

in the human population. Certainly, host genetic factors play a role in determining the clinical outcome of the infection ⁽⁵⁾. On the other hand, *H. pylori* virulence factors also play a role in pathogenesis, since virulent strains are associated with more aggressive tissue damage and an increased risk of a severe clinical outcome⁽⁶⁾. Finally, environmental factors such as nutrition are also thought to be important ⁽⁷⁾. Epidemiologic studies have shown that the prevalence of *H. pylori* varies considerably with age ⁽⁸⁾ *H.pylori* needs to have at least four basic characteristics to be able to colonize and establish an infection in the gastric mucosa: urease, flagella, a particular shape, and adhesins. *H. pylori* is able to adhere to the surface and sites of epithelial cells and to the basement membrane of gastric epithelial cells ⁽⁹⁾. When *H.pylori* is introduced in the stomach, a pH-neutral microenvironment around the bacteria is produced by exogenous shedding of urease, which converts urea to ammonia ions that neutralize the acidic gastric juice, and thereby enables *H. pylori* to survive and multiply in the stomach ⁽¹⁰⁾. Thus, the disease outcome is determined by a combination of host, bacterial, and environmental factors.

The acute *H. pylori* infection that is dominated by abdominal pain and infiltration of polymorph nuclear leucocytes (PMNs) in the gastric mucosa only lasts for a few weeks ⁽¹¹⁻¹³⁾. Thereafter, it turns into an active chronic superficial gastritis with an increased recruitment of lymphocytes and other mononuclear leucocytes. In the humoral immune response to *H. pylori* infection, IgM antibodies to *H. pylori* are produced shortly after colonization whereas IgG antibodies to *H. pylori* seem to be delayed up to 3–6 months ^(14, 15). Thus,

within a few weeks of the primary exposure to *H. pylori*, a true infection can be established.

The superficial gastritis may or may not evolve to atrophic gastritis, which later may lead to intestinal metaplasia, dysplasia, and gastric cancer ⁽¹⁶⁾. As the inflammation progresses, the specific immune response becomes more dominating and even the PMNs lose their ability to recognize the specific *H. pylori* strain in the host as a foreigner ⁽¹⁷⁾.

The diagnostic methods available for detecting *H. pylori* infection include conventional PCR and real-time PCR ^(18, 19). Rapid urease test is highly specific for *H. pylori* infection and is commonly used for the detection of *H. pylori* infection at endoscopy. It requires a high density of bacteria ⁽²⁰⁾. The sensitivity of urease test is reduced in patients who are taking proton pump inhibitors (PPI), antibiotics or bismuth compounds ^(21, 22). Any antibiotic active against *H. pylori* will cause a reduction in the numbers of bacteria in the stomach ⁽²³⁾.

Increase Iron Uptake and Utilization by Bacteria

Epidemiologic studies have shown that persons seropositive for *H. pylori* infection have a significantly lower serum ferritin level ^(24, 25, 26, 27, 28). Although *H. pylori* infection is common, iron deficiency anemia does not develop in all infected patients. The ability to cause iron deficiency anemia does not appear to be related to the virulence of the organism because ferritin levels did not differ between patients infected with cytotoxin-associated gene A (CagA)-positive and CagA-negative strains of *H. pylori* ⁽²⁴⁾. It may be possible that other bacterial virulence factors or host factors are responsible for the development of iron deficiency anemia.