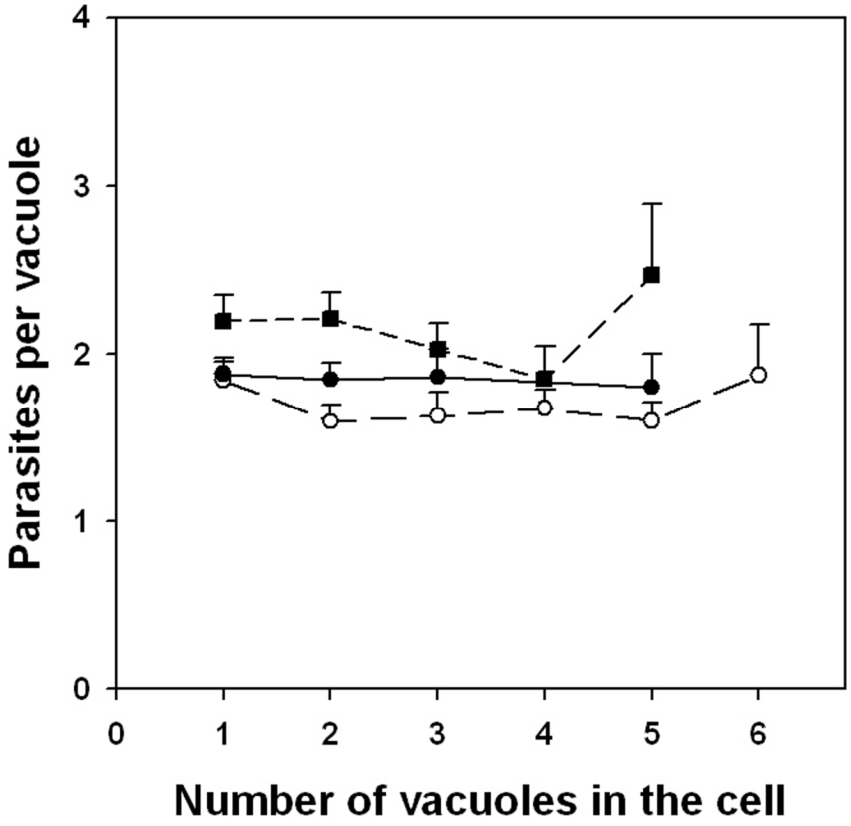

Figure S1. Vacuolar parasite content is independent of the number of vacuoles per cell. The datasets employed for Fig. 1B were examined to determine the number of parasites per vacuole as a function of the number of vacuoles in each inflammatory macrophage. Each curve represents one mouse, corresponding to one of the three mice used for the data displayed in Fig. 1B.

