



**Supplementary Materials:** Supplementary Figure S1 and Supplementary Figure S2 and their corresponding figure legends.

## **Supplementary Figure S1:**



Figure S1. *H. pylori* virulence factor CagA is implicated in TAZ overexpression and EMT. (A) Phase-contrast images of gastric epithelial cells during a time-course experiment of 2, 5 and 24 h after *H. pylori* 7.13 WT strain infection. Percentages indicated at the bottom right in each image correspond to the number of cells exhibiting a mesenchymal phenotype. (**B-E**) Investigation of the role of CagA and the T4SS in *H. pylori*-induced TAZ overexpression. Co-culture of MKN45 cells, 7.13 and P12 *H. pylori* strains either WT or invalidated for CagA ( $\Delta cagA$ ) or the T4SS ( $\Delta cagE$  and  $\Delta cagPAI$ ) after 24 h of infection. (**B**) Expression of TAZ evaluated by Western blot. (**C**) CYR61 relative mRNA expression assessed by RTqPCR. (**D**) Relative mRNA expression of Vimentin, ZEB1 and SNAI1 assessed by RTqPCR. (**E**) Percentage of cells with the mesenchymal "hummingbird" phenotype in NCI-N87 cells (**a**) and in MKN45 cells (**b**). (**c**) Phase-contrast images of MKN45 infected with the different *H. pylori* strains. Percentages at the bottom right of each image correspond to the number of cells with a mesenchymal phenotype. (**A**, **E**) Scale bars, 10 µm. (**B-E**) Bars represent means ± SEM, n=3, \**p*<0.05 *vs* uninfected control "-". # *vs* respective WT *H. pylori* strain. (**C-D**) Kruskal-Wallis with Dunn's posttest. (**E**) ANOVA test.

## **Supplementary Figure S2:**



Figure S2. Effect of TAZ silencing by siRNA on TAZ expression, TEAD-luciferase activity, ZEB1 expression, invasive and tumourigenic cells properties. (A) Validation of TAZ knock-down after siRNA silencing and *H. pylori* infection or not in MKN45, GC07 and NCI-N87 cells assessed by Western blot. Number represents the quantification of the relative TAZ protein expression compared to uninfected siCtrl cells and normalised to GAPDH or  $\alpha$ -tubulin protein expression. (B) Validation

of siTAZ silencing efficiency assessed by relative TEAD-luciferase reporter activity performed in MKN45 cells. Bars represent means  $\pm$  SEM of fold changes relative to uninfected cells. n=4, \$ *vs* uninfected siCtrl and \* *vs H. pylori*-infected siCtrl. Kruskal-Wallis test with Dunn's post-test. **(C)** Immunofluorescence of ZEB1 (in green), actin cytoskeleton stained by phalloidin (in red) and nuclei stained with DAPI (in blue) performed in MKN45 and GC07 siTAZ-transfected cells. Scale bars, 10 µm. (D) Representative images illustrating invading cells of Figure 4D. MKN45 and GC07 cells were transfected with siCtrl or 3 different siTAZ and infected with *H. pylori* for 24 h. DAPI positive staining of invasive cells was counted 18 h after seeding on type I collagen coated inserts. Scale bars, 100 µm. (E) Representative images illustrating NCI-N87 tumorspheres of Figure 4F. NCI-N87 cells were seeded in non-adherent conditions during 7 days after being transfected with siCtrl or 3 different siTAZ and infected bers, 100 µm.



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