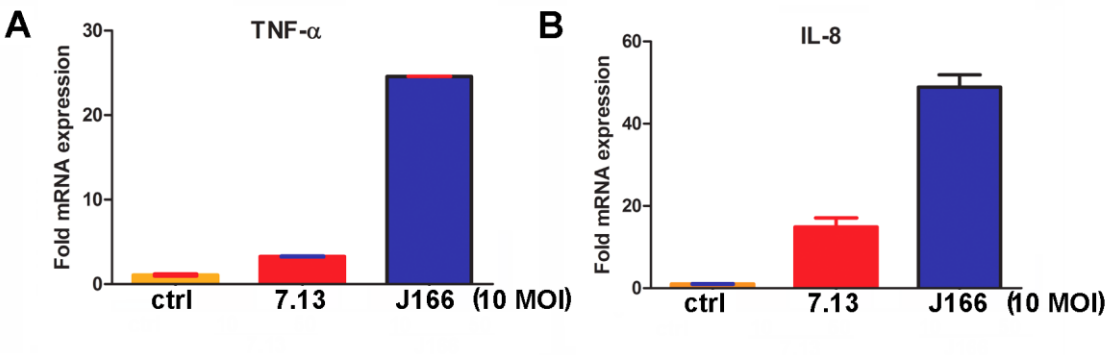
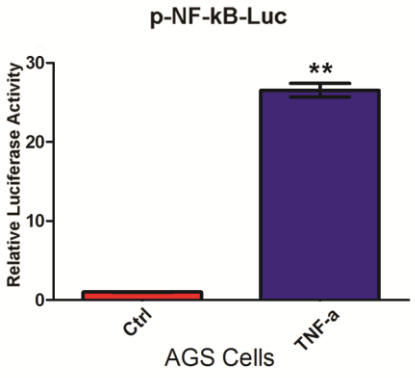
Supplemental Figure 1. H. pylori infection induces TNF-α and IL-8 expression



A-B) The qRT-PCR analysis of TNF-α and IL-8 was performed in AGS cells with H. pylori infection.

Supplemental figure 01, Zhu et al.

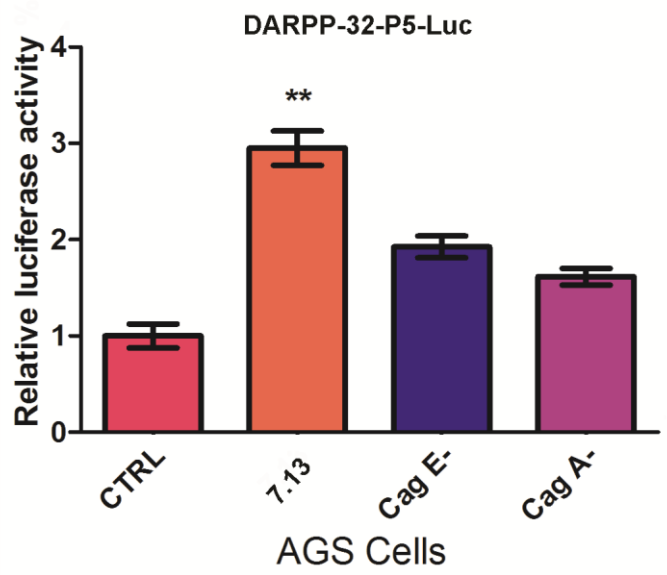
Supplemental Figure 2. TNF-α activates NF-κB



Luciferase reporter assay for p-NF-κB-luc in AGS cells with TNF-α treatment.

Supplemental figure 02, zhu et al.

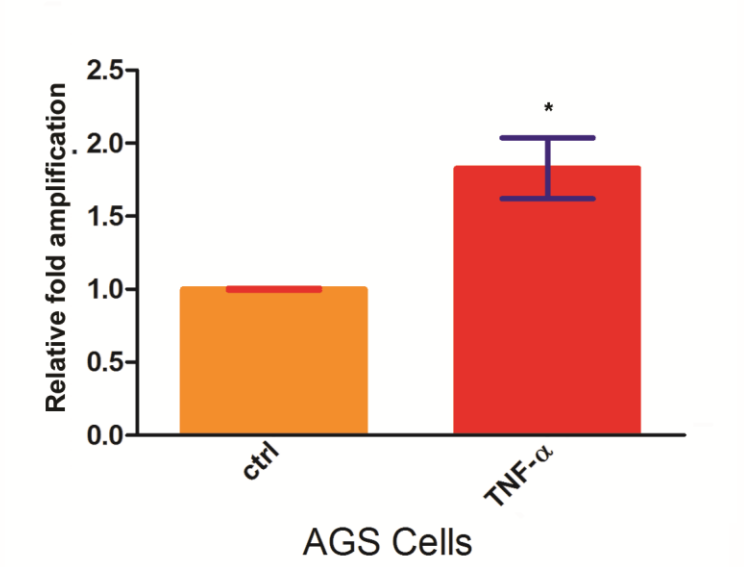
Supplemental Figure 3. H. pylori infection regulates DARPP-32 promoter activity



