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Research Article

Helicobacter Pylori Infection Triggers PERK-Associated Survivin Loss in Gastric Tissue Samples and Cell Lines

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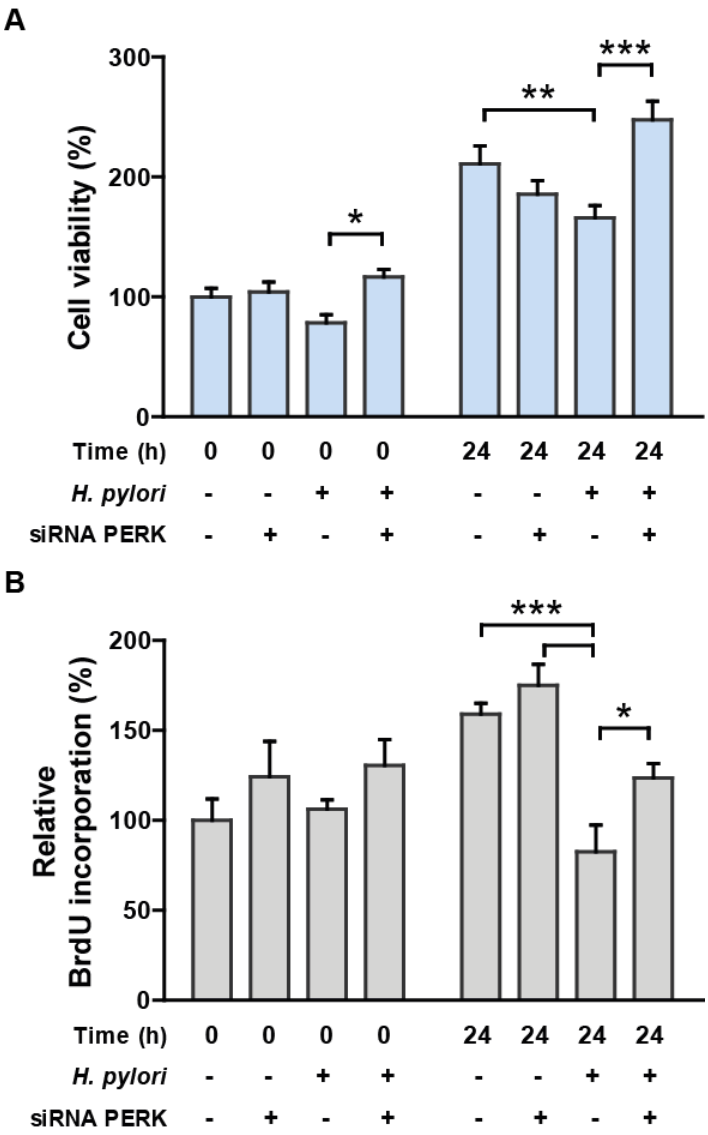
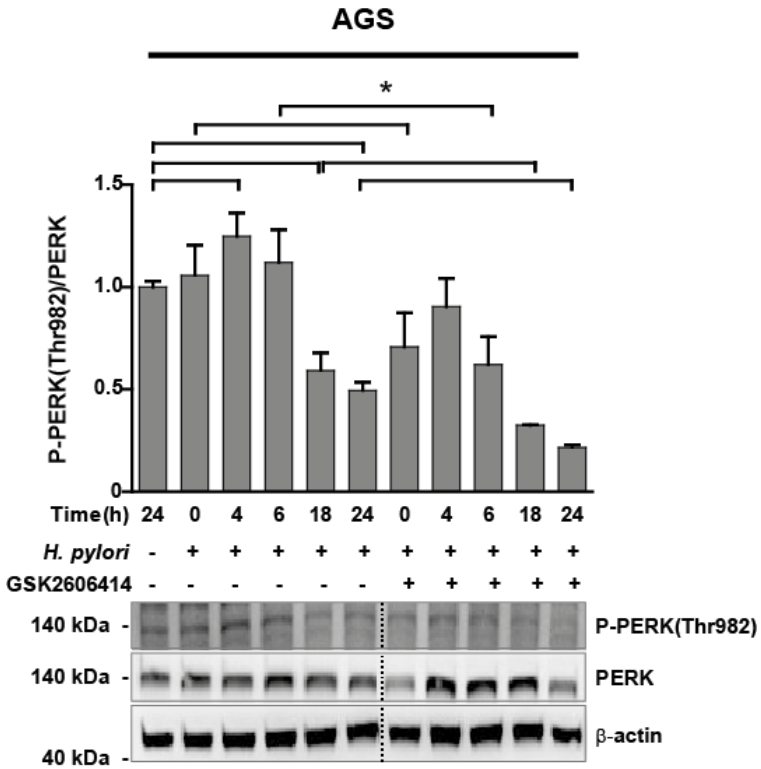
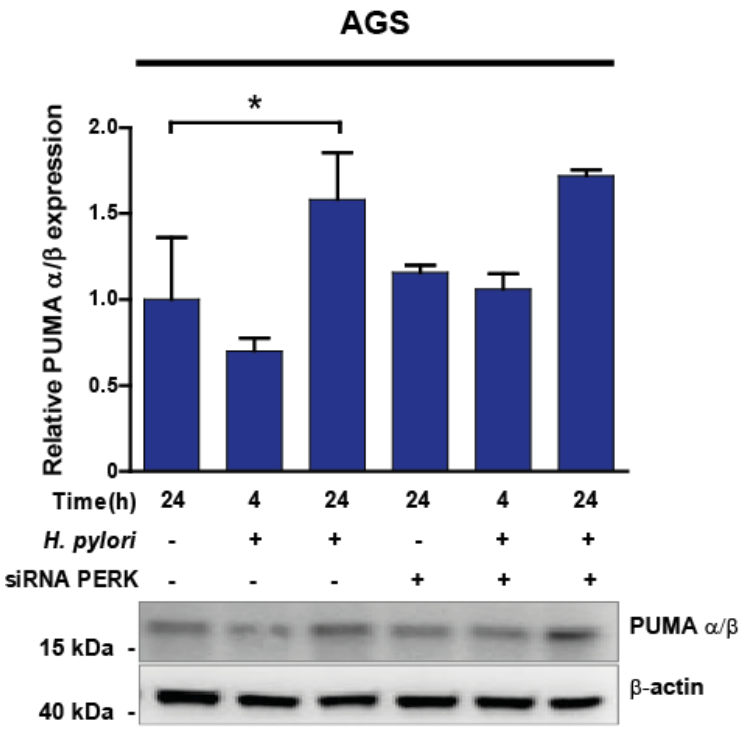


