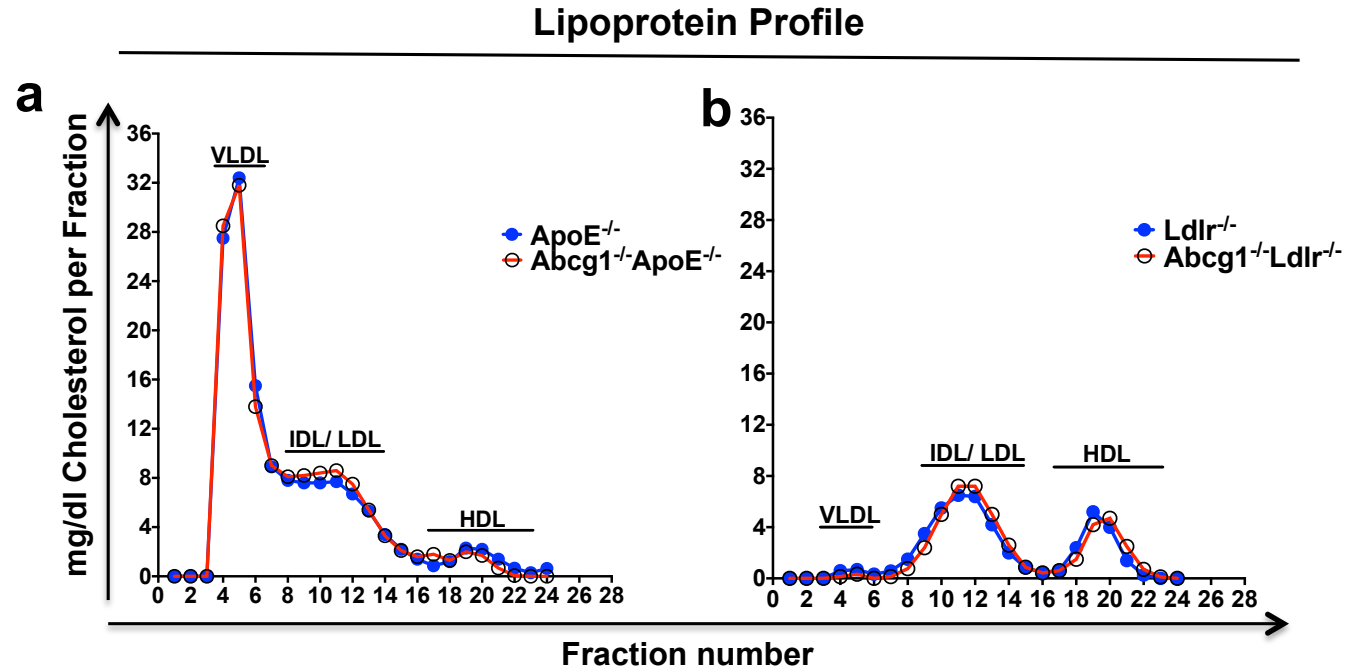


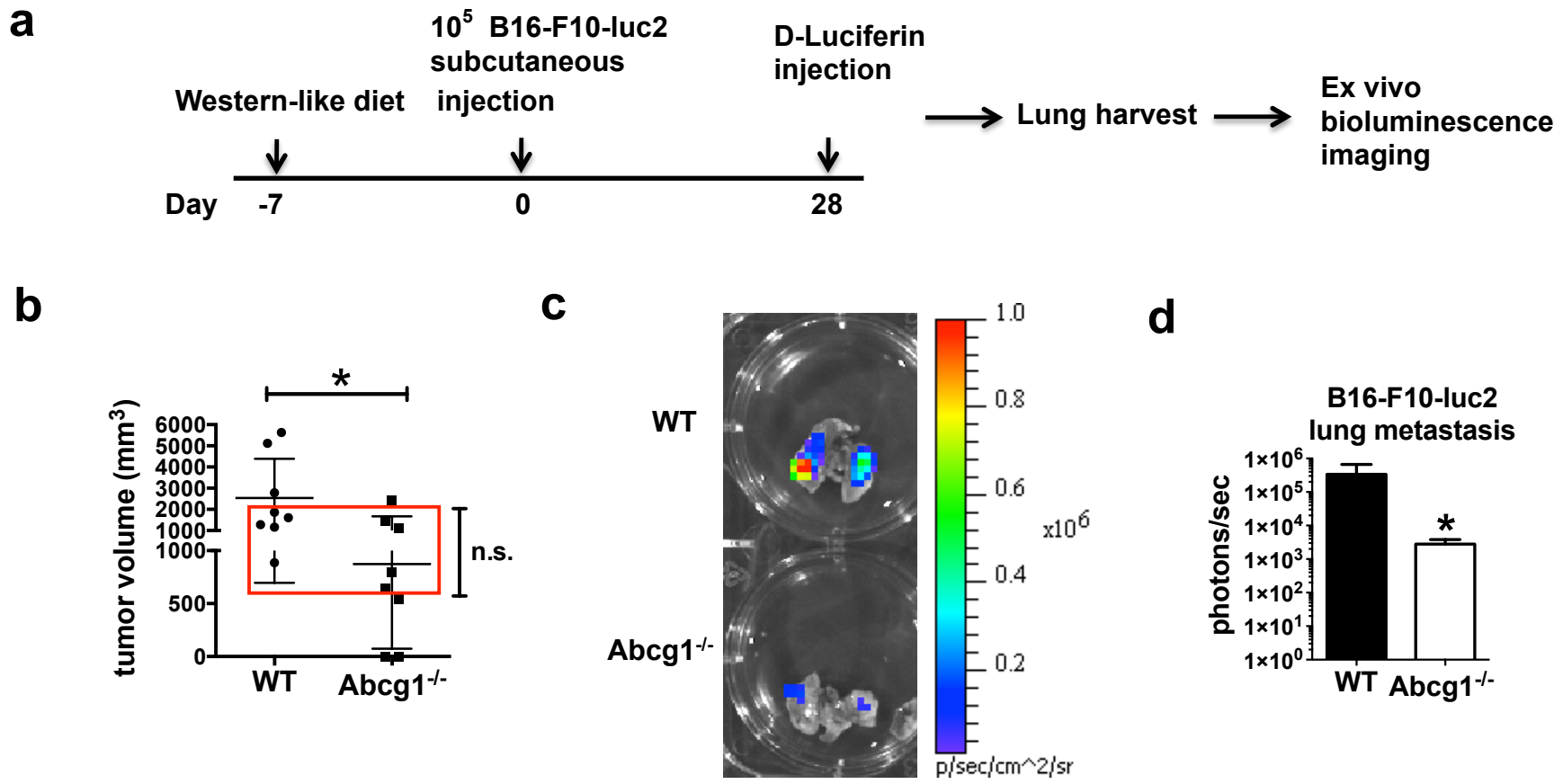
Supp. Figure 1. Hedrick



Supplementary Figure 1

The loss of ABCG1 does not change plasma lipoprotein profiles in tumor-bearing hypercholesterolemic mice. Chow diet-fed (a) ApoE^{-/-}, Abcg1^{-/-} ApoE^{-/-} and (b) Ldlr^{-/-}, Abcg1^{-/-}Ldlr^{-/-} mice were injected with MB49 tumor cells. 12 days later, blood plasma from 5 tumor-bearing mice for each group was pooled and lipoprotein profiles were analyzed by FPLC. Graph shows VLDL, IDL/LDL and HDL levels in all groups.

