Supplementary Figure 1. Deletion of IL-13Rα2, suppresses proinflammatory gene expression and protects *il10*$^{-/-}$ mice from piroxicam-induced colitis. Mice were fed piroxicam infused food for 14 days, with colitis assessed on day 14. Five animals per group were used with 1 of 2 experiments shown. Mean±SEM are shown with $p<0.05$ considered statistically significant. RNA was extracted from the colon of mice, 14 days post piroxicam exposure. Relative to HPRT, the following gene transcripts were quantified:

il17a
il17f
ifnγ
tnfα
cxcl9 (mig)
inos
mmp2

Supplementary Figure 2. IL-13Rα2 restricts IL-13-mediated gene expression in the caecum of *T. muris* infected *il10*$^{-/-}$ mice. Mice were infected with 200 *T. muris* eggs with gene expression in the caecum analyzed at day 15. Five animals per group were used with 1 of 3 experiments shown. Mean±SEM are shown with $p<0.05$ considered statistically significant. RNA was extracted from the caecum of mice, 15 days post infection.

il13
il10
ccl11 (eotaxin)
cxcl10 (IP-10)
retlnα (Fizz-1)

Supplementary Figure 3. In vitro polarized Th1 cells do not respond to IL-13. FACS-purified naïve $CD4^{+}CD62L^{hi}CD44^{lo}$ T cells were stimulated under Th1 conditions. One of 4 experiments is shown. Mean±SEM are shown with $p<0.05$ considered statistically significant.

A- Intracellular cytokine staining of IFNγ and IL-17A from Th1 polarized cells using

IL-13-responsive (IL-13Rα1$^{+/+}$) and unresponsive (IL-13Rα1$^{-/-}$) cells.

B- Th1-polarized cells cultured with rIL-13. IFNγ secretion measured by ELISA.