**Supplementary Figures**

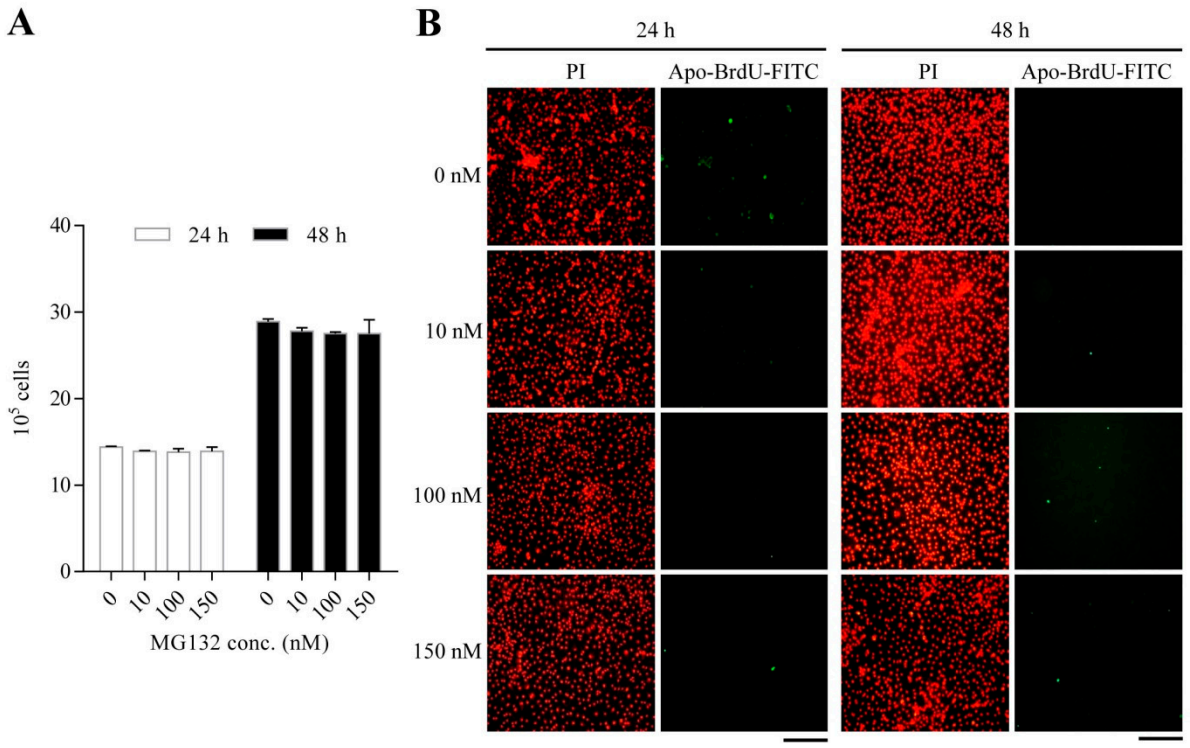


**Figure S1**. Effects of MG132 on cell proliferation and apoptotic cell death. Osteoclast progenitors were treated with the indicated concentrations of MG132 for 24 h or 48 h in the presence of M-CSF (30 ng/ml). (**A**) Cell proliferation was determined by counting the number of cells with a haemocytometer after trypan blue staining. Quantitative data are presented as means ± SD (n = 3) of three independent experiments. (**B**) Apoptotic cell death (shown in green) was identified by DNA breaks using an Apo-BrdU In Situ DNA Fragmentation Assay Kit. Red indicates propidium iodide (PI)-stained cells. Images are representative of three independent experiments. Scale bar, 100 μm.

