Journal of Medical Genetics and Clinical Biology
Homepage: https://journal.antispublisher.id/index.php/JMGCB
Email: admin@antispublisher.com

e-ISSN: 3032-1085
JMGCB, Vol. 2, No. 10, October 2025
Page 434-439

Impact of Infection by H. pylori on Gastric Parietal Cell Physiology: Review

Noor Zuhair Bakheet NZ
Jabir Ibn Hayyan University for Medical & Pharmaceutical Science, Iraq

DOI: https://doi.org/10.61796/jmgcb.v2i10.1407

Sections Info

Article history:
Submitted: June 30, 2025
Final Revised: July 08, 2025
Accepted: July 30, 2025
Published: August 18, 2025

Keywords:
Helicobacter pylori
Parietal cells
Hyperacidity
Hypoacidity
Gastric diseases

ABSTRACT

Objective: *This review aims to highlight the strategies used by Helicobacter pylori to influence the physiology of gastric parietal cells, affecting gastric acid secretion, epithelial elasticity, and the development of various gastric diseases.* ***Method:*** *The study reviews existing literature on Helicobacter pylori infection and its impact on gastric parietal cells, including mechanisms of colonization, inflammatory response, and physiological effects on the stomach and duodenum.* ***Results:*** *Helicobacter pylori infection can significantly affect parietal cells, which are essential for gastric acid production. Upon Helicobacter pylori infection, inflammatory cells replace normal glandular cells, leading to hypo- or hyperchloremia, based on the colonization site in the stomach. Chronic colonization of the gastric lumen promotes hyperacidity and duodenal ulcers, while hypoacidity resulting from long-term Helicobacter pylori infection of the gastric corpuscle and fundus increases the risk of gastric cancer and may damage these cells, gastric atrophy, and other complications.* ***Novelty:*** *In summary, Helicobacter pylori infection has a complex and multifaceted effect on the physiology of gastric parietal cells, affecting acid secretion, epithelial cell elasticity, and the development of various gastric diseases.*

INTRODUCTION

Parietal cells are specialized cells in the stomach lining, found primarily in the stomach fundus and body, where they form gastric glands. Their main functions are the secretion of hydrochloric acid and intrinsic factor [1], [2], [3]. Parietal cells produce and secrete hydrochloric acid, which is essential for digestion, breaking down food, and activating digestive enzymes such as pepsin. They also control bacteria, eliminating harmful bacteria that are digested with food. In addition, they aid in mineral absorption, facilitating the absorption of certain minerals such as iron. Parietal cells also produce intrinsic factor, a protein that binds to vitamin B12, facilitating its absorption in the small intestine [4], [5], [6].

Several mechanisms significantly regulate parietal cell secretion, including the vagus nerve, and hormonal like histamine, gastrin, somatostatin, and others. Various endocrine cells, including G-cells, D-cells, ECL (enteric chromaffin-like) cells, ghrelin cells, and others. G-cells excrete gastrin during ingestion in the stomach antrum. Gastrin stimulates ECL cells to excrete acid by liberating histamine and activating parietal cells [7], [8]. Somatostatin is produced from D-cells, and it's the main paracrine inhibitor of HCL secretion and GI motility [9], [10]. The precise regulation of parietal cells maintains the suitable secretion of hydrochloric acid. Normally, hydrochloric acid is generated by the gastric parietal cells, where water (H2O) combines with carbon dioxide (CO2) within the parietal cell cytoplasm to form carbonic acid (H2CO3). This occurs under the catalyst

of the enzyme carbonic anhydrase, and this carbonic acid quickly dissociates spontaneously into hydrogen ions (H+) and bicarbonate ions (HCO3-). The formed hydrogen ions are then transported into the gastric lumen via the H+–K+ ion pump, catalyzed by the enzyme H+-K+-ATPase expressed in parietal cells. This exchange of cytoplasmic hydrogen ions for extracellular potassium ions uses ATP as an energy source. Meanwhile, bicarbonate ions are transported out of the cell into the blood via a transporter protein called an anion exchanger, which transports bicarbonate ions outside the cell in exchange for chloride ions (Cl-). The chloride ions are transported via a chloride channel. Thus, the presence of hydrogen ions and chloride ions within the gastric lumen binds to form hydrochloric acid (HCl). The most effective method for inhibiting harmful gastric acid secretion is to inhibit the H+-K+-ATPase enzyme. Proton pump inhibitors and potassium-competitive acid antagonists are commonly used therapeutically to inhibit acid secretion. As well as their unique ability to secrete gastric acid, parietal cells play a crucial role in gastric mucosal homeostasis by secreting multiple growth factor molecules. So, gastric parietal cells play multiple roles in gastric secretion and protection, in addition to coordinating physiological repair [3].