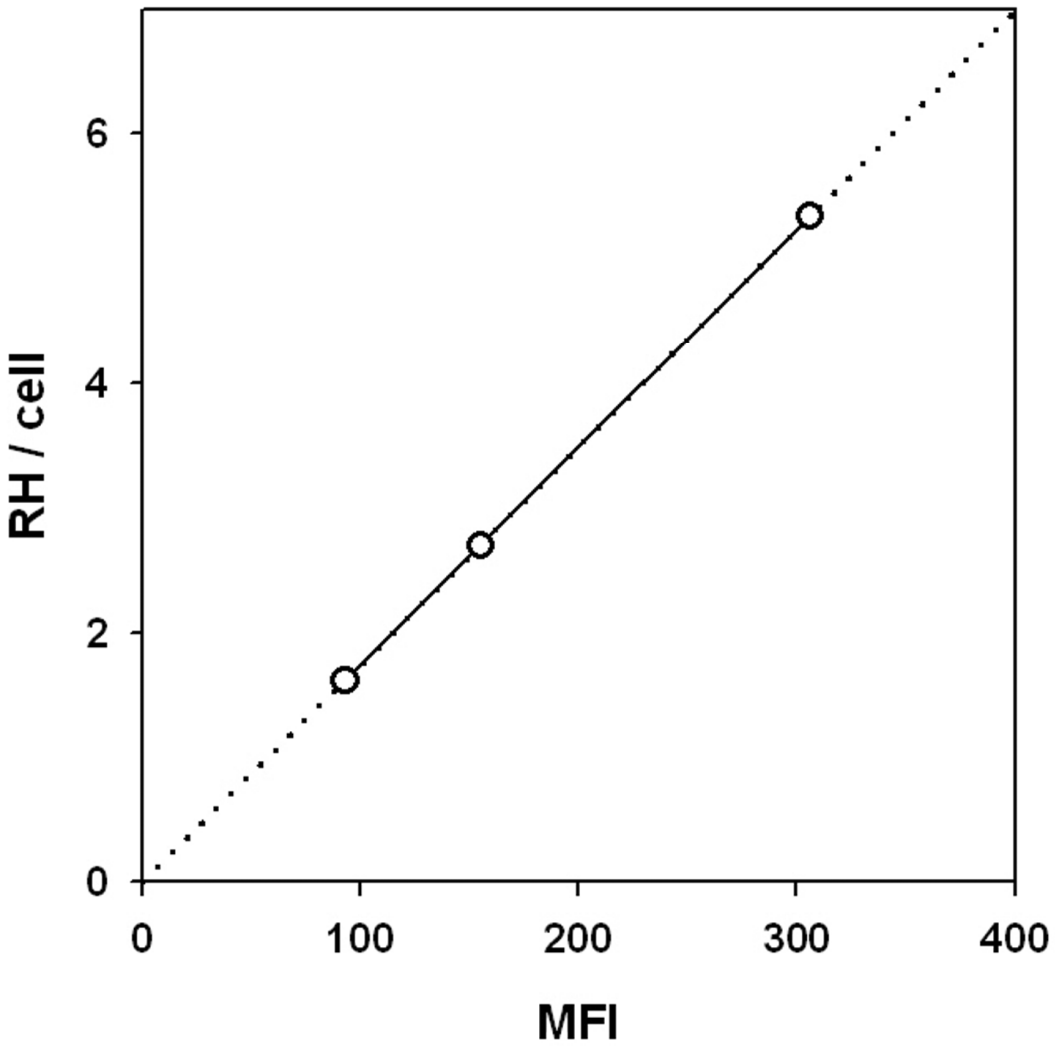

Figure S2. YFP-RH fluorescence measures intracellular parasite content. Exudate cells were collected by lavage from three mice infected 4d previously with YFP-RH. Cells were pooled, fixed with and equal volume of 4% paraformaldehyde, and three populations of different mean fluorescence (MFI) were obtained by sorting on a MoFlo cell sorter. The sorted populations were analyzed for parasite content / cell by fluorescence microscopy of cytospin preparations (total of 597 vacuoles counted). The plot displays the correlation between MFI and parasite content for the three populations ($r^2 = 1.00$). Dotted line = linear regression of the data.

