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Helicobacter pylori (redirected from *H. pylori*)

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Helicobacter pylori [helˈɪ-ko-bakˈter pi-loˈri]

a gram-negative spiral bacterium that causes gastritis and pyloric ulcers in humans; a history of *H. pylori* infection is associated with gastric carcinoma; Formerly called *Campylobacter pylori*.

Miller-Keane Encyclopedia and Dictionary of Medicine, Nursing, and Allied Health, Seventh Edition. © 2003 by Saunders, an imprint of Elsevier, Inc. All rights reserved.

Hel·i·co·bac·ter py·lor·i

a bacterial species that produces urease and causes gastritis and nearly all peptic ulcer disease of the stomach and duodenum. Infection with this organism also plays an etiologic role (probably along with dietary cofactors) in dysplasia and metaplasia of gastric mucosa, distal gastric adenocarcinoma, and non-Hodgkin lymphoma of the stomach.

Synonym(s): [Campylobacter pylori](#)

The organism was first observed in 1982 by Robin Warren and Barry J. Marshall at Royal Perth Hospital in Western Australia in biopsy specimens from patients with chronic gastritis. Originally believed to be a species of *Campylobacter*, the organism was reclassified as *Helicobacter pylori* in 1989. A curved or spiral, flagellated gram-negative bacillus, *H. pylori* colonizes the gastric mucosa, attaching itself to the surface of mucus-secreting columnar cells. The ability of the organism to survive in an acid medium is due to its production of urease, which converts urea to ammonia and alkalizes the film of mucus in which it resides. Infection with *H. pylori* is common worldwide, and the prevalence of infection increases with age, reaching about 50% among people 60 and older. Transmission is believed to be from person to person by the fecal-oral route. Familial clustering of infection and a higher incidence among blacks and Hispanics have been attributed to social rather than genetic factors. After infection occurs, it typically remains for life unless treated with antibiotics. Newly acquired infection results in transitory acute gastritis with extensive damage to parietal cells accompanied by impairment of acid production. Most people have no symptoms (possibly because some strains of *H. pylori* do not produce cytotoxins) but each year about 1% of *H. pylori*-infected adults develop peptic ulcer. The risk of progression to peptic ulcer disease is increased by cigarette smoking and long-term use of nonsteroidal antiinflammatory agents. About 70% of all patients with gastric ulcers and 90% of those with duodenal ulcers are found to be infected with *H. pylori*. In the U.S., about 500,000 new cases of peptic ulcer disease occur each year. The disease is responsible for 3-4 million physician visits and approximately 16,000 deaths annually. *H. pylori* infection has not been associated with nonulcer dyspepsia or inflammatory disorders of the digestive tract other than peptic ulceration. However, incidence of both gastric adenocarcinoma and gastric lymphoma is higher in those infected. In addition, the organism has been implicated in some cases of cholecystitis and autoimmune thyroiditis, and some studies have suggested that gastric infection with *H. pylori* may be a factor, by an unknown mechanism, in some cases of sudden infant death syndrome (SIDS). Diagnosis of *H. pylori* infection can be confirmed by identification of the organism in stained sections of gastric biopsy material, by culture from biopsy material, by testing biopsy material for urease activity, by identification of bacterial antigen in stool, by finding IgG antibody to the organism in the serum (the method of choice to confirm infection in a previously untreated patient), or by detection of urease activity with various biochemical tests. The urea breath test is more useful than serologic testing to confirm eradication of *H. pylori* after a course of treatment because IgG antibody may remain elevated for 1-5 years after eradication. Antibiotic therapy does not yield faster healing of a peptic ulcer than treatment with antisecretory agents, but it greatly reduces the likelihood

of ulcer recurrence. Recommended regimens for eradication of *H. pylori* include combinations of bismuth subsalicylate with two antibiotics (metronidazole or clarithromycin and tetracycline or amoxicillin). Acquired resistance of *H. pylori* to the macrolide and imidazole antibiotics is a growing problem. It is estimated that 30% of strains of the organism in the U.S. are resistant to metronidazole and that 10% are resistant to macrolides. A major factor in the emergence of resistant strains appears to be an inadequate or failed first course of treatment. Active vaccination by oral administration of an enzymatically inactive recombinant subunit of *H. pylori* urease combined with a mucosal adjuvant (labile toxin of *Escherichia coli*) has elicited microbiologic and clinical cure of *H. pylori* infection in animal studies and limited human trials.

