Genus: *Helicobacter*

Morphology

Helicobacter are spiral, curved, or fusiform rod-shaped Gram-negative bacteria, microaerophilic bacterium, motile with **lophotrichous** flagella. *Helicobacter* species have been isolated from the gastrointestinal and hepatobiliary tract of many different mammalian hosts, including humans.

Helicobacter pylori the most important species related to human. Unlike other bacteria, *H. pylori* can survive in the harsh acidic environment of the stomach because they produce a substance that neutralizes stomach acid.

Important Properties

H. pylori it is a **strong producer of urease**. Humans are the primary host-reservoir for *H. pylori*, the various helicobacters can be divides into two groups:

1. *Helicobacter* that primarily colonize the stomach (gastric helicobacters).
2. *Helicobacter* that colonize the intestines (enterohepatic helicobacters).

Pathogenicity and clinical findings

H. pylori is causative agent of gastritis, peptic ulcer that may be lead to gastric cancer. Acute infection yields an upper gastrointestinal illness with nausea and pain; vomiting and fever may also be present.

This bacterium able to survive in the acidic environment of the stomach and colonize the gastric mucosa in the absence of antimicrobial

treatment. While *H. pylori* grows optimally at a pH of (6–7), it would be killed or not grow at the pH within the gastric lumen (pH 1–3). Several factors contribute to the organism's ability to overcome the acidic environment of the stomach, contributing to colonization, inflammation, changes in gastric acid production, and tissue destruction. Gastric mucus is impermeable to acid and has a strong buffering capacity. On the lumen side of the mucus, the pH is low (1–3); on the epithelial side, the pH is about (6–7). After entering the stomach, *H. pylori* utilizes its urease activity to neutralize the gastric acid; intracellular urease activity as well as urease located on the bacterial cell surface allow for the breakdown of urea into ammonia and CO2. NH_3 is converted to ammonium (NH4+) and extruded from the bacterial cell leading to neutralization of the gastric acid.

Flagella-mediated motility then allows the organisms, protected from the gastric acid, to move through the gastric mucus toward the epithelium.

H. pylori is found deep in the mucous layer near the epithelial surface where it releases several **effector proteins and toxins**, including **adhesion factors, neutrophil-activating protein A, a heat shock protein, and various cytotoxins (mucinase, phospholipase) that exert tissue damage.**

Following acquisition of the *H. pylori*, individuals may develop mild to severe upper gastrointestinal diseases such as acute symptoms may last for less than 1 week or as long as 2 weeks. After the acquisition of the organisms and the initial acute stage of the infection, colonization with *H. pylori* occurs in many patients. Such colonization persists for years or even a lifetime.

Transmission

Helicobacter can transmitted by person-to-person transmission, especially within the same family, the reinfection rate will increase if there are family members affected, but also environmental contamination is possible. The bacteria can also be spread by fecal contamination of food or water. In developing countries, a combination of untreated water, crowded conditions, and poor hygiene contributes to higher *H. pylori* prevalence.

Laboratory Diagnosis

1. The organism can be seen on Gram-stained smears of biopsy specimens of the gastric mucosa.

2. Urease production is the basis for a diagnostic test called the "urea breath" test. In this test, radiolabeled urea is ingested. If the organism is present, urease will cleave the ingested urea, radiolabeled CO2 is evolved, and the radioactivity is detected in the breath.

3. A test for *Helicobacter* antigen in the stool can be used for diagnosis and for confirmation that treatment has eliminated the organism.

4. Gastroscope also, used in diagnosis.

5. Culture: *H. pylori* can grow on different solid media containing blood or blood products (blood or lysed blood agar plates).

Most studies have used **Brucella agar or Columbia agar** as the agar base with blood addition.

Treatment

Use of antibiotics to eliminate *Helicobacter* plus a drug to reduce gastric acidity. Treatment with combination of two antibiotics is used due to emergence of resistance (e.g., amoxicillin and metronidazole) and bismuth salts results in decreased recurrence rate.

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