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The Effect of *Helicobacter pylori* on the Human Digestive System

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Abstract*—Helicobacter pylori (H. pylori) is a spiral-shaped, Gram-negative bacterium that infects the human gastrointestinal tract and is considered a chronic infectious agent. A major risk posed by this bacterium is its ability to adapt to the host environment. Furthermore, it contributes to clinical disorders such as chronic gastritis, peptic ulcers, and gastric cancer. This study aims to elucidate the biological evolution of this bacterium, the mechanisms by which it alters the host environment, its interaction with the gut microbiota, and its pathogenic progression in humans. Some studies have revealed the ability of this bacterium to infect parts of the body outside the gastrointestinal tract, causing iron-deficiency anemia. It is also believed to be associated with other diseases related to the cardiovascular system, liver, and pancreas, as well as other neurological conditions. Studies have shown that children infected with H. pylori have intestinal patterns that differ from those of uninfected children, and it is believed that H. pylori infection may be eradicated with treatment using natural substances such as honey or aloe vera juice, and possibly even kill the bacteria*.

***Keywords*—** *Helicobacter pylori, digestive system, Stomach inflammation, Gut microbiota, Gastric Microbiome*.

I. Introduction

Helicobacter pylori, a spiral-shaped, gram-negative bacterium, colonizes the gastric mucosa. It is believed that more than half the world's population is infected, often without showing symptoms. Its continued presence in the human stomach is attributed to its evolutionary adaptation and its ability to alter physiological and immune processes in the stomach. Research shows that the human stomach is not a sterile environment, and that the presence or absence of *H. pylori* infection affects microbial diversity (1). The study of *H. pylori* is crucial because of its association with gastrointestinal diseases such as gastritis, peptic ulcers, and gastric cancer. This infection plays a significant role in the global burden of cancer, with *H. pylori* being a major contributor to the global prevalence of gastric cancer (2).

II. Literature Review

1-1: Origins in History and Evolution

The interaction between humans and *Helicobacter pylori* (*H. pylori*) is the product of a long-term co-evolutionary process, not mere chance. Researchers have found that *H. pylori* has migratory patterns and has adapted to regional host changes as it has evolved alongside humans originating in Africa (3). Genetic research has also revealed regional clustering among *H. pylori* strains, suggesting that human ethnic and population migrations have a significant impact on the bacteria's evolution (4). Achtemann *et al*. and Morellioz *et al*. have demonstrated the dynamic nature of the bacteria during chronic infection by confirming evidence of substantial genetic recombination across geographically diverse *H. pylori* strains (4, 5). Kodaman *et al*. have shown that host origin influences the likelihood and severity of *H. pylori*-associated gastric diseases (6)

1-2: Colonization and Transmission

Although transmission of infection between family members is still widespread, the Helicobacter pylori bacterium spreads mostly horizontally (7). Colonization of *H. pylori* begins in early childhood and may continue for life if not eliminated. According to measuring the density of bacteria in vivo, the severity of the disease and mucosal inflammation are linked to the bacterial load (8). The diversity of strains contributes to the widespread spread of infection by identifying rapidly changing genes that facilitate adaptation in different locations (9).
H. pylori's

1-3; Impact on the Gastric Microbiome

Natural stomach microbes affect the functioning of the digestive and immune systems, as *H. pylori* changes the microbial environment. Researcher Das and his colleagues showed that the bacteria modify network connections and microbial diversity within the microbial communities in the stomach. Other researchers discovered that infection affects natural and other microbes by influencing the physiological responses of the intestine (9,10,11).

1-4: The Effect of Helicobacter pylori on the Epithelial Cell Barrier

The primary function of epithelial cells is to form a barrier between the interstitial space and the intestinal lumen. Apical junctions play a crucial role in maintaining epithelial cell functions, preserving their proliferation, motility, and intercellular adhesion, as well as maintaining the apical-basal-lateral polarity of the cell. The sealing of epithelial cells is achieved through four junctions, the most important and common of which are the zonula occludens (ZO-1) protein, adhesion molecules (JAM-1), and other proteins. These junctions are targeted by many pathogenic bacteria capable of disrupting the intercellular connections. The other junctions are called adhesive junctions, vacuole junctions, and the fourth

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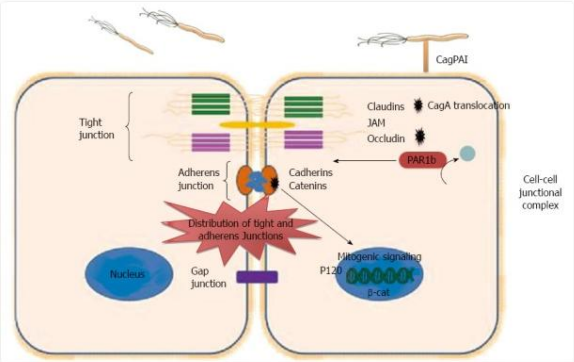
and final junction is the desmosome junction. See the figure below (12).



