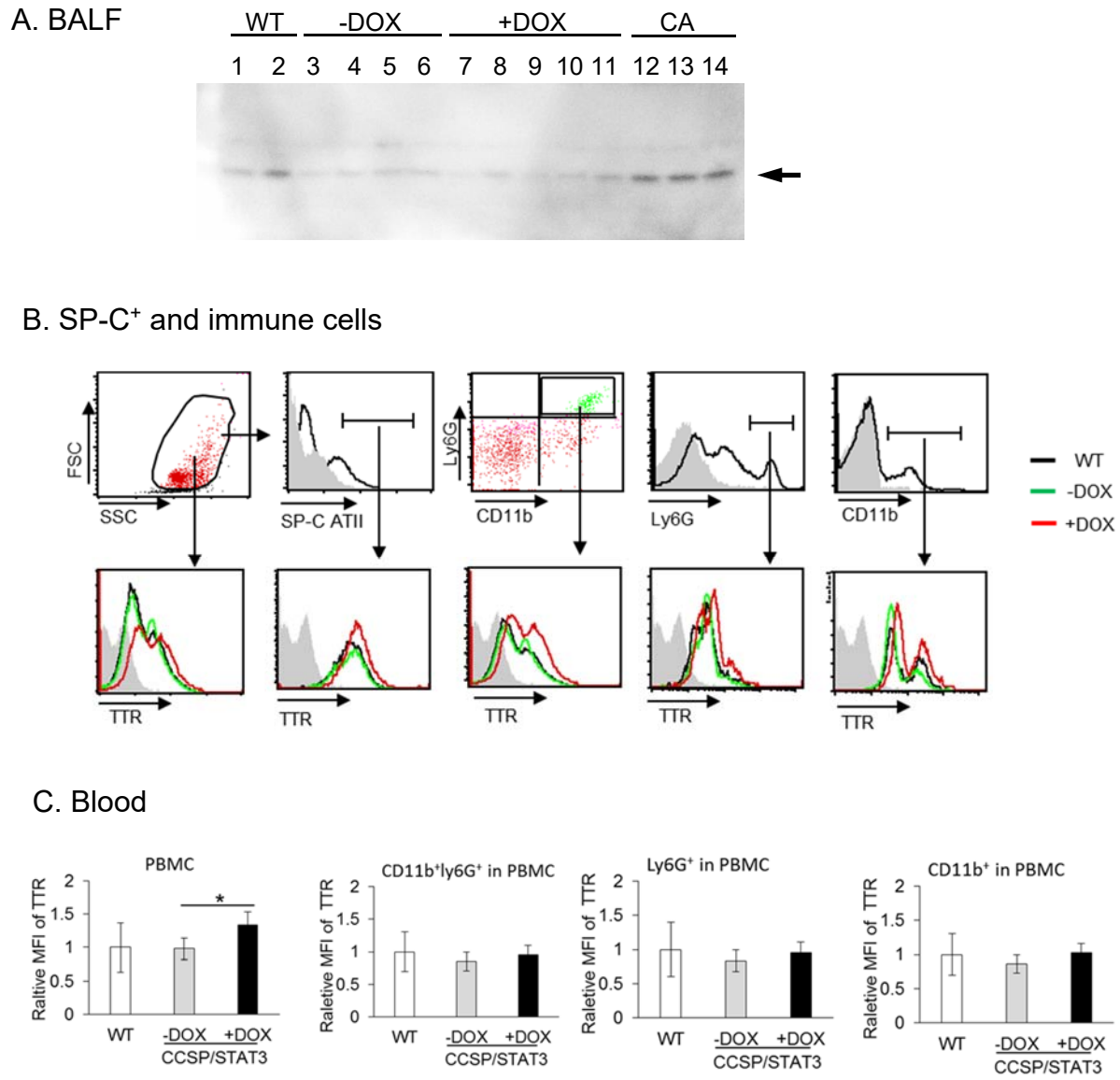
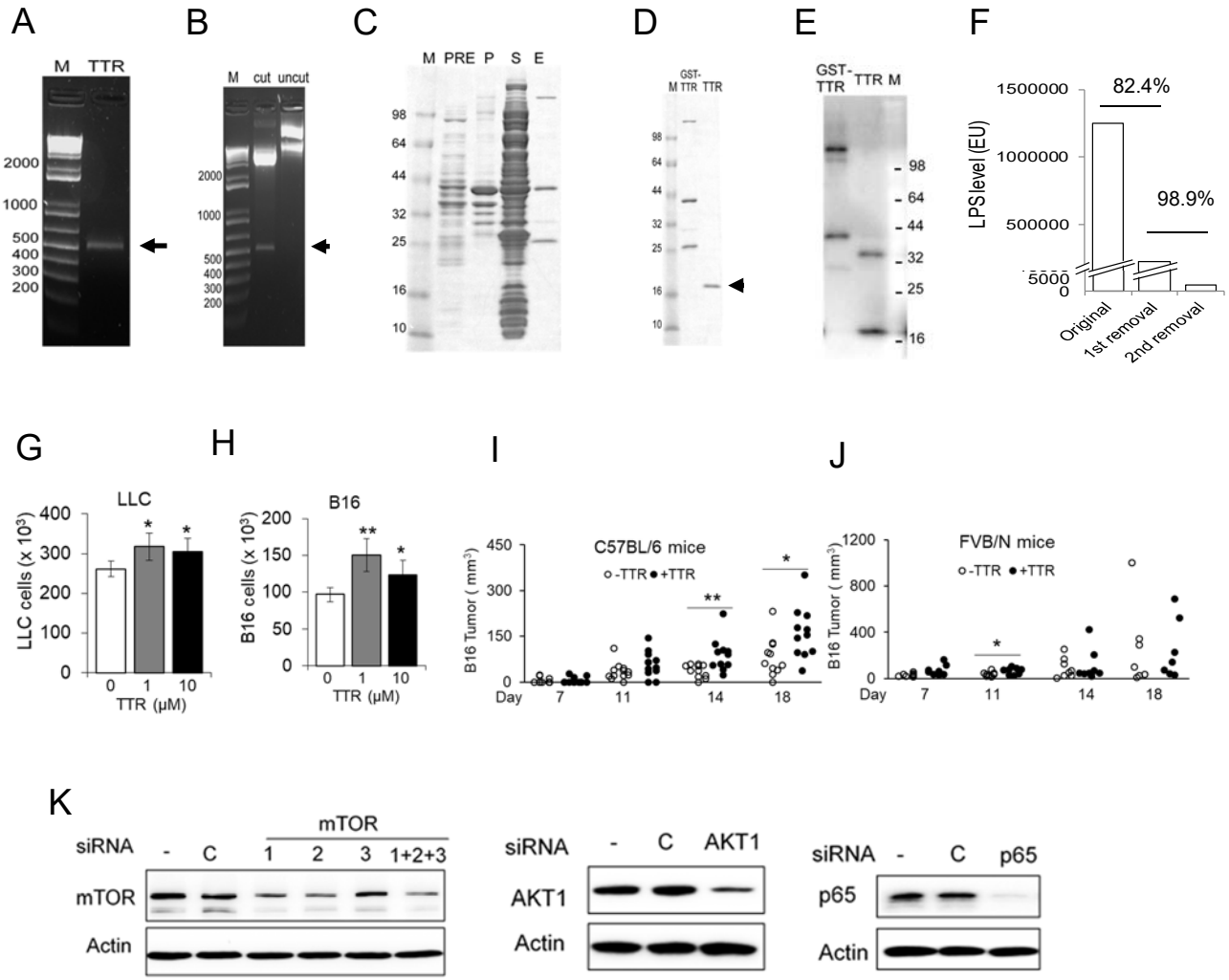
Supplemental Figure 1



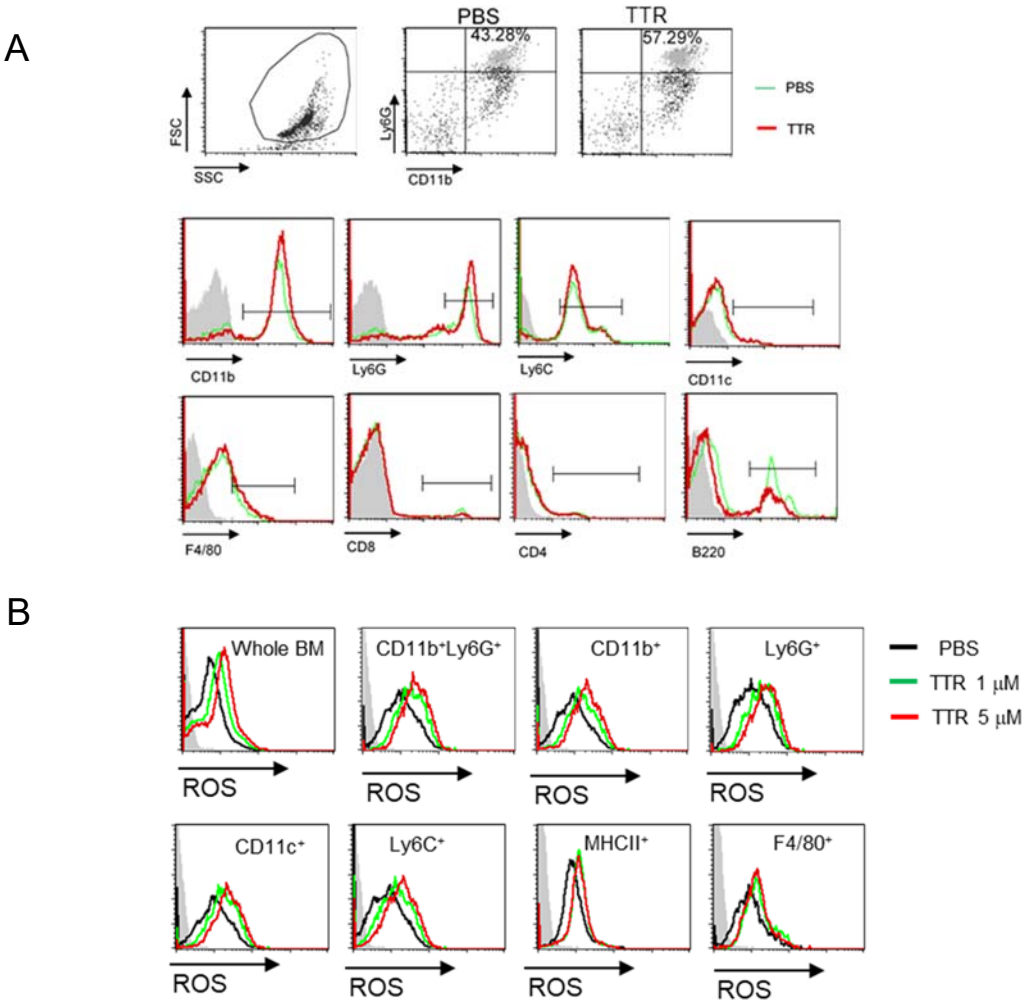
Supplemental Figure 1. **The expression level and distribution of TTR in BALF, blood and spleen of CCSP-rtTA/(tetO)$_7$-Stat3C bitransgenic mice. (A)** The expression levels of TTR in bronchoalveolar lavage fluid (BALF) of CCSP-rtTA/(TetO)$_7$-Stat3C bitransgenic mice were determined by Western blot. The arrow points to TTR. WT, wild type; -Dox, doxycycline untreated; +Dox, doxycycline treated without tumor; CA, doxycycline treated with tumor. (**B**) The gating strategies of the relative TTR expression levels in whole cells, SPC$^+$ cells, CD11b$^+$Ly6G$^+$ cells, Ly6G$^+$ cells and CD11b$^+$ cells of the lungs from WT and doxycycline treated (+DOX) and untreated (-DOX) CCSP-rtTA/(TetO)$_7$-Stat3C bitransgenic mice by flow cytometry; **(C)** The relative expression levels of TTR in whole white blood cells, CD11b$^+$Ly6G$^+$ cells, Ly6G$^+$ cells and CD11b$^+$ cells of the blood were determined by flow cytometry, n = 4. *, $p<0.05$.