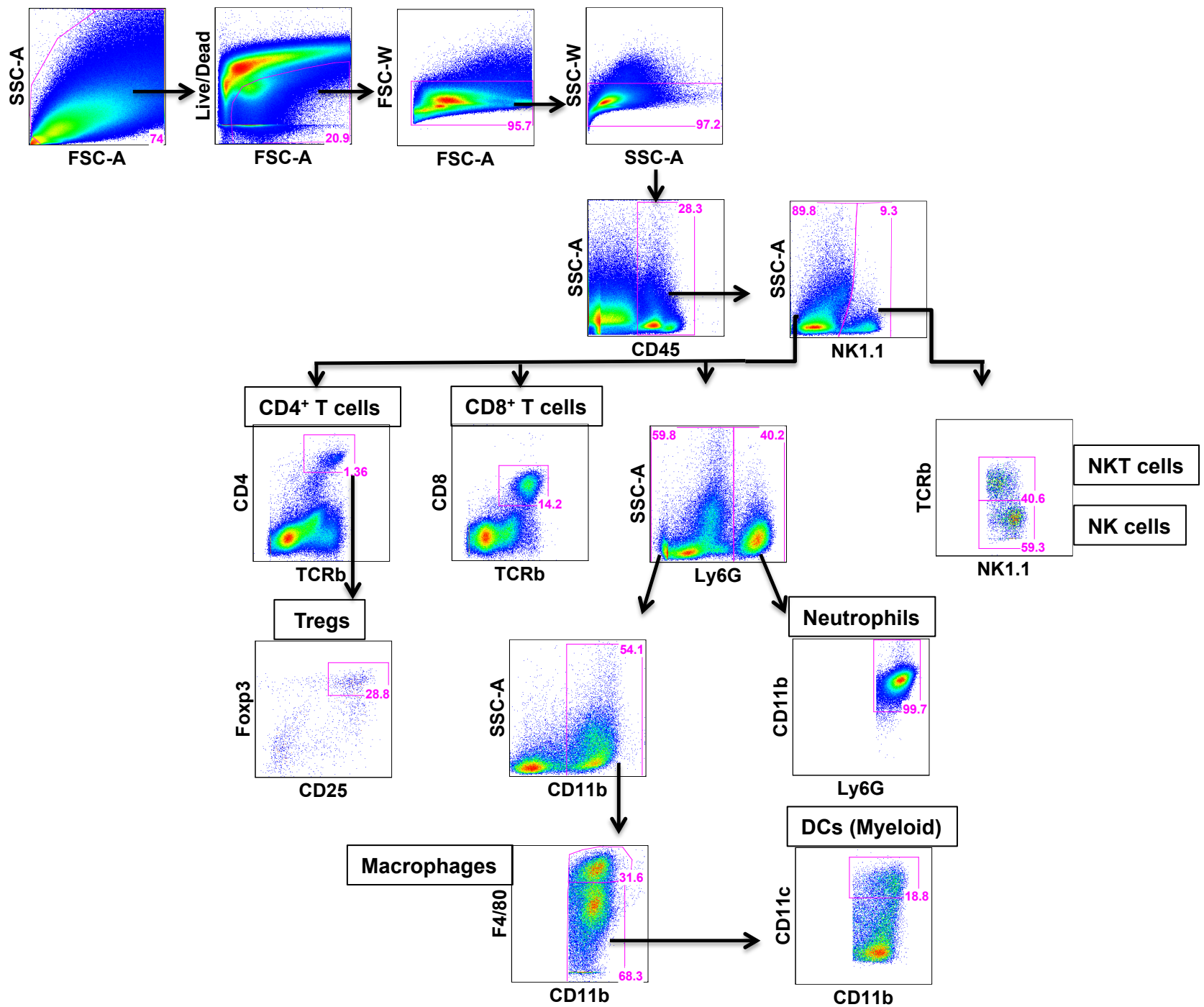
Supp. Figure 2. Hedrick



Supplementary Figure 2

***Abcg1*^{-/-} mice on Western-like diet show decreased tumor metastasis.** (a-d) Lung metastasis was quantified based on luminescence detected by using IVIS 200 Bioluminescence imager. (a) Schematic diagram of the experimental design is shown. (b) The graph shows tumor volume in Western-like diet-fed *Abcg1*^{-/-} and WT mice 28 days after injection of B16-F10-luc2 cells subcutaneously. Red box shows the mice that were picked for the spontaneous metastasis analysis. **p*<0.05, n.s.: not significant, two tailed Student's t test (c) Representative lung images and (d) Bar graph show spontaneous B16-F10-luc2 lung metastasis in *Abcg1*^{-/-} (*n*=5) and WT (*n*=5) mice. Data is pooled from 2 independent experiments