Dysregulation of epithelial junction by *Helicobacter pylori*. Intercellular junctions include tight junctions, adherens junctions, and gap. Tight junction consists of important Integral membrane proteins, such as occludin, claudins, and junctional adhesion molecules (JAMs), while adheren junction consists of Catenins and Cadherins. *Helicobacter pylori* (*H. pylori*) interact with cell-cell junctions and disrupt the polarity of polarized epithelial cells. Translocated CagA binds to PAR1b and inhibits its activity and blocks its phosphorylation by atypical protein kinase C, eventually causing junctional and polarity defects. CagA interacts with E-cadherin leads to the release of E-cadherin from the E-cadherin/β-catenin adherent complex, results in buildup of β-catenin at the cytoplasm and nuclear translocation of β-catenin and p120, and activation of β-catenin-mediated signaling that induces transformation of gastric epithelial cells. CagPAI: Cag pathogenicity island.

1-5: Pathogenicity and Disease Mechanisms

PAI is a genetic island, which is one of the most important pathogenic characteristics of Helicobacter pylori and is closely linked to high virulence and the risk of gastrointestinal diseases. Researchers have found that most isolates taken from infected people have a defect in cag-PAI, and this defect leads to diversity in bacterial strains and thus a diversity of disease mechanisms, the most important of which are manipulation of stomach acid, damage to epithelial cells, chronic inflammation of the stomach, and then the development of ulcers (13). Focus Hatakeyama focused on the molecular processes that lead to gastric cancer and discovered a genetic mechanism linked to *H. pylori*. In addition, infections are associated with adverse immune responses and inflammatory modulation, which may lead to damage to the gastrointestinal tract. Helicobacter pylori infection is a significant risk factor for peptic ulcer disease (14).

1-6: Gastrointestinal Diseases Associated with H. pylori

Gastrointestinal diseases associated with Helicobacter pylori. There is a clear relationship between disease severity and bacterial density. Gastritis and gastric ulcers occur in cases of persistent infection and damage to the gastric mucosa (14, 15). Helicobacter pylori is a leading cause of gastric cancer worldwide (16). Virulence factors of Helicobacter pylori are linked to the toxin gene (CagA), which affects the balance of gut microbiota (17), while cytotoxin A (VacA) promotes the death of gastric epithelial cells after damage (18).

1-7:Host-Microbe Dynamics and Coevolution

Numerous studies indicate that the genetic compatibility of the bacteria and the host influences the severity of the sickness and the results of digestion. Human-*H. pylori* coevolution decreases disease severity in genetically matched groups but increases illness in mismatched populations, as demonstrated by Kodaman *et al*. This coevolutionary hypothesis explains why certain populations experience severe illness(19).

1-8: Clinical Consequences for Digestive Health

Clinical gastroenterology requires an understanding of *H. pylori*. Risk factors and treatment approaches are determined by the presence or lack of certain virulence genes, strain lineage, and microbial interactions. Important clinical insights consist of: Not every infection results in illness. Genetic variation influences the course of infection The makeup of the microbiome affects the effectiveness of treatment. Research examining the genomes of isolates from Peru and Colombia (Devi *et al*. and Gutiérrez-Escobar *et al*.) shows geographical heterogeneity that might affect the effectiveness of therapy (20).

The gastric epithelium, composed of a single layer of cells covered with mucus, forms gastric glands or foci, which may be basal, cardiac, or pyloric (see figure below). The cardiac glands are lined with mucus-secreting cells and are located near the esophagus. The basal glands are located in the fundus and body of the stomach and consist of three parts: (1) principal cells that produce pepsinogen, (2) parietal cells that secrete hydrochloric acid and intrinsic factor, and (3) enterochromaffin-like cells responsible for histamine secretion. The pyloric glands are located in the antrum of the stomach and contain two types of cells: G cells, responsible for gastrin production, and D cells, responsible for somatostatin production. Helicobacter pylori primarily colonizes the antrum, which lacks parietal cells. Protecting underlying tissues from pathogenic organisms that may enter the gastric lumen is an additional function of the gastric epithelial cells, in addition to their primary function of digestion (21)

