MARSZAŁEK, Dominika, CZUDOWSKA, Michalina, ZAWADZKA, Magdalena, KURZĄTKOWSKA, Klaudia, BYSTROS, Aleksandra, DROZDOWSKA, Marta, BORYCHOWSKA, Emilia, OCIMEK, Aleksandra, GWÓŹDŹ, Karolina, and MIERZEJEWSKA, Zofia Aneta. Management of *Heliobacter pylori* Infection: Current Standards and Future Perspectives. Journal of Education, Health and Sport. 2026;87:67506. eISSN 2391-8306.
https://dx.doi.org/10.12775/JEHS.2026.87.67506
https://apcz.umk.pl/JEHS/article/view/67506

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).© The Authors 2024;
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The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 15.12.2025. Revised: 21.12.2025. Accepted: 10.01.2026. Published: 11.01.2026.

Management of *Helicobacter pylori* Infection: Current Standards and Future Perspectives

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ABSTRACT

Helicobacter pylori infection remains a major clinical challenge due to its high global prevalence, rising antibiotic resistance, and the increasingly limited effectiveness of traditional treatment approaches. Although several diagnostic and therapeutic strategies are well established, eradication rates continue to decline in many regions. Effective management now requires careful selection of treatment based on local resistance patterns, adequate acid suppression, and attention to treatment adherence. Current guidelines support the use of bismuth-containing quadruple therapy, concomitant therapy, and susceptibility-guided approaches where available. Emerging treatment options—including potassium-competitive acid blockers, rifabutin-containing therapies, antimicrobial peptides, microbiome-modulating interventions, and vaccine candidates—show promising potential to improve future outcomes. Continued progress in diagnostics and access to newer therapies may enable more individualized, effective, and preventive strategies in the management of *H. pylori* infection.

Materials and methods

The article is a result of the review of the scientific literature searched by keywords "*Helicobacter pylori* ", "eradication", "antibiotic resistance", "bismuth quadruple therapy", and "potassium-competitive acid blockers" available in the PubMed database. This overview focuses on current standards, guidelines, and selected emerging therapies important for a comprehensive strategy for managing *H. pylori* infection.

Aim of the study

The aim of the study is to review the current challenges and evidence-based strategies for *Helicobacter pylori* eradication, with a particular focus on optimizing treatment in the era of increasing antibiotic resistance and evaluating future therapeutic options.

Keywords: *Helicobacter pylori*, radication, antibiotic resistance, bismuth quadruple therapy, potassium-competitive acid blockers, vaccine development

1. Introduction

Helicobacter pylori (H. pylori) remains one of the most significant bacterial pathogens worldwide, with an estimated global prevalence of nearly 50% [1], accompanied by substantial regional variation shaped by socioeconomic conditions, hygiene standards, and early-life exposure patterns [2]. Epidemiological data from Poland also indicate persistently high infection rates among adults, with a considerable proportion of cases observed in pediatric populations [3–5], underscoring the ongoing need for effective and accessible diagnostic and therapeutic strategies [11,12].

Despite the long-standing availability of antibiotic-based eradication regimens, the clinical management of *H. pylori* has become increasingly difficult. The reduced effectiveness of traditional triple therapy is primarily driven by rising antimicrobial resistance to clarithromycin, metronidazole, and fluoroquinolones [6–8]. Monitoring data from Europe and Asia consistently show increasing antibiotic resistance, which reduces treatment success and requires updates to standard therapeutic recommendations [6,7]. As a result, recent international guidelines now favor bismuth-containing quadruple therapy, non-bismuth concomitant regimens, and susceptibility-guided approaches as preferred first-line strategies [11]. These global trends have also influenced current national recommendations in Poland [12].

At the same time, advances in pharmacotherapy have introduced new therapeutic opportunities. Potassium-competitive acid blockers (PCABs), such as vonoprazan, offer more stable and potent acid suppression than proton pump inhibitors and have demonstrated high eradication rates across different clinical settings [14]. Additional antimicrobial options, including rifabutin-based rescue therapies, show promise for patients with multidrug resistant infections [15]. Beyond antibiotics, emerging research focuses on innovative modalities such as antimicrobial peptides, microbiome-modulating interventions, nanotechnology-enhanced drug delivery, and next-generation vaccine candidates [18–21,24,25]. Overall, these developments are moving clinical practice toward individualized therapy based on resistance data, patient factors, and local epidemiology [16,17].

Given the increasing complexity of therapeutic decision making and the ongoing risk posed by rising antimicrobial resistance, an updated synthesis of current standards is essential. This review provides an overview of current evidence based therapies, key clinical challenges, and emerging treatment strategies for *H. pylori.*

2. Epidemiology

The prevalence of *Helicobacter pylori* varies widely across the world and is strongly influenced by socioeconomic conditions, sanitation, and early-life living environments [2]. In many low- and middle-income countries, infection rates may reach 70–80%, largely due to early childhood exposure, crowded living conditions, and limited access to clean and safe water [2].

