**Helicobacter pylori infection** is one of the most common bacterial infections in the world. It is responsible for most cases of chronic gastritis and peptic ulcer disease, and the main risk factor for gastric cancers (adenocarcinoma and gastric mucosa-associated lymphoid tissue lymphoma). Recent studies suggest an increased **H. pylori** prevalence in patients with various extra gastrointestinal disorders, including skin, cardiovascular, rheumatic, liver diseases [1,2] and respiratory disorders, including chronic obstructive pulmonary disease (COPD), bronchiectasis [3,4] and active pulmonary tuberculosi. The observed associations might be explained by a potential etiopathogenetic role of **H. pylori** infection in these disorders. Estimates suggest that half the world is infected with the bacteria, with an especially high rate of infection in Asia [5]. We know that **H. pylori** causes ulcers, but do all strains of **H. pylori** cause ulcers and why some people experience symptoms while others remain asymptomatic? If we can understand that we may be more efficient at treating and avoiding development of **H. pylori** antibiotic resistance. **Helicobacter pylori** are a spiral-shaped, microaerophilic and Gram-negative bacterium discovered by Marshall and Warren [6]. This bacterium possesses strain-specific virulence factors cytotoxin associated gene A (CagA) and vacuolating cytoxin gene A (VacA) that allow the organism to colonize the gastric mucosa, evade host defense and, finally, damage host tissue [7,8]. **H. pylori** colonization of the gastric mucosa stimulates the release of a variety of proinflammatory cytokines, including interleukin-1, IL-8 and tumor necrosis factor-a. Moreover, a crossmolecular mimicry between bacterial and host antigens exists in **H. pylori**-infected patients. Therefore, **H. pylori** might have a pathogenetic role in diseases characterized by abnormal activation of inflammatory mediators and/or induction of autoimmunity [9,10]. Diagnosis of **H. pylori** infection is based on noninvasive tests, such as serological methods, 14C urea breath test, and bacterial DNA sequences or bacterial antigen detection in stool by the **H. pylori** stool antigen (HpSA) test [11] which were preferred due to their simplicity and rapidity. In contrast, the direct detection and culturing of **H. pylori** from gastric biopsy specimens requires intensive gastroendoscopy [12]. Culture methods require an incubation period of at least 4-7 days. Several factors cause difficulty with culturing of the organism: patchy distributions of the organism on tissues, proper transportation to avoid oxygen and maintain a temperature of 4°C [13]. The presence of **H. pylori** or resistance to clarithromycin can be investigated on gastric tissue samples with molecular methods, such as polymerase chain reaction (PCR) and fluorescence in situ hybridization (FISH) [14]. Optimal treatment for **H. pylori** remains controversial, due to differences in number and type of drugs, dosing, and length of treatment. Quadruple therapy (QT) using omeprazole twice daily with bismuth subcitrate, metronidazole, and tetracycline 4 times daily for 10 days were reported by Korenowyk and Kolber [15]. Rizzato et al. [16] analyzed the genetic variability in cagA and someother selected genes of the **H. pylori** cytotoxin-associated genes pathogenicity island (cagC, cagE, cagL, cagT, cagV and cag Gamma) using DNA extracted from frozen gastric biopsies of 95 cagA+ patients with chronic gastritis or gastric cancer in Venezuela and Mexico by sequencing reactions. In this study they reported that polymorphisms in genes coding for energy-supply protein CagE and for the β-1 integrin recognizing CagL may also affect virulence, because they are necessary for a functional secretory system.

**References**


*Corresponding author: Mona Z Zaghloul, Microbiology Unit, Department of Clinical Pathology, Ain Shams University Hospitals, Cairo, Egypt, Tel. 02-24023494; E-mail: monazaki_810@hotmail.com*

**Accepted February 08, 2012; Published February 10, 2012**

**Citation:** Zaghloul MZ (2012) **Helicobacter pylori**. J Medical Microbiol Diagnosis 1:104. doi:10.4172/2161-0703.1000e104

**Copyright:** © 2012 Zaghloul MZ. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.