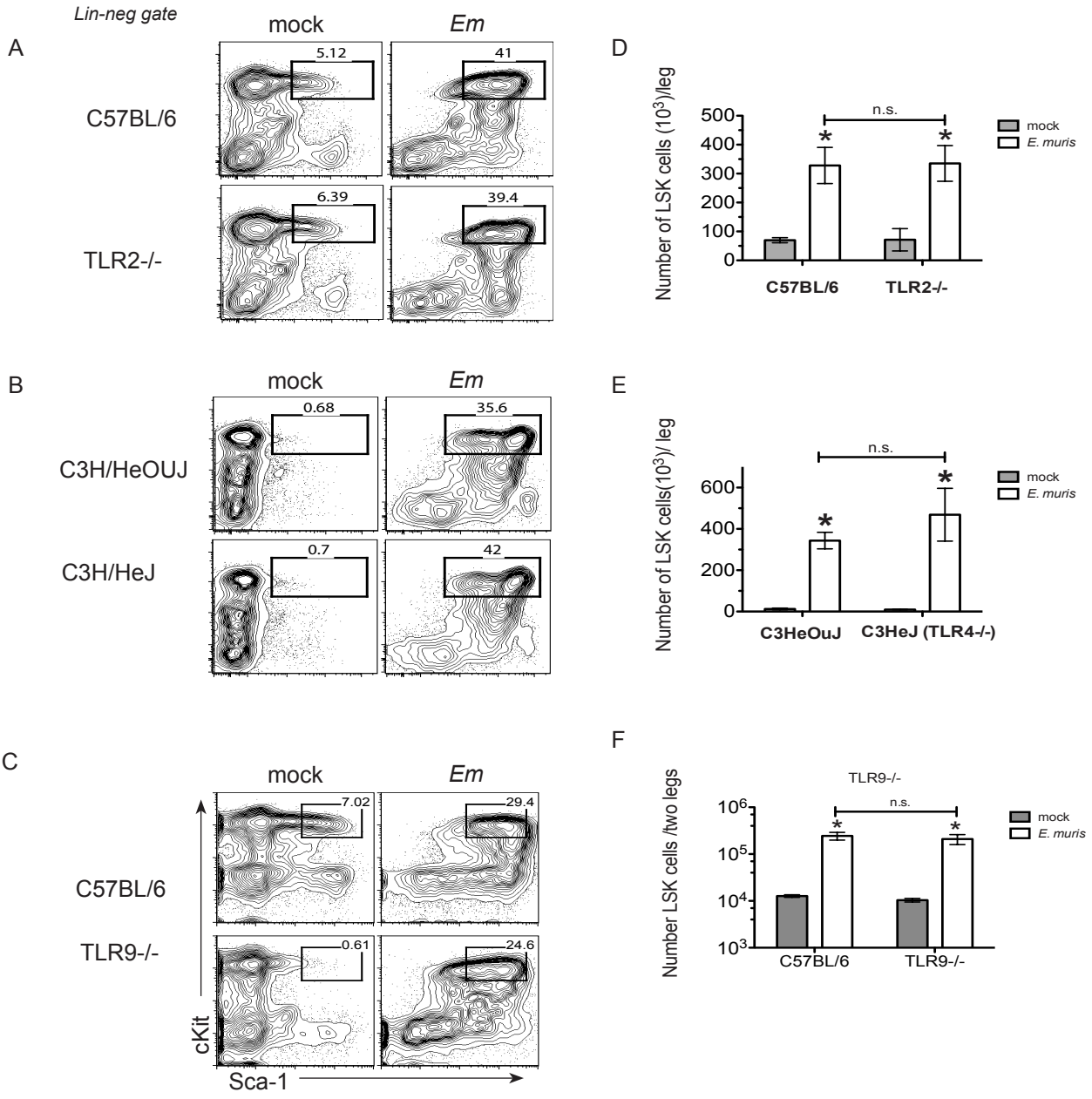
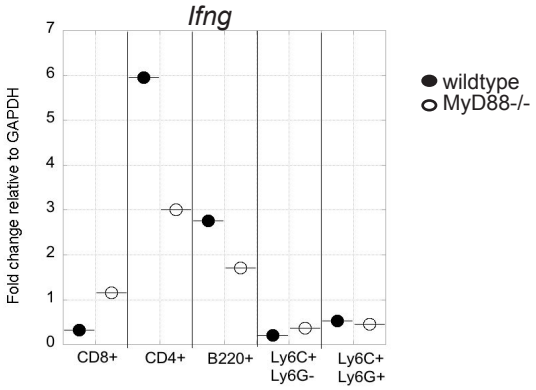
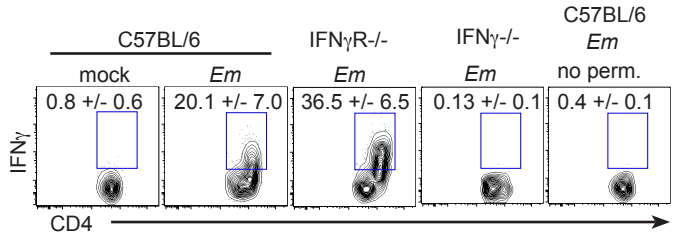


