Supplementary materials

Fig. 1 Attenuated diseases in *L. braziliensis- or L. major*-infected IFNAR-/- mice. WT and IFNAR-/- mice (5 per group) were s.c. injected with $2 \times 10^6 L$. *braziliensis* or *L. major* promastigotes in the hind foot. Lesion sizes were shown weekly for *L. braziliensis* (A) and for *L. major* (B). ** (p < 0.01) indicates statistically significant difference between the two groups.

Fig. 2 The expression of inflammatory cytokine and chemokine genes in *L. amazonensis*-infected tissues. WT and IFNAR^{-/-} mice were s.c. injected with 1×10^6 *La* promastigotes in the inner side of the ear. The infected area from each ear were excised at 1 and 3 d post-infection and immediately frozen in liquid nitrogen. The expression of inflammatory cytokine and chemokine genes was analyzed by Oligo Microarray (OMM-011, SABiosciences) and normalized with non-infected controls. Genes with more than 2-fold increase are listed here.

Fig. 3 The detection of three different subsets of peritoneal cells following injection with *L. amazonensis* parasites. WT mice were i.p. injected with $2 \times 10^7 La$ promastigotes. Peritoneal cells were collected at 24 h post-infection and stained for Gr-1 and CD11b expression. Three different cell subsets were identified by FACS. Some cells were sorted, cytospun onto slides, and stained by a Diff-Quik assay.

Fig. 4 Neutrophil and monocyte recruitment following injection with *L. major* **parasites.** WT and IFNAR^{-/-} mice (2 per group) were i.p. injected with $2 \times 10^7 L$. *major* promastigotes. At 24 h post-infection, peritoneal cells were collected, stained with Ly-6G, Ly-6C and CD11b. The percentages of CD11b⁺Ly-6G⁺ neutrophils and CD11b⁺Ly-6C⁺ inflammatory monocytes from WT and IFNAR^{-/-} mice were assayed by FACS. Shown are the representative results from two independent repeats.

Fig. 5 The analyses of blood neutrophils and monocytes and lymph node monocytes during infection. WT and IFNAR^{-/-} mice were s.c. injected with 1×10^6 *L. amazonensis* promastigotes in the inner side of the ear. (A) At 2 d post-infection, the percentages of blood neutrophils and monocytes were measured by FACS based on the expression of CD11b, Ly-6C and Ly-6G. The pooled data (3~4 mice per group) from two repeats were shown as mean ± SD. (B) At 7 d post-infection, the percentages of CD11b^{high}Ly-6C⁺ monocytes and CD11b^{low}Ly-6C⁺ dendritic cells gated on CD11c⁺ lymph node (LN) cells were measured by FACS. Shown are the representative results from two independent repeats.

Fig. 6 Similar patterns of cytokine production from WT and IFNAR^{-/-} **neutrophils.** WT and IFNAR^{-/-} mice were i.p. injected with $2 \times 10^7 L$. *amazonensis* promastigotes. Peritoneal cells were collected at 6 h post-infection and restimulated with GolgiPlug for 4 h. The levels of intracellular cytokine (IFN- γ , IL-10, TNF- α , and IL-12p40) in Ly-6G⁺ neutrophils were analyzed by FACS. Shown are the representative results from three independent repeats.

Fig. 7 Neutrophil apoptosis in *L. major* **infection.** TG-elicited peritoneal neutrophils were purified from WT and IFNAR^{-/-} mice and infected with *L. major* promastigotes in the absence or presence of IFN- α (200 U/ml) at the indicated cell-to-parasite ratios at 37°C for 18 h. Neutrophil apoptosis in Gr-1-gated cells were assayed by staining with Annexin V and 7-AAD, respectively. The numbers indicate the percentages of positively stained cells. Shown are the representative results from two independent repeats. Similar trends were observed for infection performed at 33°C (data not shown).

