Research paper helicobacter pylori

Helicobacter pylori| Previously named Campylobacter pyloridis, is a Gram-negative, microaerophilic bacterium found in the stomach. | | Microbiology (B1325) Research Paper| Detailed Introduction Helicobacter pylori are a species of epsilon proteobacteria which colonizes the harshenvironmentof the human stomach. Its name refers to both its spiral shape (Helicobacter) and the area of the lower stomach which it habitually colonizes: the gateway (pylorus) between the stomach and small intestine (Meyers, 2007).

This bacterium is thought to be present within up to 50% of the human population and has been linked to the development of a number of different medical conditions (Chalmers et al. 2004). This report will provide information about the discovery of H. pylori as well as its morphological characteristics, taxonomic information, biochemical/metabolic characteristics, chemotherapeutic methods of control/treatment/eradication, immunological responses, pathological information, and epidemiology information. Morphological Characteristics Helicobacter pylori are a spiral-shaped, Gram-negative rod approximately 0. x 3. 0 micrometers in size. It is catalase-positive organism which has 4-6 sheathed flagella attached to one pole which allow for motility. It lives in the human stomach and duodenum. H. pylori possess five major outer membrane protein (OMP) families. The largestfamilyincludes known and putative adhesions. The other four families include 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 O antigen of LPS may be fucosylated and mimic Lewis blood group antigens found on the gastric epithelium. The outer membrane also contains cholesterol glucosides, which are found in few other bacteria. H. pylori has four to six lophotrichous flagella; all gastric and enterohepatic Helicobacter species are highly motile due to flagella. The characteristic sheathed flagella filaments of Helicobacter are composed of two copolymerized flagellants, FlaA and FlaB. [1](From Wikipedia, the free encyclopedia). Taxonomic Information Helicobacter pylori are a gram-negative, spiral-shaped organism associated with gastrointestinal disease in humans.

It has a worldwide prevalence, with approximately 50% of the world's population infected. Before the first isolation and documentation of this organism from the human stomach in 1982, it was assumed that the human stomach was a sterile environment because of the high levels of acid, which would exclude it as an ecologic niche for any organism. This bacterium is the human-adapted Helicobacter primarily found in the gastric mucosa and areas of gastric metaplasia in the duodenum and occasionally in Meckel's diverticulum and the rectum. It has been cultured rarely from feces, blood, and saliva.

It can be detected by polymerase chain reaction (PCR) in dental plaque and feces. In the latter instances, the viability of the bacteria is in question. H. pylori also have been found in nonhuman primates and cats. H. pylori detection in animals is not common and could be due to human contact with animals. To date, no environmental reservoir has been shown. [2](http://www. gastro. theclinics. com/article/S0889-8553(05)70135-7/abstract) Biochemical/Metabolic

Characteristics The genus Helicobacter was created in 1989 with H. pylori as the type species. Since then the genus has expanded to include about 18 species.

Some species were reclassified from Campylobacter, but most were newly discovered microorganisms from gastric or intestinal sites in mammalian host animals. The essential property of almost all helicobacter is the presence of sheathed flagella. Most species possess strong ureolytic ability, particularly those associated with gastric mucosa, and exhibit considerable diversity in cell morphology withrespectto cell length, number and location of flagella, and presence of periplasmic fibrils. H. pylori have a global distribution and infect human gastric mucosa exclusively but there is some evidence for infection in cats.

Genomes of isolates from different individuals are unusual in their diversity in gene order and sequences within individual genes. 'H. heilmannii1 is another gastric spiral shaped organism less frequently infecting humans but commonly found in cat and dog gastric tissue. H. felis is important in the mouse model of infection. A range of conventional phenotypic tests as well as some new PCR based assays are available for identifying isolates of Helicobacter from clinical specimens. [11](http://bmb. oxfordjournals. org/content/54/1/17. full. pdf) Chemotherapeutic Methods of Control, Treatment, and Eradication

Treatment If you are found to have Helicobacter pylori infection, you may wish to have antibiotic treatment of some kind. Treatment of Helicobacter pylori is usually simple ; straight forward. However, occasional patients need repeated endoscopies, biopsies, breath tests and several courses of

treatment with different antibiotic combinations. After treatment of H. pylori, it is necessary to repeat one of these tests to see if the germ has been killed or eradicated for good. Only breath tests or endoscopy with biopsy can be used to prove that the bacterium has been eradicated.

The blood tests *(serology) is not suitable to monitor H. pylori eradication because antibodies to H. pylori may remain positive for months or even years after successfully killing the H. pylori. [3] (http://www. helico. com/? q= TreatmentForHelicobacterPylori) Eradication Prolonging the treatment period is a possible strategy for improving H. pylori eradication rates. Several studies have been published that tested this approach, including this paper by Calvet et al. These authors studied the value of extending PPI-based triple therapy from 7 to 10 days and found no additional benefit for patients with peptic ulcers.