RESEARCH METHOD

Helicobacter pylori (*H. pylori*) is a Gram-negative bacterium that selectively colonizes the epithelium of the human stomach [11], [12]. *Helicobacter pylori* is a type of bacteria that attacks the lining of your stomach and duodenum (the first part of your small intestine). While most people don't have symptoms. While most people don't exhibit symptoms, the bacteria can still cause irritation and inflammation, leading to conditions such as peptic ulcers and gastritis [13], [14]. It is transmitted through contaminated food and water, but human-to-human transmission is more common [15]. *Helicobacter pylori* colonization is primarily trapped in the gastric lumen, which is free from parietal cells and is abundant in gastric epithelial cells [16]. Although it participates in digestive processes, the primary role of mucosal epithelial cells is to keep the underlying tissues from pathogenic microorganisms that may enter the lumen [17]. The most popular outcome of infection with *Helicobacter pylori* is chronic gastritis, while chronic infection can drive clinically significant gastric and duodenal diseases [18], [12]. *H. pylori* utilizes a variety of mechanisms that help it adapt to the harsh environment of the gastric to survive and keep chronic infection [19]. Numerous studies have demonstrated the multiple effects of *Helicobacter pylori* on stomach epithelial cells, including the encouragement of apoptosis, cell proliferation, and the devastation of junctions in epithelial cells [20], [21]. Upon *Helicobacter pylori* infection, inflammatory cells replace normal glandular cells, resulting in hypo- or hyperchloremia based on the colonization site in the stomach [22]. Therefore, *Helicobacter pylori* has been identified as the primary pathogen of gastric ulcer [13]. Chronic colonization in the antrum of the stomach triggers hyperacidity and duodenal ulcer, while hypoacidity due to long-term *Helicobacter pylori* infection in the stomach fundus and corpuscle increases the risk of gastric cancer [16], [13], [23], [24]. Therefore, it is crucial to regulate gastric acid secretion with acid-lowering

drugs, which are the mainstay of treatment for gastrointestinal diseases, as HCL plays a crucial role in the evolution of diseases of the gastrointestinal system [25].

RESULTS AND DISCUSSION

Helicobacter pylori infection can significantly affect parietal cells, which are essential for gastric acid production. The infection can lead to a decrease in parietal cell numbers and a reduced ability to produce acid [26]. This occurs through various mechanisms. The first involves direct cell damage and disruption of signaling pathways that regulate parietal cell activity, affecting their ability to produce acid in response to stimuli. As for parietal cell damage and loss, *H. pylori* can damage E-cadherin, a protein essential for cell-to-cell adhesion of parietal cells, leading to their loss [27].The infection can contribute to gastric atrophy, causing a decrease in the overall number of parietal cells in the stomach lining. With parietal cell loss, mucus-secreting cells may replace the affected areas of the gastric glands [28]. The second mechanism involves bacteria that affect acid secretion. *Helicobacter pylori* interferes with the expression of the H+ and K+-ATPase, a proton pump essential for acid secretion, leading to impaired acid secretion. The overall effect is a reduction in the amount of acid produced by the stomach, which may have implications for digestion and contribute to some gastrointestinal disorders [29]. The third mechanism involves functional and morphological changes. Studies have appeared that *Helicobacter pylori* infection can lead to alterations in the appearance of parietal cells, including dilatation of small canals and vacuole-like structures, which are not typically seen in healthy cells. Importantly, the morphological changes and functional abnormalities in parietal cells are reversible after successful eradication of the *Helicobacter pylori* infection [30]. In essence, *Helicobacter pylori* infection can impair the structure and function of parietal cells, leading to decreased acid production and possibly contributing to various gastric diseases.

