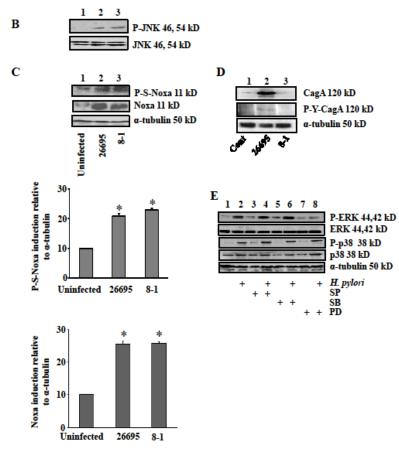
MPGKKARKNAQPSPARAPAELEVECATQLRRFGDKLNFRQKLLNLISKLFCSGT



Supplemental Fig. S2A. Amino acid sequence of human Noxa. Noxa has five residues which can be phosphorylated (S and T, boldfaced and in italics). But, only  $S^{13}$  appears to be a potential kinase-target as predicted by GPS2.1 program.

Supplemental Fig. S2B. JNK and its active form P-JNK were equally induced by both 26695 and 8-1 strains. Western blot analysis of whole cell lysates prepared from AGS cells infected with 200 MOI of H. pylori cag PAI(+) strain (26695) and cag PAI negative strain (8-1) or left uninfected for 5h showing equal expression of JNK and P-JNK. α–tubulin was used as a loading control.

**Supplemental Fig. S2C.** Noxa and P-S-Noxa induction by 26695 and 8-1 strains. Western blot analysis of whole cell lysates prepared from AGS cells infected with 200 MOI of *H. pylori cag* PAI(+) strain (26695) and *cag* PAI negative strain (8-1) or left uninfected for 5h showing equal expression of Noxa and P-S-Noxa.  $\alpha$ -tubulin was used as a loading control. Bars depict P-S-Noxa and Noxa expression normalized to  $\alpha$ -tubulin (mean ± SEM, n = 4), \**P*<0.05 compared with uninfected cells.

**Supplemental Fig. S2D.** CagA and its phosphorylated form are only induced in *H. pylori* 26695 infected AGS cells. Western blot analysis of whole cell lysates showed CagA expression and its phosphorylation in 26695-infected cells but not in cells infected with strain 8-1.

**Supplemental Fig. S2E.** Expression status of P-ERK, P-p38, total ERK and p38 in AGS cells infected (5 h) with 200 MOI of *H. pylori*. Pretreatment with 25  $\mu$ M JNK inhibitor II (SP600125), 25  $\mu$ M p38 MAPK inhibitor (SB203580) and MEK 1/2 inhibitor (PD98039) were done as indicated. This data showed that JNK inhibition by SP had no effect on ERK and p38 MAPK phosphorylation. SB partially inhibited p38 MAPK phosphorylation and PD inhibited ERK phosphorylation.

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