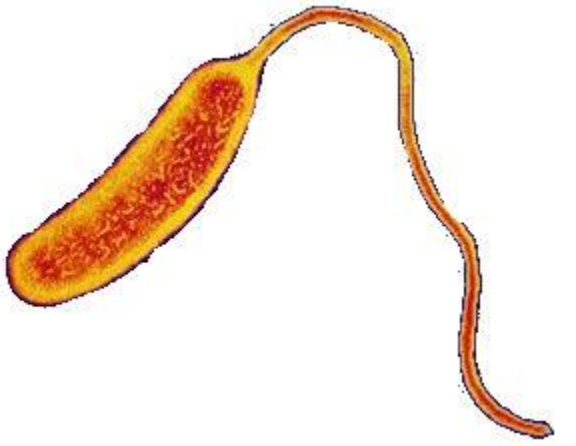
Vibrio cholerae



Growth Characteristics

V cholerae regularly ferments sucrose and mannose but not arabinose. A positive oxidase test is a key step in the preliminary identification of *V cholerae* and other vibrios. Most *Vibrio* species are halotolerant, and NaCl often stimulates their growth. Some vibrios are halophilic, requiring the presence of NaCl to grow. Another difference between vibrios and aeromonas is that vibrios grow on media containing 6% NaCl, whereas aeromonas does not.

Antigenic Structure & Biologic Classification

Many vibrios share a single heat-labile flagellar H antigen. Antibodies to the H antigen are probably not involved in the protection of susceptible hosts.

V cholerae has O lipopolysaccharides that confer serologic specificity. There are at least 139 O antigen groups. *V cholerae* strains of O group 1 and O group 139 cause classic cholera; occasionally. Antibodies to the O antigens tend to protect laboratory animals against infections with *V cholerae.*

The El Tor biotype produces a hemolysin, gives positive results on the Voges-Proskauer test, and is resistant to polymyxin B. Molecular techniques can also be used to type *V cholerae.* Typing is used for epidemiologic studies, and tests generally are done only in reference laboratories.

Vibrio cholerae Enterotoxin

V cholerae produce a heat-labile enterotoxin with a molecular weight of about 84,000. Diarrhea occurs—as much as 20–30 L/day— with resulting dehydration, shock, acidosis, and death. The genes for *V cholerae* enterotoxin are on the bacterial chromosome. Cholera enterotoxin is antigenically related to *Escherichia coli* and can stimulate the production of neutralizing antibodies. However, the role of antitoxic and antibacterial antibodies in protection against cholera is not clear.

Clinical Findings

About 60% of infections with classic *V cholerae* are asymptomatic. The incubation period is 1–4 days for persons who develop symptoms, depending largely upon the size of the inoculum ingested. There is a sudden onset of nausea and vomiting and profuse diarrhea with abdominal cramps. Stools, which resemble "rice water," contain mucus, epithelial cells, and large numbers of vibrios. There is rapid loss of fluid and electrolytes, which leads to profound dehydration, circulatory collapse, and anuria. The mortality rate without treatment is between 25% and 50%. The diagnosis of a full-blown case of cholera presents no problem in the presence of an epidemic. However, sporadic or mild cases are not readily differentiated from other diarrheal diseases.

Immunity

Gastric acid provides some protection against cholera vibrios. An attack of cholera is followed by immunity to reinfection, but the duration and degree of immunity are not known. In experimental animals, specific IgA antibodies occur in the lumen of the intestine. Similar antibodies in serum develop after infection but last only a

few months. Vibriocidal antibodies in serum (titer 1:20) have been associated with protection against colonization and disease. The presence of antitoxin antibodies has not been associated with protection.

Diagnostic Laboratory Tests

Specimens

Specimens for culture consist of mucus from stools.

Smears

The microscopic appearance of smears made from stool samples is not appear. Dark-field or phase contrast microscopy may show the rapidly motile vibrios.

Culture

Growth is rapid in peptone agar, on blood agar with a pH near 9.0 or on TCBS agar, and typical colonies can be picked in 18 hours. For enrichment, a few drops of stool can be incubated for 6–8 hours in taurocholatepeptone broth (pH 8.0–9.0); organisms from this culture can be stained or subcultured.

Specific Tests

V cholerae organisms are further identified by slide agglutination tests using anti-O group 1 or group 139 antisera and by biochemical reaction patterns.

Treatment

The most important part of therapy consists of water and electrolyte replacement to correct the severe dehydration and salt depletion. Many antimicrobial agents are effective against *V cholerae*. Oral tetracycline tends to reduce stool output in cholera and shortens the period of excretion of vibrios. In some endemic areas, tetracycline resistance of *V cholerae* has emerged.

