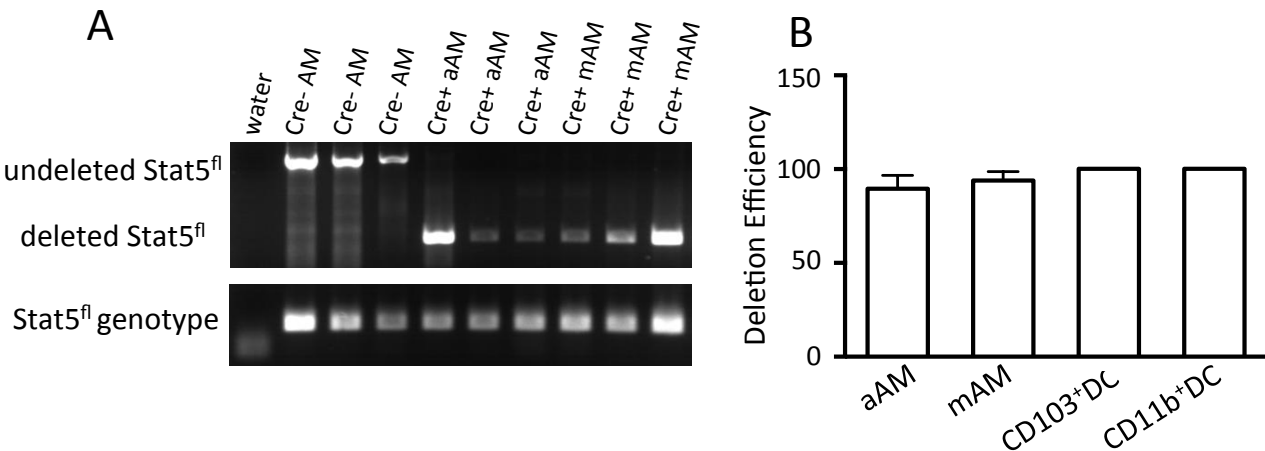
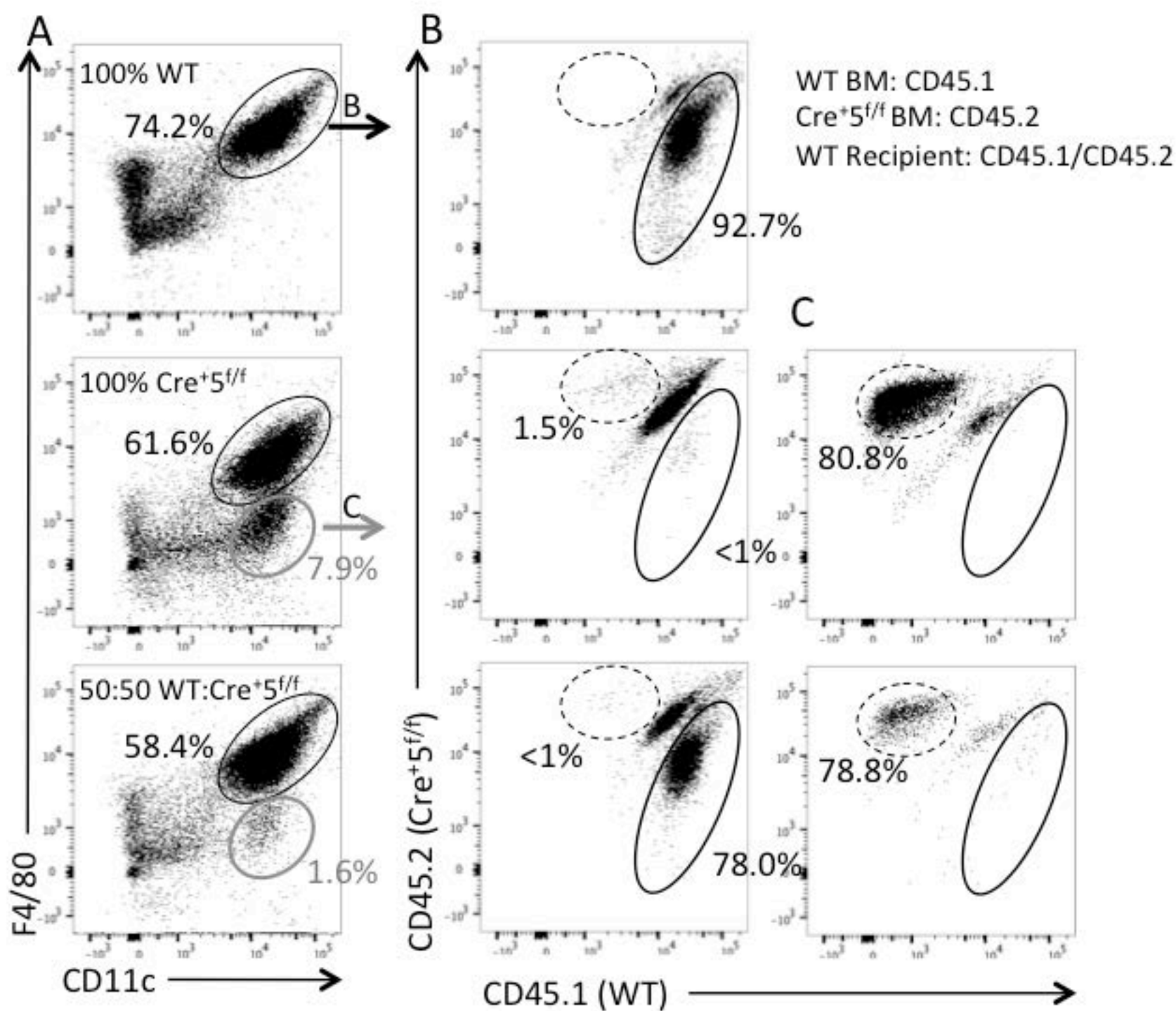


Supplemental Figure 1



Supplemental Figure 1. Deletion Efficiency of Stat5^{fl} in CD11c⁺ cells in the lung. Lungs cells from Cre⁻ and Cre⁺Stat5^{fl} mice were processed for flow cytometry and cell sorting of SigF^{int}CD11c^{int}CD11b^{high} (aAM), SigF^{high}CD11c^{high}CD11b^{low} (mAM), CD11c⁺CD103⁺ (CD103⁺DC), and CD11b⁺ DC cell populations. (A, B) PCR was performed on the DNA isolated from these cells to measure the deletion efficiency of Stat5. (n=3 mice/group)

Supplemental Figure 2



Supplemental Figure 2. Failure of Stat5-deficient bone marrow to reconstitute alveolar macrophage populations. Bone marrow chimeras were generated in CD45.1/CD45.2 wild-type recipient mice using CD45.1 wild-type, CD45.1 Cre^{5f/f} or 50:50 mixed donor marrow cells. (A, B) At 3 months, the origin of F4/80⁺CD11c⁺ alveolar macrophages was determined by CD45.1 and CD45.2 immunostaining. Mice that received WT marrow cells (top row) had a majority of alveolar macrophages of donor origin. Mice that received Cre^{5f/f} marrow cells (middle row) had a majority of alveolar macrophages of host origin and fewer than 2% of donor origin. Mice that received a 50:50 mix of WT and Cre^{5f/f} marrow cells (bottom row) had a majority of alveolar macrophages of WT donor origin and less than 1% of Cre^{5f/f} origin. (C) Mice that received Cre^{5f/f} marrow cells had persistence of donor cells that failed to mature into alveolar macrophages.