Helicobacter pylori

Helicobacter pylori is a spiral (helical), a microaerophilic, gram-negative bacterium that colonizes the gastric mucosa.

The prevalence of H.pylori infection in our population is estimated at 30–55%. The prevalence increases with the age of the population. H. pylori infection is present in 90–95% of patients with duodenal ulcers and in 60–80% of patients with gastric ulcers. The bacterium was first identified by Barry Marshall and Robin Warren (2005 Nobel Prize in Physiology or Medicine).

Helicobacter pylori is classified as a **WHO Group 1 Carcinogen** However, there is no evidence that its eradication reduces the risk of stomach cancer.[1]

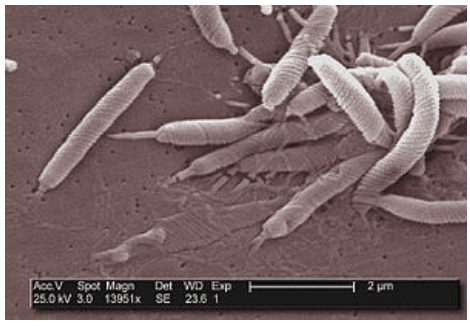
Ethiology and Epidemiology

- discovered in 1982
- **movable**, curved microbe with **flagellum**
- lives in the mucus **the gastric mucosa**
- high **urease** production
- has a significant diversity of proteins and DNA - typing of species and subspecies
- a purely **human pathogen**, has not been detected in animals or soil
- about 50% of the population is estimated to be infected; the incidence is lower in developed countries
- **transmission** − oro-oral or orofecal; direct and indirect transmission from person to person is possible (contaminated food, cutlery, dishes)
- most infections are acquired in childhood - the most common is direct transmission from an infected mother; but there are also new infections in adulthood, especially people with impaired immunity

Signs and Symptoms

Helicobacter colonizes mainly the mucosa of the **antrum** of the stomach, later the body, but also the cardia. The settlement is focal, not diffuse; therefore, a larger number of endoscopic biopsies is required for capture. Colonization of the gastroduodenal mucosa is accompanied by the development of **chronic gastritis**, which represents a heterogeneous group of inflammatory processes of various etiologies. Prolonged chronic gastritis caused by *H. pylori* can lead to mucosal atrophy and **intestinal metaplasia**, the most common precursor of intestinal gastric adenocarcinoma.

H. pylori infection has a causal relationship to the **peptic ulcer of the gastroduodenum**.

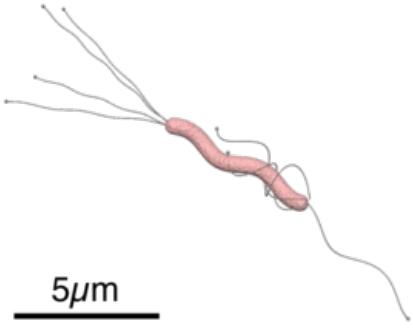


Helicobacter pylori in an electron microscope

The bacteria form a protein that stimulates gastrin production, which increases HCl secretion. They also produce mucus-disrupting proteases and phospholipases. They are also equipped with the enzyme catalase, which impairs the local ability of phagocytosis of cells of the immune system.

Disruption of the mucosal barrier defense mechanisms (surfactant, mucus, basement membrane of gastric epithelial cells) by bacteria is followed by the release of inflammatory metabolites of epithelial cells and is one of the most important factors in the pathogenesis of chronic gastritis and peptic ulcers. It is not known to what extent the degree of inflammation is affected by the characteristics of the host (genetic factors) or pathogen (phenotype, genotypes).

Eradication of *H. pylori* infection leads to **healing of the ulcer**.



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In addition, infection is associated with extraintestinal diseases and syndromes, such as:

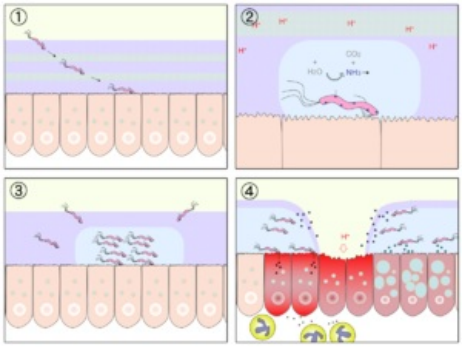
- **autoimmune disease** (idiopathic trombocytopenic purpura, autoimmune thyreoiditis)
- **skin condition** (acne, rosacea, idiopathic chronic urticaria)
- **endocrine disorders** (thyreopatia)
- **neurological disorders** (migraine)
- **hepatobiliary disease**
- **cardiac** (ICHS), **vascular** (Raynaudova choroba)
- **recurrent dyspepsia/discomfort**
- **Iron-deficiency anemia**
- **growth disorders**
- **larynx disorders**[2]
    1. benign: chronic laryngitis (incidence of infection in almost 46% of patients in the research), vocal cord polyps (singing nodule)

2. malignant: laryngeal tumors (incidence of infection in 46% of patients in the research)

Sufficient valid studies are still lacking to verify these connections.

Diagnosis

- a stool polymerase chain reaction (PCR) test
- Serological examination of antibodies against Helicobacter pylori by ELISA method
- Endoscopically obtained biopsy specimens can be tested by rapid urease test, histologically, or by culture (*Skirowa's soil*)
- ^{13}C-labeled urea breath test
- * Serological detection of preneoplastic markers may be useful in preventing gastric cancer

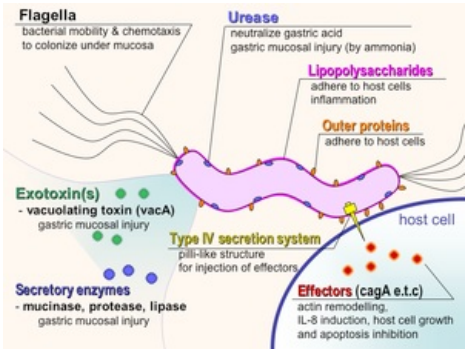


Mechanism of gastric ulcer formation

Treatment

- The gold standard of eradication is a triple combination of drugs given for 7 days:
  - **omeprazole + amoxicillin + clarithromycin (or any other macrolide)**
- If the patient has a penicillin allergy: omeprazole + metronidazole + clarithromycin

In recent years, there has been a decline in the successful eradication of H.p. after standard triple combination therapy with increased resistance to antibiotics, especially clarithromycin. Where there is increasing resistance to clarithromycin, bismuth quadruterium (bismuth) is recommended. Probiotics can reduce the incidence of side effects of standard eradication. The most successful eradication treatment is now the subject of numerous studies.



Helicobacter pylori virulence factors

Summary Video

Resources

Related articles

- Serological examination of antibodies against Helicobacter pylori by ELISA method
- 13C-labeled urea breath test
- Examination of Helicobacter pylori antigen in stool
- Peptic ulcer disease

External links

- Helicobacter pylori (Czech wikipedia)
- Helicobacter pylori (English wikipedia)
- Current view on the eradication of Helicobacter pylori (https://web.archive.org/web/20160331222721/http://zdravi.e15.cz/clanek/priloha-lekarske-listy/soucasny-pohled-na-eradikaci-helicobacter-pylori-450196)

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