



## ***Review Article: Epidemiological Study of Helicobacter Pylori Bacteria in Wasit Governorate.***

Noor Majeed Abdulhasan<sup>1</sup>, Shahad kadhim Jaafar<sup>2</sup>

<sup>1</sup>*Department of Forensic Evidence, College of Science, University of Wasit, Iraq*

<sup>2</sup>*Department of Biology, College of education for pure Science, Wasit University, Iraq .*

### **Article Information**

Received: 1-3-2026

Accepted: 12-3-2026

Published: 1-4-2026

### **Abstract**

The study involved surveying recorded results of H. pylori infection in Wasit Governorate and its districts and subdistricts to identify the prevalence of this bacterium, the diseases it causes, and whether there is a relationship with seasonal climate changes. The research also includes a statistical analysis of infection rates over the past three years, from 2021 to 2024. The spread of H. pylori infection is closely linked to water quality, personal and public hygiene, as well as avoiding the use of items belonging to the infected person. Studies and research have found medicals treatment for H. pylori infection, known as triple therapy, to eradicate this bacterium.

The aim of the study is to review the high rates of Helicobacter pylori infection in developing countries, including Iraq. This study aims to review the prevalence of H. pylori\* bacteria in Wasit Governorate over the past three years, 2021-2023. . It is one of the highly significant pathogenic bacteria in recent years, as it can remain in water for more than 96 hours, which is enough time for the spiral bacteria to be transmitted. The environment and seasons play an important role in infections, as this type of bacteria can survive in water and food for certain periods, influenced by heat and environmental factors.

**Keyword:** Helicobacter pylori, Gram-negative, Infection; and bacteria

### **Introduction**

#### **1-1 Helicobacter pylori:**

Helicobacter pylori is a Gram-negative and spiral, multi-flagellated (5-7 flagella), is responsible for various upper gastrointestinal disorders, including chronic gastritis, peptic ulcer disease, and gastric carcinoma, microaerophilic bacterium that resides in the mucus layer above the gastric epithelial layer in humans (Sun, Q., Yuan et al.,2023).

## 2-1 History of *Helicobacter pylori*:

Discovery *H. pylori* was first discovered by the Australian scientists Marshall and Warren in 1982, when a large number of spiral or twisted cells were found in biopsies taken from the stomach walls of individuals with chronic gastritis. This bacterium was found in the gastric mucosal wall and closely associated with the surface of the gastric epithelial layer. It was previously believed that the acidic environment of the stomach prevented bacterial colonization in this medium (Marshall, B 2002). The hypothesis of the relationship between *Helicobacter pylori* bacteria and stomach ulcers was met with skepticism. Therefore, to prove his hypothesis, scientist Marshall drank a Petri dish containing a colony of organisms extracted from a patient's stomach, and he soon developed gastritis. The symptoms disappeared within two weeks, but he took antibiotics to eliminate the remaining bacteria at his wife's insistence, as bad breath is one of the symptoms of the infection. In 1984, the study was published in the Australian Medical Journal and is considered one of the most cited articles. In 2005, the Karolinska Institute in Stockholm awarded him the Nobel Prize in Physiology or Medicine for discovering the role of *Helicobacter pylori* in causing gastritis and stomach ulcers (Morena et al., 2007).

**3-1 Overview of *Helicobacter pylori* Bacteria** Classification of *Helicobacter pylori* (*H. pylori*) *Helicobacter pylori* was previously classified based on morphology and biochemistry as *Campylobacter pyloridis*. Later, the name of the bacterium was changed to *H. pylori* (Warren 1983 and Marshall). *Helicobacter pylori* was classified based on laboratory test results conducted after the bacterium was recorded using DNA sequencing techniques of deoxyribonucleic acid and ribosomal DNA. It was classified as the first species in a new genus independent of *Campylobacter* (Chey et al., 2017; Lee et al., 2021)..Strains of *H. pylori* bacteria There are two important strains of *H. pylori* bacteria:

1- The first type: It is pathogenic and contains the genes (Vaca, caga), and this type causes the appearance of disease symptoms.