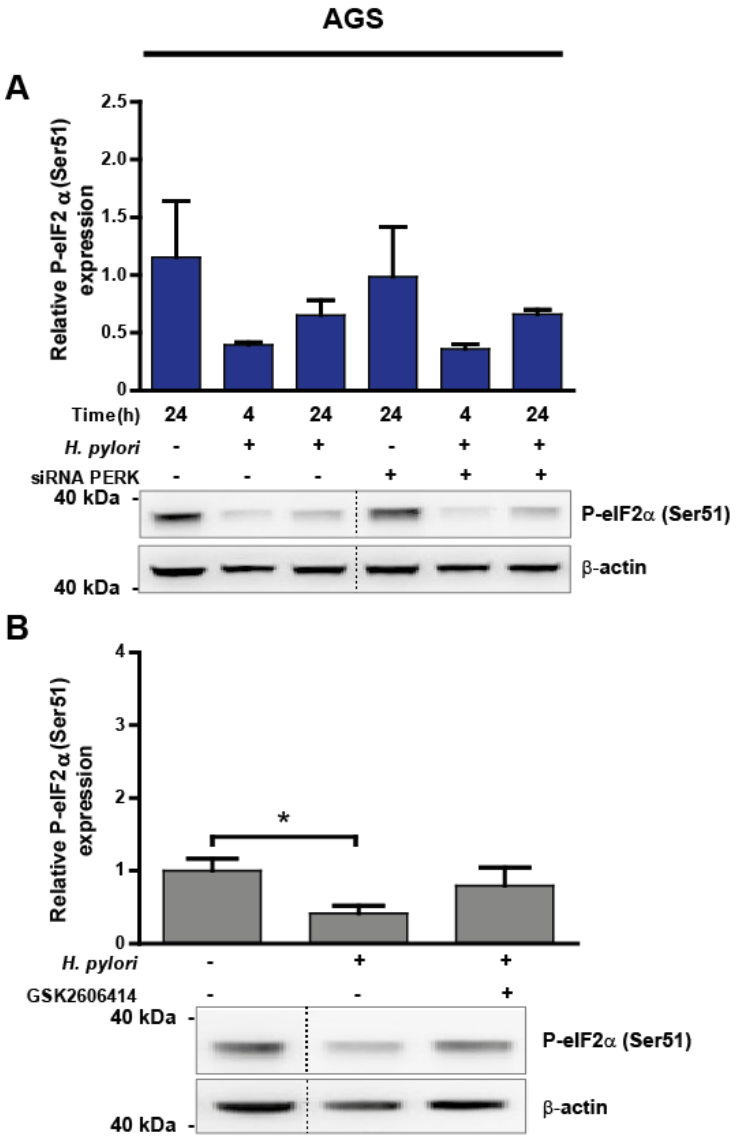
Suppl. Figure 1: (A) Viability in uninfected (*H. pylori* -) and *Hp*-infected (*H. pylori* +) AGS cells. Viability was measured using the MTS assay after 24 h *Hp* infection. PERK silencing (siRNA PERK) was achieved by transfecting AGS cells with 100 nM PERK siRNA 16 h before *Hp* infection. Non-specific siRNA (100 nM) was used as control (-). n=5, *p<0.05, **p<0.01, ***p<0.001; mean + SEM; (B) Proliferation in uninfected (*H. pylori* -) and *Hp*-infected (*H. pylori* +) AGS cells. Proliferation was detected 24 h after *Hp* infection by measuring BrdU incorporation using a colorimetric BrdU assay. AGS cells were transfected with 100 nM PERK siRNA (siRNA PERK) 16 h prior *Hp* infection. Non-specific siRNA (100 nM) was used as control (-). n=3, *p<0.05, ***p<0.001; mean + SEM.



