

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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