Effect of *Helicobacter pylori* on acid secretion; In acute infection, *Helicobacter pylori* infection often leads to a temporary reduction in acid secretion, possibly due to the bacteria's ability to interfere with proton pump activity and histamine release [31]. In chronic infection, it can lead to either increased or decreased acid secretion. Increased acid secretion due to *H. pylori* infection is a main risk factor for duodenal ulcers [32]. *H. pylori* can stimulate gastrin secretion and decrease somatostatin secretion, leading to increased acid production and hyperchlohydria. Decreased acid secretion, often associated with gastric atrophy, is a risk factor for gastric cancer [22]. *Helicobacter pylori* can promote decreased HCL due to parietal cell loss and the inflammatory effects of infection. *Helicobacter pylori* bacteria can cause gastric atrophy, resulting in a decreased number of parietal cells and consequently decreased acid secretion [28]. Finally, cytokines secreted during gastritis caused by *Helicobacter pylori* directly inhibit parietal cell function and histamine release from enterochromaffin-like cells, further impacting acid secretion [32]. *Helicobacter pylori* can regulate proton pump secretion, affecting gastric acidity and possibly influencing the composition of the gut microbiota [33]. This infection can alter the elasticity and homeostasis of gastric epithelial cells, contributing to

the development of gastric ulcers, atrophy, and possibly tumors [34]. *Helicobacter pylori* can disrupt the balance of gastrin and somatostatin, the hormones that regulate acid secretion, leading to changes in acid production [7].

This review aims to highlight the strategies used by *Helicobacter pylori* to influence the physiology of gastric parietal cells, affecting acid secretion, epithelial elasticity, and the development of various gastric diseases and complications resulting from secretory dysfunction. For example, autoimmune destruction of parietal cells by *Helicobacter pylori* leads to decreased vitamin B12 absorption and pernicious anemia. It also impairs iron absorption, which requires an acidic gastric environment and may contribute to iron deficiency anemia. Gastroenteritis is caused by a deficiency in gastric acid production (achlorhydria), which can be caused by damage to parietal cells, impairing digestion and increasing the risk of gastroenteritis. Gastritis is an inflammation of the stomach lining that can affect parietal cells. Gastroesophageal reflux disease (GERD) is a disorder of acid secretion that can contribute to the backflow of gastric acid into the esophagus and cause heartburn [35], [36], [37].

CONCLUSION

Fundamental Finding : This review aims to highlight the strategies used by *Helicobacter pylori* to influence the physiology of gastric parietal cells, affecting acid secretion, epithelial cell elasticity, and the development of various gastric diseases. *Helicobacter pylori* (*H. pylori*) infection can significantly affect parietal cells through proton pump expression, epithelial cell elasticity, and gastrin-somatostatin balance, which are essential factors for gastric acid production. **Implication :** All of this has led to *H. pylori's* ability to damage parietal cells, leading to decreased or increased acid secretion and potentially causing gastric atrophy and other complications, such as duodenal ulcers, gastric cancer, and iron deficiency anemia. The identification of these mechanisms emphasizes the clinical significance of *H. pylori* infection in altering gastric physiology and underscores the need for early diagnosis and targeted therapies. **Limitation :** Although the review provides a comprehensive overview of *H. pylori's* strategies in affecting parietal cells, it remains limited in addressing patient variability and host immune response that may modulate the degree of parietal cell damage. Additionally, the complexity of interactions between bacterial virulence factors and gastric microenvironment is not fully captured in the existing findings. **Future Research :** Future research should focus on investigating the molecular pathways through which *H. pylori* disrupts parietal cell function with greater precision, particularly in relation to host genetic susceptibility and immune modulation. Longitudinal clinical studies are also necessary to establish stronger causal links between these cellular changes and the progression toward specific gastric diseases.

REFERENCES

[1] C. D. Varela and N. Segura, "Histology, parietal cells," *StatPearls* [Internet]. StatPearls Publishing, May 1, 2023.

[2] M. Hsu, A. O. Safadi, and F. Lui, "Physiology, stomach," *StatPearls* [Internet]. StatPearls Publishing, Jul. 17, 2023.

