**Supplementary Inforamtion**

The supplementary information file consists of five supplementary figures.

**Supplementary Figure**

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**Figure S1.** Expression and distribution of LAMP2 in AGS cells treated with *LAMP1* siRNA.(**A**) AGS cells were transfected with control, *LAMP1* siRNA-1, or *LAMP1* siRNA-2, infected with *H. pylori* for 5 h (multiplicity of infection [MOI] 50) and incubated in a medium containing antibiotic for 24 h. Then, LAMP2 and phalloidin staining were performed. Nuclei (blue) were stained with DAPI. Scale bar: 20 μm. (**B**) AGS cells, which had been transfected with pTet-On and TRE2hyg-mCherry-EGFP-LC3 plasmids, were transfected with control, *LAMP1* siRNA-1, or *LAMP1* siRNA-2, infected with *H. pylori* for 5 h (MOI 50), and incubated in a medium containing antibiotic for 24 h, and then immunostained for LAMP2. Scale bar: 20 μm.



**Figure S2.** Confirmation of interaction of GTF2I and CAPZA1 with LRP1-ICD by western blot analysis of the immunoprecipitates.(**A**) AGS cells were treated with 200 nM leptomycin B overnight, infected with *H. pylori* for 5 h (MOI 50), and incubated in a medium containing antibiotic for the indicated times. Then, subcellular fractionation of these cells indicated the localization of LRP1-ICD in cytoplasmic (Cyt) and nuclear (Nuc) extracts. (**B and C**) AGS cells were infected with *H. pylori* for 5 h (MOI 50) and incubated in a medium containing antibiotic for 0 and 24 h. An immunoprecipitation assay was performed on these cells with an anti-LRP1-carboxyterminal end antibody. GTF2I (**B**) and CAPZA1 (**C**) were detected. (**D**) AGS cells were infected with *H. pylori* for 5 h (MOI 50) and incubated in a medium containing antibiotic for 0 and 24 h. After subcellular fractionation of these cells, an immunoprecipitation assay was performed with an anti-LRP1-carboxyterminal end antibody. The localization of GTF2I was detected in cell membrane (Mem), cytoplasmic (Cyt) and nuclear (Nuc) extracts.

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**Figure S3.** Effect of CAPZA1 overexpression on cytoplasm-nuclear transport of LRP1-ICD.AGS cells were transfected with pCMV-control or pCMV-*CAPZA1* plasmids, infected with *H. pylori* for 5 h (MOI 50), and incubated in a medium containing antibiotic for the indicated times. Then, subcellular fractionation of these cells indicated the localization of LRP1-ICD in cytoplasmic (Cyt) and nuclear (Nuc) extracts.

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**Figure S4.** Staining intensity of CAPZA1 in CagA-staining cells and cell invasion ability of CAPZA1-overexpressing AGS cells.(**A**)The staining intensity of CagA-positive cells (red arrowhead) and their adjacent CagA-negative cells (white arrowhead) were quantified by using the ImageJ program. Data are presented as the mean ± SD of 5 cells. \**P* < 0.05. Scale bar: 20 μm. (**B**) AGS cells were transfected with pCMV-control or pCMV-*CAPZA1* plasmids and infected with *H. pylori* for 5 h (MOI 50). The cells were added to the top chambers of 24-well Trans-well plates, which were either coated with Matrigel® barriers or uncoated. After 24-h incubation, invaded cells were stained with hematoxylin and eosin. Scale bar: 20 μm. (**C**) The numbers of invading cells were counted and fractions of invaded cells were calculated. Data are presented as the mean ± SD of 3 independent assays. \*\**P* < 0.01, NS, not significant.



**Figure S5.** HDAC activity in AGS cells treated with SAHA or trichostatin A. AGS cells were incubated with SAHA or trichostatin A for 24 h, and HDAC activities were measured. Data are presented as the mean ± SD of 3 independent assays. \*\**P* < 0.01.