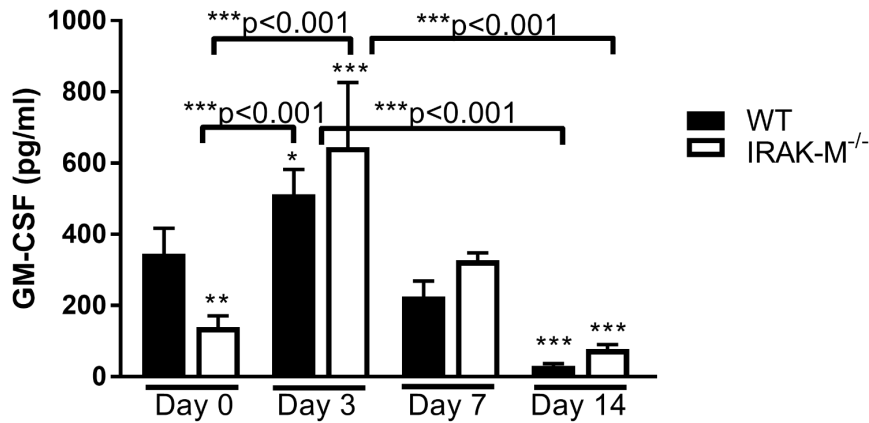
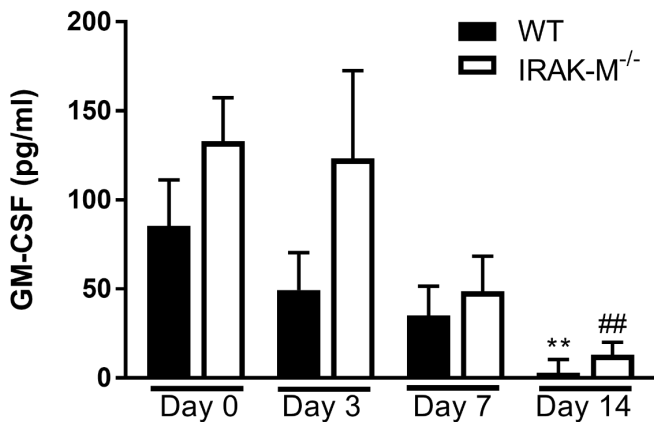
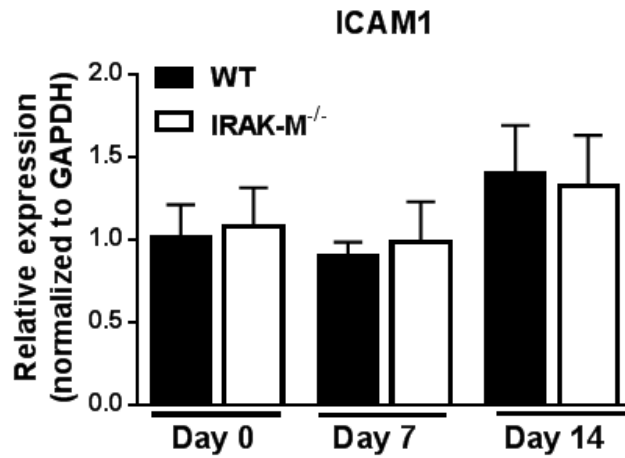
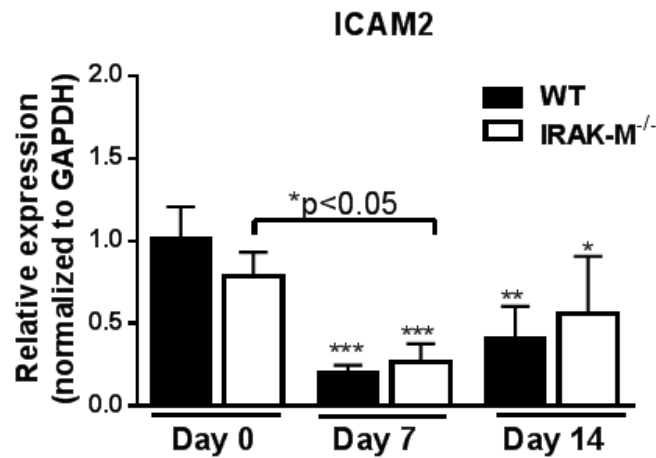
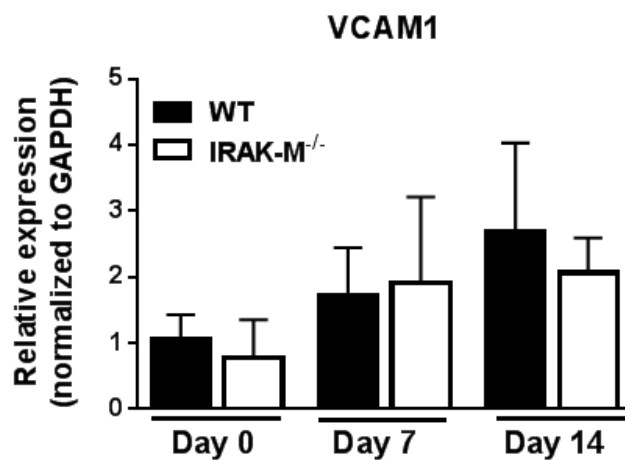