Helicobacter pylori

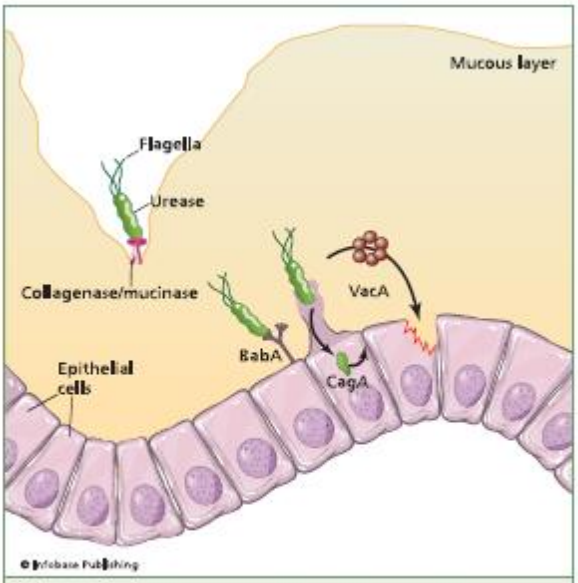
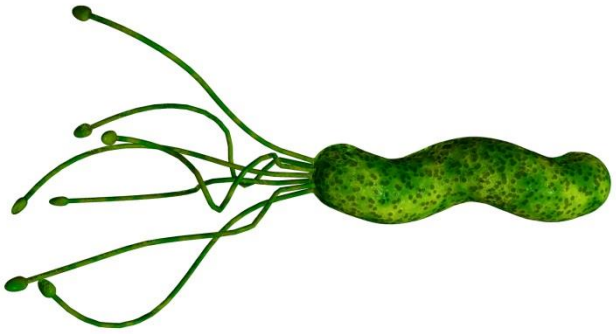


Figure 3.5 The accessory proteins that *H. pylori* uses to establish and maintain its presence in the stomach.



Helicobacter pylori is a spiral-shaped gram-negative rod. *H pylori* is associated with antral gastritis, duodenal (peptic) ulcer disease, gastric ulcers, and gastric

carcinoma. Other *Helicobacter* species that infect the gastric mucosa exist but are rare.

Morphology & Identification

Typical Organisms

H pylori has many characteristics in common with campylobacters. It has multiple flagella at one pole and is actively motile.

Culture

Culture sensitivity can be limited by prior therapy, contamination with other mucosal bacteria, and other factors. *H pylori* grows in 3–6 days when incubated at 37°C in a microaerophilic environment, as for *C jejuni*. The media for primary isolation include Skirrow's medium with vancomycin, polymyxin B, and trimethoprim, chocolate medium, and other selective media with antibiotics (eg, vancomycin, nalidixic acid, amphotericin). The colonies are translucent and 1–2 mm in diameter.

Growth Characteristics

H pylori is oxidase-positive and catalase-positive, has a characteristic morphology, is motile, and is a strong producer of urease.

Clinical Findings

Acute infection can yield an upper gastrointestinal illness with nausea and pain; vomiting and fever may be present also. The acute symptoms may last for less than 1 week or as long as 2 weeks. Once colonized, the *H pylori* infection persists for years and perhaps decades or even a lifetime. About 90% of patients with duodenal ulcers and 50–80% of those with gastric ulcers have *H pylori* infection. *H pylori* also may have a role in gastric carcinoma and lymphoma.

Diagnostic Laboratory Tests

Specimens

Gastric biopsy specimens can be used for histologic examination or minced in saline and used for culture. Blood is collected for determination of serum antibodies.

Smears

The diagnosis of gastritis and *H pylori* infection can be made histologically. A gastroscopy procedure with biopsy is required. Routine stains demonstrate gastritis, and Giemsa or special silver stains can show the curved or spiraled organisms.

Culture

As above.

Antibodies

Several assays have been developed to detect serum antibodies specific for *H pylori.* The serum antibodies persist even if the *H pylori* infection is eradicated, and the role of antibody tests in diagnosing active infection or following therapy is therefore limited.

Special Tests

Rapid tests to detect urease activity are widely used for presumptive identification of *H pylori* in specimens. Gastric biopsy material can be placed onto a urea-containing medium with a color indicator. If *H pylori* is present, the urease rapidly splits the urea (1–2 days) and the resulting shift in pH yields a color change in the medium. In vivo tests for urease activity can be done also. ^{13}C- or ^{14}C-labeled urea is ingested by the patient. If *H pylori* is present, the urease activity generates labeled CO_2 that can be detected in the patient's exhaled breath.

Detection of *H pylori* antigen in stool specimens is appropriate as a test of cure for patients with known *H pylori* infection who have been treated.

Immunity

Patients infected with *H pylori* develop an IgM antibody response to the infection. Subsequently, IgG and IgA are produced, and these persist, both systemically and at the mucosa, in high titer in chronically infected persons. Early antimicrobial treatment of *H pylori* infection blunts the antibody response; such patients are thought to be subject to repeat infection.

Treatment

Triple therapy with metronidazole and either bismuth subsalicylate or bismuth subcitrate plus either amoxicillin or tetracycline for 14 days eradicates *H pylori* infection in 70–95% of patients. An acid-suppressing agent given for 4–6 weeks enhances ulcer healing. Proton pump inhibitors directly inhibit *H pylori* and appear to be potent urease inhibitors. Either 1 week of a proton pump inhibitor plus amoxicillin and clarithromycin or of amoxicillin plus metronidazole also is highly effective.