In contrast, most high-income countries report prevalence below 30%, reflecting improvements in hygiene, public health infrastructure, and overall living standards, with a continued decline observed in Western Europe, North America, and East Asia [1,2].

Polish epidemiological data show a similar downward trend. Earlier studies reported adult prevalence exceeding 70% [3], whereas more recent findings indicate lower infection rates, although *H. pylori* remains present across all age groups, including children [4,5]. Despite decreasing prevalence, the infection continues to be clinically relevant, especially given the rising challenge of antibiotic resistance [6–8].

3. Pathogenesis and Mechanisms of Colonization

3.1 Virulence factors

1. *Helicobacter pylori* expresses several virulence factors that allow it to adapt to the acidic gastric environment and establish long-term colonization [13]. One of the most important is urease, an enzyme that neutralizes gastric acid and creates conditions that support bacterial survival. Flagella provide motility, enabling the bacterium to move through the gastric mucus layer and reach the epithelial surface [13].

2. Adhesins such as BabA and SabA facilitate binding to gastric epithelial cells, supporting stable colonization and promoting persistent infection [13]. Two additional virulence-associated proteins, CagA and VacA, play major roles in gastric injury and disease progression [13]. After translocation into host cells, CagA disrupts normal signaling pathways and triggers inflammatory responses, while VacA induces epithelial vacuolization, mitochondrial dysfunction, and immune evasion [13].

3. Together, these mechanisms support chronic gastric inflammation and increase the risk of progressive mucosal damage and malignant transformation [22].

4. Diagnosis of *Helicobacter pylori* Infection

Accurate diagnosis of *Helicobacter pylori* infection is essential for selecting the appropriate treatment, confirming eradication, and reducing the long-term risk of complications such as peptic ulcer disease and gastric cancer [11]. Diagnostic tools include non-invasive tests, which are generally preferred during initial evaluation, and invasive endoscopic methods, which offer additional clinical information and allow assessment of antibiotic resistance [11,12].

4.1 Non-Invasive Diagnostic Methods

The urea breath test (UBT) is considered the most reliable non-invasive method due to its high sensitivity and specificity [11]. When ^13C-labelled urea is used, UBT can accurately detect active infection and is recommended for both initial diagnosis and post-treatment confirmation [11].

Stool antigen testing using monoclonal antibodies provides diagnostic accuracy comparable to UBT. It is a practical option when breath testing is not available. To reliably confirm eradication, testing should be performed at least four weeks after completing therapy [11].

Serological assessment detects IgG antibodies but cannot distinguish active from past infection. Therefore, it is not recommended for confirming eradication. However, serology may be useful in selected cases, such as in patients with gastric atrophy or in those taking proton pump inhibitors, where other diagnostic methods may give false-negative results [11].

Although stool antigen testing is currently the main non-invasive alternative to UBT, ongoing technological development may lead to more advanced stool-based diagnostic tools in the future. These could offer improved detection accuracy and provide additional information relevant for personalized treatment approaches [11].

4.2 Invasive Diagnostic Methods

Endoscopy with biopsy is indicated in patients presenting with alarm symptoms, gastrointestinal bleeding, or an increased risk of gastric cancer [11]. Biopsy samples allow several complementary diagnostic procedures:

- Rapid urease test (RUT) – provides quick and specific detection of urease activity [11].
- Histology – essential for evaluating gastritis, intestinal metaplasia, dysplasia, and neoplastic changes [11].

- Culture with antibiotic susceptibility testing (AST) – considered the reference standard for assessing resistance, although its use is limited by technical challenges and variable availability in many centers [6–8].

PCR-based genotyping of gastric tissue can identify mutations linked to clarithromycin and fluoroquinolone resistance [7,8]. Although not routinely performed in all clinical settings, this method is becoming increasingly relevant in regions with rising antimicrobial resistance [6–8].

5. Current Treatment Strategies

Therapeutic strategies for *Helicobacter pylori* infection have evolved considerably in recent years, mainly due to rising antibiotic resistance and the declining effectiveness of clarithromycin-based triple therapy [6–8,11]. Current guidelines emphasize quadruple therapy, effective acid suppression, and susceptibility-guided treatment when available [11]. Successful eradication depends on choosing appropriate antibiotics, ensuring adequate treatment duration, and maintaining stable gastric acid suppression [11,14,16].