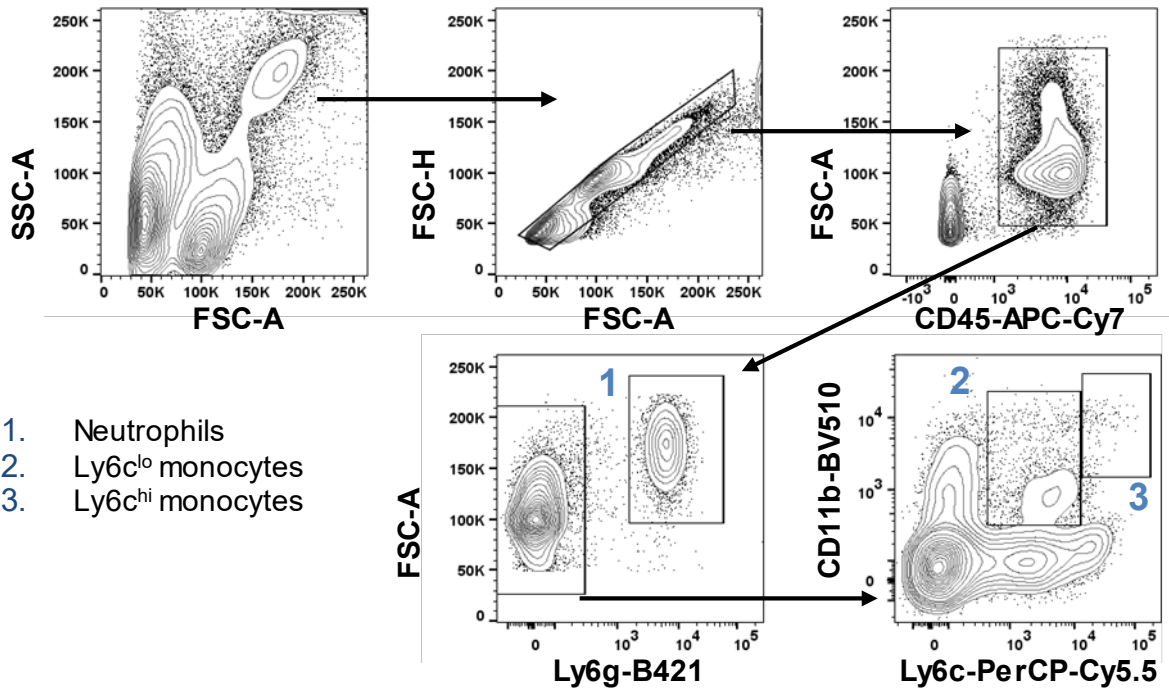
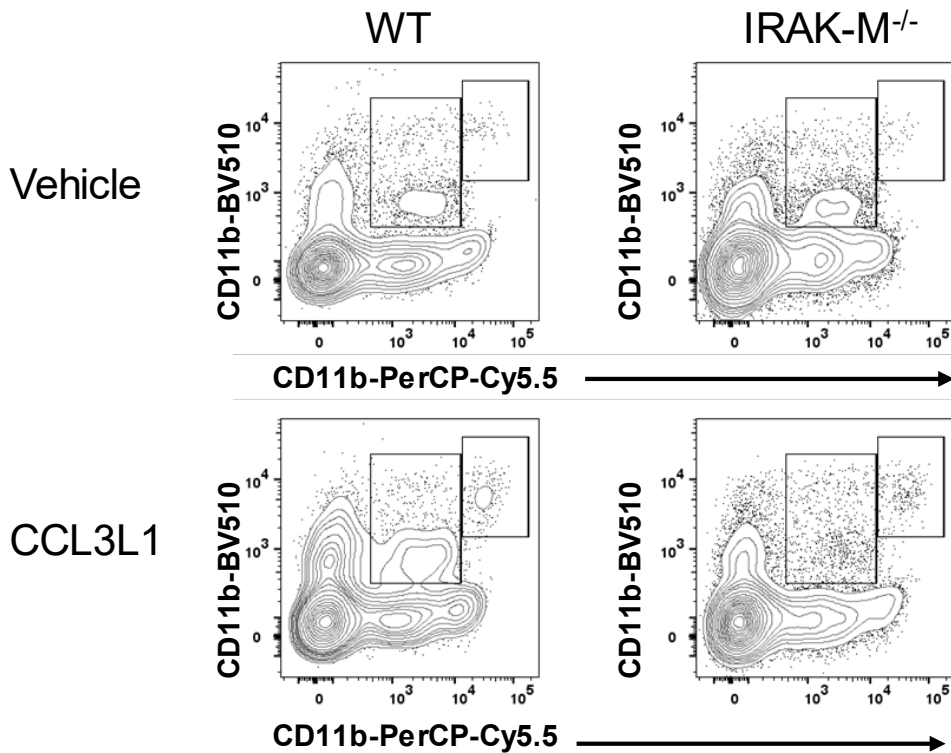
**Supplemental Figure 1: Flow gating scheme for identification of neutrophils, macrophages and monocytes in the lungs.** WT mice were challenged with bleomycin (i.t.) and whole lung tissue was digested and expression of various myeloid cell populations were determined by flow cytometry as described in the material and methods section and specific markers and fluorescent labels are detected in the representative blots. (A) Using an untreated WT mouse as a representative group, cells were first gated to exclude debris, singlets and CD45<sup>+</sup> leukocytes. Then neutrophils (1) were selected as CD45<sup>+</sup>CD64<sup>+</sup>Ly6g<sup>+</sup>. Monocytes were classified as inflammatory monocytes (2) which express CD45<sup>+</sup>Ly6g<sup>+</sup>MHCII<sup>+</sup>CD64<sup>+</sup>CD11b<sup>+</sup>Ly6c<sup>+</sup> or classical monocytes (3) which express CD45<sup>+</sup>Ly6g<sup>+</sup>MHCII<sup>+</sup>CD64<sup>+</sup>CD11b<sup>+</sup>Ly6c<sup>-</sup>. In addition, macrophages were classified as tissue-resident (TR)-alveolar macrophages (5) which express CD45<sup>+</sup>Ly6g<sup>+</sup>CD11c<sup>+</sup>CD64<sup>+</sup>SigF<sup>hi</sup>, monocyte-derived (Mo)-alveolar macrophages (4) which express CD45<sup>+</sup>Ly6g<sup>+</sup>CD11c<sup>+</sup>CD64<sup>+</sup>SigF<sup>lo</sup>, or interstitial macrophages (6) which express CD45<sup>+</sup>Ly6g<sup>+</sup>CD64<sup>+</sup>SigF<sup>-</sup>MHCII<sup>+</sup>CD11b<sup>+</sup>. (B) A representative figure depicting the changes in TR-AMs and Mo-AMs populations in a WT mouse after bleomycin challenge.

