colistin alone. Combinations of polymyxins with a carbapenem look more promising and are being evaluated in prospective clinical trials. Fosfomycin has poor activity against *Acinetobacter* and should not be relied upon for treatment. Clearly, new treatment options are needed for serious *Acinetobacter* infections.

COMPLICATIONS AND PROGNOSIS

Given the propensity of *A. baumannii* to cause infections in seriously ill patients in ICUs, it is not surprising that *A. baumannii* infections are associated with high mortality rates. Thus a pertinent question is whether *A. baumannii* infections are associated with high attributable mortality rates after the severity of illness is controlled for. A number of studies have addressed this issue but have had disparate results. Whether the discrepant results can be explained purely by methodologic differences is unknown at present.

PREVENTION

Multidrug-resistant *A. baumannii* clearly causes outbreaks of infection and then establishes endemicity. In endemic situations, a small number of strain types predominate. In the 1991–1992 outbreaks in New York City, for example, two strain types accounted for more than 80% of carbapenem-resistant isolates. This "oligoclonality" plainly demonstrates the potential importance of infection control interventions in response to outbreaks of multidrug-resistant *A. baumannii* infection.

The hospital environment is an important reservoir of organisms capable of colonizing patients and causing infection. Environmental sources of *A. baumannii* include computer keyboards, glucometers, multidose medication vials, IV nutrition, inadequately sterilized reusable arterial pressure transducers, ventilator tubing, suction catheters, humidifiers, containers of distilled water, urine collection jugs, and moist bedding articles. Pulsatile-lavage wound treatment—a high-pressure irrigation system used to debride wounds—has been associated with an outbreak of *A. baumannii* infection.

Contaminated inanimate objects should be removed from the patient-care environment or subjected to enhanced environmental cleaning. Although contact-isolation procedures (use of gloves and gowns when dealing with colonized patients or their environment), accommodation of patients in single rooms, and improved hand hygiene are critical, attention to the patient-care environment may be the only measure that leads to control of outbreaks of *A. baumannii* infection. One study found that *Acinetobacter* can be cultured from the air in rooms of patients with *A. baumannii* infection; the infection-control implications are not yet clear.

188 *Helicobacter pylori* Infections

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DEFINITION

Helicobacter pylori colonizes the stomach in ~50% of the world's human population, essentially for life unless eradicated by antibiotic treatment. Colonization with this organism is the main risk factor for peptic ulceration (Chap. 348) as well as for gastric adenocarcinoma and gastric mucosa-associated lymphoid tissue (MALT) lymphoma (Chap. 109). Treatment for *H. pylori* has revolutionized the management of peptic ulcer disease, providing a permanent cure in most cases. Such treatment also represents first-line therapy for patients with low-grade gastric MALT lymphoma. Treatment of *H. pylori* is of no benefit in the treatment of gastric adenocarcinoma, but prevention of *H. pylori* colonization could potentially prevent gastric malignancy and peptic ulceration. In contrast, increasing evidence indicates that lifelong *H. pylori* colonization may offer some protection against complications of gastroesophageal reflux disease (GERD), including esophageal adenocarcinoma. Recent research has focused on whether *H. pylori* colonization is also a risk factor for some extragastric diseases and whether it is protective against some recently emergent medical problems, such as childhood-onset asthma and obesity.

ETIOLOGIC AGENT

Helicobacter pylori *H. pylori* is a gram-negative bacillus that has naturally colonized humans for at least 100,000 years, and probably throughout human evolution. It lives in gastric mucus, with a small proportion of the bacteria adherent to the mucosa and possibly a very small number of the organisms entering cells or penetrating the mucosa; the organism's distribution is never systemic. Its spiral shape and flagella render *H. pylori* motile in the mucus environment. The organism has several acid-resistance mechanisms, most notably a highly expressed urease that catalyzes urea hydrolysis to produce buffering ammonia. *H. pylori* is microaerophilic (i.e., requires low levels of oxygen), is slow-growing, and requires complex growth media in vitro.

Other *Helicobacter* Species A very small proportion of gastric *Helicobacter* infections are due to species other than *H. pylori*, possibly acquired as zoonoses. These non-*pylori* gastric helicobacters are associated with low-level inflammation and occasionally with disease. In immunocompromised hosts, several nongastric (intestinal) *Helicobacter* species can cause disease with clinical features resembling those of *Campylobacter* infections; these species are covered in Chap. 192.

EPIDEMIOLOGY

Prevalence and Risk Factors The prevalence of *H. pylori* among adults is <30% in most parts of the United States and in other developed countries as opposed to >80% in most developing countries. In the United States, prevalence varies with age: up to 50% of 60-year-old persons, ~20% of 30-year-old persons, and fewer than 10% of children are colonized. *H. pylori* is usually acquired in childhood. The age association is due mostly to a birth-cohort effect whereby current 60-year-olds were more commonly colonized as children than are current children. Spontaneous acquisition or loss of *H. pylori* in adulthood is uncommon. Childhood acquisition explains why the main risk factors for infection are markers of crowding and social deprivation in childhood.

Transmission Humans are the only important reservoir of *H. pylori*. Children may acquire the organism from their parents (most often the primary caregiver) or from other children. The former is more common in developed countries and the latter in less developed countries. Whether transmission takes place more often by the fecal-oral or the oral-oral route is unknown, but *H. pylori* is easily cultured from vomitus and gastroesophageal refluxate and is less easily cultured from stool.

PATHOLOGY AND PATHOGENESIS

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 these features do not correlate with microscopic findings or even with the presence of *H. pylori*.) 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