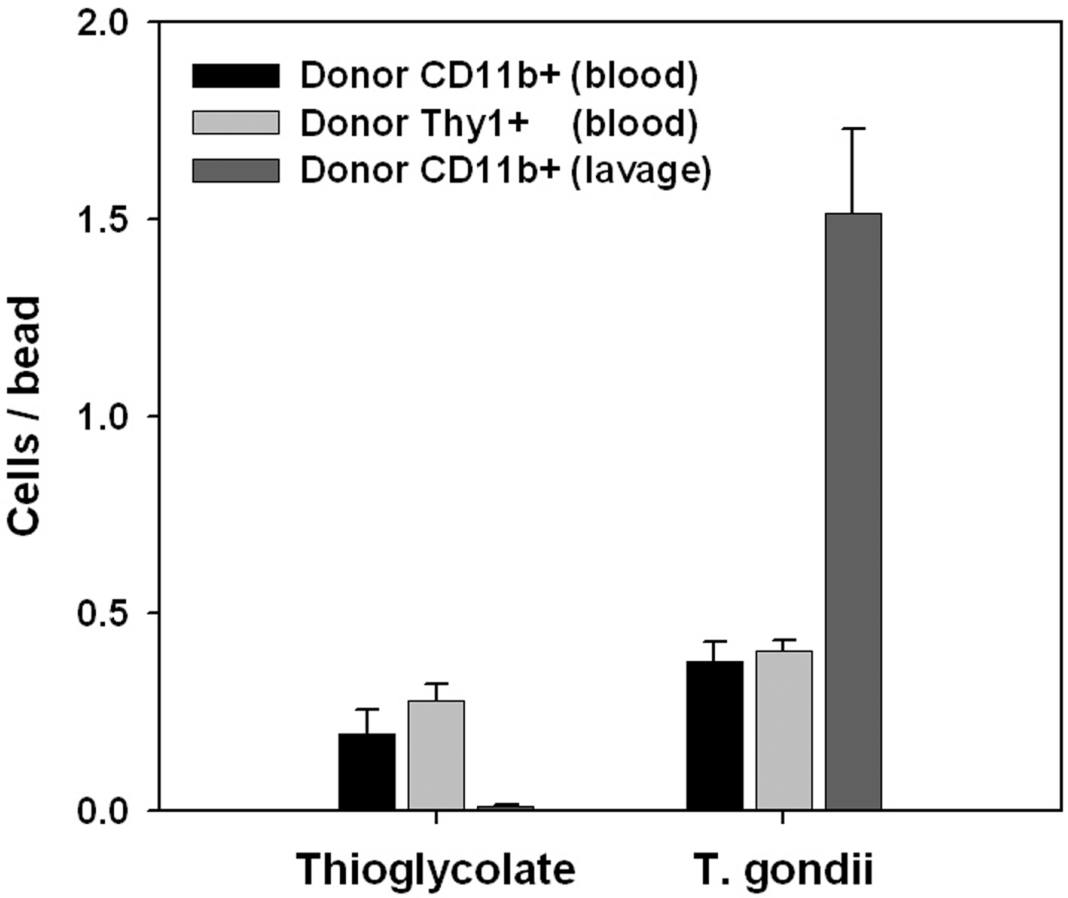

Figure S3. Enhanced recruitment of myeloid cells to peritonitis induced by *T. gondii* compared to thioglycolate. Groups of 5 recipient mice (C57BL/6; CD45.1) were either infected with wild-type RH or else injected with thioglycolate. After 5d, pooled donor blood was obtained from 3 SJL mice (CD45.2) by cardiac puncture into EDTA-saline and transfused into recipient mice by tail vein injection (0.2 ml). After 3h, blood and peritoneal lavage samples were obtained and analyzed by flow cytometry after hypotonic lysis of red blood cells, followed by incubation for 1 h with Fc block, FITC-anti-CD45.1, PE-anti-CD454.2 and either 647-anti-Thy1 to detect T cells or 647-anti-CD11b to detect myeloid cells. F4/80 was not used since it is not sufficiently expressed on monocytes (1)(data not shown). Samples were fixed with an equal volume of 4% paraformaldehyde and fluorescent beads were added to normalize the analyzed volume. No Thy1+ donors were detected in lavage. The results show that while chimerism (as indicated by donor Thy1+ levels) was comparable in the two groups, the distribution of donor myeloid cells was strongly shifted from blood to the peritoneal cavity in infected, compared to thioglycolate-treated hosts.

1. Gordon, S., Lawson, L., Rabinowitz, S., Crocker, P.R., Morris, L. and V.H. Perry. 1992. Antigen markers of macrophage differentiation in murine tissues. *Curr. Top. Microbiol. Immunol.* 181: 1-37.

