Figure S1. Gene set enrichment rug panel plot. Enrichment (red) or depletion (blue) of indicated Gene Ontology gene sets was determined by CERNO testing in the *tmod* package and a ranked list of genes determined by differential gene expression analysis. Bar width represents effect size (area under the curve for CERNO testing). Proportion of genes upregulated or downregulated in first group vs. second group represented by red or blue shading, respectively. Gray indicates proportion of genes for which direction of regulation could not be determined based on pre-defined thresholds (absolute $\log_2$ fold change <0.5, false discovery rate >5%). Only gene sets with a false discovery rate less than $1x10^{-6}$ are shown. Pathways highlighted in red were referenced in main text.

Figure S2. IPA upstream regulator analysis for each two-way comparison at 24 and 48 hours post infection. Shown is a heatmap of IPA upstream regulator z-scores indicating predicted activation (red shading), predicted inhibition (blue shading), or no predicted regulation (white) of indicated genes in first group relative to second group of each column label. Only upstream regulators with a P value <0.01 are shown. Absolute z-score values less than 2 were set to 0. Rows are ordered by absolute z-scores starting with the first column, followed by the second column and sort forth for easier comparative visualization. Genes highlighted in red were referenced in main text.

Figure S3. *C. burnetii* is sensitive to IL-17 treatment during early stages of infection

Macrophages were infected with WT *C. burnetii* and treated for 24 hours with the indicated concentrations of IL-17A at either 24 hpi or 48 hpi. The number of viable bacteria was determined using fluorescent foci forming unit (FFU) assay and normalized to untreated control. IL-17A leads to a significant decrease in bacterial viability in a dose-dependent manner, with *C. burnetii* being more sensitive at 24 hpi. Shown is the average +/- SEM from three independent experiments done

in duplicate. *p<0.05 and **p<0.01 as determined by one-way ANOVA with Dunnett's posthoc test, compared to untreated.

Figure S4. MH-S cells were infected with WT (black bars) or *dotA* (white bars) for 24 hours, followed by treatment for 24 hours with either **(A-B)** IL-17 alone or **(C-D)** IL-17 and an IL-17A receptor blocking antibody (2 µg/ml). hMDMs **(E-F)** were infected for 24 hours and treated with human-IL-17A for 24 hours. The number of recoverable bacteria was determined using colony forming unit (CFU) assay, and shown here as raw CFU numbers compared to normalized numbers shown in Figure 5. Error bars indicate the mean +/- SEM from four individual experiments. *p<0.05, **p<0.01 and ***p<0,005 as determined by one-way ANOVA with Dunnett's posthoc test compared to untreated controls.

Supplementary Data Set 1. Differentially expressed genes for each comparison for each time point using thresholds of $|\log_2$ fold-change$|>0.585$ (1.5 in linear space) and false discovery <5%. Details of the analysis are found in the Methods.

Supplementary Table 1. Self-contained gene set testing for differential expression of an IL-17 pathway gene set (*Ccl5*, *Il17rc*, *Lcn2*, *Traf6*, *Il17ra*, *Nfkb1*, *Nfkb2*, *Ccl2*, and *Ccl3*). P value and false discovery rate (FDR) represent two-sided directional significance, whereas P value Mixed and FDR Mixed represent non-directional significance. Details of the analysis are found in the Methods.