with similar results. (mean ± s.e.m., **P*< 0.05, Wilcoxon-matched-pairs signed rank test)

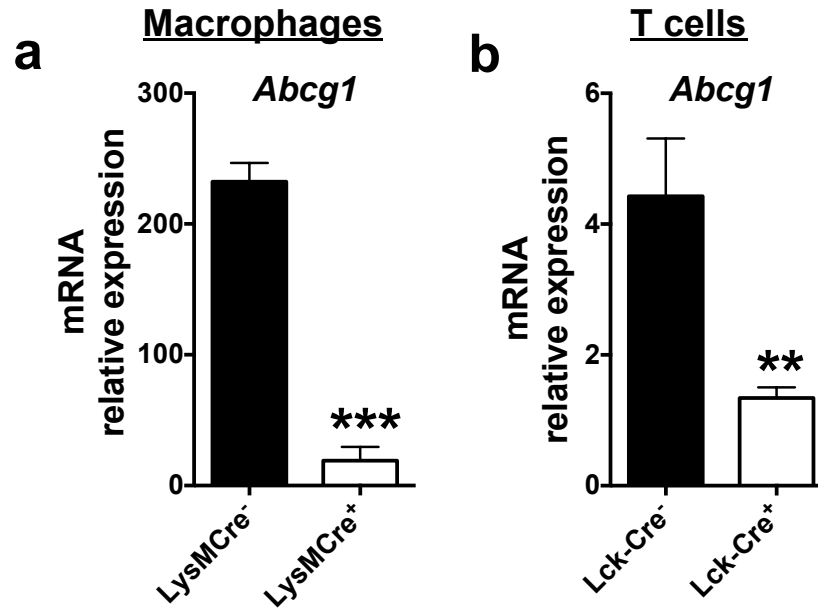
Supp. Figure 3. Hedrick



Supplementary Figure 3

Flow cytometry gating strategy to identify immune cell subsets in the tumor. Single-cell suspension from tumor was stained with different fluorophore-conjugated antibodies and analyzed by flow cytometry. Among single cells, the live cells, singlets and CD45⁺ cells were selected for further analysis to identify NKT cells (NK1.1⁺ TCRβ⁺), NK cells (NK1.1⁺ TCRβ⁻), CD4⁺ T cells (NK1.1⁻ TCRβ⁺, CD4⁺), CD8⁺ T cells (NK1.1⁻, TCRβ⁺ CD8⁺), Tregs (NK1.1⁻, TCRβ⁺, CD4⁺, CD25⁺, Foxp3⁺), neutrophils (NK1.1⁻, Ly6G⁺, CD11b⁺), macrophages (NK1.1⁻, Ly6G⁻, CD11b⁺, F4/80^{high}) and myeloid dendritic cells (NK1.1⁻, Ly6G⁻, F4/80⁻, CD11b⁺ CD11c⁺).

