HELICOBACTER PYLORI

GENERAL CHARACTERISTICS

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Helicobacter pylori, previously known as ***Campylobacter pylori***, is a gram-negative, microaerophilic, spiral (helical) bacterium usually found in the stomach. Its helical shape (from which the genus name, helicobacter, derives) is thought to have evolved in order to penetrate the mucoid lining of the stomach and thereby establish infection. The bacterium was first identified in 1982 by the Australian doctors Barry Marshall and Robin Warren. *H. pylori* has been associated with cancer of the mucosa-associated lymphoid tissue in the stomach, esophagus, colon, rectum, or tissues around the eye (termed extranodal marginal zone B-cell lymphoma of the cited organ), and of lymphoid tissue in the stomach (termed diffuse large B-cell lymphoma)

Morphology

Helicobacter pylori is a helix-shaped (classified as a curved rod, not spirochaete) Gram-negative bacterium about 3 μm long with a diameter of about 0.5 μm. *H. pylori* can be demonstrated in tissue by Gram stain, Giemsa stain, haematoxylin–eosin stain, Warthin-Starry silver stain, acridine orange stain, and phase-contrast microscopy. It is capable of forming biofilms and can convert from spiral to a possibly viable but nonculturable coccoid form.

Helicobacter pylori has four to six flagella at the same location; all gastric and enterohepatic *Helicobacter* species are highly motile owing to flagella. The characteristic sheathed flagellar filaments of *Helicobacter* are composed of two copolymerized flagellins, FlaA and FlaB.

H. pylori infection usually has no symptoms but sometimes causes gastritis . The infection is also associated with the development of certain cancers. Many investigators have suggested that *H. pylori* causes or prevents a wide range of other diseases, but many of these relationships remain controversial

Some studies suggest that *H. pylori* plays an important role in the natural stomach ecology, e.g. by influencing the type of bacteria that colonize the gastrointestinal tract Other studies suggest that non-pathogenic strains of *H. pylori* may beneficially normalize stomach acid secretion,] and regulate appetite In 2015, it was estimated that over 50% of the world's population had *H. pylori* in their upper gastrointestinal tracts] with this infection (or colonization) being more common in developing countries. In recent decades, however, the prevalence of *H. pylori* colonization of the gastrointestinal tract has declined in many countries

Physiology

Helicobacter pylori is microaerophilic – that is, it requires oxygen, but at lower concentration than in the atmosphere. It contains a hydrogenase that can produce energy by oxidizing molecular hydrogen (H_2) made by intestinal bacteria. It produces oxidase, catalase, and urease.

H. pylori possesses five major outer membrane protein families. The largest family includes known and putative adhesins. The other four families are porins, iron transporters, flagellum -associated proteins, and proteins of unknown function. Like other typical Gram-negative bacteria, the outer membrane of *H. pylori* consists of phospholipids and lipopolysaccharide (LPS).

. The outer membrane also contains cholesterol glucosides, which are present in few other bacteria.

Adaptation of *H. pylori* to high acidity of stomach

As mentioned above, *H. pylori* produce large amounts of urease to produce ammonia as one of its adaptation methods to overcome stomach acidity. Helicobacter pylori arginase, a bimetallic enzyme binuclear Mn2-metalloenzyme arginase, crucial for pathogenesis of the bacterium in human stomach, a member of the ureohydrolase family, catalyzes the conversion of L-arginine to L-ornithine and urea, where ornithine is further converted into polyamines, which are essential for various critical metabolic processes.

This provides acid resistance and is thus important for colonization of the bacterium in the gastric epithelial cells. Arginase of *H. pylori* also plays a role in evasion of the pathogen from the host immune system mainly by various proposed mechanisms, arginase competes with host-inducible nitric oxide (NO) synthase for the common substrate L-arginine, and thus reduces the synthesis of NO, an important component of innate immunity and an effective antimicrobial agent that is able to kill the invading pathogens directly