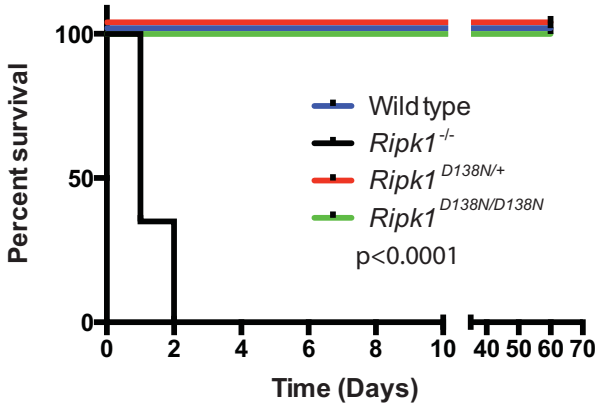
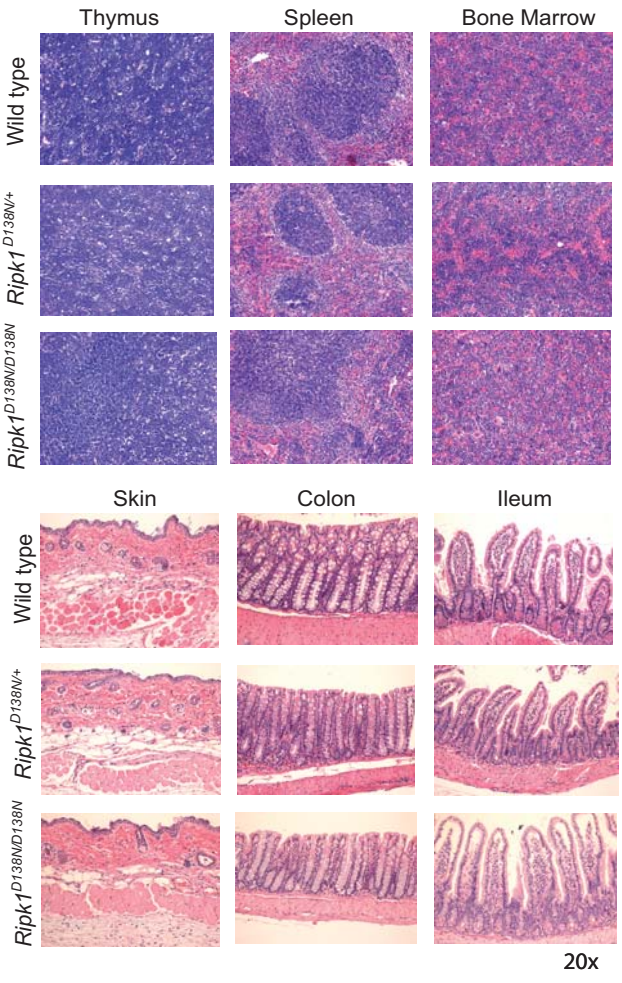
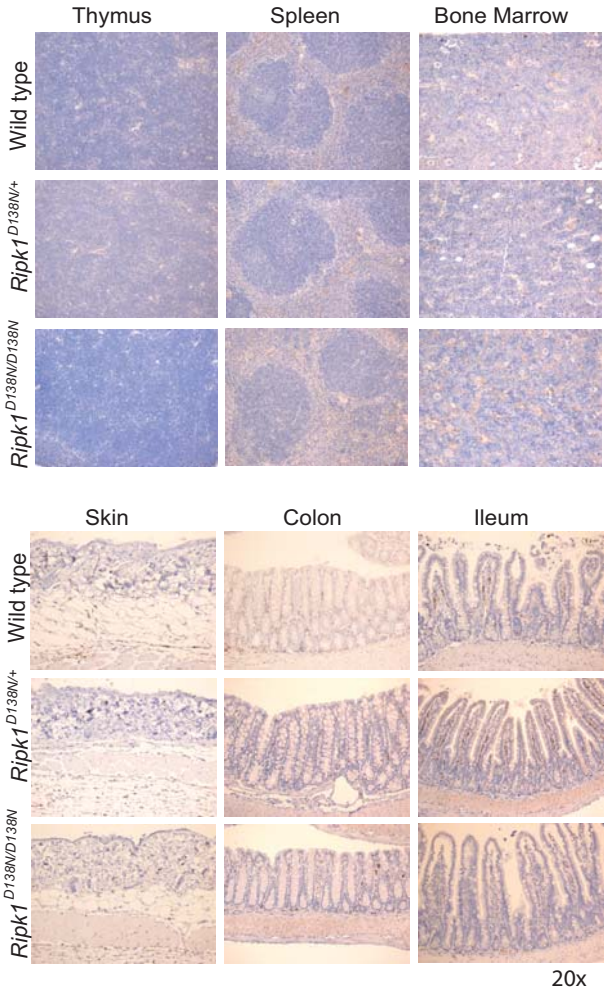
A



