

Supplementary Material

Supplementary Figures



Supplementary Figure 1. Infection with *H. pylori* had no effect on endothelium-independent relaxation. Aortas from mice with one week or 12 weeks of $CagA^+H$. *pylori* infection exhibited no change in nitroglycerin (NTG)-induced endothelium-independent relaxation responses compared with control (A, B). Similarity, there was no change in NTG-induced endothelium-independent relaxation of aortas from mice with one week or 12 weeks of infection with $CagA^-H$. *pylori* compared with control (C, D). NC(1/2): normal control; NTG: nitroglycerin. Data are presented as mean±SEM, N=8-10 mice for each group at each time point.



Supplementary Figure 2. CagA⁺ H. pylori, not CagA⁻ H. pylori infection, increased ROS production in LDLR^{-/-} mice. Representative fluorescent images and quantification of ROS formation in the aorta of male LDLR^{-/-} mice with CagA⁺ H. pylori, CagA⁻ H. pylori or PBS gavage. CagA⁺ H. pylori, not CagA⁻ H. pylori infection significantly increased aortic ROS production in LDLR^{-/-} mice with three or five weeks of HFD compared with their controls (A, B). There was no significant difference in aortic ROS production in LDLR^{-/-} mice with CagA⁺ H. pylori infection, or with CagA⁻ H. pylori infection after 12 weeks of HFD (C). NC(1/2): normal control. Data are presented as mean±SEM; *P < 0.05, **P < 0.01 by t-test, N=8-10 mice for each group at each time point.



Supplementary Figure 3. Treatment with GW4869 had no effect on atherosclerosis in mice without *H. pylori* infection. After 3 or 5 weeks of high-fat diet (HFD), treatment of LDLR^{-/-} mice with GW4869 had no effect on early atherosclerotic plaque formation in the aorta (A-C) and aortic root (D-F) compared with control mice. Control: LDLR^{-/-} mice without *H. pylori* infection on HFD without GW4869 treatment; GW4869: LDLR^{-/-} mice without *H. pylori* infection on HFD without GW4869. Data are presented as mean \pm SEM. N=8-10 mice for each group at each time point.