**Supplementary Figure Legends**

**Fig. S1. Myeloid cells, CCL9 and its effect on tumor phenotype**. (A) Cytokine array of Gr-1+CD11b+ myeloid cells sorted from lungs or spleens of tumor-bearing mice 14 days after tumor cell injection. Pooled samples from 3 mice were used for each group. The myeloid cells were cultured in tumor-conditioned media, and the culture supernatant was used for array. (B) CCL9 ELISA of proteins extracted from the organs of mice bearing 4T1 on day 14 after tumor injection, at which time the premetastatic niche was established. (C) Lung metastasis of 4T1 cells co-injected with Gr-1+CD11b+ or RAW246.7 cells. n=9-10 mice per group. (D) Representative pictures of lung metastasis of tumor cells co-injected with RAW246.7 cells deficient in CCL9 expression. Mice received tail vein co-injection of 5X105 67NR and 2X105 RAW264.7 cells. Mice were sacrificed 4 weeks after cell injection. Quantitative data in Fig. 2E. (E) Primary tumor growth of 67NR co-injected with Gr-1+CD11b+ myeloid cells with or without CCL9 knockdown. n=8-10 mice per group. \*P<0.05, \*\*P<0.01.

**Fig. S2. CCR1 expression in tumor cells.** (A) RT-qPCR of CCR1 expression in B16F1 and B16F10 cell lines representing different capability of metastasis. Shown is one of two experiments performed. (B & C) RT-qPCR showed CCR1 over-expression in 67NR cells (B) and B16F1 cells (C). One experiment with triplicates.

**Fig. S3. CCL23, the human CCL9 orthologous.** (A) Family tree that identified by online tool Treefam. mouse CCL9 and human CCL23 are closely related. (B) Human CCL23 (Phy000875R) is identified as one of the two orthologues of mouse CCL9. Data obtained from online tool mrtaPhOrs.

**Fig. S4. CCL9 promotes tumor cell survival and colonization.** (A) SCVM imaging showing a majority of tumor cells die within 24 hours after tail vein injection. Left panels: Representative pictures. Right panel: Quantitative data indicates fluorescence signal per field. (B) A decreased tumor cell survival (upper panel, SCVM and TUNEL) or metastatic colonization (lower panel, PUMA) when co-injected with Gr-1+CD11b+ cells with CCL9 knockdown. (C) Deletion of CCL9 in Gr-1+CD11b+ cells did not affect tumor cell extravasation. 67NR-GFP tumor cells were evaluated under confocal microscope against red blood vessel (Dextran labeled). (D) Flow cytometry analysis of tumor cells stained with Annexin V and 7AAD after co-culture with Gr-1+CD11b+ cells. Triplicates for each group, one representative experiment from 2 performed is shown. rmCCL9: recombinant mouse CCL9; CoSN: myeloid-tumor co-culture supernatant; CCL9 NeuAB: CCL9 neutralizing antibody. (E) B16F1 cells express higher level of CCR1 compared with 67NR cells, by RT-qPCR. (F) MTT assay showing that CCL9 partially rescue doxorubicine-induced apoptosis. Recombinant CCL9 was added to 4T1 cells before overnight treatment of doxorubicine. Shown is one of the two experiments performed. \*P<0.05, \*\*P<0.005, \*\*\*P<0.001, #P>0.05.

**Fig. S5. CCL9 regulation by TGFβ-p38 pathway.** (A) ELISA showing overexpression of CCL9 in TβRII KO Gr-1+CD11b+ cells. (B) Western blot of p-ATF2, a substrate of p38 kinase. Gr-1+CD11b+ cells with or without TβRII expression were treated with p38 inhibitor SB203580 at indicated doses. Jurkat cells treated with RPS were used as a positive control for P-ATF2. \*P<0.05, \*\*P<0.005, \*\*\*P<0.001, #P>0.05.

**Fig. S6. Fold increase in myeloid cell subsets in the lungs.** Single cell suspensions were prepared from lungs of tumor bearing mice. n=3 mice for each group. Fold changes of cell number were calculated using day 0 or non-tumor bearing mice as a base line.