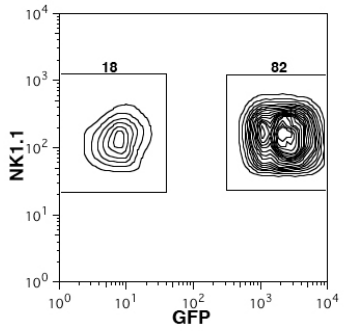
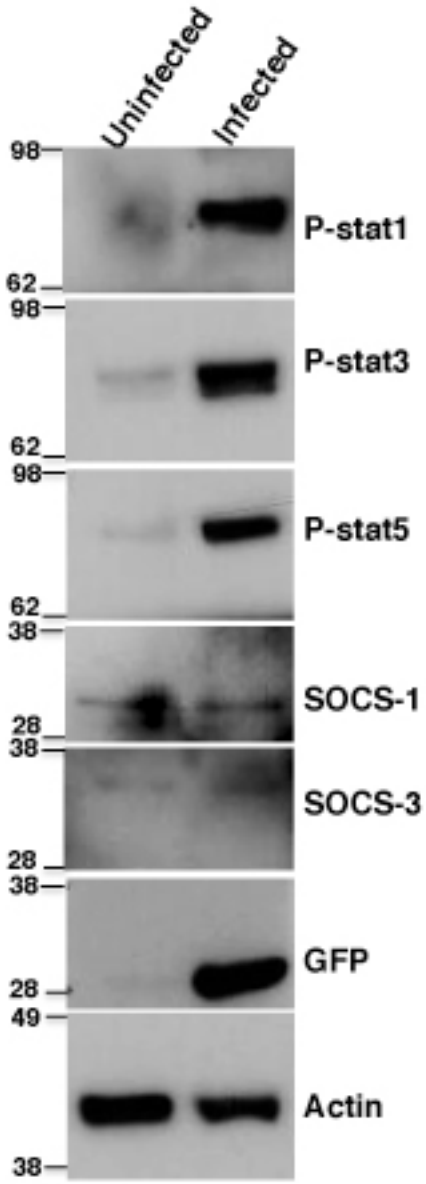


Fig S1 Percentage of infected NK cells used in the conjugation assay shown in FIG 2.

Fig S2 Induction of STAT phosphorylation upon *T. gondii* infection of NK cells.



Increased phosphorylation of STATs was observed in NK cells infected with T. gondii. However there did not appear to be large differences in the expression of SOCS1 or SOCS3 upon T. gondii infection.

NK cells incubated with Toxoplasma for 2 hours and then stimulated with IL-12 and IL-18. After one hour, cells were collected and sorted by gating on live GFP^+ or GFP^- $NKp46^+$ cells After collection(1), cells were lysed with Christea Buffer (20 mM HEPES, pH 7.4, 110 mM potassium acetate, 2 mM magnesium chloride, 0.1% (vol/vol) Tween 20, 1% (vol/vol) Triton X-100, 0.5% (wt/vol) sodium deoxycholate, and 500 mM sodium chloride (1) supplemented with xomplete protease inhibitor (ThermoFisher) and PhosSTOP phosphatase inhibitor cocktails (ThermoFisher) on ice. Then the lysates were mixed with 4×reducing NuPAGE lithium dodecyl sulfate (LDS) sample buffer (Life Technologies), followed by SDS-PAGE and Western blotting analysis for the indicated proteins. Membranes were blocked in TBST with 5% (w/v) nonfat dry milk. Antibodies were applied as: P-stat1 (Cell Signaling 9171, 1:500), P-stat3, (Cell Signalling 9145, 1:500), P-stat5 (Cell Signaling 4322, 1:500), SOCS-1 (Santa Cruz sc-9021, 1:500), SOCS-3 (Cell signalling 2932, 1:500), actin (Santa Cruz sc-1616, 1.500) GFP (Abcam ab290, 1:3000).