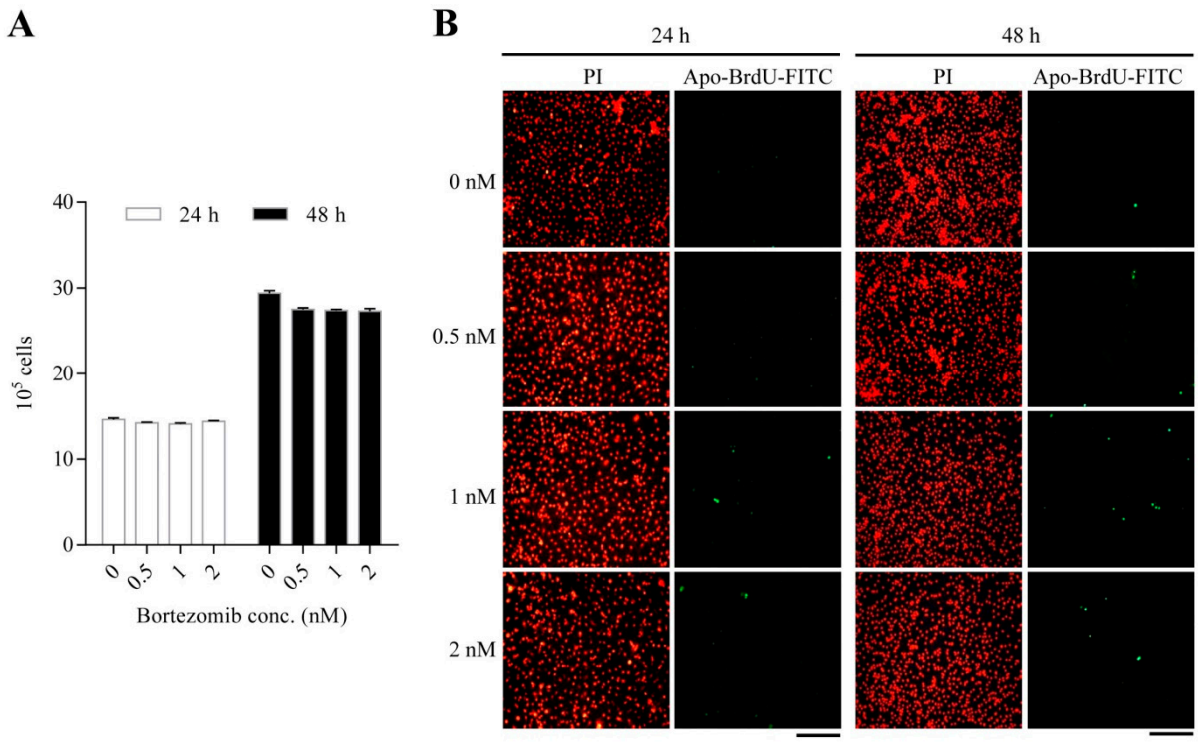


**Figure S2.** Effects of bortezomib on cell proliferation and apoptosis. After osteoclast progenitors were treated with the indicated concentrations of bortezomib for 24 h or 48 h in the presence of M-CSF (30 ng/ml), cell proliferation (**A**) and apoptosis (**B**, green color) were identified as described in Fig. S1. Red indicates propidium iodide (PI)-stained cells. Quantitative data are presented as means ± SD (n = 3) of three independent experiments. Images are representative of three independent experiments. Scale bar, 100 μm.