Supp. Figure 4. Hedrick

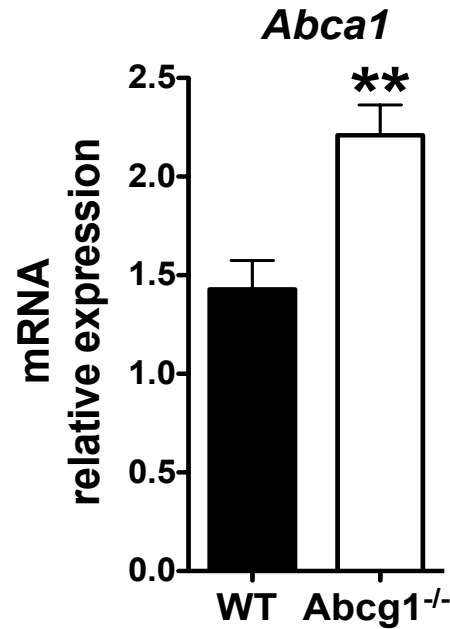


Supplementary Figure 4

Deletion of ABCG1 in macrophages from *Abcg1*^{fl/fl}-LysM-Cre⁺ mice and T cells from *Abcg1*^{fl/fl}-Lck-Cre⁺ mice.

(a) Macrophages were FACS-sorted from peritoneal lavage of *Abcg1*^{fl/fl}-LysM-Cre⁻ and *Abcg1*^{fl/fl}-LysM-Cre⁺ mice. (b) T cells were FACS-sorted from spleen of *Abcg1*^{fl/fl}-Lck-Cre⁻ and *Abcg1*^{fl/fl}-Lck-Cre⁺ mice. Expression of *Abcg1* was assessed by qPCR. (mean \pm s.e.m., ** $P < 0.01$, *** $P < 0.001$, two-tailed Student's t test).

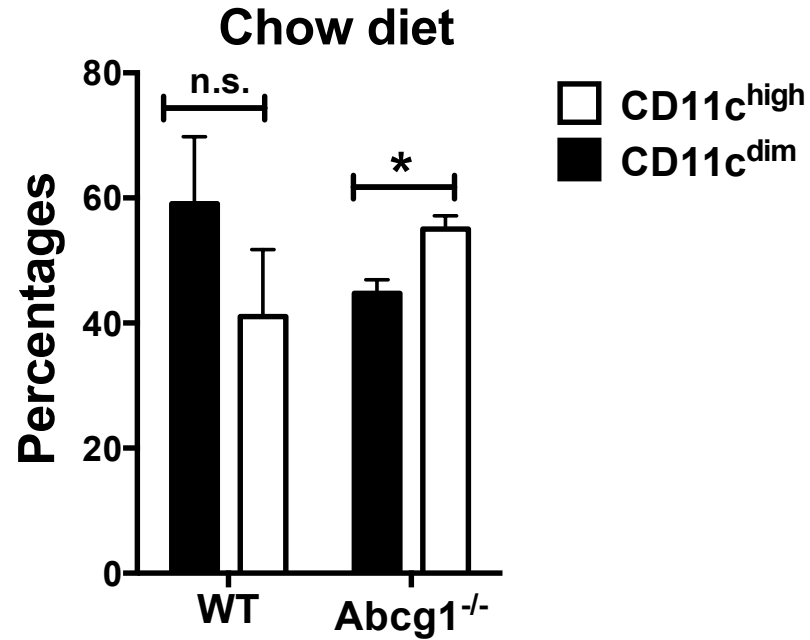
Supp. Figure 5. Hedrick



Supplementary Figure 5

ABCG1-deficient tumor macrophages display increased expression of *Abca1*. Macrophages were FACS-sorted from tumors from Western-like diet-fed *Abcg1*^{-/-} ($n=6$) and WT ($n=6$) mice 20 days after inoculation of MB49 cells. Expression of *Abca1* was measured by qPCR. Data are pooled from 2 independent experiments with similar results. (mean \pm s.e.m., ** $P < 0.01$, two-tailed Student's t test).