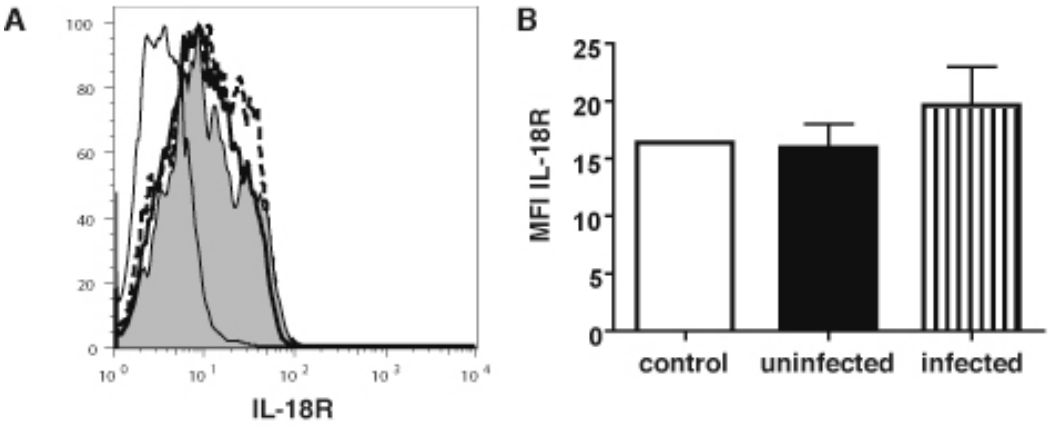


Fig S3 Expression of IL-18R on NK cells is not affected by *T. gondii* infection.
(A) Expression of IL-18R on control NK cells (*dashed line*), bystander NK cells (*solid line*) and infected NK cells (s*haded*). (B) Expression levels from 2 experiments.

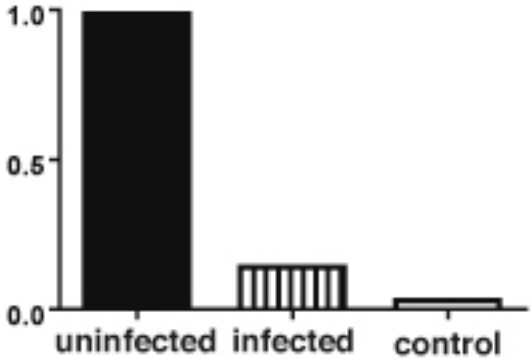


Fig S4 TNF is inhibited in *T. gondii infected* NK cells.

qPCR of TNF RNA, comparing the relative expression of TNF between uninfected NK cells (*filled columns*) with infected NK cells (*lined columns*) from the same culture stimulated with IL-12 and IL-18, and NK cells that were not stimulated with IL-12 and IL-18 (control). NK cells infected with RH-LDM *T. gondii* were activated with IL-12 and IL-18 as outlined above and then sorted by flow cytometry based on the expression of GFP. RNA was isolated from TRIReagent (Sigma-Aldrich)-treated samples according to the manufacturer's instructions. Total RNA was reverse transcribed (RevertAid First Strand cDNA Synthesis kit, Thermofisher) and levels of TNF were quantified by real-time PCR (SYBR Green Jumpstart Taq Ready Mix, Sigma-Aldrich) using the following sequences: forward GGC TGC CCC GAC TAC GT

reverse GAC TTT CTC CTG GTA TGA GAT AGC AAA. The relative amount of transcripts was calculated with the 2-(ΔΔCt) method in relation to HPRT or U6 (for miRNA) and calibrated to uninfected cells. Specificity of obtained products was confirmed with dissociation curves.

Reference

1. **Cristea IM, Carroll JW, Rout MP, Rice CM, Chait BT, MacDonald MR.** 2006. Tracking and elucidating alphavirus-host protein interactions. J Biol Chem **281:**30269-30278.