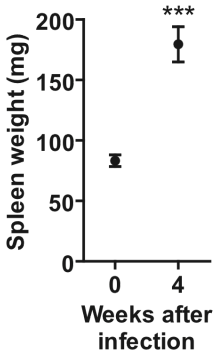
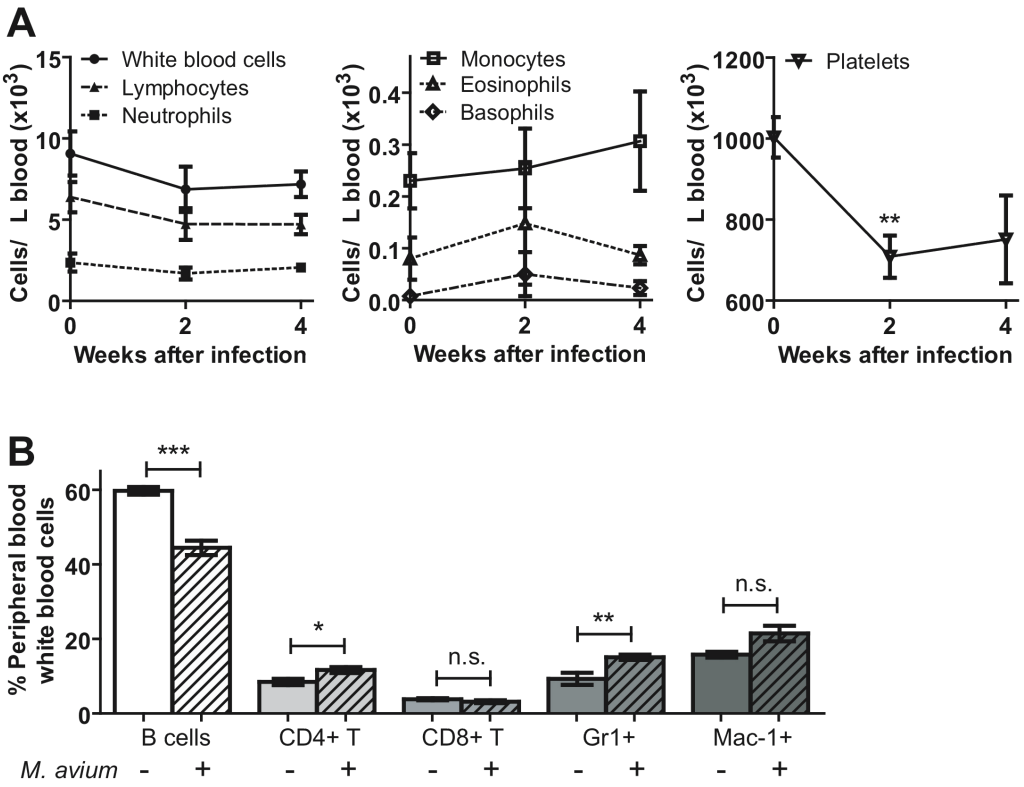
SUPPLEMENTARY INFORMATION