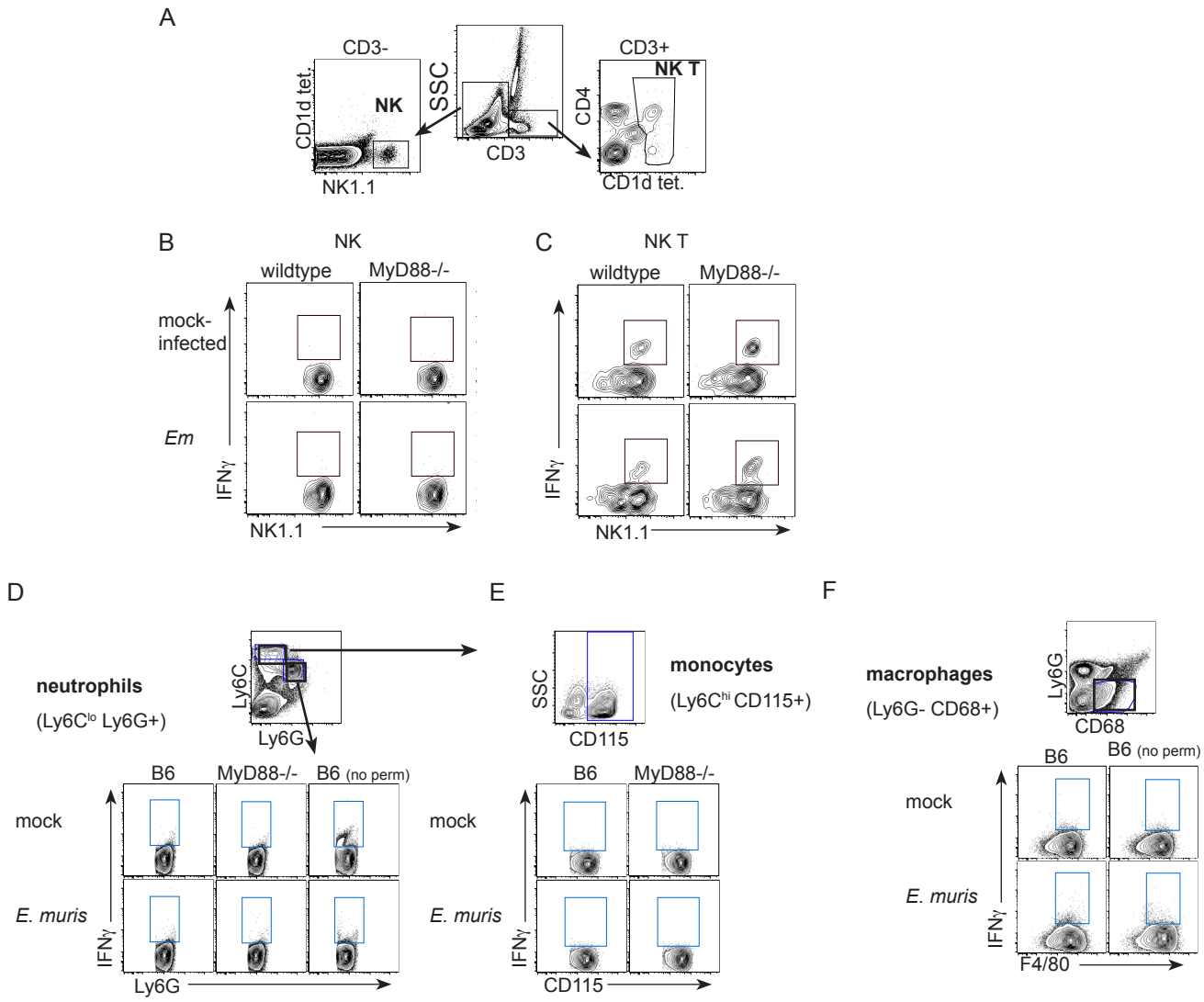
Supplemental Figure 1. LSK expansion occurs in the absence of TLR2, 4, and 9.
