Microarrays can be used to demonstrate differences in genetic content between *Helicobacter pylori* strains, giving a foretaste of how research may be conducted in the near future.

*Helicobacter pylori* enhances the risk for ulcer disease and gastric cancer, yet only a minority of *H. pylori*-colonized individuals develop disease. We examined the ability of two *H. pylori* isolates to induce differential host responses in vivo or in vitro, and then used *H. pylori* whole genome microarray to identify bacterial determinants related to pathogenesis. Gastric ulcer strain B128 induced more severe gastritis, proliferation, and apoptosis in gerbil mucosa than did duodenal ulcer strain G1.1, and gastric ulceration and atrophy occurred only in B128+ gerbils. In vitro, gerbil-passaged B128 derivatives significantly increased IL-8 secretion and apoptosis compared with G1.1 strains. DNA hybridization to the microarray identified several strain-specific differences in gene composition including a large deletion of the cag pathogenicity island strain G1.1. Partial and complete disruption of the cag island strain B128 attenuated induction of IL-8 in vitro and significantly decreased gastric inflammation in vivo. These results indicate that the ability of *H. pylori* to regulate epithelial cell responses related to inflammation depends on the presence of an intact cag pathogenicity island. Use of an *H. pylori* whole genome microarray is an effective method to identify differences in gene content between *H. pylori* strains that induce distinct pathological outcomes in a rodent model of *H. pylori* infection.
could only detect major differences in genetic content whereas in the near future microarrays will be able to detect small differences in individual genes. That will be the next leap forward.

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REFERENCES


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The chips are down for *Helicobacter pylori*

J C Atherton

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