Supplementary Figures

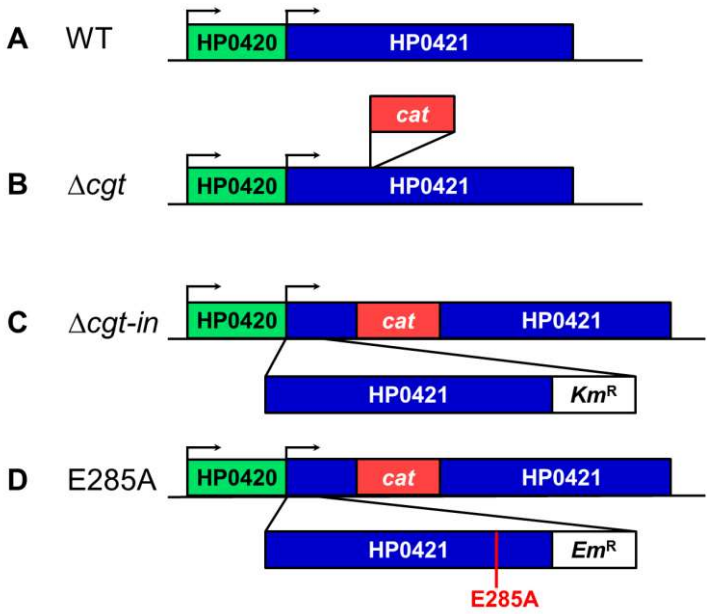

Figure S1. Construction of isogenic *H. pylori* mutants. (A) Wild-type *H. pylori* genes with normal *hp0420* and *hp0421* are shown. (B) Chloramphenicol acetyltransferase (*cat*) was inserted into WT *H. pylori hp0421* to generate a *cgt* knockout strain (Δ*cgt*). (C) The full length of *hp0421*, followed by a kanamycin-resistant cassette (Km^R) was inserted into Δ*cgt H. pylori hp0421* with to generate a *cgt* knockin strain (Δ*cgt*-in). (D) The full length of *hp0421*, along with a point mutation (E285A) followed by an erythromycin-resistance cassette (Em^R) was inserted into Δ*cgt H. pylori hp0421* with to generate a *cgt*-dead mutant strain (*cgt*-dead mutant). E285A: glutamate was mutated and changed to alanine at the amino acid sequence of 285, which is indicated in red.

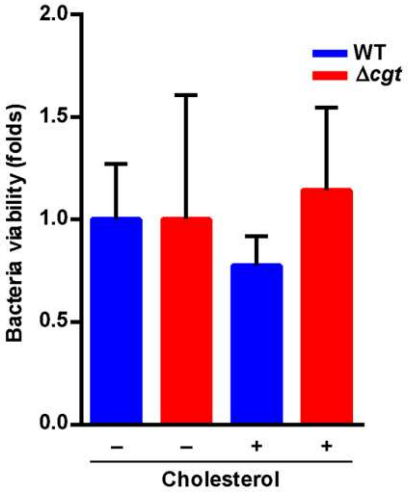

Figure S2. Exogenous cholesterol coating *H. pylori* does not affect bacterial viability. WT and Δ*cgt* were incubated with water-soluble cholesterol (5 mg/ml) for 1 h. The cholesterol-treated bacteria were plated by serial dilution onto blood agar plates and incubated for 5 days and the CFUs of viable *H. pylori* were counted.

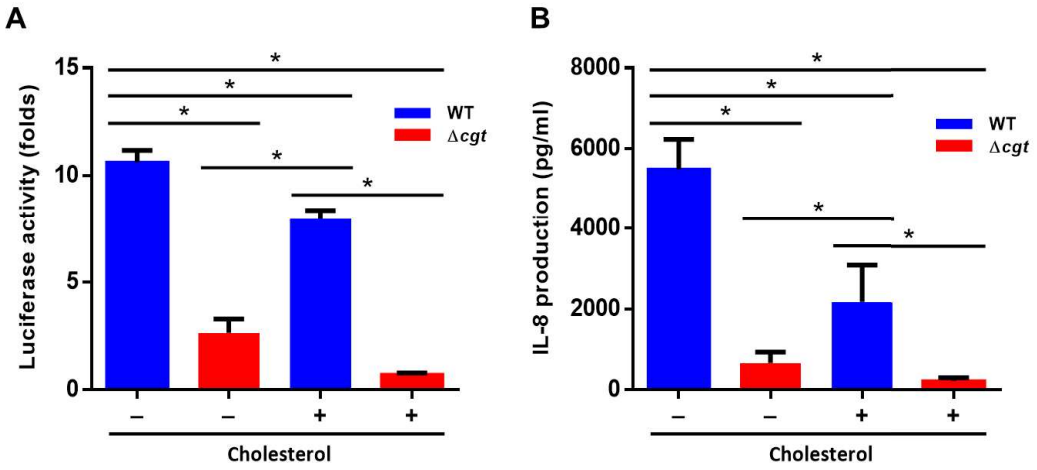

Figure S3. Excess exogenous cholesterol dampens *H. pylori* adhesion to cells. *H. pylori* was pretreated with water-soluble cholesterol (5 mg/ml) for 1 h prior to infecting AGS cells at an MOI of 100 for 6 h. (A) The level of NF-κB activation was determined by luciferase assay. (B) IL-8 production was assessed using ELISA. *, $P < 0.05$.