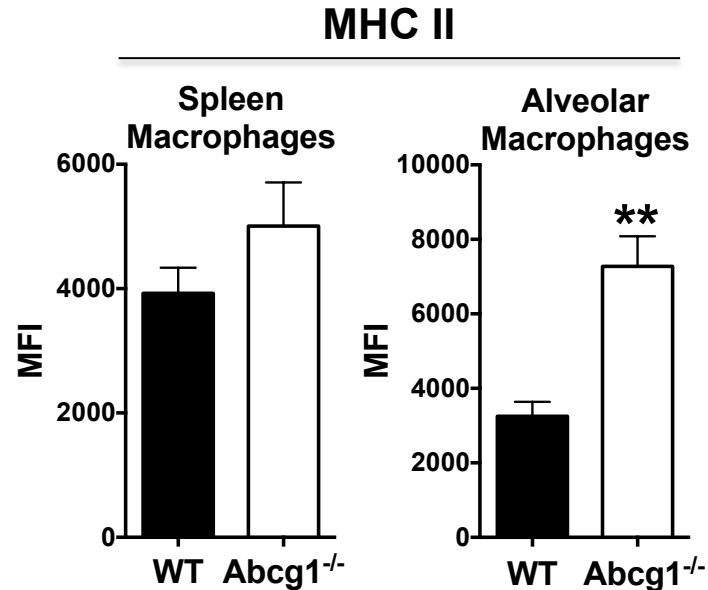
Supp. Figure 6. Hedrick



Supplementary Figure 6

Chow diet-fed *Abcg1*^{-/-} mice display a slightly higher percentage of CD11c^{high} (M1-like) macrophages in the tumor. Tumor cells from Western diet-fed *Abcg1*^{-/-} ($n=3$) and WT mice ($n=3$) mice were analyzed by flow cytometry 20 days after injection of MB49 cells. Bar graph shows percentages of CD11c^{high} (M1-like) and CD11c^{dim} (M2-like) macrophages (CD45⁺, NK1.1⁻, Ly6G⁻, CD11b⁺, F4/80^{high}) in the tumor. Data is representative of 2 independent experiments with similar results (mean \pm s.e.m., * $P < 0.05$, two-tailed Student's t test).

Supp. Figure 7. Hedrick

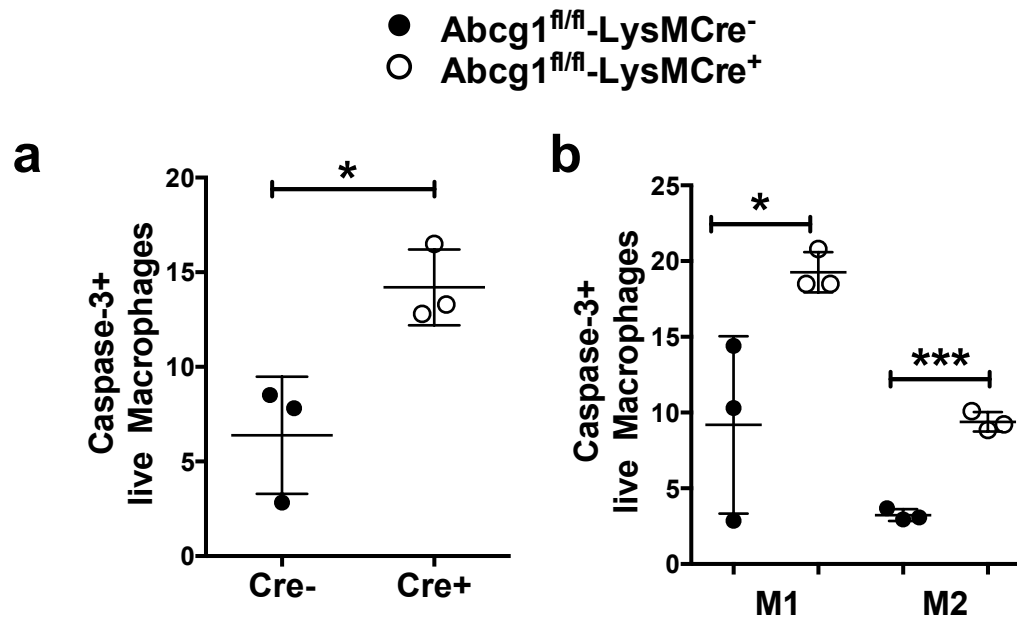


Supplementary Figure 7

Alveolar macrophages in Western-like diet-fed tumor-bearing *Abcg1*^{-/-} mice display an M1 bias.