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Helicobacter pylori *Campylobacter pylori* GI disease A curved gram-negative microaerophilic bacillus which causes most cases of gastritis; it is present in 10-50% of healthy young persons and up to 60% of those ≥ age 60
Epidemiology *H. pylori* may be spread by houseflies—hand washing may ↓ transmission, by intrafamilial contact, or domestic cats Note: 90% of Pts with intestinal-type gastric CA have been infected by *H. pylori*, which may be a co-factor for gastric CA, acting either to stimulate production of cellular mutagens or induce ↑ proliferation after cell damage; it has been implicated in antibiotic-responsive duodenal ulcers, and linked to other gastric lesions, benign and malignant Diagnosis Gastric Bx with special—eg, Diff-Quik stain, serologic tests—90-95% accuracy, urea breath test Disease course *H. pylori* infection → chronic superficial gastritis, peptic ulcer disease, lymphoproliferative disease, ± → gastric lymphoma, chronic atrophic gastritis, ± → gastric adenoCA Management Proton-pump inhibitors—eg, omeprazole, lansoprazole, esomeprazole; antibiotics—amoxicillin, clarithromycin, metronidazole; ranitidine; bismuth subcitrate. See [Duodenal ulcer](#), [Gastric lymphoma](#), [Urea breath test](#).

McGraw-Hill Concise Dictionary of Modern Medicine. © 2002 by The McGraw-Hill Companies, Inc.

Hel·i·co·bac·ter py·lor·i (hel'i-kō-bak'tēr pī-lō'rī)

A bacterial species that produces urease and is associated with several gastroduodenal diseases, including gastritis and peptic ulcer. The type species of the genus *Helicobacter*.

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Helicobacter pylori An organism found in the stomach and duodenum in at least half of the world's population. Whether or not it causes disease depends on several factors including the presence or absence in the organism of the *babA2* (bacterial adhesion) gene. In people with [PEPTIC ULCER](#) *H. pylori* with this gene is more than three times as common as in people without peptic ulceration. The bacterium has also been linked with other gastrointestinal diseases including gastric cancer and gastric mucosa-associated lymphoid tissue (MALT) lymphoma. Destruction of the organisms with [METRONIDAZOLE](#) or clarithromycin used in combination with a proton pump inhibitor drug such as [OMEPRAZOLE](#) is often followed by ulcer healing and appears to prevent recurrence. Eradication of the infection can be confirmed with a urea breath test. It is not yet clear whether this organism actually causes peptic ulcer but it certainly causes a diffuse gastritis. The genome of *H. pylori* has been sequenced.

Collins Dictionary of Medicine © Robert M. Youngson 2004, 2005

Helicobacter pylori a spiral-shaped Gram-negative (see [GRAM'S STAIN](#) bacterium, motile by way of polar [FLAGELLA](#), that causes most peptic ulcers. About 20% of those under 40 years and 50% of those over 60 carry the bacterium in their stomachs, but most infected people do not develop ulcers. *H. pylori* weakens the protective mucous

coating of the **STOMACH** and **DUODENUM**, allowing acid to irritate the lining. Together with the ability of the bacterium to burrow into the lining, the acid causes an ulcer. *H. pylori* is able to survive in the stomach because it secretes enzymes, such as **UREASE** that lead to neutralization of stomach acid. Peptic ulcers caused by *H. pylori* are treated with **ANTIBIOTICS** to kill the bacteria and drugs to reduce stomach acid and hence protect the stomach lining.

Originally named *Campylobacter pyloridis*, due to similar structure to other *Campylobacter* species, afterwards renamed *C. pylori*, then *H. pylori*.

Collins Dictionary of Biology, 3rd ed. © W. G. Hale, V. A. Saunders, J. P. Margham 2005

Helicobacter pylori

A gramnegative rod-shaped bacterium that lives in the tissues of the stomach and causes inflammation of the stomach lining.

Mentioned in: **Indigestion**, **Ulcers (Digestive)**

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Hel·i·co·bac·ter py·lor·i (hel'i-kō-bak'ter pī-lō'rī)

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