Supplemental Figure 2



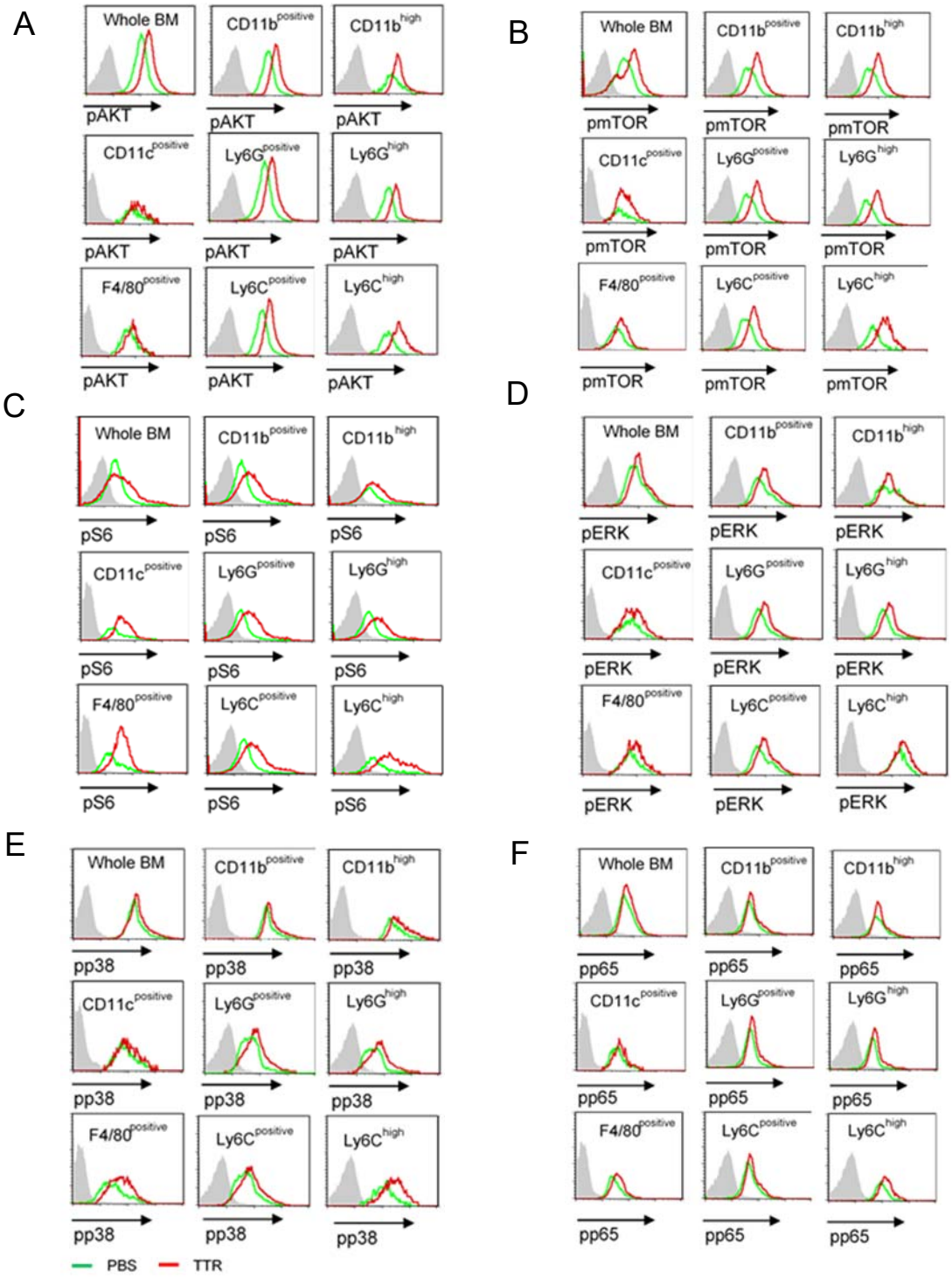
Supplemental Figure 2. **Molecular cloning, protein expression of mouse transthyretin (TTR) cDNA, and effect of TTR on tumor cell proliferation and tumor growth. (A)** The TTR cDNA was visualized in agarose gel. **(B)** The TTR-Flag cDNA insert in the pGEX-4T-1-TTR-Flag plasmid vector was confirmed by Not I and Xma I restriction enzyme digestion. **(C)** The expression of GST-TTR-Flag fusion protein (~42 kDa) in pre-induction (PRE), post induction pellet (P), supernatant (S) and GST column elution (E) were analyzed by SDS-PAGE and Coomassie blue staining. The TTR-Flag fusion protein (17 kDa) was digested by thrombin, visualized by Coomassie staining (**D**), and confirmed by Western blot analysis by using anti-FLAG antibody (**E**). (**F**) The LPS level in purified TTR-Flag fusion protein was measured before and after the removal procedure by endotoxin assay kit. In **A-E**), M, molecular weight marker of DNA or protein. Cut, vector digested by NotI and XmaI. Uncut, pGEX-4T-1-TTR plasmid. Recombinant TTR stimulate Lewis lung carcinoma (LLC) cells **(G)** and B16 melanoma cells **(H)** proliferation in vitro**.** n=4-5. Recombinant TTR stimulates B16 melanoma *in vivo* growth in syngeneic recipient C57BL/6 mice (n=11) (**I**) and allogeneic recipient FVB/N mice (n=8) (**J**). for G-J, *, $p<0.05$. **, $p<0.01$. (**K**) Expression levels of mTOR, AKT, and NFκB after siRNA knockdown respectively were measured by Western blot analyses.