[3] A. C. Engevik, I. Kaji, and J. R. Goldenring, "The physiology of the gastric parietal cell," *Physiol. Rev.*, vol. 100, no. 2, pp. 573–602, 2020.

[4] P. N. Rathore, S. Gupta, R. Madhariya, and A. Ram, "Vitamin B12 malabsorption: The pathophysiological impacts on neurological and hair physiology," *Int. J. Gastroenterol. Hepatol. Dis.*, vol. 4, no. 1, p. E26662906322948, Jan. 2025.

[5] R. Heda, F. Toro, and C. R. Tombazzi, "Physiology, pepsin," *StatPearls* [Internet]. StatPearls Publishing, May 1, 2023.

[6] H. P. Festen, "Intrinsic factor secretion and cobalamin absorption: physiology and pathophysiology in the gastrointestinal tract," *Scand. J. Gastroenterol.*, vol. 26, sup188, pp. 1–7, 1991.

[7] M. L. Schubert and J. F. Rehfeld, "Gastric peptides—gastrin and somatostatin," *Compr. Physiol.*, vol. 10, no. 1, pp. 197–228, Jan. 2020.

[8] J. H. Walsh, "Role of gastrin as a trophic hormone," *Digestion*, vol. 47, Suppl. 1, pp. 11–16, 1990.

[9] M. L. Schubert, "Hormonal regulation of gastric acid secretion," *Curr. Gastroenterol. Rep.*, vol. 10, pp. 523–527, 2008.

[10] G. Cui and H. L. Waldum, "Physiological and clinical significance of enterochromaffin-like cell activation in the regulation of gastric acid secretion," *World J. Gastroenterol.*, vol. 13, pp. 493–496, 2007.

[11] Y. Y. Cheok et al., "An overview of Helicobacter pylori survival tactics in the hostile human stomach environment," *Microorganisms*, vol. 9, no. 12, p. 2502, Dec. 2021.

[12] R. M. Peek and J. E. Crabtree, "Helicobacter infection and gastric neoplasia," *J. Pathol.*, vol. 208, pp. 233–248, 2006.

[13] V. E. Reyes, "Helicobacter pylori and its role in gastric cancer," *Microorganisms*, vol. 11, no. 5, p. 1312, May 2023.

[14] A. Elbehiry et al., "Helicobacter pylori infection: current status and future prospects on diagnostic, therapeutic and control challenges," *Antibiotics*, vol. 12, no. 2, p. 191, Jan. 2023.

[15] O. S. Joy, E. F. Aluko, O. R. Ayanbolade, and O. A. Olowe, "Public health perspectives of zoonotic potential of H. pylori," *Adv. Anal. Pathol.*, vol. 1, pp. 22–38, 2025.

[16] E. A. Marcus and D. R. Scott, "Gastric colonization by H. pylori," in *Helicobacter pylori*. Singapore: Springer Nature Singapore, 2024, pp. 25–37.

[17] L. E. Wroblewski and R. M. Peek, "Targeted disruption of the epithelial-barrier by Helicobacter pylori," *Cell Commun. Signal.*, vol. 9, p. 29, 2011.

[18] Z. Hua et al., "Helicobacter pylori infection altered gastric microbiota in patients with chronic gastritis," *Front. Cell. Infect. Microbiol.*, vol. 13, p. 1221433, Aug. 2023.

[19] L. Zhang, X. Chen, B. Ren, X. Zhou, and L. Cheng, "Helicobacter pylori in the oral cavity: current evidence and potential survival strategies," *Int. J. Mol. Sci.*, vol. 23, no. 21, p. 13646, Nov. 2022.

[20] D. K. Sah, A. Arjunan, B. Lee, and Y. D. Jung, "Reactive oxygen species and H. pylori infection: a comprehensive review of their roles in gastric cancer development," *Antioxidants*, vol. 12, no. 9, p. 1712, Sep. 2023.