2- The second type: It is non-pathogenic as it has been found not to contain the genes (Vaca, caga), and this type does not cause the appearance of disease symptoms. It has been found that the appearance of symptoms is associated with the type of bacterial strain, which explains the presence of the bacteria without the appearance of disease symptoms. Studies have shown that about 2% of the world's population carry the bacteria without showing disease symptoms (Liu et al., 2021; Zhang et al., 2022)

## 4-1 Microbiological characteristics of (*H. pylori*) bacteria:

*H. pylori* is an aerobic, microscopic, spiral-shaped bacterium with a length of 2.5 to 10  $\mu\text{m}$  and a width of 0.5 to 1  $\mu\text{m}$ . It has 4-6 polar flagella, each approximately 2.5 microns long and about 30 nanometers thick, which efficiently colonize the human stomach mucosa. It is Gram-negative, resistant to high acidity, inhabits the stomach mucosa, and overcomes gastric acid by producing alkaline ammonia around it. It attaches to the stomach wall after secreting special enzymes that protect it from high acidity (Farinha & Gascoyne, 2005). This bacterium causes inflammation of the stomach lining (gastritis) that may progress to ulceration and is also among the causes of stomach cancer (Fleisher et al., 2006). Infection with this bacterium has been recorded in up to 90% of patients suffering from ulcers, and infection with this bacterium is very common, especially in the Gulf and Arabian Peninsula regions due to many factors.

**5-1 Virulence factors of (*H. pylori*) bacteria** This type of bacteria is able to survive in the human stomach because it is uniquely equipped with defense mechanisms that protect it from gastric acid. These mechanisms include:

1- This bacterium secretes large amounts of the enzyme urease, which helps in the production of ammonia that neutralizes stomach acidity. (Ferwana *et al.*, 2015)

2- The microbe surrounds itself with a wall made of ammonia that protects it from stomach acid, which could otherwise kill it (Nakashima *et al.*, 2018)

3- The microbe acts like a corkscrew or a plug remover, twisting itself into the mucus layer that lines the stomach and protects it from digestive gastric juices. (Malfertheiner *et al.*, 2017)

### **6-1 Causes of the spread of H. pylori bacteria.**

Although about 50 percent of the world's population is colonized by *Helicobacter pylori*, the prevalence rate varies greatly depending on age, as well as the country's background, ethnicity, and socio-economic conditions (Czinn 2005). There is increasing and abundant evidence indicating that the spread of *H. pylori* infection is closely related to water quality. Nevertheless, there is no widespread practice of detecting *Helicobacter pylori* capable of surviving and spreading in aquatic environments. In developing countries, *H. pylori* can be spread through water, including sources such as streams and rivers (Akcem *et al.* 2000; Zatwarka *et al.* 2007). It has also been confirmed that childhood drinking water sources are a risk factor for *H. pylori* infection, with the highest prevalence recorded when the local river was the drinking water source and the lowest when the water was filtered or boiled.

In addition, Goodman and colleagues (1996) found that swimming in streams that serve as a source of drinking water and frequently consuming raw vegetables washed in contaminated water increases the likelihood of contracting duodenal infections. Other studies also support the link between *Helicobacter pylori* infection and the consumption of untreated water or untreated spring water (Carballo *et al.*, 1997). Developing countries bear the greatest burden, with more than 70% of children over the age of 15 being affected (Czinn, 2005).

In contrast, the developed world has witnessed, since the 1950s, a decrease in the prevalence of *Helicobacter pylori* (Rothen & Brenner 2003). Currently, about 20% to 30% of individuals are affected, and this decline in infection in the developed world is attributed to improvements in socioeconomic conditions, personal hygiene, and enhancements in drinking water quality. Although the natural habitat of *Helicobacter* is the human stomach, widespread infection can occur in the host through the bacteria's survival in the external environment (Brown 2000).