5.1 First-Line Treatment Options

Current guidelines recommend the following first-line therapies for *H. pylori* infection [11]:

- Bismuth quadruple therapy (BQT):
  PPI + bismuth + tetracycline + nitroimidazole; recommended for 14 days and effective even where metronidazole resistance is common [11].
- Non-bismuth concomitant therapy:
  PPI + amoxicillin + clarithromycin + nitroimidazole; suitable only in regions with clarithromycin resistance <15% [11].
- PCAB-based triple therapy (vonoprazan regimens):
  Vonoprazan + amoxicillin ± clarithromycin; offers stronger acid suppression and higher eradication rates compared with traditional PPI-based regimens, including in settings with moderate clarithromycin resistance [14,26,27]. PCAB-based therapies are currently available only in some countries and remain limited in many regions [14].

These therapies represent the main evidence-based options currently recommended for initial eradication [11,16].

5.2 Second-Line and Rescue Therapies

When first-line therapy fails, current guidelines recommend choosing regimens that contain antibiotics not previously used [11,16].

- Levofloxacin-based triple therapy
  A possible option when local fluoroquinolone resistance is low, although its usefulness has declined due to increasing resistance worldwide [6–8].
- Rifabutin-based triple therapy
  PPI + amoxicillin + rifabutin; an effective rescue regimen with high eradication rates, including in multidrug-resistant infections [15].
- Susceptibility-guided therapy
  Culture-based or molecular testing (e.g., PCR for clarithromycin or quinolone resistance) improves eradication outcomes and is preferred when available [11].

These approaches provide alternatives after first-line treatment failure and support more individualized therapy [11,16,17].

5.3 Optimizing acid suppression and treatment duration

Adequate acid suppression is essential for improving antibiotic effectiveness and overall treatment outcomes [11,14]. PCABs provide more stable gastric pH control than PPIs and help reduce variability related to CYP2C19 metabolism [14,26]. Most current guidelines recommend 14-day regimens for both bismuth and non-bismuth therapies, as shorter treatment durations are consistently associated with lower eradication rates [11].

5.4 Treatment Recommendations in Poland

Current treatment practice in Poland follows the Maastricht VI recommendations, adjusted for local antimicrobial resistance patterns [11,12]. Because clarithromycin resistance in Poland clearly exceeds 15%, clarithromycin-based triple therapy is no longer recommended as a first-line option [6–8].

The preferred first-line treatment is:

- Bismuth quadruple therapy (14 days) — PPI + bismuth + tetracycline + nitroimidazole [11].
- Non-bismuth concomitant therapy (14 days) – may be used when bismuth is not available or not tolerated [11,12].

For patients who fail first-line treatment, treatment should include antibiotics not previously used [11,16]. Levofloxacin-based therapy may be considered in selected cases,

although rising fluoroquinolone resistance limits its usefulness [6–8]. Rifabutin-based therapy and susceptibility-guided approaches are recommended when available [11,15].

6. Challenges in Current Therapy

Several treatment options for *Helicobacter pylori* are well established, but achieving successful eradication is still difficult in clinical practice. The effectiveness of treatment may be reduced by factors such as increasing antibiotic resistance, variable acid suppression, and challenges with adherence [6–8,11,16]. Resistance levels vary between regions, which can make it challenging to apply one universal treatment approach [6–8]. New therapies are emerging, but limited access and early evidence of resistance show that important challenges remain [14,26,27].

6.1 Antibiotic resistance

Antibiotic resistance is widely recognized as the leading cause of treatment failure. Clarithromycin resistance now exceeds the 15% threshold in most parts of the world, including Europe, and metronidazole resistance is particularly common in low- and middle-income countries [6–8]. Fluoroquinolone resistance has also increased due to widespread use of these drugs in general medicine [7,8].

These patterns reduce overall eradication rates and often lead to repeated treatment attempts, higher healthcare use, and a need for alternative therapeutic options [6–8,16].

6.2 Acid Suppression Variability and Treatment Adherence

Effective eradication depends on maintaining adequate gastric pH to support antibiotic activity. Proton pump inhibitors can provide inconsistent acid suppression, partly because CYP2C19 metabolism varies between individuals [11,14,16]. Potassium-competitive acid blockers (PCABs) offer more stable pH control and have shown improved outcomes in clinical studies, but access to these agents remains limited in many countries [14,26,27].

Treatment adherence is another important factor that affects eradication success. Standard therapies often require multiple daily doses and may cause gastrointestinal side effects, which can discourage patients from completing the full course of treatment [11,16]. Even small deviations from dosing schedules can significantly reduce treatment effectiveness [16].

6.3 Regional differences in resistance

Resistance profiles vary markedly between countries and even between local populations. Polish studies have demonstrated high rates of clarithromycin and metronidazole resistance,

which limit the usefulness of traditional triple therapy [9,10]. Limited access to culture or molecular susceptibility testing in many clinical settings further complicates individualized treatment decisions and often forces clinicians to rely on empirical choices [9,10].