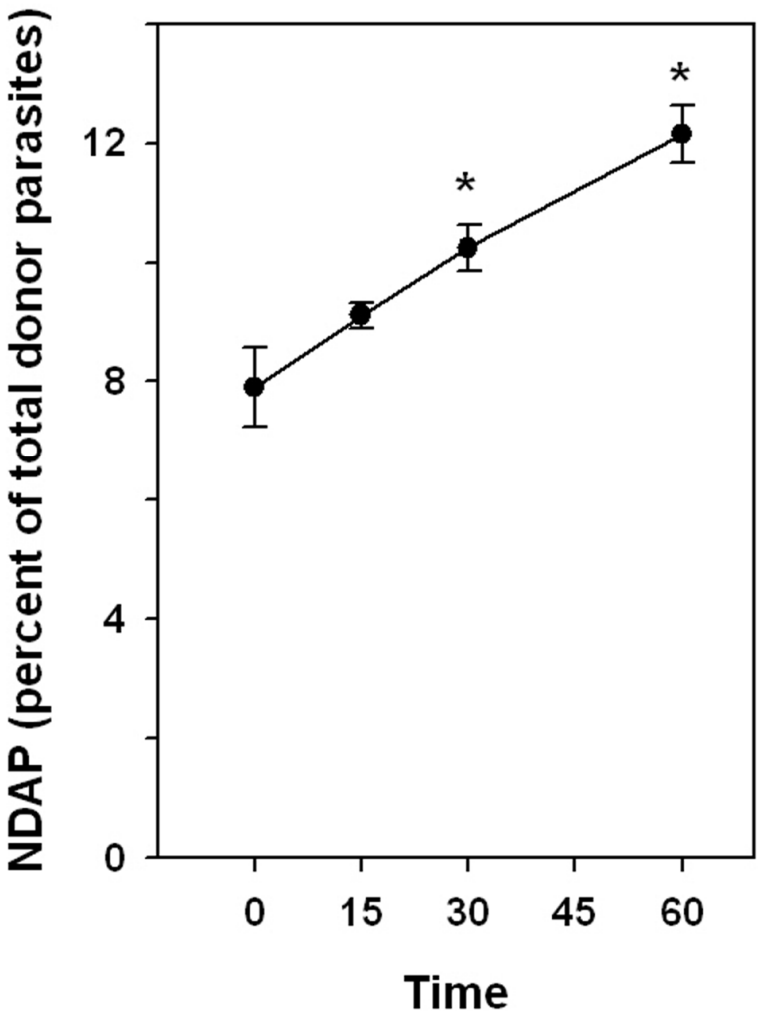

Figure S4. Time-dependence of ETE. ETE assays were performed as described in the legend to Fig. 7E, except that the time of co-culture was varied as indicated. Data represent the mean ± S.E. of four replicates. *, $p < 0.05$ compared to time 0.