Luciferase reporter assay for DARPP-32-P5-luc in AGS cells co-cultured with H. pylori 7.13 strains (wildtype, cagA− and cagE− mutants).

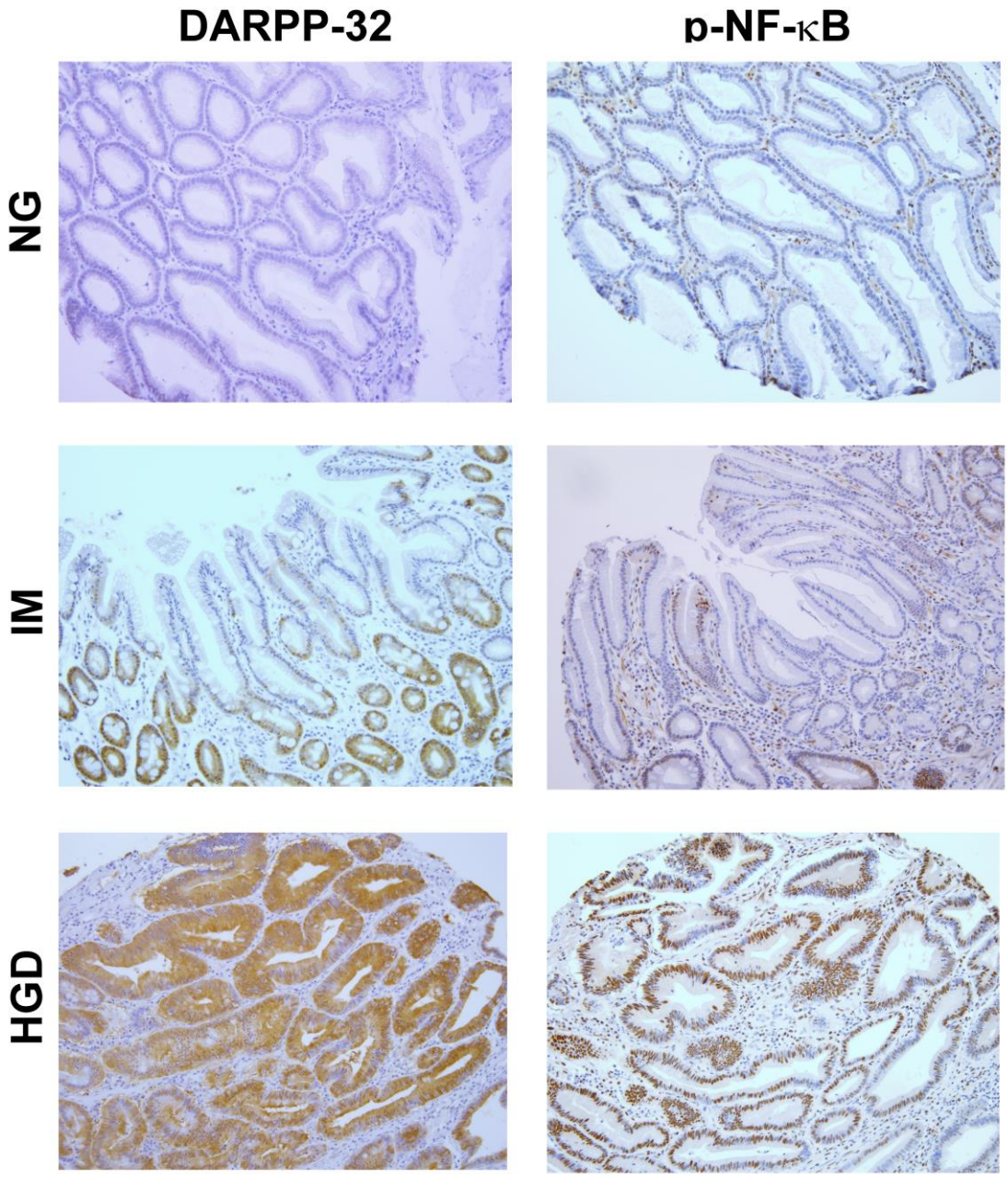
Supplemental figure 03, zhu et al.

