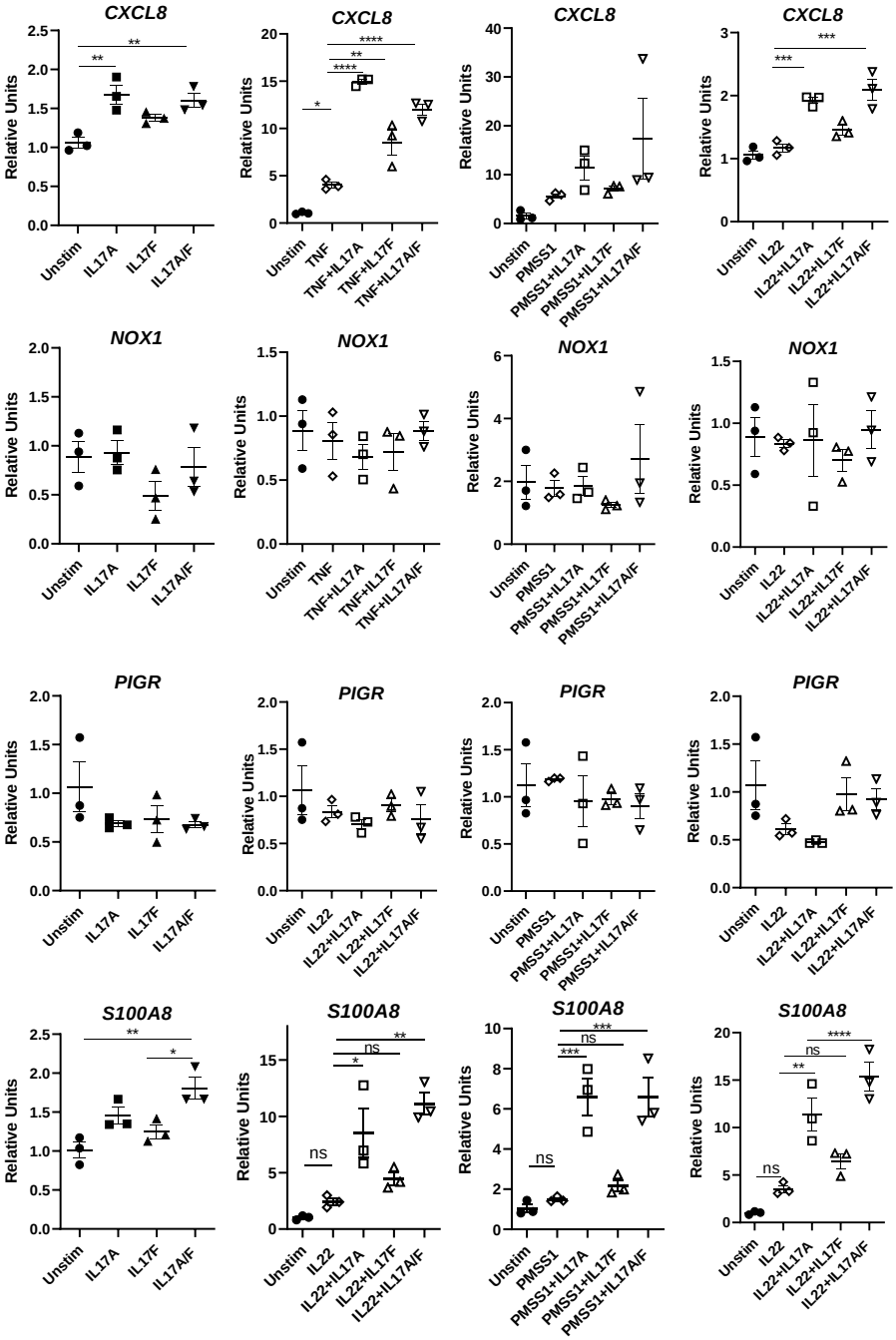
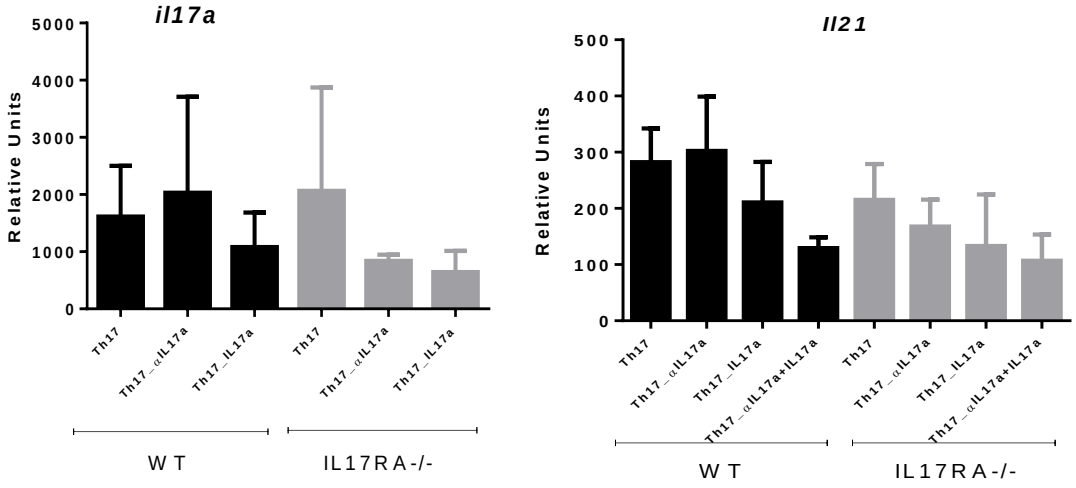