**A****B**

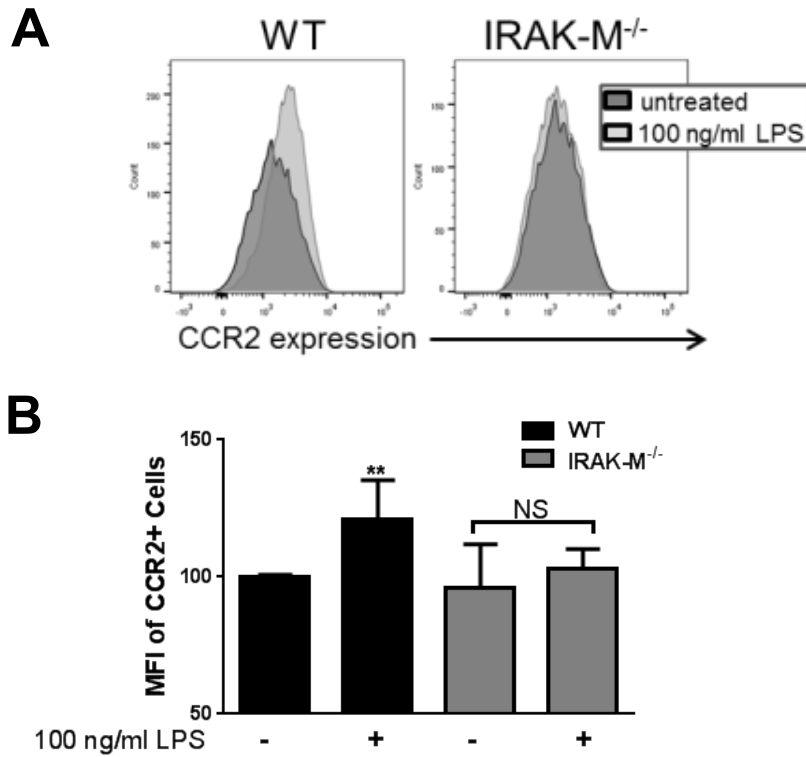
**Supplemental Figure 2: No difference in production of GM-CSF in BALF or by airway epithelial cells (AECs) from WT and IRAK-M<sup>-/-</sup> mice after bleomycin challenge.** WT and IRAK-M<sup>-/-</sup> mice were challenged with bleomycin (i.t.). Production of GM-CSF was assessed by specific ELISA in (A) isolated alveolar epithelial cells (AEC) and (N=5 samples/group) and (B) BALF (N=4 samples/group) at the time points indicated. Statistics were determined by one-way ANOVA with Bonferroni correction. \*p<0.05 \*\*p<0.01, \*\*\*p<0.001 when compared to WT Day 0 and ##p<0.01 when compared to IRAK-M<sup>-/-</sup> Day 0, unless otherwise noted.

