quantitation of the percentage of bone surface covered by osteoclasts in ankles of each group of mice (*p<0.001 as compared to serum (+)). (E) Circulating tartrate-resistant acid phosphatase 5b (TRACP 5b) levels (*p<0.001 compared to serum (+), #p<0.001 compared to serum (-)). (F). Representative TRAP stained histological sections of proximal tibia of each group of mice. TRAP reaction product identifying osteoclasts is red (n=4 mice/group).

Figure 10. Anti-c-Fms mAb blocks TNF-α-induced osteoclastogenesis and bone resorption, in vivo (A) Histological sections of calvariae, excised from mice after 5 daily supra-calvarial injections of TNF-α with or without intraperitoneal injection of anti-c-Fms mAb were stained for TRAP activity (red reaction product). (B) The percentage of bone/marrow interface covered by osteoclasts was histomorphometrically determined (*p<0.001 as compared to TNF-α alone, #p<0.001 compared to control). (C) Circulating TRACP 5b levels were determined by ELISA (*p<0.001 compared to TNF-α alone, #p<0.001 compared to control). (D) Abundance of CD11b positive osteoclast precursors in marrow recovered from TNF-injected mice with (red line) or without (green line) anti-c-Fms mAb. The black line represents FITC-conjugated isotype mAb. The illustration is representative of four separate experiments. (n=4 in all groups).

Supplemental Figure 1. Chimeric mice selectively express TNFRs on osteoclast precursors. WT>WT, WT>KO, KO>WT and KO>KO bone marrow cells were cultured with M-CSF for 3 days. The resultant BMMs were incubated with purified anti-TNFR1 mAb or PE-conjugated anti-TNFR2 hamster mAb. The cells exposed to anti-TNFR1 mAb were further incubated with FITC-conjugated anti-hamster IgG. TNFR expression was determined by FACS. TNFR1 black lines represent cells incubated with purified hamster IgG and FITC-conjugated anti-hamster IgG. TNFR2 black lines represent cells incubated with PE hamster IgG.