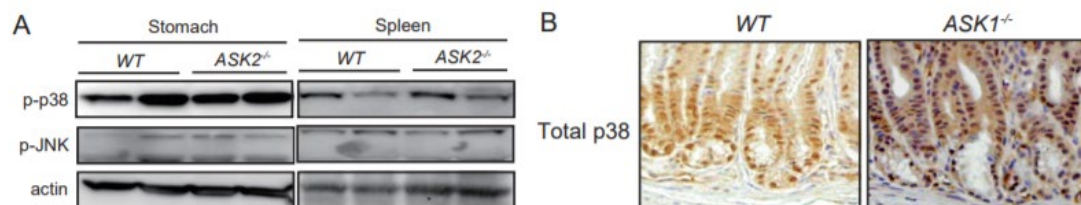
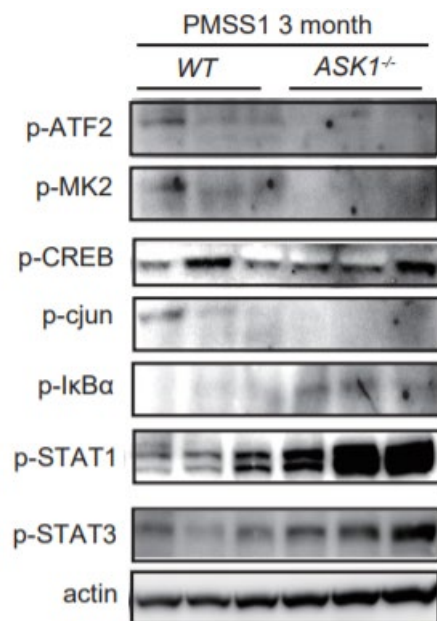


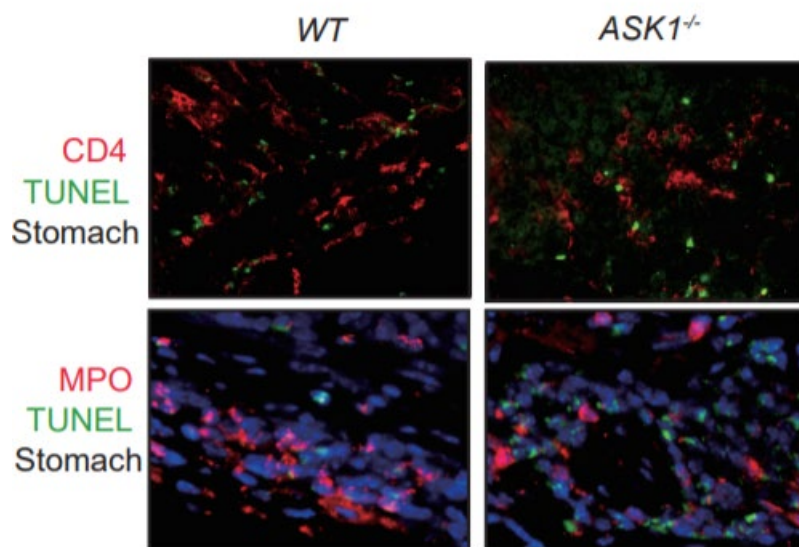
Supplementary Figure S1. PMSS-1 infection experiments. **(A)** Representative H&E staining of WT and ASK1^{-/-} mouse stomach with *H. pylori* PMSS-1 infection. Original magnification, ×100. **(B)** Histological scoring of PMSS-1-infected WT and ASK1^{-/-} mouse stomach. Inf., inflammation; atr., atrophy; met., metaplasia; hyp., hyperplasia. Data are shown as mean ± SD. **P* < 0.05 compared to infected WT mice. **(C)** Colony-forming units of *H. pylori* isolated from the infected WT and ASK1^{-/-} mouse stomach.



Supplementary Figure S2. ASK1, not ASK2, is responsible for downstream MAPK activation in the stomach. **(A)** Immunoblotting of WT and ASK2^{-/-} mouse stomach and spleen infected with SS-1. **(B)** Total p38 staining in SS-1-infected WT and ASK1^{-/-} stomach. Original magnification, ×200.



Supplementary Figure S3. ASK1 deficiency induced NF-κB and STAT1 activation in the PMSS-1 infected stomach. Immunoblotting of PMSS-1-infected WT and ASK1^{-/-} mouse stomachs.



Supplementary Figure S4. ASK1 deficiency did not promote apoptosis in T cells and neutrophils. Immunofluorescent staining combined with TUNEL assay in SS-1-infected WT and ASK1^{-/-} stomach and spleen. Original magnification, ×200.