**A****B****C**

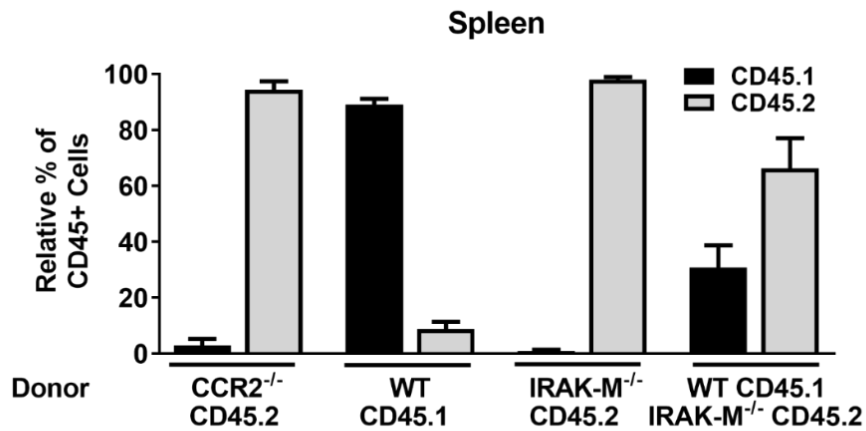
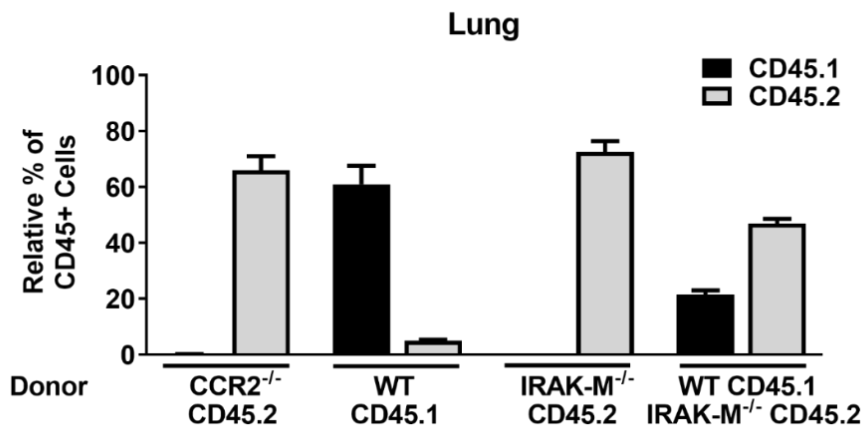
**Supplemental Figure 3: No difference in expression of ICAM and VCAM integrin expression in lungs isolated from WT and IRAK-M<sup>-/-</sup> mice after bleomycin challenge** WT and IRAK-M<sup>-/-</sup> mice were challenged with bleomycin (i.t.) and mRNA expression of (A) ICAM1, (B), ICAM2, and (C) VCAM1 (N=5 samples/group) was assessed by qPCR. Statistics were determined by one-way ANOVA with Bonferroni correction. \*p<0.05 \*\*p<0.01, \*\*\*p<0.001 when compared to WT Day 0.

**A****B**

**Supplemental Figure 4: Flow gating scheme for identification of neutrophils and monocytes in the blood.** Blood was collected from the descending aorta via heparinized needles and RBCs were lysed as described in the material and methods. (A). Cell populations from a WT untreated mouse were determined by staining of specific antibodies to differentiate neutrophils (CD45<sup>+</sup>Ly6g<sup>+</sup>), classical monocytes (CD45<sup>+</sup>Ly6g<sup>-</sup>CD11b<sup>+</sup>Ly6c<sup>lo</sup>) and inflammatory monocytes (CD45<sup>+</sup>Ly6g<sup>-</sup>CD11b<sup>+</sup>Ly6c<sup>hi</sup>). (B). WT and IRAK-M<sup>-/-</sup> mice were treated with CCL3L1 (3 μg) or vehicle control and blood was collected and stained for flow cytometry using the gating scheme listed above. A depiction of a representative blot for monocyte populations is provided for WT and IRAK-M<sup>-/-</sup> mice that were treated with either CCL3L1 or vehicle.



**Supplemental Figure 5: Presence of IRAK-M resulted CCR2 upregulation after LPS stimulation in BMDMs after LPS stimulation.** BMDMs were derived from WT and IRAK-M<sup>-/-</sup> mice as described in the material and methods. At day 6, cells were lifted and either stimulated with LPS (100 ng/ml) or left as untreated controls. The presence of CCR2 was measured by flow cytometry and expressed either as a (A) histogram or MFI (B) when compared to untreated controls (N=5 samples/group). Statistics were determined by one-way ANOVA with Bonferroni correction. \*\*p<0.01

**A****B**

**Supplemental Figure 6: Relative reconstitution rates of competitive bone marrow transplant model.**

The relative reconstitution rates of the competitive BM chimera mice were determined by measuring the relative number of CD45.1 vs. CD45.2 cells present in the spleen (A) and lung (B). N=4 samples/group statistics