### **7-1 Mechanisms of Helicobacter pylori Transmission.**

The precise mechanisms involved in the transmission of *Helicobacter pylori* are not exactly known, but it is clear that bacterial entry into the human stomach can lead to infection. Several modes of *Helicobacter pylori* transmission have been proposed, including ingestion through the mouth (Raymond *et al.* 2000) and through feces from animal sources (Fox 1995) and via food and water. Despite the debate in this area, there is increasing evidence suggesting that the bacteria are transmitted through feces. Accordingly, bacterial colonies present on the surface of feces may reach water sources, turning these surfaces into transportable sources (Hult *et al.* 1996, Herrera 2004). Clinical studies (Xia & Telley 1997) indicate several causes and factors for the emergence of gastric bacteria, including:

1- Poor personal hygiene. 2- Using shared personal items. 3- The bacteria can be transmitted through saliva, making it easy for infection to spread among family members. 4- Poor sanitation, especially in developing countries suffering from inadequate sanitation services. 5- Consuming food and water contaminated with bacteria makes infection more common in crowded places.

6- The stomach bacteria are transmitted through contact with an infected person via certain negative behaviors such as sneezing or through contaminated feces, but they do not spread through blood.

### **8-1 Bacterial pathology (H. pylori)**

The bacteria enter the human body through the mouth and settles in the stomach and intestines. Over time, *Helicobacter pylori* can stimulate increased production of stomach acid, which causes many diseases affecting the upper digestive system. Among these diseases are gastritis and peptic ulcers, and in some cases, it can progress to stomach cancer (Fleisher et al. 2006). However, the main disease caused by *Helicobacter pylori* infection is peptic ulcer, and the most common symptoms of peptic ulcers are:

1- Burning or aching pain in the middle of the abdomen, vomiting blood that may appear bright red or dark brown with a grainy appearance resembling ground coffee.

2- Dark stool with a sticky, tar-like consistency, in addition to nausea, vomiting, loss of appetite, weight loss, abdominal bloating, and belching.

2-8: Laboratory Identification of *H. pylori* Bacteria This bacterium can be identified through several characteristics, including:

1- The properties of the oxidase enzyme, catalase enzyme, urease enzyme, and nitrate reduction.

2- H<sub>2</sub>S production in triple sugar agar and resistance to Thalidixic acid. The strong urease activity and unique carbohydrate properties help distinguish *H. pylori* from the genus *Campylobacter* (Nikolaos and Sibylle Koletzko, 2001).

### **9-1 Urease Enzyme Activity of Bacteria**

In 1924 B.C., the bioactivity of urease enzyme in gut of human were was recorded for the first time. At that time, it was believed that existence of enzyme action in the gastric wall remained not related to the presence of bacteria in the stomach. This association was not confirmed until 1968, when the absence of urease activity was observed in the stomachs of bacteria-free animals. To clarify further, urease activity is absent in the cell of mammals; therefore, existence of this enzyme in human gut indicates the presence of bacteria (Seiichi Kato et al., 2014).

### **10-1 Signs and symptoms of the disease that appear in the patient**

1. Abdominal pain: There is intestinal pain strongly associated with eating meals in the case of duodenal ulcers, and this pain appears three hours after eating. 2. Abdominal bloating and water retention: Saliva is produced quickly after vomiting to reduce acid in the esophagus, although this is more associated with gastroesophageal disease. 3. Nausea and severe vomiting. 4. Loss of appetite and weight. 5. Bloody vomiting: This can occur due to direct bleeding from a stomach ulcer or severe damage (Feldman M et al., 2016). After studies and research, a treatment called triple therapy was discovered to eradicate *Helicobacter pylori*. The triple therapy is applied for one week and consists of proton pump inhibitors such as the drugs omeprazole, clarithromycin, and amoxicillin. However, this may lead to increased bacterial resistance to antibiotics, resulting in the failure of the first stage of treatment and the need for the patient to repeat antibiotic therapy. Another method was used, such as quadruple therapy, which targets resistant strains, like the drug Salicylate–Bismuth, and Levofloxacin may also be used (Chey WD et al., 2017).

