less easily cultured from stool. Most acquisition of *H. pylori* is during the early years of childhood.

■ PATHOLOGY AND PATHOGENESIS

Long-term *H. pylori* colonization induces *chronic superficial gastritis*, a tissue response in the stomach that includes infiltration of the mucosa by both mononuclear and polymorphonuclear cells. (The term *gastritis* should be used specifically to describe histologic features; it has also been used to describe endoscopic appearances and even symptoms, but only magnification endoscopy correlates with microscopic findings or even with the presence of *H. pylori*, and even this is insufficient for diagnosis.) Although *H. pylori* is capable of numerous adaptations that prevent excessive stimulation of the immune system, colonization is accompanied by a considerable persistent local and systemic immune response, including the production of antibodies and cell-mediated responses. However, these responses are ineffective in clearing the bacterium. This inefficient clearing appears to be due in part to *H. pylori*'s downregulation of the immune system, which fosters its own persistence.

Most *H. pylori*–colonized persons do not develop clinical sequelae. That some persons develop overt disease whereas others do not is related to a combination of factors: bacterial strain differences, host susceptibility to disease, and environmental factors.

Bacterial Virulence Factors Several *H. pylori* virulence factors are more common among strains that are associated with disease than among those that are not. The *cag* island is a group of genes that encodes a bacterial type IV secretion system. Through this system, an effector protein, CagA, is translocated into epithelial cells, where it may be activated by phosphorylation and induces host cell signal transduction; proliferative, cytoskeletal, and inflammatory changes in the cell result. The protein at the tip of the secretory