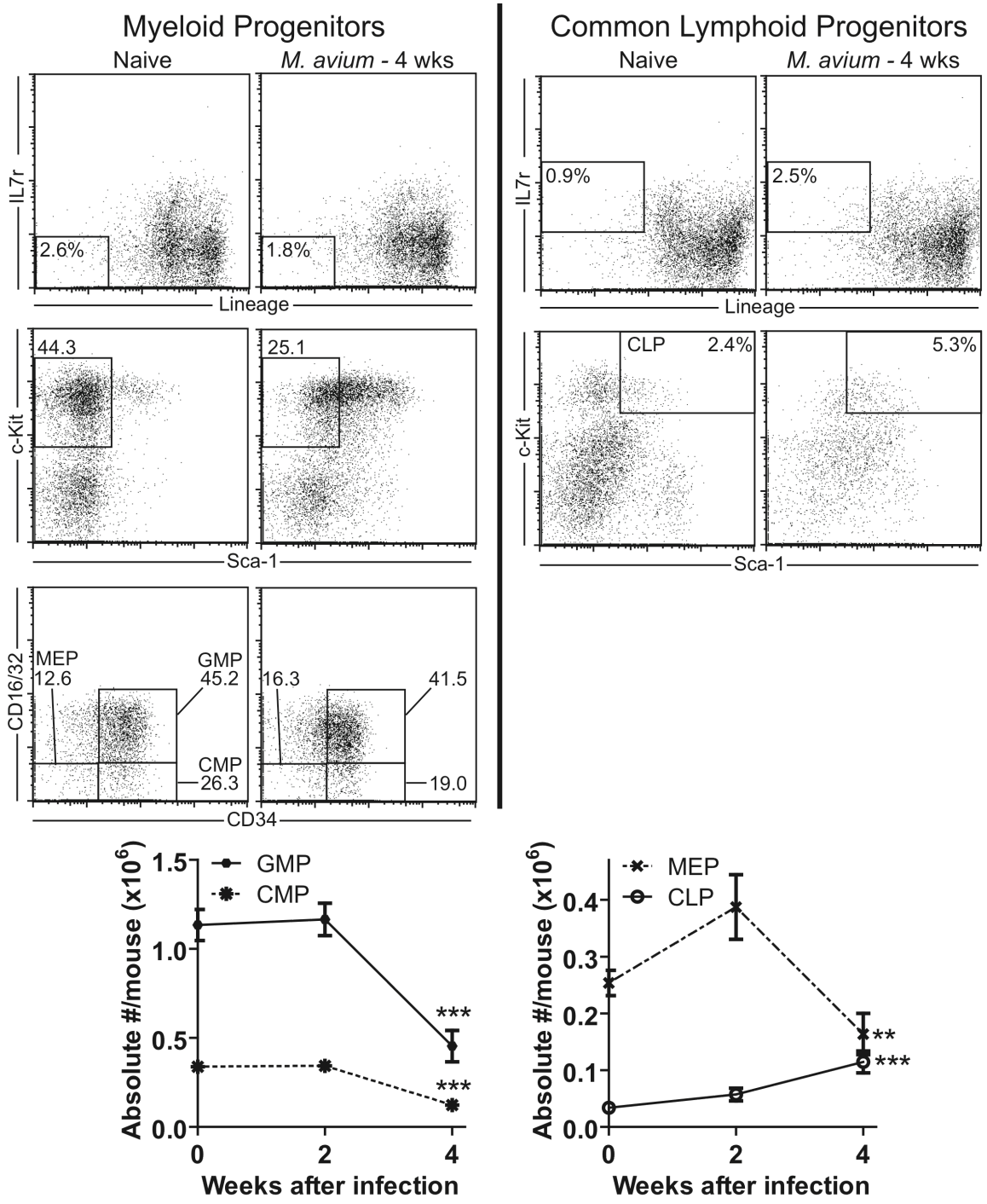
Supplementary Figure 1. Spleen weights increase with infection.

Spleen weight was determined 4 weeks after *M. avium* infection of mice. n=4 to 6.



Supplementary Figure 2. Peripheral blood homeostasis is maintained during *M. avium* infection.

