



## Alterations in gastric mucin synthesis by *Helicobacter pylori*

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### Abstract

**AIM:** To determine the role of *Helicobacter pylori* in altering gastric mucin synthesis and define how this process relates to *H. pylori*-related diseases.

**METHODS:** Analyses of human gastric tissues using immunohisto-

chemistry and in situ hybridization document the role of *H. pylori* in altering the composition and distribution of gastric mucins.

**RESULTS:** These data indicate a decrease in the product of the MUC5 (MUC5AC) gene and aberrant expression of MUC6 in the surface epithelium of *H. pylori*-infected patients. A normal pattern was restored by *H. pylori* eradication. Inhibition of mucin synthesis including MUC5AC and MUC1 mucins by *H. pylori* has been established *in vitro* using biochemical and Western blot analyses. This effect is not due to inhibition of glycosylation, but results from inhibition of synthesis of mucin core structures. In vitro experiments using inhibitors of mucin synthesis indicate that cell surface mucins decrease adhesion of *H. pylori* to gastric epithelial cells.

**CONCLUSION:** Inhibition of mucin synthesis by *H. pylori in vivo* can disrupt the protective mucous layer and facilitate bacterial adhesion, which may lead to increased inflammation in the gastric epithelium.

**Key words:** Mucins; Glycoproteins; Gastric mucin/biosynthesis; Gastric mucosa; *Helicobacter pylori*; Glycosylation; *In vitro*; Immunohistochemistry; *In situ* hybridization

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