中文題目：胃幽門螺旋桿菌感染藉由肝癌衍生生長因子上調在小鼠模式中造成慢性胃發炎

英文題目：*Helicobacter pylori* infection induces chronic gastritis mediated upregulation of hepatoma derived growth factor in mice

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Background: *Helicobacter pylori* (HP): HP is a Gram-negative bacterium, specialized in the colonization of the human stomach. HP infection is associated with chronic gastritis, peptic ulcer, and gastric cancer. The inflammatory response toward HP is characterized by the recruitment of different immune cells, mainly dendritic cells (DCs), neutrophils, macrophages, and B and T lymphocytes to the site of infection. **Hepatoma-derived growth factor (HDGF):** HDGF is a protein of 240 amino-acid isolated from the cultured supernatants of human hepatoma cells. HDGF is mainly localized in nucleus and stimulates the proliferation in various types of cells including fibroblasts, endothelial cells and hepatoma cells. HDGF is overexpressed in a variety of human cancers and correlated with poor prognosis in patients of many solid tumors including gastric cancer.

Method and Material: *In vitro*: AGS gastric cancer cells were infected by HP, and the expression and secretion of HDGF ware analyzed by Western blot, qRT-PCR, immunoflourecence staining. *In vivo*: After HP infection to wild-type (WT) and HDGF knock-out (KO) mice. And, the effects of HDGF on the infiltration of immune cells in gastric tissues were examined by hematoxylin and eosin staining.

Result: HDGF protein level was increased in AGS cells lysate and culture medium during H. pylori infection associated with elevated the expression of HDGF mRNA, and HDGF was also upregulated in gastric tissues of H. pylori-infected mice. Moreover, HDGF deficiency suppressed the infiltration of immune cells in gastric tissues during H. pylori infection, and the upregulation of TNFα, NFκB and COX-2 in gastric tissues during H. pylori infection was blocked in HDGF KO mice. The markers of cell apoptosis and proliferation in gastric cells during H. pylori infection was increased, and this indicated H. pylori infection accelerated cell turnover rate in gastric tissues. Interestingly, HDGF deficiency delayed H. pylori-promoted cell turnover.

Conclusion: HDGF protein level was increased in AGS cells lysate and culture medium during HP infection associated with elevated the expression of HDGF mRNA, and HDGF was also upregulated in HP-infected gastric tissues. Moreover, HDGF deficiency suppressed the infiltration of immune cells in gastric tissues during *H. pylori* infection. HDGF maybe play a positive role in HP-induced gastritis, even carcinogenesis.