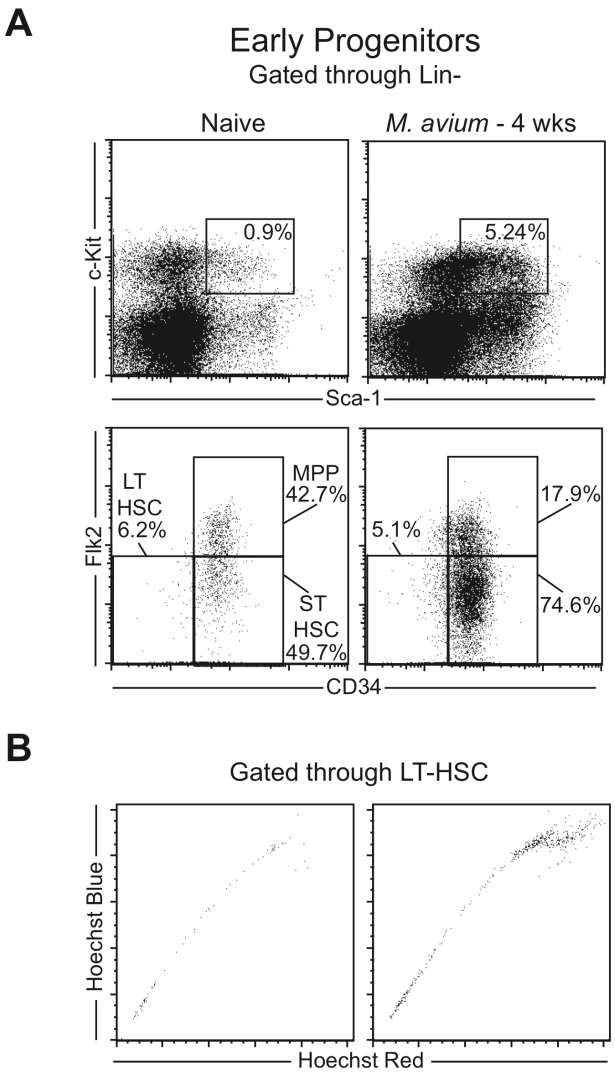
(A) Peripheral blood composition, with the exception of platelets, remains stable over 4 weeks of *M. avium* infection. n=3-8. (B) The relative percentage of B cells declines 4 weeks postinfection whereas percentages of CD4+ T-cells and granulocytes increase in mice infected with *M. avium*. n=4 or 5.



Supplementary Figure 3. Infection stimulates changes in myeloid and lymphoid progenitor compartments.

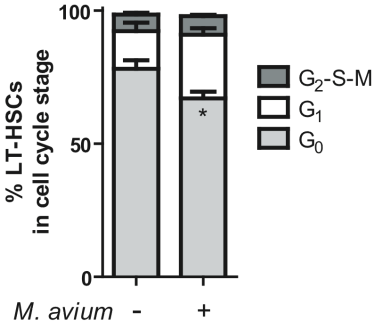
Whole bone marrow was isolated from naïve and M. avium-infected mice 4 weeks postinfection. Progenitor populations shown were previously gated as live cells. For

myeloid progenitor gating, GMPs (granulocyte-macrophage progenitors) are Il7rα^-, Lin$^-$, c-kit$^+$, Sca-1$^-$, CD16/32$^+$, and CD34$^+$, CMPs (common myeloid progenitors) are Il7rα^-, Lin$^-$, c-kit$^+$, Sca-1$^-$, CD16/32$^-$, and CD34$^+$, and MEPs (macrophage-erythroid progenitors) are Il7rα^-, Lin$^-$, c-kit$^+$, Sca-1$^-$, CD16/32$^-$, and CD34$^-$. CLPs (common lymphoid progenitors) are Il7rα^+, Lin$^-$, c-kit$^+$, and Sca-1$^+$. Absolute numbers of progenitor populations are shown below. n=3-7.

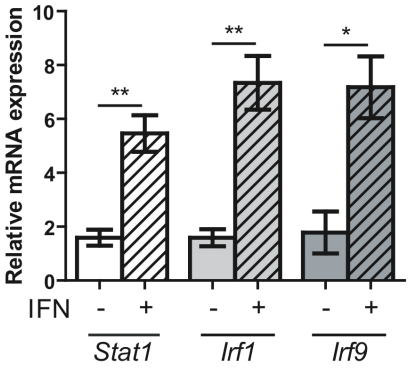


Supplementary Figure 4. Infection stimulates expansion of early progenitor compartments.

(A) Whole bone marrow was isolated from naïve and *M. avium*-infected mice 4 weeks postinfection. Progenitor populations shown were previously gated as Lin- live cells. MPPs (multipotent progenitors) are Lin^{-}, c-kit^{+} Sca-1^{+}, $Flk2^{+}$, and $CD34^{+}$. ST-HSCs (short-term HSCs) are Lin^{-}, c-kit^{+}, Sca-1^{+}, $Flk2^{-}$, and $CD34^{+}$. LT-HSCs are Lin^{-}, c-kit^{+}, Sca-1^{+}, $Flk2^{-}$, and $CD34^{-}$. (B) LT-HSCs were gated to Hoechst Blue and Red to view the Side Population. n=3-7.