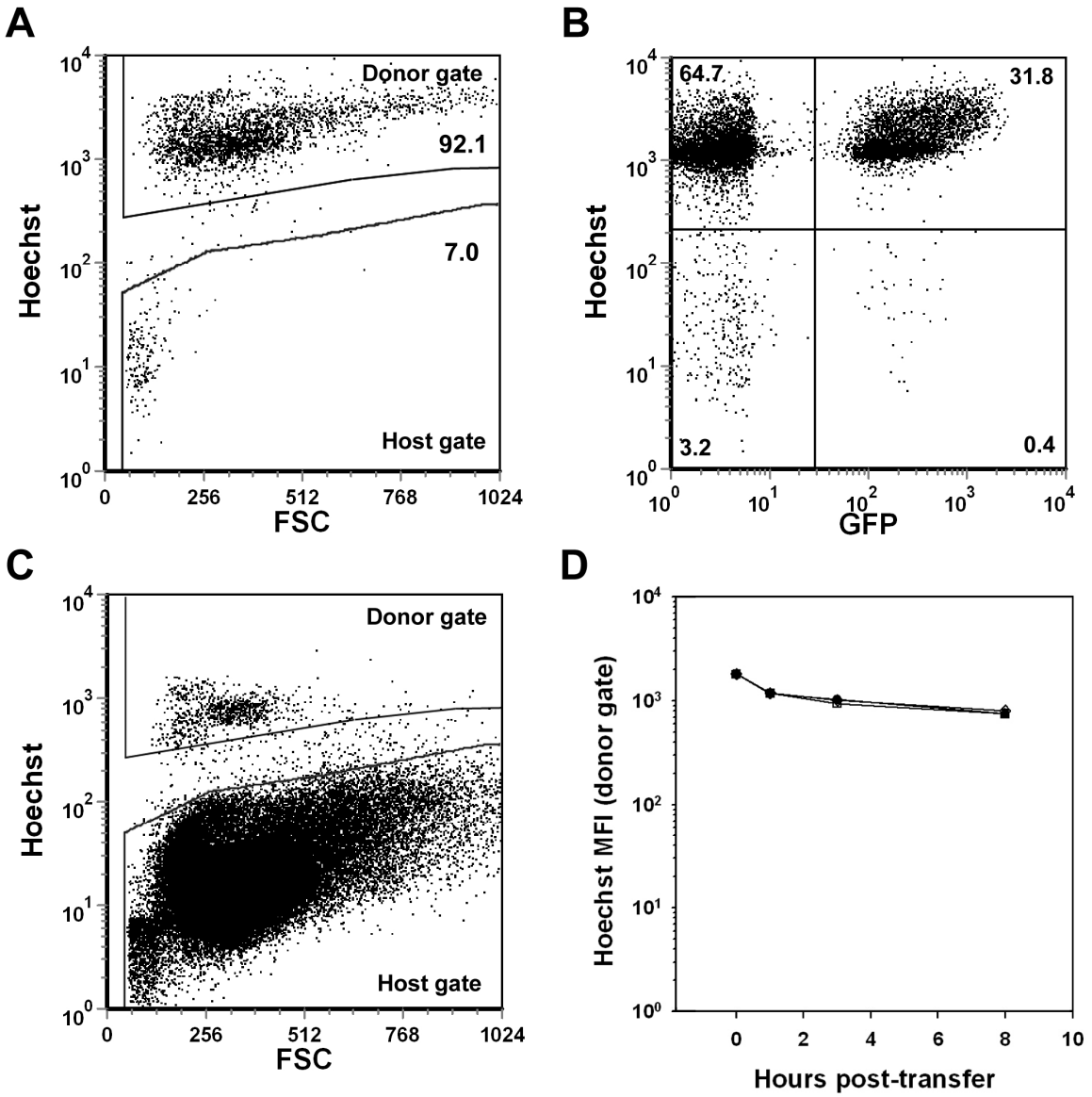

Figure S5. Uniformity and stability of Hoechst 33342 labeling of donor cells. Data are derived from the experiment displayed in Fig. 2. **(A)** Dot plot (gated on total viable cells, excluding free parasites) displaying the Hoechst 33342 labeling in untransferred donor cells from a GFP-RH-infected donor mouse. The donor and host gates used for egress analysis are shown, as well as the percent of donor cells in each gate. **(B)** Uniformity of labeling in infected donor cells. The sample displayed in panel A was analyzed for Hoechst staining as a function of GFP-RH content. Infected donor cells (GFP+) are 99 percent labeled. **(C, D)** Stability of Hoechst 33342 labeling. Panel C (gated as for panel A) illustrates the retention of label in donor cells in a sample removed at 8 h post-transfer from a host mouse injected with the donor cells displayed in panel A. Panel D displays the mean Hoechst 33342 intensity (MFI) of cells in the donor gate at 0 - 8 h post-transfer. Three curves are displayed, each corresponding to a host animal injected with the donor cells displayed in panel A.