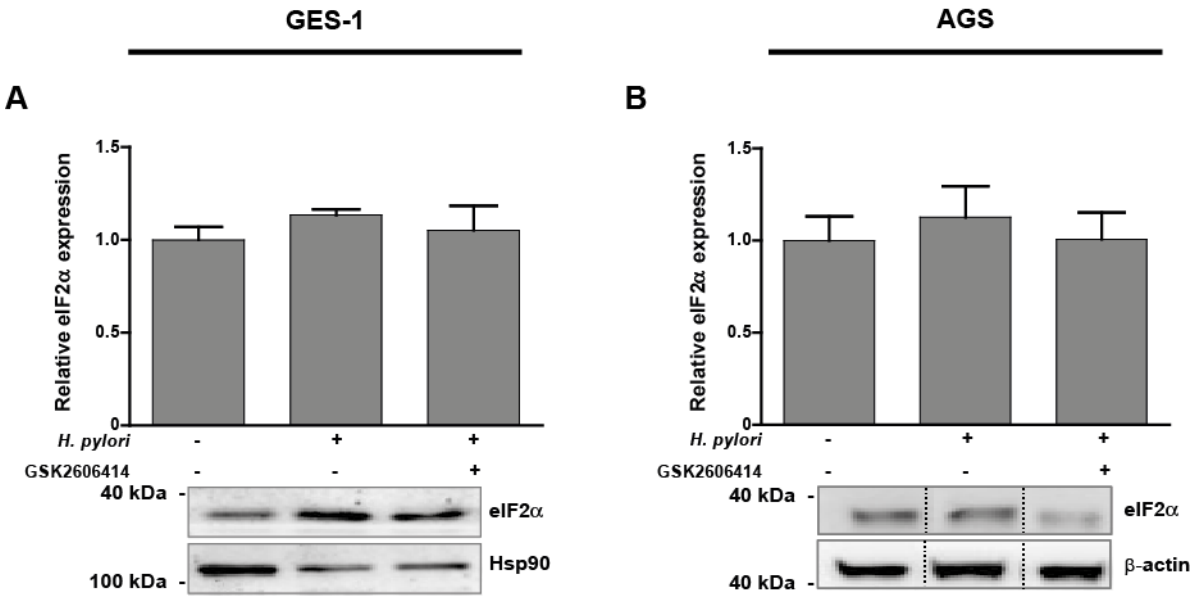
Suppl. Figure 2: Time course of P-PERK(Thr982) protein levels in *Hp* infected (*H. pylori* +) AGS cells at 0, 4, 6, 16 and 24 h. In parallel, uninfected control cells were kept in medium for 24 h (*H. pylori* -). Representative immunoblots are shown together with the corresponding P-PERK(Thr982)/PERK ratios, whereby P-PERK(Thr982) and PERK were normalised to β-actin abundance. AGS cells were pre-incubated for 1 h with 25 μM GSK2606414 before *Hp* infection. n=2, *p<0.05, mean + SEM. Dotted lines indicate where lanes, from the same immunoblot, have been removed for clarity.



