Supplementary Figure Legends

Supplementary Figure 1. Cdx2 expression in human gastric epithelial cell lines. Total RNA was collected from human gastric cancer cell lines and subjected to RT-PCR specific for Cdx2, MUC1, MUC2 and MUC5AC. Expression of GAPDH was also assessed as a loading control. Result shown is the representative of two independent experiments.

Supplementary Figure 2. The expression of Cdx2 was enhanced by *H. pylori* infection in gastric epithelial cell. AGS cells were infected with *H. pylori*. Cell lysates were collected at the indicated time points after infection and subjected to western blotting. Result shown is the representative of two independent experiments.

Supplementary Figure 3. Scheme of the Cdx2 promoter-luciferase reporter plasmids used in this study. Two luciferase reporter constructs (cdx2-luc containing NF-κB binding site and cdx2-N0-luc not containing this

binding site) are depicted.

Supplementary Figure 4. BAY11-7082 inhibits NF-κB activation. GCIY cells were transfected with NF-κB luciferase reporter plasmid together with pRL-TK vector. The cells were then infected with *H. pylori* in the presence or absence of the indicated concentrations of BAY11-7082, and relative luciferase activity was measured. Result indicated as mean±S.D., **P*<0.05 as compared to *H. pylori*-infected cells. Resultsshown are the representative of two independent experiments.

Supplementary Figure 5. NOD1 siRNA reduced the expression of NOD1. AGS cells were transfected with either control-siRNA or human NOD1 specific-siRNA. Expression of NOD1 was evaluated by western blot and Q-PCR.

Supplementary Figure 6. Cdx2 and MUC2 expression in GSM06 cells. Total RNA was collected from normal murine gastric epithelial GSM06 cells. The

expression of Cdx2 and MUC2 was evaluated by RT-PCR. RNAs isolated from mouse colon and stomach, were used as positive and negative controls, respectively. Result shown are the representative of two independent experiments.

Supplementary Figure 7. Pretreatment with iE-DAP reduces *H. pylori* infection-induced Cdx2 expression. AGS cells were cultured for 24 hours in the presence and the absence of NOD1 ligand. Total RNA was subjected to Q-PCR for Cdx2. Result shown in means±S.D. *P<0.05 as compared to *H. pylori*-infected AGS cells. Result shown is the representative of three independent experiments.

Supplementary Figure 8. iE-DAP alone does not alter Cdx2 expression. AGS cells and GCIY cells were cultured in the presence and the absence of NOD1 ligand iE-DAP for 24 hours. Then total RNA was extracted from the cells and subjected to Q-PCR for Cdx2.

Supplementary Figure 9. Inflammatory scores of the stomach did not differ between NOD1-intact and NOD1-deficient mice. NOD1-deficient and NOD1-intact mice were infected with *cag*PAI-positive *H. pylori*; one year after infection the mice were sacrificed and gastric tissues were collected and subjected to HE staining (n=5/group). The degree of inflammatory cell infiltration was scored according to the criteria developed by Rogers et al. (see Methods). Result shown is the representative of five independent experiments.

Supplementary Figure 10. TNF-α was induced in the stomachs of *H. pylori*-infected mice. NOD1-deficient and NOD1-intact mice were infected with *cag*PAI-positive *H. pylori*; one year after infection the mice were sacrificed and gastric tissues were collected and subjected to Q-PCR for TNF-α (n=5/group). Result shown in means±S.D. **P*<0.05 as compared to *H. pylori*-uninfected mice. Result shown is the representative of five independent experiments.