### **11-1 Epidemic or Disease Status of the Bacteria:**

Scientific studies have indicated that more than half of the world's population carries *H. pylori* bacteria. It was found that in industrialized countries, 50-60% of people carry it, with most cases occurring between

the ages of 50-60. In developing countries, however, infection begins at an earlier age and reaches high levels. Antibodies of the type IgG against *H. pylori* were found in the serum of people under 20 years old, meaning they are high in developing countries (86% in Vietnam, 85% in Nigeria, 54% in Taiwan) and low in industrialized countries (37% in the USA, 30% in the UK). About 15% of those infected develop it into genetic tumors. It is well established that the prevalence of infection increases with age and is higher among manual laborers. *H. pylori* is found in the human stomach in almost all countries worldwide, with nearly half of the global population infected. It has also been commonly isolated from non-human organisms such as the Mongolian gerbil (Takeshi W. et al., 1998). The infection rate has decreased alongside the industrial developments in the field of industry, and it is considered that the *H. pylori* infection rate in developing countries ranges between 70-90% of the population, while this rate decreases to 25-50% in developed countries, as illustrated in the figure (Barik. A. S., 2009). Statistical data were collected from the Wasit Health Department in Al-Kut, as recorded in the official records, to study the outcomes of *Helicobacter pylori* (*H. pylori*) infection for the years 2021-2023. Information on infected individuals was recorded, and numerous cases were documented through positive *H. pylori* test results, indicating the extent of the disease's prevalence.

The recorded cases were based on confirmed blood tests of patients to detect antibodies against the bacteria. Based on the recorded cases in Wasit Governorate, the infection was more common among females than males. The infection rates for males and females in 2021 were 20.5% and 35.16%, respectively, while in 2022 they were 47.83% and 51.66%, respectively. The number of infected individuals increased with age, reaching 85.16% for males and 106.75% for females in 2023. This is attributed to increased pollution levels in both drinking water and air, as well as the growing popularity of restaurants and fast food, which has impacted human health and led to a rise in *Helicobacter pylori* infections.. The findings of this study align with the research done by Khoder et al.,(2019) and Albadri,(2024),who discovered a notable correlation between *H. pylori* infections and factors such as gender, age, career, household overcrowding, drinking water source, and participants' gastrointestinal characteristics.

### **12-1 Genes Responsible for Causing the Disease**

There are two types of genes in bacteria responsible for causing the disease: (VacA) and (CagA). Studies have shown that there is a relationship between bacterial genes and the acute symptoms of the disease. It was found that the secretion of the Vacuolating toxin by the VacA gene is responsible for destroying cells and damaging epithelial cells, followed by the effect of the CagA gene, which is responsible for stomach and duodenal ulcers and stomach cancer. Many scientists have confirmed the close relationship between these genes and the genetic composition of the bacteria, meaning the different genes (Hatakeyama, 2004).

In order for bacteria to cause disease, they must evade the immune system through two main mechanisms: 1- Producing chemical components in their cell walls that are similar to molecules found in host cells, so the immune system does not recognize them because it is designed to ignore molecules present in the host, allowing the bacteria to escape. 2- Entering the mucous membrane of the stomach, which enables them to escape the immune system. This has been confirmed by several studies (Gabriel, et al., 2011).

### **13-1 Risk Factors**

Children are the most susceptible to developing *Helicobacter pylori* infection, and their risk is generally higher due to the lack of proper hygiene.

The risk of infection depends partially on the environment and living conditions, and it is higher if they:

- Live in developing countries.
- Share a home with other infected individuals.
- Live in crowded housing.
- Long-term use of NSAIDs (non-steroidal anti-inflammatory drugs) increases the risk of developing peptic ulcers( Mohammed Muhanad,2025)

#### **14-1 Prevention and Treatment of H. Pylori Bacteria Prevention**

There are several ways to prevent stomach bacteria, the most important of which are:  
1- We must wash our hands thoroughly with water and soap immediately after using the bathroom, as hygiene is considered one of the most important methods for preventing all diseases, not just stomach bacteria.

2- Maintaining home cleanliness, killing and controlling insects.

3- Limiting kissing when greeting people to prevent the transmission of infection.

4- Avoiding the use of personal items of an infected person to prevent the spread of infection.