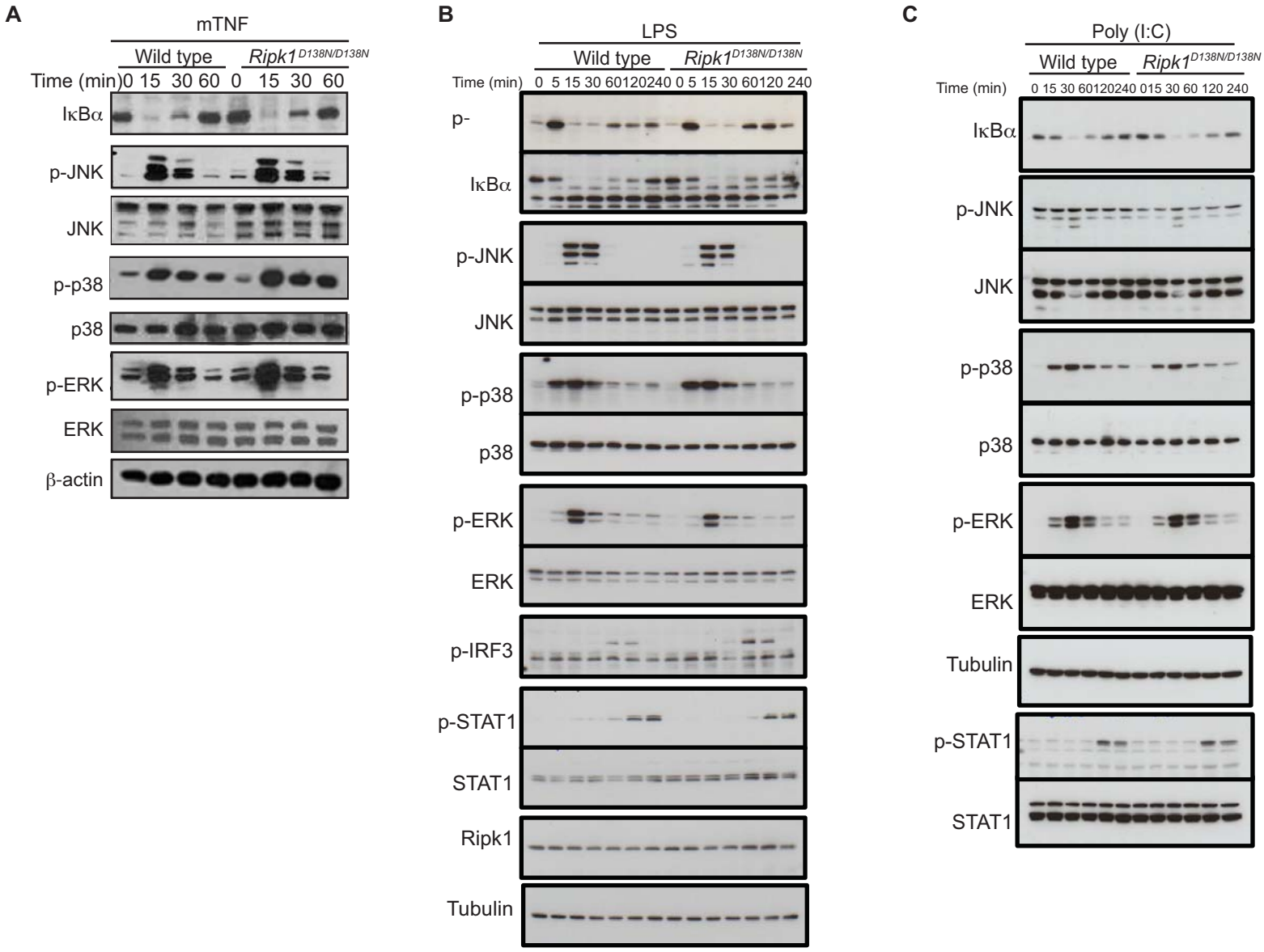
B



C



Supplemental Figure 1. A. Survival of mice from intercrosses of *Ripk1*$^{D138N/+}$ or *Ripk1*$^{+/-}$ animals. Survival of RIPK1 knockout mice was compared to Wild type, *Ripk1*$^{D138N/+}$ or *Ripk1*$^{D138N/D138N}$ mice. p<0.0001. **B and C. Histological examination of tissues from Wild type, *Ripk1*$^{D138N/+}$ and *Ripk1*$^{D138N/D138N}$ mice.** Thymus, spleen, bone marrow, colon, ileum and skin from Wild type, *Ripk1*$^{D138N/+}$ and *Ripk1*$^{D138N/D138N}$ mice were fixed in 10% formalin and stained with hematoxylin and eosin (B) or cleaved caspase 3 antibody (C).



Supplemental Figure 2. The kinase activity of Ripk1 is not essential for TNF- or TRIF-dependent activation of NF-κB or MAP kinase signaling. A. Wild type and *Ripk1*$^{D138N/D138N}$ mouse embryonic fibroblasts were left untreated or were treated with 10ng/ml mTNF for the time periods indicated and protein lysates were probed with phospho-specific antibodies for JNK, p38 MAPK and ERK. Lysates were also probed with antibodies specific for total IκBα, JNK1/2, p38α and ERK. **B and C.** Wild type and *Ripk1*$^{D138N/D138N}$ BMDM were left untreated or stimulated with LPS (100ng/ml) (B) or poly (I:C) (25μg/ml) (C) for the time periods indicated and lysates probed with antibodies to phospho-IκBα, -p38 MAPK, -JNK, -ERK, -IRF3 or -STAT1. Lysates were also probed with antibodies specific for total IκBα, JNK1/2, p38α, ERK or STAT1.