Supplementary Figure S1. AZD1480 abrogates STAT3 DNA binding while targeting JAK2 or STAT3 has little effect on viability or proliferation in cultured cells. STAT3 activation and DNA binding was stimulated by treatment of OVCAR-5 and OVCAR-8 cells with 50/µg/ml IL-6. IL-6 stimulated cells were treated with AZD1480 (0, 1.0 or 0.1 µmol/L) and subjected to EMSA analysis to determine the effects of AZD1480 on pSTAT3 DNA binding. DNA binding was reduced in the presence of 0.1 µmol/L AZD1480, and abrogated in the presence of 1.0 µmol/L AZD1480 in both cell lines. B) The viability of cultured murine ovarian carcinoma cells (MOVCAR) treated with increasing concentrations of AZD1480 was determined showing inhibition at concentrations of 5 or 10 µmol/L. C) Proliferation was assayed in MOVCAR cells expressing STAT3 shRNA or vector alone showing no difference in proliferation in cells with STAT3 knockdown. D) The viability of MOVCAR cells was reduced in the presence of concentrations of 5 or 10 µmol/L of the small molecule STAT3 inhibitor STATTIC.
Supplementary Figure S2. Pharmacodynamics of AZD1480 treatment. Mice treated with vehicle or AZD1480 were euthanized 2, 6 and 24 hours after drug treatment. Protein lysates of ovarian tumor tissue was analyzed by immunoblot for detection of pSTAT3\textsuperscript{Y705} and total STAT3. pSTAT3\textsuperscript{Y705} levels are reduced at 2 and 6 hours and recover by 24 hours post-treatment.
Supplementary Figure S3. Flow cytometry analysis of immune cell subpopulations in tumors and peritoneal washes from vehicle- and AZD1480-treated mice. A) Analysis of tumors showed the fraction of immune cells in tumors were small, the majority of infiltrating cells were CD45^+ F4/80^+ CD11b^+ macrophages (shown as normalized to CD45^+ leukocytes). B) Cell suspensions harvested from peritoneal washes were analyzed for CD45^+ CD19^+ B cells, CD45^+ F4/80^+ CD11b^+ macrophages, CD45^+ CD11b^+ Gr-1^+ MDSCs. The peritoneal fraction of B cells, macrophages and MDSC are unaffected by drug treatment.