5- We must wash vegetables and fruits well with water to clean them from the microbes that may be attached to them (Abadi A.T.B ,2017).

#### **Treatment**

When the presence of *Helicobacter pylori* bacteria is detected in a person with a peptic ulcer, the usual procedure is to eliminate the bacteria and allow the ulcer to heal (Abadi, A.T. Bet al.,2017).

#### **Conclusions**

1. Through the research, it was observed that the most common sources of infection are contaminated water as well as contaminated food, so attention must be given to the cleanliness of water and food to avoid infection.

2. The environment and seasons play an important role in infections, as this type of bacteria can survive in water and food for certain periods, influenced by heat and environmental factors.

#### **Recommendations**

1- A doctor should be consulted if disease symptoms appear to prevent the progression of infection in patients.

2- Other treatment methods should be found for this bacteria due to its widespread prevalence, as it has become more resistant to antibiotics.

3- Alternative natural treatment methods should be explored through the use of herbs and natural substances whose effectiveness has been proven.

#### **Reference**

**Abadi, A. T. B. (2017).** *Helicobacter pylori* treatment: New perspectives using current experience. *Journal of global antimicrobial resistance*, 8, 123-130.

**Albadri, M. (2024).** Study of epidemiological *Helicobacter pylori* infection among Iraqi patients. *Technium BioChemMed*, 8, 12-16.

- Azevedo, N. F., Almeida, C., Fernandes, I., Cerqueira, L., Dias, S., Keevil, C. W., & Vieira, M. J. (2008).** Survival of gastric and enterohepatic *Helicobacter* spp. in water: implications for transmission. *Applied and Environmental Microbiology*, 74(6), 1805-1811.
- Barik, A. S. (2009).** *Helicobacter pylori* Infection in Developing Countries :The Burden for how long . The Saudi Journal of Gastroenterology, 15(3) :201-207.
- Bellack, N. R., Koehoorn, M. W., MacNab, Y. C., & Morshed, M. G. (2006).** A conceptual model of water's role as a reservoir in *Helicobacter pylori* transmission: a review of the evidence. *Epidemiology & Infection*, 134(3), 439-449.
- Chey, W. D., Leontiadis, G. I., Howden, C. W., & Moss, S. F. (2017).** ACG clinical guideline: treatment of *Helicobacter pylori* infection. Official journal of the American College of Gastroenterology|ACG, 112(2), 212-239.
- Chey, W. D., Leontiadis, G. I., Howden, C. W., & Moss, S. F. (2017).** ACG clinical guideline: treatment of *Helicobacter pylori* infection. Official journal of the American College of Gastroenterology|ACG, 112(2), 212-239.
- Del Bello, B., Valentini, M. A., Mangiavacchi, P., Comporti, M., & Maellaro, E. (2004).** Role of caspases-3 and-7 in Apaf-1 proteolytic cleavage and degradation events during cisplatin-induced apoptosis in melanoma cells. *Experimental cell research*, 293(2), 302-310.
- Di Bartolomeis, S. M., & Moné, J. P. (2003).** Apoptosis: a four-week laboratory investigation for advanced molecular and cellular biology students. *Cell biology education*, 2(4), 275-295.
- Feldman, M., Sleisenger, M. H., Friedman, L. S., & Brandt, L. J. (2016).** Sleisenger and Fordtran's gastrointestinal and liver disease: pathophysiology, diagnosis, management.
- Ferwana, M., Abdulmajeed, I., Alhajiahmed, A., Madani, W., Firwana, B., Hasan, R., ... & Knawy, B. (2015).** Accuracy of urea breath test in *Helicobacter pylori* infection: meta-analysis. *World journal of gastroenterology: WJG*, 21(4), 1305.
- Fleischer, A. ; Ghadiri, A. ; Dessauge, F. ; Duhamel, M. and Rebollo, M.P., et.al . (2006).** Modulating apoptosis as a target for effective therapy . *Mollmmunal.* , 43 (8) , pp .1079-1065 .
- Gabriel Arismendi-MorilloI; Ileana HernándezI; Edgardo MengualI ; Alisbeth, F. UenmayorII; GiselaRomeroIII;& Maribel Lizarzába. (2011).** Comparison of three methods based on endoscopic gastricbiopsiesbiopsies for diagnosis of *Helicobacter pylori* active infection in a clinical setting . *Arq. Gastroenterol.* vol.48 no.3.
- Giao, M. S. ; Azevedo, N. F. ; Wilks, S. A. ; Vieira, M. J . and Keevil, C. W. (2008).** Persistence of *Helicobacter pylori* in heterotrophic drinking water biofilms. *Appl. Environ . Microbiol.* 74(19), 5898–5904.
- Hatakeyama Akopyants, N. S. ;Clifton, S. W.and Kersulyte D. (2004) .** Oncogenic mechanisms of the *Helicobacter pylori* CagA protein. *Nat. Rev Cancer.*
- Khoder, G., Muhammad, J. S., Mahmoud, I., Soliman, S. S., & Burucoa, C. (2019).** Prevalence of *Helicobacter pylori* and its associated factors among healthy asymptomatic residents in the United Arab Emirates. *Pathogens*, 8(2), 44.
- Lee, D. H., Ha, J. H., Shin, J. I., Kim, K. M., Choi, J. G., Park, S., ... & Jung, M. (2021).** Increased risk of severe gastric symptoms by virulence factors *vacAs1c*, *alpA*, *babA2*, and *hopZ* in *Helicobacter pylori* infection. *Journal of Microbiology and Biotechnology*, 31(3), 368.