Supplemental Figure 4. TNF-α enhances NF-κB binding to DARPP-32 promoter



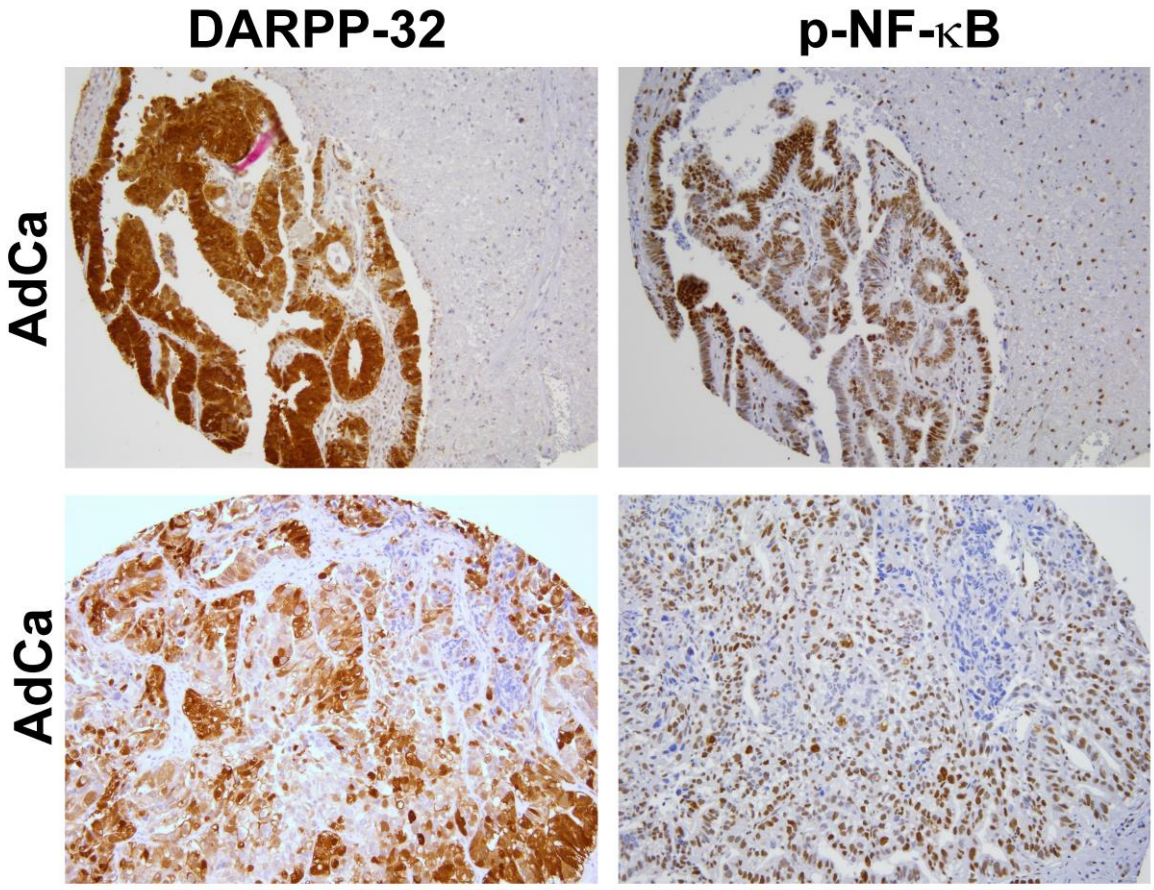
ChIP assay using a specific antibody against NF-kB-p65 for immunoprecipitation of formaldehyde-fixed chromatin in AGS cells with control or TNF-a treatment. qRT-PCR was performed using primers designed to amplify the NF-kB-p65 binding site on DARPP-32 promoter region.

Supplemental figure 04, zhu et al.



A) Immunohistochemical staining of DARPP-32 and NF-kB in serial tissue samples from human gastric mucosa with normal histology (NG), intestinal metaplasia (IM), and high-grade-dysplasia (HGD).

Supplemental figure 05, zhu et al.



B) Immunohistochemical staining of DARPP-32 and NF-kB in serial tissue sections from human gastric mucosa showing intestinal-type (upper panels) and diffuse-type (lower panels) adenocarcinomas.

Supplemental figure 05, zhu et al.