Wildtype and *Tlr*-deficient mice were inoculated with *E. muris*. Bone marrow cells were purified and examined for surface expression of lineage markers, c-Kit, and Sca-1. Lineage-negative cells were analyzed for expression of c-Kit+, Sca-1+. (**A-C**)Square gates are drawn on Lin-negative cells expressing both c-Kit and Sca-1 (LSK) in mock-infected (left column) and *E. muris*-infected (right column) mice on day 11 post-infection in Tlr-deficient or control mice, as indicated. Numbers above the LSK gate represent the frequency of c-Kit+ Sca-1+ cells among total Lin-negative cells. (**D-F**) Numbers of LSK cells the bone marrow is shown for mock- (gray bars) and *E.muris*-infected (open bars) mice. The data represent the mean and standard error of the data. At least four mice are examined for each group. The asterisks reflect significant differences between mock and infected groups and # reflect differences between different strains of mice; * or # $p < 0.05$.



Supplemental Figure 2. *Ifng* expression in MyD88-deficient and wildtype bone marrow-derived cell populations. Cell poulations were purified by flow cyotmetric sorting from the bone marrow of mock-infected and *E. muris*-infected wildtype and MyD88-deficient mice. RNA was extracted and real time PCR was performed to determine the relative gene expression of *Ifng*. Data represents the fold change between mock and infected samples, relative to *Gapdh* expression. Closed circles represent cells from wildtype mice and open circles from MyD88-deficient mice.



Supplemental Figure 3. **Detection of *in vivo* production of IFNγ**. Bone marrow of wild type, IFNγ-deficient and IFNγR-deficient mice was analyzed for populations producing IFNγ in response to mock or *E. muris* infection (day 11 post-infection). Mice that lacked the *Ifngr* gene exhibited increased IFNγ production, relative to infected wildtype mice. Cells that were not permabealized exhibited little IFNγ staining.



Supplemental Figure 4. **NK, NK T, and myeloid cells are not major producers of IFNγ during ehrlichial infection.** Bone marrow was isolated from mock and *E. muris* infected mice and surface stained for NK, NK T, and myeloid populations. ICCS was performed directly ex vivo. Bone marrow cells were harvested from mock and *E. muris*-infected C57BL/6 and MyD88-deficient mice on day 11 post-infection. (**A**) Schematic for gating NK and NK T cells is shown. NK cells were identified as CD3- NK1.1+ cells and NK T cells were identified by expression of CD3+ CD1d tetramer+. Representative flow cytometric plots of IFN expression among NK1.1+ NK cells (**B**) and NK T cells (**C**). Numbers above the gated region represent the average frequency and standard deviation of IFNγ+ cells among the population. (**D**) Ly6C^{lo} Ly6G+ cells were identified as neutrophils and analzyed for intracellular IFNγ using flow cytometry. Control samples were not permeabilizaed to detect surface-bound IFNγ. (**E**) Ly6C+ cells were further analyzed for expression of CD115 and intracellular IFNγ to identify IFNγ-producing monocytes. (**F**) Ly6G- CD68+ cells were further analyzed for F4/80 and IFNγ to identify IFNγ producing macrophages. At least four independent mice were analyzed for mock and infected groups.