[21] H. H. Xia and N. J. Talley, "Apoptosis in gastric epithelium induced by Helicobacter pylori infection: implications in gastric carcinogenesis," *Am. J. Gastroenterol.*, vol. 96, pp. 16–26, 2001.
[22] S. K. Bashir and M. B. Khan, "Overview of Helicobacter pylori infection, prevalence, risk factors, and its prevention," *Adv. Gut Microbiome Res.*, vol. 2023, no. 1, p. 9747027, 2023.
[23] R. M. Genta, "Helicobacter pylori, inflammation, mucosal damage, and apoptosis: pathogenesis and definition of gastric atrophy," *Gastroenterology*, vol. 113, no. 6 Suppl., pp. S51–S55, 1997.
[24] A. Lee et al., "Local acid production and Helicobacter pylori: a unifying hypothesis of gastroduodenal disease," *Eur. J. Gastroenterol. Hepatol.*, vol. 7, pp. 461–465, 1995.
[25] H. Patel, A. Kwamboka, D. Magembe, E. Manyinsa, and K. Ouma, "Valuation of prescription patterns of Helicobacter pylori drugs in outpatient department, Meru Teaching and Referral Hospital," *Afr. J. Health Sci.*, vol. 36, no. 6, pp. 733–738, 2023.
[26] X. Yao and A. J. Smolka, "Gastric parietal cell physiology and Helicobacter pylori–induced disease," *Gastroenterology*, vol. 156, no. 8, pp. 2158–2173, Jun. 2019.
[27] M. A. Ferraz et al., "Downregulated expression of e-cadherin and tp53 in patients with gastric diseases: the involvement of H. pylori infection and its virulence markers," *J. Gastrointest. Cancer*, vol. 47, no. 1, pp. 20–26, Mar. 2016.
[28] Y. K. Wang et al., "How does Helicobacter pylori infection cause gastric mucosal atrophy," *Infect. Drug Resist.*, vol. 15, pp. 3619–3629, Jan. 2022.
[29] H. Aslam, A. U. Khan, N. G. Qazi, F. Ali, S. S. Hassan, and S. Bungau, "Pharmacological basis of bergapten in gastrointestinal diseases focusing on H+/K+ ATPase and voltage-gated calcium channel inhibition: A toxicological evaluation on vital organs," *Front. Pharmacol.*, vol. 13, p. 1005154, Nov. 2022.
[30] Y. K. Wang et al., "Histopathological features of Helicobacter pylori infection in gastric mucosa," *J. Inflamm. Res.*, vol. 15, pp. 6231–6243, Jan. 2022.
[31] R. Veysey-Smith et al., "Effects of proton pump inhibitor therapy, H. pylori infection and gastric preneoplastic pathology on fasting serum gastrin concentrations," *Front. Endocrinol.*, vol. 12, p. 741887, Nov. 2021.
[32] J. Geibel, "Gastric secretions," in *Yamada's Textbook of Gastroenterology*. Hoboken, NJ: Wiley, Apr. 2022, pp. 313–333.
[33] A. Minalyan, L. Gabrielyan, D. Scott, J. Jacobs, and J. R. Pisegna, "The gastric and intestinal microbiome: role of proton pump inhibitors," *Curr. Gastroenterol. Rep.*, vol. 19, no. 8, p. 42, Aug. 2017.
[34] Y. Huang, Q. L. Wang, D. D. Cheng, W. T. Xu, and N. H. Lu, "Adhesion and invasion of gastric mucosa epithelial cells by Helicobacter pylori," *Front. Cell. Infect. Microbiol.*, vol. 6, p. 159, Nov. 2016.
[35] S. K. Motupalli and T. L. Oroszi, "The nexus between Helicobacter pylori infection and anemia—a systematic review," *Front. Hematol.*, vol. 3, p. 1423494, Aug. 2024.
[36] H. Aljaberi et al., "Current understanding of the transmission, diagnosis, and treatment of H. pylori infection: A comprehensive review," *Int. J. Med. Pharm. Drug Res.*, vol. 7, p. 2, 2023.
[37] V. V. Lupu et al., "Iron deficiency anemia in pediatric gastroesophageal reflux disease," *Diagnostics*, vol. 13, no. 1, p. 63, Dec. 2022.

***Noor Zuhair Bakheet NZ (Corresponding Author)**
Jabir Ibn Hayyan University for Medical & Pharmaceutical Science, Iraq
Email: noor.z.bakheet@jmu.edu.iq