Suppl. Figure 3: PUMA α/β protein abundance in uninfected (*H. pylori* -) and *Hp*-treated (*H. pylori*) AGS cells after 4 and 24 h. Representative immunoblots with corresponding graphic quantifications showing mean relative abundance of PUMA α/β protein (normalised to β-actin abundance). Gastric cells were infected with *Hp* for 4 and 24 h; in parallel, uninfected control cells were kept in medium for 24 h. PERK silencing (siRNA PERK) was achieved by transfecting AGS cells with 100 nM PERK siRNA 16 h before *Hp* infection. Non-specific siRNA (100 nM) was used as control (-). n=2, **p*<0.05, mean + SEM.



Suppl. Figure 4: (A) eIF2α protein activation, measured as P-eIF2α(Ser51), in uninfected (*H. pylori* -) and *Hp*-treated (*H. pylori* +) AGS cells after 4 and 24 h. Representative immunoblots with corresponding graphic quantification showing mean relative protein abundance of P-eIF2α(Ser51) (normalised to β-actin abundance). Gastric cells were infected with *Hp* for 4 and 24 h; in parallel, uninfected control cells were kept in medium for 24 h. PERK silencing (siRNA PERK) was achieved by transfecting AGS cells with 100 nM PERK siRNA 16 h before *Hp* infection. Non-specific siRNA (100 nM) was used as control (-). n=3; mean + SEM. (B) P-eIF2α(Ser51) abundance in uninfected (*H. pylori* -) and *Hp*-treated (*H. pylori* +) AGS cells after 24 h. Representative immunoblots with corresponding graphic quantification showing mean relative protein abundance of P-eIF2α(Ser51) (normalised to β-actin abundance). AGS cells were pre-incubated with 25 μM GSK2606414, a selective PERK inhibitor, 1 h before *Hp* infection. n=3, **p*<0.05; mean + SEM. Dotted lines indicate where lanes, from the same immunoblot, have been removed for clarity.



Suppl. Figure 5: (A-B) eIF2α protein abundance in uninfected (*H. pylori* -) and *Hp*-infected (*H. pylori* +) GES-1 (A) and AGS (B) cells after 24 h. Representative immunoblots are shown together with the corresponding quantification of eIF2α protein normalised to Hsp90 (A) or β-actin (B) abundance. GES-1 and AGS cells were pre-incubated for 1 h with 25 μM GSK2606414, a selective PERK inhibitor, before *Hp* infection. (A) n=2; mean + SEM. (B) n=2; mean + SEM. Dotted lines indicate where lanes, from the same immunoblot, have been removed for clarity.