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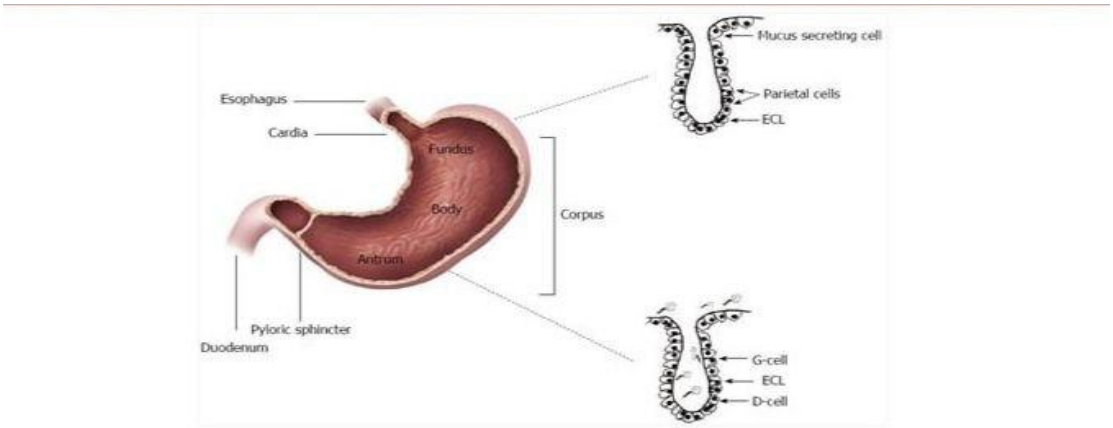
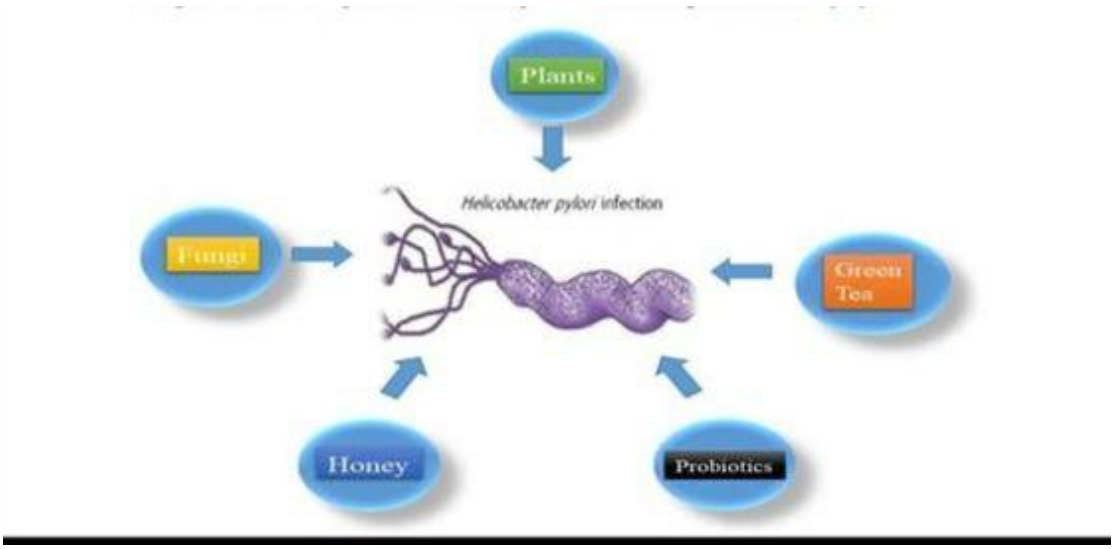


Helicobacter pylori colonization the antrum of the stomach. The gastric epithelium consists of a single layer of cells that invaginate in order to form cardiac, oxyntic or/and pyloric gland. Cardiac glands are located closest to the esophagus, while the oxyntic glands located in the fundus and corpus of the stomach and contain chief cells, parietal cells and enterochromaffine-like cells (ECL). Pyloric glands which located in the antrum contain G and D cells. *H. pylori* colonization is only limited to the antrum of the stomach.



III. Natural treatment

Studies have shown that natural remedies can eliminate Helicobacter pylori and alleviate gastritis. Bifid bacteria have positive effects on the digestive system due to their ion-producing properties. A study on mice demonstrated that green tea reduces the number of Helicobacter pylori bacteria in infected mice. The gel in aloe vera inhibits the growth of Helicobacter pylori strains and may even kill them. The antioxidants in honey also have antibacterial properties against Helicobacter pylori(22) The figure below illustrates the elimination of Helicobacter pylori using natural remedies in addition to probiotics(23)

IV. Conclusion

The ongoing evolution of Helicobacter pylori *(H. pylori*) is a cause for concern, as this bacterium plays a significant role as a highly evolved microbial partner, possessing a broad capacity to modify organ functions and the microbial environment. *H. pylori* causes significant gastrointestinal disturbances, ranging from simple gastritis to cancer, affecting mucosal integrity and immune responses. Understanding this *H. pylori* evolution requires a multidisciplinary scientific approach that integrates clinical medicine with molecular science, genetics, and other immunology disciplines to develop advanced methods for treating and preventing gastrointestinal infections. This review highlights the biological activity of this bacterium and the beneficial natural therapies for treating inflammation and eradicating the bacteria. It also reveals that children infected with H. pylori have intestinal patterns that differ from those of uninfected children. We recommend intensive study and research into the main mechanisms by which Helicobacter pylori alters the host epithelium to transform it into an immunosuppressive agent and the development of a protective vaccine to inhibit and control the bacteria.

Declaration of Conflict of Interest

There is no conflict of interest in this business

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