There was, however, a significant benefit for nonulcer dyspepsia patients (an increase from 66% to 77% in the intention-to-treat analysis and from 73% to 91% in the per-protocol analysis). The authors concluded that the treatment period should be extended from 7 to 10 days for patients with nonulcer dyspepsia. As most eradication therapy, however, is given to patients with uninvestigated dyspepsia, it is not unreasonable to argue that longer therapy should be given to all subjects. Distinguishing between patients with ulcer and nonulcer dyspepsia is therefore ratheracademicand impractical.

The most obvious one is that existing PPI-based triple therapy regimens are not perfect. In the community at large, up to 30% of patients might fail this therapy. If clinicians prescribe triple therapy it should therefore be prescribed for longer than 7 days. This runs the risk of decreased patient

compliance, more side effects and a greater cost, but ultimately it boils down to local and national guidelines, which vary from one country to another. Alternatively, clinicians might consider some of the newer eradication approaches, such as use of fluoroquinolone-based therapy or sequential treatment.

The latter comprises quadruple therapy over a 10-day period, starting with a PPI plus amoxicillin (1, 000 mg twice daily) for the first 5 days, followed by PPI plus clarithromycin (500 mg twice daily) and tinidazole (500 mg twice daily) for another 5 days. Intention-to-treat analysis eradication rates of 97%, 92%, and 94% have been reported in children, adults and elderly patients, respectively. Ultimately, clinicians should still strive towards a much simpler eradication strategy, but this will require investment in novel antibiotic discovery or a better understanding of the pathogenesis of H. ylori. Either way, there is much to be gained from continued interest in this little organism. [4](http://www. medscape. com/viewarticle/525100_2) Immunological Responses Lifelong Helicobacter pylori infection and its associated gastric inflammation underlie peptic ulceration and gastric carcinogenesis. The immune and inflammatory responses to H. pylori are doubly responsible: gastric inflammation is the main mediator of pathology, and the immune and inflammatory response is ineffective, allowing lifelong bacterial persistence.

However, despite inducing gastric inflammation, most infections do not cause disease, and bacterial, host and environmental factors determine individual disease risk. Although H. pylori avoid many innate immune receptors, specific virulence factors (including those encoded on the cag

pathogenicity island) stimulate innate immunity to increase gastric inflammation and increase disease risk. An acquired T helper 1 response up regulates local immune effectors. The extent to which environmental factors (including parasite infection), host factors and H. ylori itself influence T-helper differentiation and regulatory T-cell responses remains controversial. Finally, effective vaccines have still not been developed: a better understanding of the immune response to H. pylori may help. [5](http://www. ncbi. nlm. nih. gov/pubmed/17382275) Pathological Information Until the discovery of Helicobacter in 1982, ulcers were thought to be caused bystress. Now it is known that ulcers, in addition to gastritis, are caused by a bacterial infection of H. pylori. Though relatively easy to treat with antibiotics, H. ylori can be a risk factor for gastric cancer if it becomes a long-term infection [6] (Stated by D. J. Kelly, 2004. The University of Sheffield). The body's natural defenses cannot combat H. pylori because white and killer T cells cannot easily get through the stomach lining. The defense cells eventually die, spilling their superoxide radicals on stomach lining cells, on which H. pylori can feed[6] (Stated by Helicobacter Foundation, 2004). Epidemiology Information The frequency of H pylori infection in the United States may be linked to race. White persons account for 29% of cases, and Hipic persons account for 60% of cases.

Internationally, H pylori are a ubiquitous organism. At least 50% of all people are infected, but an exact determination is not available, mostly because exact data are not available from developing countries. H pylori may be detected in approximately 90% of individuals with peptic ulcer disease; however, less than 15% of infected persons may have this disease. The

mortality rate related to H pylori infection is not precisely known, but it seems to be minimal (i. e. , approximately 2-4% of all infected people). Mortality is due to the complications of the infection, uch as gastric ulcer perforation or MALTomas of the GI tract. Otherwise, the morbidity of H pylori infection can be very high. [7](http://emedicine. medscape. com/article/176938-overview#a0199) The pathogenetic role of H pylori may differ depending on geography and race. White persons are infected with H pylori less frequently than persons of other racial groups. The prevalence rate is approximately 20% in white persons, 54% in African American persons, and 60% in Hipic persons. No sex predilection is known; however, females have a higher incidence of reinfection (5-8%) than males.

