Supplemental Figure Legends

**Figure S1. SEMA3A does not affect tumor cell metabolism**

(A-D) Cells were incubated in CO2-free incubator for 1 hour in medium containing glutamine (2 mM) and sodium pyruvate (1 mM). The (A) ECAR and (C) OCR were measured 3 times at intervals of 6 minutes at baseline and after the sequential addition of glucose, oligomycin, FCCP and rotenone/antimycin A. (B) Basal and glucose-stimulated ECAR were calculated from the last measurement at baseline and after the injection of glucose. (D) OCR was calculated from the last measurement at baseline, after the injection of glucose and after the injection of FCCP (maximal respiration). Data are shown as means ± SD from 5 independent experiments and are normalized to total protein content.

 **Figure S2. SEMA3As effect on monocytes and macrophages**

(A) 4T1-3A+ and 4T1-CTR tumors were analyzed by flow cytometry. Histograms display (A) MHCIIlowLy6Chigh monocytes, (B) classical CCR2+Ly6C+CD11b+ monocytes and (C) non-classical CX3CR1+Ly6C+CD11b+ monocytes from 4T1-3A+ and 4T1-CTR tumors (n = 7).

(D-E) Blood from 4T1-3A+ and 4T1-CTR tumor bearing mice was analyzed by flow cytometry. Histograms display classical (D) CCR2+CD115+Cd11b+ monocytes and (E) non-classical CX3CR1+CD115+CD11b+ (n = 6).

(F-G) Matrigel with 4T1 tumor cells ±rmSEMA3A were injected below the mammary fat pad of BALB/c mice. (F) Tumor volume and (G) tumor weight were measured at the end of the experiment (n=5, \*\*p < 0.01).

(H-J) SEMA3A and CTR matrigel tumors were analyzed by flow cytometry. (H) Dot plots display 1) MHCIIlowLy6Chigh monocytes, 2) immature MHCIIlowLy6Cintermediate, 3) MHCIIhighLy6Chigh/intermediate, 4) pro-tumoral M2- (Ly6ClowMHCIIlow) and 5) anti-tumoral M1- (Ly6ClowMHCIIhigh) Mφs gated from CD11b+Ly6G- cells from representative 3A+ and CTR tumors. Graphs display quantification of (I) M2-Mφs and (J) M1-Mφs (n = 5, \*p < 0.05).

**Figure S3. Increased accumulation of lymphocytes in SEMA3A tumors**

(A–B) Flow cytometry analyses display the percentage of (A) CD8+ T cells and (B) CD4+ T cells out of all CD45+ cellsin4T1-3A+ and 4T1-CTR tumors (n = 6, \*\*p < 0.01).

(C-D) Flow cytometry analyses display (C) the ratio between CD8+ T cells and CD4+ T cellsout of the total CD45+ population, and (D) NK cells (n = 5, \*p < 0.05) in SEMA3A and CTR matrigel tumors.

Data are presented as the mean + SD, and all data represent one out of two (C-D) or three (A-B) independent experiments.

**Figure S4. Macrophage depletion by anti-CSF1**

(A) Dot plots show the percentage of macrophages in representative 4T1-3A+ and 4T1-CTR tumors from vehicle and anti-CSF1 treated mice.

**Figure S5. SEMA3A does not regulate the gene signature in M0-, M1- or M2-educated BMDMs**

(A-L) Total RNA was extracted from un-polarized (M0) BMDMs or BMDMs polarized to an M1 or M2 phenotype and supplemented with rmSEMA3A. Gene expression levels were analyzed by qPCR. All levels were normalized to β-actin and are presented as the fold change compared to (A-C, E-L) M0-CTR or (D) M1-CTR (n = 3, \*p < 0.05, \*\* p < 0.01).

**Figure S6. NP1 expression is increased in M2-Mφ**

(A) MFI for NP1 expression in M1- and M2-polarized BMDMs.