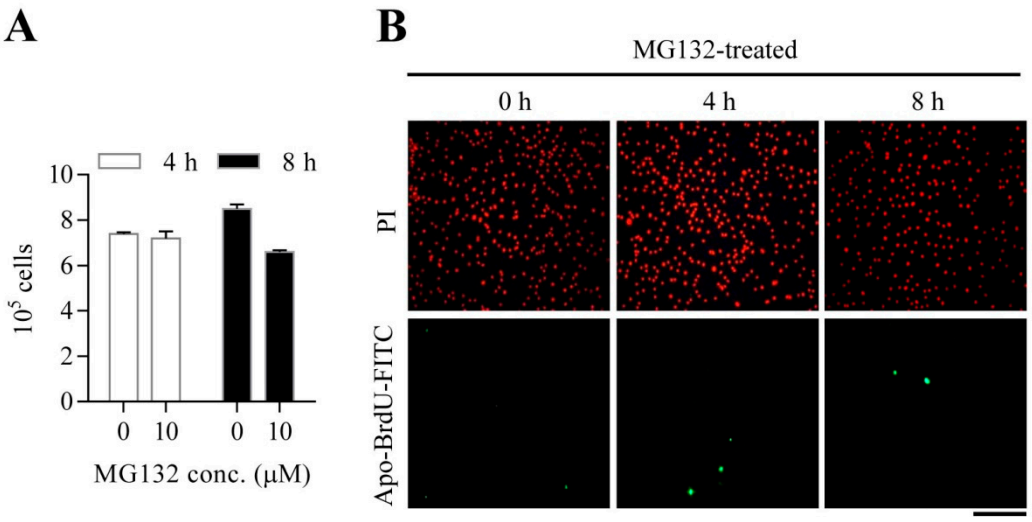


**Figure S3.** Effects of MG132 (10 μM) on cell proliferation and apoptosis. After osteoclast progenitors were treated with MG132 (10 μM) for 4 h or 8 h in the presence of M-CSF (30 ng/ml), cell proliferation (**A**) and apoptosis (**B**, green) were assessed as in Fig. S1. Red represents propidium iodide (PI)-stained cells. Quantitative data are presented as means ± SD (n = 3) of three independent experiments. Images are representative of three independent experiments. Scale bar, 100 μm.

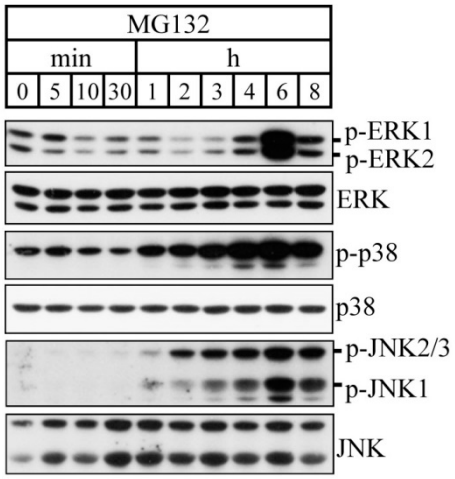


**Figure S4.** MAPKs (ERK, JNK, and p38) activation by MG132. Osteoclast progenitors starved of M-CSF for 4 h were cultured with MG132 (10 μM) for the indicated times, and whole cell lysates were subjected to immunoblot analysis.

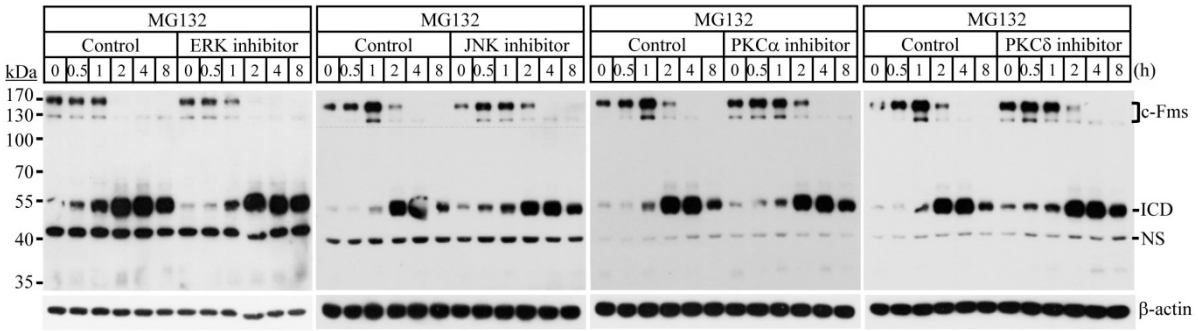


**Figure S5.** MG132-induced c-Fms degradation is independent of ERK, JNK, PKCα, and PKCδ signalling. Osteoclast progenitors were incubated with PD98059 (10 μM; a specific inhibitor of ERK), SP600125 (20 μM; a specific inhibitor of JNK), Gö6976 (1 μM; a specific inhibitor of PKCα), and rottlerin (1 μM; a specific inhibitor of PKCδ) for 30 min. Cells were then treated with MG132 (10 μM) for the indicated times. Total cell lysates were subjected to immunoblotting with antibodies against c-Fms or β-actin (loading control). ICD, intracellular domain of c-Fms; NS, nonspecific bands. Data are representative of three independent experiments.