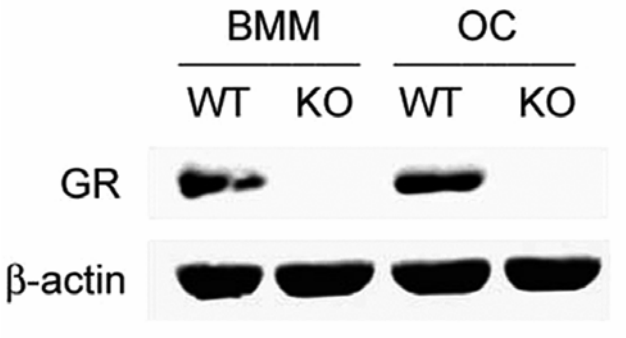
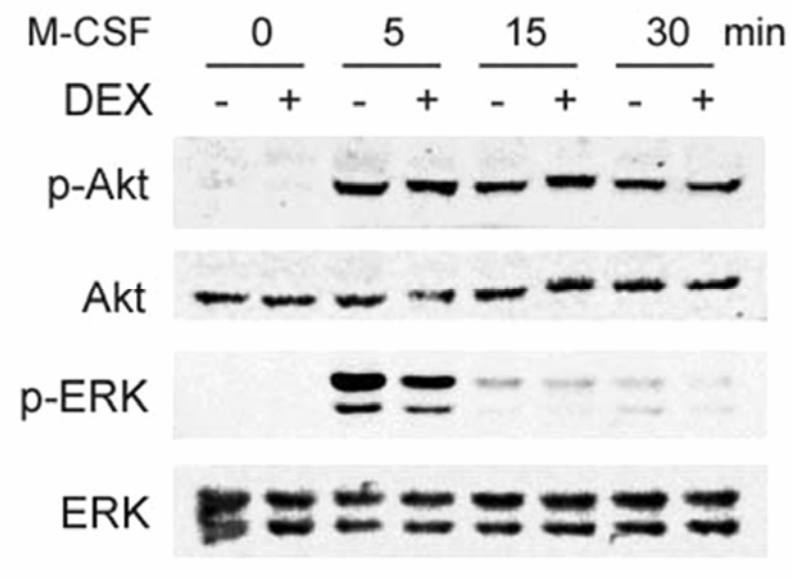
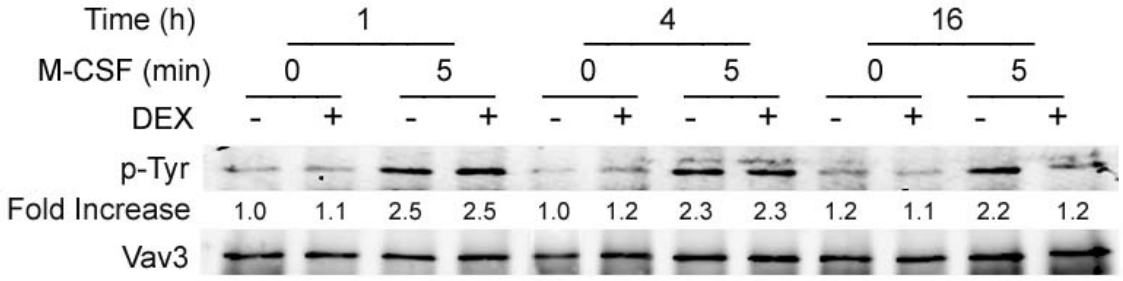
Supplemental Figure 1



Supplemental Figure 2



Supplemental Figure 3.



Supplemental Figure 1. Absence of GR protein in $GR^{OC-/-}$ BMMs and osteoclasts. Total cell lysates of BMMs and mature osteoclasts (OCs) WT and $GR^{OC-/-}$ (KO) mice were analyzed for GR expression by Western blotting. β-actin serves as protein loading control.

Supplemental Figure 2. DEX does not alter M-CSF-induced Akt and ERK activation. WT BMMs were cultured with M-CSF and RANKL for three days and exposed to DEX (100 nM) or carrier for 16 hrs. Cells were stimulated with M-CSF (50 ng/ml) with time. Activation of Akt and ERK was evaluated by Western blotting using phospho-specific antibodies for Akt and p42/p44 MAPK.

Supplemental Figure 3. DEX suppression of M-CSF-activated Vav3 requires 16 hrs exposure to the steroid. WT BMMs, maintained in M-CSF and RANKL for 3 days, were exposed to DEX (100 nM) or carrier with time, and then stimulated with M-CSF (100 ng/ml) for 5 min. Vav3 was immunoprecipitated from the cell lysate and immunoblotted for phosphotyrosine or Vav3 content. The blot was subject to quantitative densitometry. The numbers represent quantity of tyrosine phosphorylated Vav3 relative to first band (1.0).