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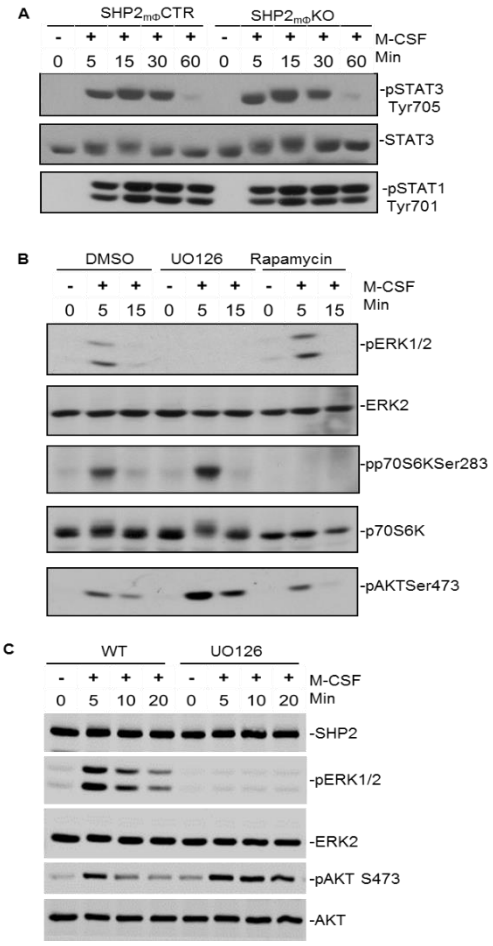

Figure S2. (**A**) M-CSF-evoked STAT1 and STAT3 activation are unaffected by SHP2 deficiency. $SHP2_{m\Phi}CTR$ and $SHP2_{m\Phi}KO$ BMMs were starved, and then either left unstimulated (-) or stimulated with M-CSF (+) for the indicated times. Lysates were resolved by SDS-PAGE and subjected to immunoblotting with the indicated antibodies. (**B**) Blocking M-CSF-evoked mTOR activation by Rapamycin treatment has no apparent effect on AKT activation. Wild type BMMs were starved, then treated with DMSO, UO126 (10µM) or Rapamycin (10 nM) for 1 hour, and then either stimulated with M-CSF (30 ng/ml) for the indicated times (+) or left unstimulated (-). Cell lysates were subjected to SDS-PAGE and immunoblotting with the indicated antibodies. ERK2 (on a duplicate blot) served as an internal loading control. (C) Blockade of ERK activation by UO126 enhanced M-CSF-evoked AKT activation in wild type (WT) BMMs. BMMs were starved, pretreated with UO126 (10µM) and stimulated with M-CSF (30ng/ml) for the indicated times (+) or left unstimulated (-). Cell lysates were resolved by SDS-PAGE and immunoblotted with the indicated antibodies. Data shown (A-C) are a representative of the three (n=3) independent experiments.