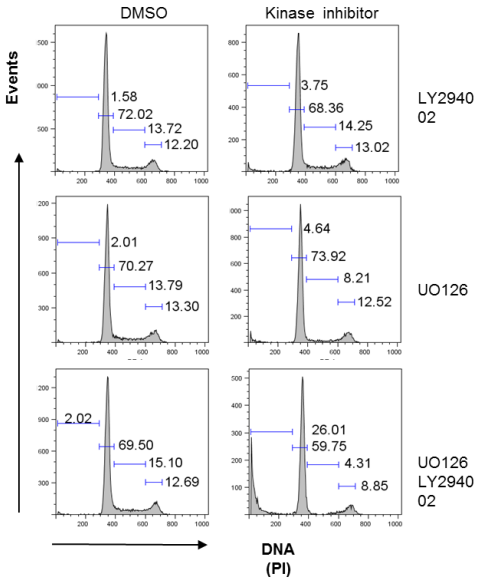

Figure S5. M-CSF-evoked ERK and PI-3 kinase activation are required for BMM survival. Randomly growing WT BMMs were treated with DMSO, UO126 (10μM) and LY294002 (10 μM) for 14 hours. Cells were then harvested, stained with propidium iodide and analyzed by flow cytometry. Note the relative insensitivity of BMM to U0126 or Ly294002 treatment, and the marked increase in apoptotic (sub-G1) cells in the presence of both inhibitors.

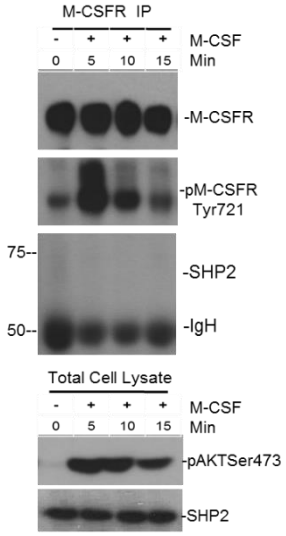

Figure S6. SHP2 is undetectable in the M-CSF-evoked M-CSFR immunocomplex. Wildtype BMMs were starved and stimulated with M-CSF (30ng/mL) for indicated time points. M-CSFR was immunoprecipitated from total cell lysates and SHP2 association with M-CSFR was visualized by immunoblotting using antibodies against SHP2. Note that M-CSF stimulation induced robust phosphorylation of M-CSFR on Tyr721 and AKT on Ser473. However, SHP2 wasn't detected in the M-CSFR immunocomplex.