H pylori infection may be acquired at any age. According to some epidemiologic studies, this infection is acquired most frequently duringchildhood. Children and females have a higher incidence of reinfection (5-8%) than adult males. [7](http://emedicine. medscape. com/article/176938-overview#a0199) Cultural Characteristics Approximately two-thirds of the world’s population is infected with H. pylori. In the United States, H. pylori are more prevalent among older adults, African Americans, Hipics, and lower socioeconomic groups. It is not known how H. ylori are transmitted or why some patients become symptomatic while others do not. The bacteria are most likely spread from person to person through fecal-oral or oral-oral routes. Possible environmental reservoirs include contaminated water sources. Iatrogenic spread through contaminated endoscopes has been documented but can be prevented by proper cleaning of equipment. [8](Centers for disease and control prevention)Case Study1 Title: Correlation

of Helicobacter pylori and gastric carcinoma. Authors: Khanna, AK, Seth, P, Nath, G, Dixit, V K, Kumar, M Issue Date: 26-Jan-2002

Citation: Khanna AK, Seth P, Nath G, Dixit VK, Kumar M. Correlation of Helicobacter pylori and gastric carcinoma. Journal of Postgraduate Medicine. 2002 Jan-Mar; 48(1): 27-8 Language: Eng. Type: Journal Article Abstract: BACKGROUND: Difference of opinion about the prevalence of H. pylori association with gastric cancer exists in the literature. AIMS: To study the correlation of Helicobacter pylori (H. pylori) to gastric carcinoma. METHODS: 50 proved cases of gastric cancer were studied by rapid urease test, culture, histopathology and ELISA test for H. pylori IgG.

RESULTS: 68% of cases of gastric cancer were found to be positive for H. pylori infection as compared to 74% of healthy controls. CONCLUSIONS: The prevalence rate of H. pylori infection in our patients of gastric cancer was lower than in the control population though statistically not significant, suggesting that H. pylori may not be responsible for gastric carcinogenesis in this population. Source URI: http://www. jpgmonline. com URI: http://imsear. hellis. org/handle/123456789/116058 MeSH: * Adult * Case-Control Studies * Enzyme-Linked Immunosorbent Assay * Female Helicobacter Infections --complications * Helicobacter pylori --isolation & purification * Humans * Male * Middle Aged * Prevalence [9](http://imsear. hellis. org/handle/123456789/116058Stomach Neoplasms –microbiology) Case Study 2 Title: Helicobacter pylori in dental plaque of children and their family members. Authors: Gill, H H, Shankaran, K, Desai, H G Issue Date: 1-Sep-1994 Citation: Gill HH, Shankaran K, Desai HG. Helicobacter pylori in dental

plaque of children and their family members. Journal of the Association of Physicians of India. 1994 Sep; 42(9): 719, 721 Language: Eng.

Type: Journal Article Abstract: A prospective study was undertaken to determine the presence of Helicobacter pylori in the dental plaque of children and their family members. 22 children (age range: 2-12 years; males: 16) admitted to the pediatric ward for various disorders and 17 healthy family members (age range: 7-40 years; males: 9) of 13 of these children were screened for presence of Helicobacter pylori in the dental plaque by the rapid urease test. H. pylori were detected in dental plaque of 82% (18/22) children and 88% (15/17) of family members.

In 85% (28/33) of the positive cases the rapid urease test was positive within 1 hour. Our observations indicate that Helicobacter pylori are present in the dental plaque of majority of children and their family members. Source URI: http://www. japi. org URI: http://imsear. hellis. org/handle/123456789/95238 MeSH: * Child * Child, Preschool * Dental Plaque --microbiology * FamilyHealth* Female * Helicobacter pylori --isolation & purification * Humans * Male * Prospective Studies Appears in Collections: Journal of the Association of Physicians of India [10](http://imsear. ellis. org/handle/123456789/95238) Conclusion The author covered morphological characteristics, taxonomic information, biochemical/metabolic characteristics, chemotherapeutic methods of control/treatment/eradication, immunological responses, pathological information, and epidemiology in this paper. The overwhelming conclusion is that it is critical to survival of the human race that hygiene andeducationwill be the best possible steps to overcome an increasing body of bacteria in our world. References 1. From

Wikipedia, the free encyclopedia 2. http://www. gastro. theclinics. om/article/S0889-8553(05)70135-7/abstract) 3. http://www. helico. com/? q= TreatmentForHelicobacterPylori 4. http://www. medscape. com/viewarticle/525100_2 5. http://www. ncbi. nlm. nih. gov/pubmed/17382275 6. Stated by D. J. Kelly, 2004. The University of Sheffield 7. http://emedicine. medscape. com/article/176938-overview#a0199 8. Centers for disease and control prevention 9. http://imsear. hellis. org/handle/123456789/116058Stomach Neoplasms – microbiology 10. http://imsear. hellis. org/handle/123456789/95238 11. http://bmb. oxfordjournals. org/content/54/1/17. full. pdf