- Liu, W., Tian, J., Hui, W., Kong, W., Feng, Y., Si, J., & Gao, F. (2021).** A retrospective study assessing the acceleration effect of type I *Helicobacter pylori* infection on the progress of atrophic gastritis. *Scientific Reports*, 11(1), 4143.
- Malfertheiner, P., Mégraud, F., O'Morain, C. A., Gisbert, J. P., Kuipers, E. J., Axon, A. T., ... & El-Omar, E. M. (2017).** Management of *Helicobacter pylori* infection—the Maastricht V/Florence consensus report. *Gut*, 66(1), 6-30.
- Marshall, B. (2002).** The discovery that *Helicobacter pylori*, a spiral bacterium, caused peptic ulcer disease. In *Helicobacter pioneers: Firsthand accounts from the scientists who discovered helicobacters, 1892-1982* (pp. 165-202). Blackwell.
- Mohammed, M. M. (2025).** Prevalence of *Helicobacter pylori* Infection Among Some Children Under 16 Years in Maysan Province, Iraq. *Journal of Medical and Life Science*, 7(3), 447-454.
- Nikolaos Konstantopoulos, HolgerRüssmann, Claudia Tasch , Thorsten Sauerwald, Hans Demmelmair, Ingo Autenrieth&SibylleKoletzko. (2001).** Evaluation of the *Helicobacter pylori* stool antigen test (HpSA) for detection of *Helicobacter pylori* infection in children *The American Journal of Gastroenterology* 96, 677–683.
- Raymond, J. ; Bergeret, M. & Kalach, N. (2008).** *Helicobacter pylori* infection in children. *Presse. Med.* 37, 513–518.
- Seiichi, Kato, Noriko Furuyama, Kyoko Ozawa,; Kenji Ohnuma ; and KazuieIinuma, (2014).** Long-term Follow-up Study of Serum Immunoglobulin G and munoglobulin A Antibodies After *Helicobacter pylori* Eradication. *Journal of the American Academy of Pediatrics* Print ISSN: 0031-4005. Online ISSN: 1098-4275.
- Sun, Q., Yuan, C., Zhou, S., Lu, J., Zeng, M., Cai, X., & Song, H. (2023).** *Helicobacter pylori* infection: a dynamic process from diagnosis to treatment. *Frontiers in cellular and infection microbiology*, 13, 1257817.
- Zhang, X., Li, C., Chen, D., He, X., Zhao, Y., Bao, L., ... & Xie, Y. (2022).** H. pylori CagA activates the NLRP3 inflammasome to promote gastric cancer cell migration and invasion. *Inflammation Research*, 71(1), 141-155.