BMPR1 α -deficient cells activated in the presence of TGF- β or IL-6. (A and B) Flow cytometry analysis of IFN- γ and IL-17 production by purified WT and *BMPR1\alpha^{T-}* naive CD4⁺ T cells that were stimulated with TGF- β (5 ng/ml) (A) or IL-6 (20 ng/ml) (B) alone for 4 days. Contour plots are representative of three independent experiments. The frequency of cytokine producing cells are means ± SD from all experiments.



Fig. S3. Antigen specific BMPR1a-deficient CD4⁺ T cells generate increased proportion of proinflammatory cells. (A to C) Flow cytometry analysis of IFN- γ and IL-17 in purified CD4⁺ T cells from WT and *BMPR1a^{T-}* mice expressing a transgenic TCR and stimulated with antigenic peptide presented by splenocytes alone (A), in the presence of LPS (B) or in Th17 polarizing conditions (C), for 4 days. Data are representative of four independent experiments. The frequency of cells are means \pm SD pooled from all experiments. *p < 0.05, as determined by Student's t-test.



Fig. S4. Transcriptional profiles of naive versus activated CD4⁺ T cells. (A and B) Volcano plots of RNAseq analysis of sorted naive and activated WT (A) or BMPR1 α -deficient (B) CD4⁺ T cells. Plots show analysis of pooled RNAseq data for all replicates. (C) Fold change expression of signature genes that define Th1, Th2, Th17 and aT_{reg} cell subsets for naive WT and activated WT and *BMPR1\alpha^{T-}* CD4⁺ T cells. Closed circles represent average fold change gene expression difference between naive WT and activated WT and open circles represent average fold change differences between naive WT and activated *BMPR1\alpha^{T-}* CD4⁺ T cells. Bars represent standard deviations.



Fig. S5. Membrane proximal signaling in BMPR1 α -deficient and -sufficient CD4⁺ T cells. Western blot analysis of JNK and p38 phosphorylation in lysates of WT CD4⁺ T cells stimulated in medium alone or in the presence of DMH1 or dorsomorphin. Blots (left) are representative of three independent experiments. Normalized band intensity (right) are means \pm SD from all experiments.



Fig. S6. DSS-induced colitis is more severe in *BMPR1a^{T-}* **mice.** (**A** to **D**) WT and *BMPR1a^{T-}* mice were treated with DSS. Change in body weight (A), bleeding (B), stool consistency (C), and total clinical score (D) were monitored over 6 days. (E) On day 7 after DSS treatment, colon tissue was isolated from WT and *BMPR1a^{T-}* mice. Representative images of colon length (left) rounded to nearest 1.0 mm were quantified (right). (**F** and **G**) Histological analysis colonic cross-sections from control (F) and DSS treated (G) WT and *BMPR1a^{T-}* mice on day 7 after treatment. Scale bars = 100 µm. (**H**) qRT-PCR analysis of *IL-6*, *IL-1β*, *IL-17* and *IFN-γ* mRNA transcripts in CD4⁺ T cells isolated from the colons of WT and *BMPR1a^{T-}* mice on day 7. (**I**) Flow cytometry analysis of the expression of activation markers, Rorc and cytokines by CD4⁺ T cells isolated from the colons of DSS treated WT and *BMPR1a^{T-}* mice on day 7. All results are representative of 3 independent experiments. All quantified data are means ± SD of 6 mice per group pooled from all experiments. *P< 0.05, **P< 0.01, ***P< 0.001, ****P< 0.0001, as determined by Student's t-test.