Spleen and lung cells from Western-like diet-fed *Abcg1*^{-/-} ($n=5$) and WT mice ($n=5$) were analyzed by flow cytometry 20 days after injection of MB49 cells. Bar graphs show the MFI of MHC II on spleen macrophages (CD19⁻, NK1.1⁻, Ly6G⁻, CD11b⁺, F4/80⁺) (left) and alveolar macrophages (CD45⁺, CD19⁻, NK1.1⁻, Ly6G⁻, CD11c⁺, SiglecF⁺) (right). Data are representative of 2 independent experiments with similar results (mean \pm s.e.m., ** $P < 0.01$, two-tailed Student's t test).

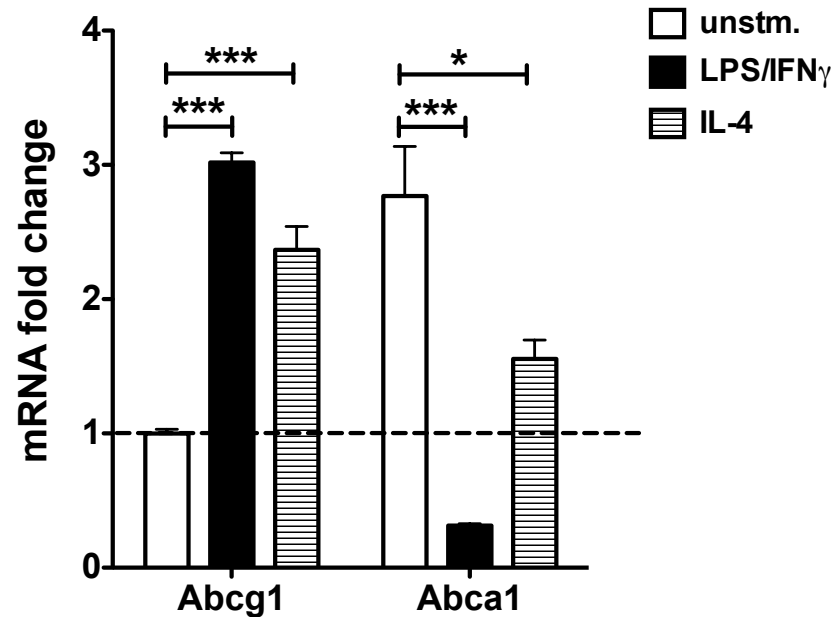
Supp. Figure 8.Hedrick



Supplementary Figure 8

ABCG1-deficient macrophages display enhanced apoptosis in the tumor. Tumor cells from Western-like diet-fed *Abcg1*^{fl/fl}-LysM-Cre⁺ ($n=3$) and *Abcg1*^{fl/fl}-LysM-Cre⁻ ($n=3$) mice were analyzed for apoptosis by Caspase-3 staining and flow cytometry 12 days after injection of MB49 cells. Dot plot show percentages of apoptotic (Caspase-3⁺ live) (a) total macrophages and (b) M1 (CD11c^{high}) and M2 (CD11c^{dim}) macrophages in the tumor. Data are representative of 2 independent experiments with similar results (mean \pm s.e.m., * $P < 0.05$ *** $P < 0.001$, two-tailed Student's t test).

Supp. Figure 9. Hedrick



Supplementary Figure 9

Polarized WT macrophages exhibit enhanced ABCG1 and reduced ABCA1 expression. WT bone marrow-derived macrophages were polarized to an M1 phenotype by IFN γ /LPS stimulation or to an M2 phenotype by IL-4 stimulation, *in vitro*. Expression of *Abcg1* and *Abca1* was assessed by qPCR. Data are representative of 2 independent experiments with similar results (mean \pm s.e.m., * P < 0.05, *** P < 0.001, two-tailed Student's t test).