Supplemental Figure 1. MKN cell response to IL-17A, IL-17F or IL-17A/F. A. MKN cells were simulated with IL-17A, IL-17F or IL-17A/F (100ng/mL). B. Various stimuli (IL-17 cytokines and/or TNFα at 2ng/ml, IL-22 at 200ng/mL, or PMSS1 at an MOI of 50) were added to serum-starved MKN cells for 8 hours. qPCR was performed to measure expression of *CXCL8, S100A8, PIGR* and *NOX1* (Relative units and is relative to expression from cells treated with equal concentration of the carrier (BSA)). Data shown as ± SEM and are representative of 2 independent experiments. Statistical analysis was performed with one-way ANOVA and Tukey's test for multiple comparisons.



Supplemental Figure 2. In vitro Th17 differentiation assays do not suggest IL-17 impacts *il17a* and *Il21* expression. T cells were differentiated as described in the methods and then either anti-IL-17A (200 ng/ml) or rIL-17A (10 ng/ml) was added at 48 hrs. Gene expression was assessed by qPCR at 24 hours later.

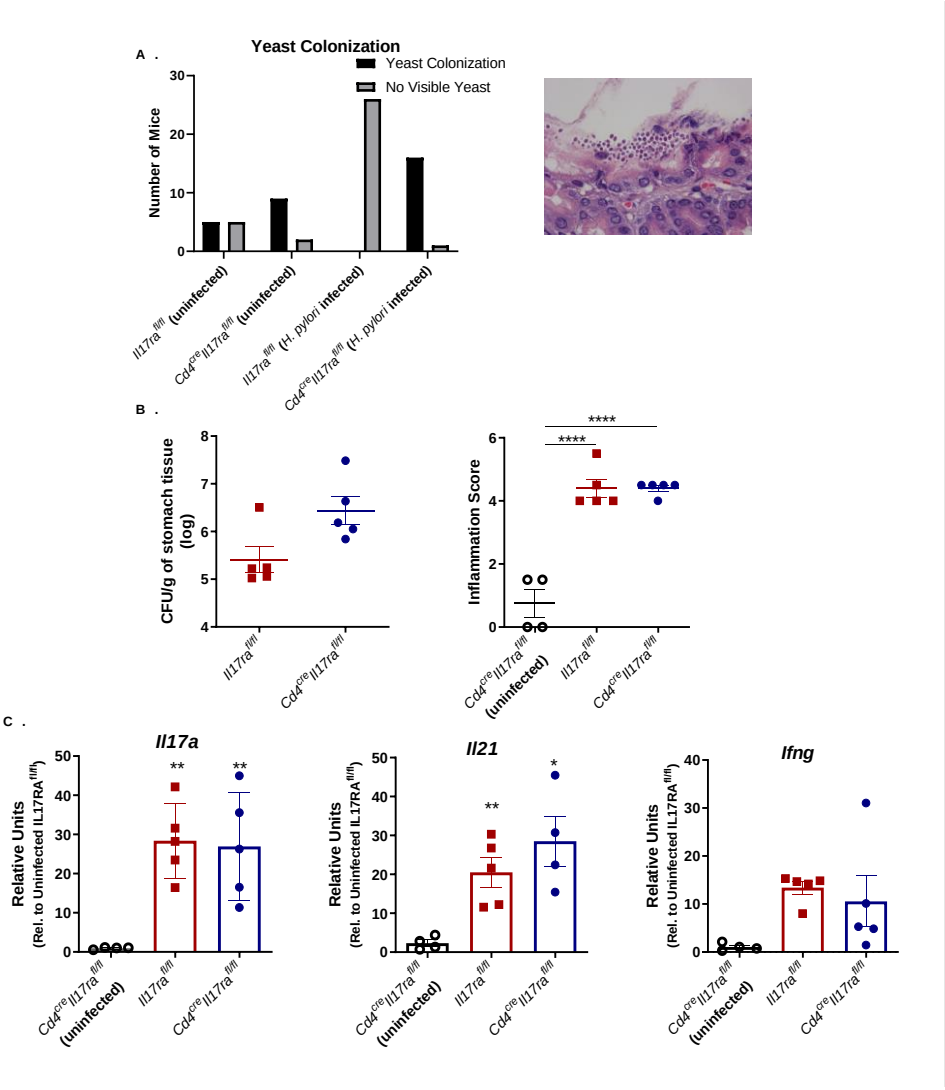
Supplemental Table I. Nanostring (Ms_Immunology Panel) was used to determine the Ratio and Log2Fold change of genes associated with innate lymphoid cells and myeloid cells in *Il17ra*$^{-/-}$ vs WT (C57BL/6J) gastric tissue at 3 months post infection with PMSS1 strain of *H. pylori*. Values highlighted in red were significantly different between genotypes.