Supplemental Figure 3



Supplemental Figure 3. Gating strategies of myeloid cells from the bone marrow after TTR injection *in vivo* and ROS assay after TTR treatment *in vitro*. (A) The TTR effect on differentiation of bone marrow lineage cells *in vivo*. TTR (320 μg / mouse) was i.v. injected into wild type mice twice a week for two weeks, and PBS was used as control. Single cells from the bone marrow were analyzed by flow cytometry. **(B)** ROS assay after TTR treatment *in vitro*. Fresh bone marrow cells ($1x10^6$) from wild type mice were recovered in RPMI 1640 medium (10% FBS) at 37^{o}C for 1 hour, followed by treatment with or without TTR (1, 5 μM) in PMB (50 μg/ml) for 1 hour. Treated cells were stained with myeloid lineage specific surface markers and 2 μmol/L 2', 7'-dichlorofluorescein diacetate (Invitrogen). The ROS level was measured by flow cytometry in gated areas using a LSRII machine (Becton Dickinson).

Supplemental Figure 4



Supplemental Figure 4. Gating strategies of signaling molecules involved in differentiation of bone marrow myeloid lineage. Whole bone marrow cells were isolated from wild type mice and cultured *in vitro* with TTR (5 μM) treatment or PBS control for 2 days. Activation of signaling molecules were analyzed by antibodies against phosphorylated Akt (**A**), mTOR (**B**), S6 (**C**), ERK (**D**), p38 (**E**), and NFκB p65 (**F**) in myeloid lineage cells by flow cytometry.