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*H. pylori*strain differences influence host responses

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Patients with chronic gastritis produced by *Helicobacter pylori* are at risk of duodenal and gastric ulceration and gastric cancer. The majority of *H. pylori*-colonized individuals remain asymptomatic, however, and the mechanism of this resistance is not fully understood. A study published in the March issue of *Journal of Clinical Investigation* shows that genetic differences between strains of *Helicobacter pylori* influence host inflammatory responses (J Clin Invest 2001, **107**:611-620).

Dawn Israel and colleagues examined in a gerbil model the ability of duodenal and gastric ulcer strains of *H. pylori* isolates to induce differential host responses *in vivo* or *in vitro*. Gastric ulcer strain B128 induced more severe gastritis than did duodenal ulcer strain G1.1. DNA hybridization to a whole *H. pylori* genome microarray identified a large deletion of the *cag* pathogenicity island in duodenal ulcer strain G1.1. Partial and complete disruption of the *cag* island in gastric strain B128 attenuated induction of IL-8 *in vitro* and significantly decreased gastric inflammation *in vivo*, suggesting that the ability of *H. pylori* strains to induce epithelial inflammation is dependent on the presence of an intact *cag* pathogenicity island.

The authors conclude that "Genotypic markers could be developed not only to identify individuals at risk for specific clinical sequelae of infection, but also to permit selective targeting of therapy for disease prevention."

References

1. Israel DA, Salama N, Arnold CN, *et al*: Helicobacter pylori strain-specific differences in genetic content, identified by microarray, influence host inflammatory responses. *J Clin Invest* 2001, 107:611-620., [http://www.jci.org/cgi/content/abstract/107/5/611]