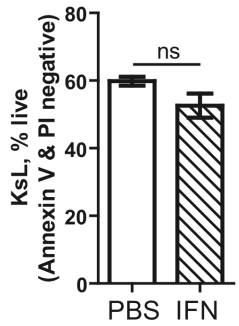


Supplementary Figure 5. *M. avium* infection stimulates increased HSC cycling. Hoechst and Pyronin Y staining was used to determine the cell cycle status of LT-HSCs (SP^{KLS}) isolated from uninfected and *M. avium*-infected WT mice. n=3-4.



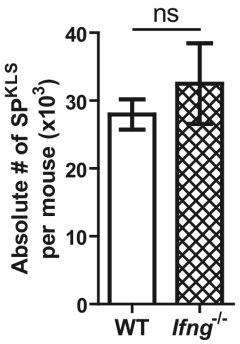
Supplementary Figure 6. Canonical IFN pathway genes are stimulated in HSCs with IFNγ exposure.

Real-time RT-PCR was used to determine relative expression of *Stat1, Irf1,* and *Irf9* mRNA in HSCs (SP^{KLS}) with or without IFNγ treatment. n=2-3 independent samples.



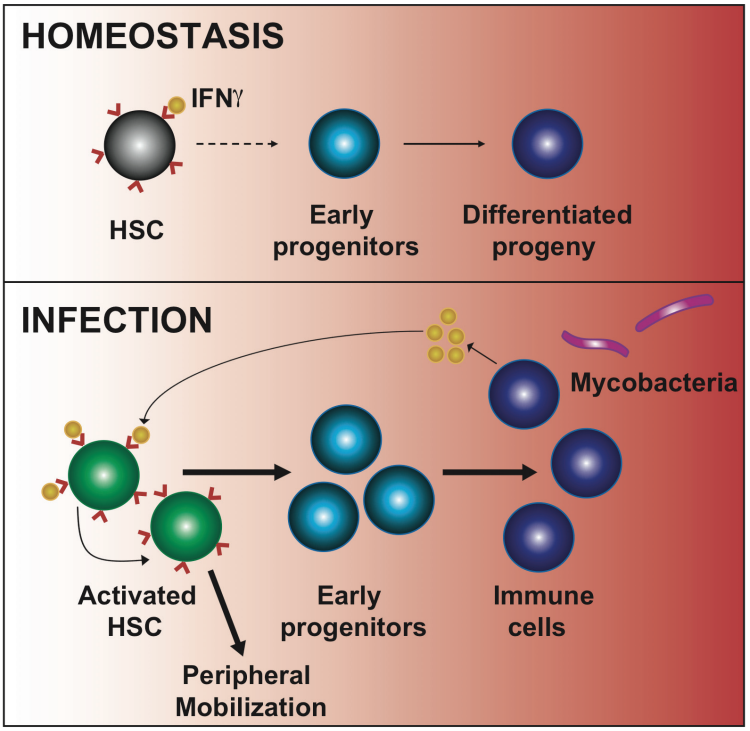
Supplementary Figure 7. IFNγ does not cause increased cell death of hematopoietic progenitors.

The percentage of live KSL cells after 6 hours incubation with PBS or IFNγ was determined using Annexin V and propidium iodide staining. n=5.



Supplementary Figure 8. The absolute number of HSCs is unchanged in *Ifng*-deficient mice.

Absolute numbers of SP^{KLS} from wild-type and *Ifng*$^{-/-}$ mice were determined. n=3-4, data representative of two independent experiments.



Supplementary Figure 9. Model for IFNγ-mediated regulation of hematopoietic stem cells.

Infection triggers HSC proliferation and mobilization via interferon signaling. Under homeostatic conditions, most HSCs are dormant and generate differentiated progeny at a low rate. Basal levels of IFNγ may contribute to HSC cycling. During infection, IFNγ is generated by macrophages, NK cells, and lymphocytes that sense pathogens such as mycobacteria. After circulation through the bloodstream, IFNγ can activate HSCs in the bone marrow, thereby promoting proliferation and mobilization to replenish immune cell populations.