ILC Markers (innate lymphoid cells)

ILC	GENE	% SAMPLES ABOVE THRESHOLD	RATIO	LOG2 FOLD CHANGE	P-VALUE
	klrb1 (encodes NK1.1)	0 %	-1.09	-0.12	0.73608887
	Kit (encodes CD117)	100 %	1.1	0.14	0.60356021
NK CELLS	*Fcgr3 (encodes Fc Receptor gamma 3)*	100 %	1.31	0.39	0.1695715
	Klrd1 (encodes CD94)	100 %	-1.08	-0.11	0.71743888
	Ncam1 (encodes CD56)	100 %	-1.01	-0.02	0.89048237

Myeloid Cell Markers

CELL TYPE	GENE	% SAMPLES ABOVE THRESHOLD	RATIO	LOG2 FOLD CHANGE	P-VALUE
NEUTROPHILS	*S100a8*	100 %	-14.02	-3.81	0.00169777
	S100a9	100 %	-5.5	-2.46	0.00158813
MACROPHAGES	*Cd14*	100 %	-1.33	-0.41	0.23644663
	CD163	100 %	1.03	0.05	0.787763
	Emr1	100 %	1.41	0.49	0.02996789
	Itgam (endodes CD11b)	100 %	1.27	0.35	0.04950276
DENDRITIC CELLS	*Itgax (endodes CD11c)*	100 %	1.38	0.47	0.17926033
GENERAL MARKERS	*Ccr2*	100 %	1.33	0.41	0.079329



Supplemental Figure 3. In the presence of yeast colonization, IL-17RA expression on T cells is required to control bacterial burden but not required to control gastric inflammation in the mouse model. A) Yeast infection was visible much more frequently in *Cd4*^{cre}*Il17ra*$^{fl/fl}$ than *Il17ra*$^{fl/fl}$. When yeast was visible in *Il17ra*$^{fl/fl}$ (uninfected mice, 5) these were littermates of the *Cd4*^{cre}*Il17ra*$^{fl/fl}$ mice. Yeast observed in the stomachs of a number of *Cd4*^{cre}*Il17ra*$^{fl/fl}$ mice are pictured (based on morphology and literature search, the yeast may be Kazachstania pintolopesii.B) Bacterial burden was measured in *Il17ra*$^{fl/fl}$ (WT) and *Cd4*^{cre}*Il17ra*$^{fl/fl}$ mice that were infected with PMSS1 for 3 months. CFU per gram of stomach was calculated and is presented ±SEM. Statistical analysis was performed using Mann Whitney U unpaired t test on log transformed CFU/gram data. No significant differences between the groups of infected mice (p=0.056). Total inflammation is presented. Statistical analysis was performed using Kruskal-Wallis test's and the Dunn's multiple comparisons test. Significantly more inflammation present compared to the uninfected controls (mean ± SEM). C) qPCR was used to measure *Il21*, *Il17a* and *Ifng* transcripts in the gastric tissue of *H. pylori*–infected *Il17ra*$^{fl/fl}$ (WT) and *Cd4*^{cre}*Il17ra*$^{fl/fl}$ mice. Relative units are calculated relative to *Gapdh* and calibrated to uninfected WT mice. Statistical analysis was performed using an ANOVA analysis and Tukey's multiple comparisons test. Error bars represent ± SEM; *$P \leq 0.05$, **, $P \leq 0.01$ compared to the uninfected group.