7. Emerging and Future Therapies

Growing antibiotic resistance and the limited effectiveness of traditional treatment options have encouraged the development of new approaches for *Helicobacter pylori* [20,21]. Current research focuses on improved acid suppression, alternative antimicrobials, microbiome-based strategies, vaccine candidates, and advanced drug-delivery systems [20,21]. Although many of these methods remain under investigation, several show meaningful potential for future clinical use [20,21].

7.1 Potassium-Competitive Acid Blockers (P-CABs)

Potassium-competitive acid blockers, such as vonoprazan, provide stronger and more stable acid suppression than proton pump inhibitors [14,26,27]. Improved pH control enhances the activity of antibiotics, particularly amoxicillin and clarithromycin. Clinical studies have reported higher eradication rates with vonoprazan-based therapies compared with standard PPI-based options, including in populations with elevated clarithromycin resistance [14].

These findings suggest that PCABs may become an important part of future treatment strategies, although their use remains limited in many regions due to restricted availability [14,26].

7.2 Alternative Antibiotics and Adjunctive Strategies

7.2.1 Rifabutin-based therapy

Rifabutin-containing therapy shows high effectiveness in patients with multidrug-resistant infections, with eradication rates frequently above 80% [15]. Resistance to rifabutin remains low, making it an important rescue option and a potential future alternative when traditional antibiotics fail [15].

7.2.2 Sitafloxacin

Sitafloxacin, a fourth-generation fluoroquinolone, has demonstrated high efficacy against *H. pylori* in studies from Asia, including against strains resistant to other fluoroquinolones [20]. However, concerns regarding adverse effects and emerging resistance currently limit its broader international application [20].

4.

7.2.3 Probiotics and Microbiome Modulation

Probiotics such as *Lactobacillus, Saccharomyces boulardii*, and *Bifidobacterium* may reduce treatment-related gastrointestinal symptoms and modestly improve eradication rates when used alongside standard therapy [18,19]. While they are not recommended as independent treatment, they may support adherence and tolerability [18]. Broader microbiome-targeted approaches are also being explored to restore gastric microbial balance and potentially decrease the risk of reinfection [18–21].

Antimicrobial peptides (AMPs) disrupt the bacterial membrane and have shown activity against *H. pylori* in experimental models [20,21]. Although still far from clinical application, they may offer future alternatives to conventional antibiotics with a potentially lower risk of resistance development [20].

7.2.4 Vaccine Development

Vaccination is considered one of the most promising long-term strategies for preventing *H. pylori* infection and reducing gastric cancer risk [24,25]. Several vaccine candidates have shown good immunogenicity and acceptable safety in early clinical trials [24,25]. An oral recombinant vaccine tested in children demonstrated reduced colonization and strong mucosal immune responses, suggesting meaningful preventive potential [24]. Although no vaccine is yet available for clinical use, continued progress may eventually shift management from treatment toward prevention [24,25].

7.2.5 Host-Directed Therapies

A deeper understanding of host immune mechanisms has prompted interest in therapies that modulate inflammatory pathways, enhance mucosal healing, or strengthen innate immunity [20–22]. Early studies suggest that host-directed approaches may help limit disease progression, especially in patients with chronic gastritis or precancerous lesions [22]. These strategies remain investigational but represent an important direction beyond traditional antibacterial therapy [20–22].

8. Conclusions

Helicobacter pylori infection remains an important clinical and public health challenge. Despite the availability of several established therapeutic approaches, eradication rates continue to decline due to increasing antibiotic resistance, variability in acid suppression, and difficulties

with treatment adherence. These factors highlight the importance of evidence-based treatment selection and the need to strengthen access to diagnostic tools, including susceptibility testing.

Bismuth-containing and concomitant therapies remain effective first-line options in many regions, particularly where clarithromycin resistance is high. Although newer acid-suppressive agents and alternative antimicrobials show promising results, their limited availability restricts their wider use in routine care.

Emerging strategies—including PCAB-based approaches, rifabutin therapy, innovative antimicrobials, microbiome-modulating interventions, and vaccine development—may help address current limitations. Continued research, together with improved diagnostic capacity, will support more individualized and effective treatment pathways.

Progress in these areas may ultimately lead to better eradication outcomes and contribute to long-term reductions in complications associated with *H. pylori* , including peptic ulcer disease and gastric cancer.

Author Contribution

All authors have read and agreed with the published version of the manuscript.

Funding Statement

This research received no external funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

The data presented in this study are available on request from the corresponding author.

Acknowledgments

Not applicable.

Conflict of Interest Statement

The authors declare no conflicts of interest.

References

1. Hooi JKY, Lai WY, Ng WK, et al. Global prevalence of *Helicobacter pylori* infection: systematic review and meta-analysis. Gastroenterology. 2017;153(2):420–429. doi:10.1053/j.gastro.2017.04.022.
2. Eusebi LH, Zagari RM, Bazzoli F. Epidemiology of *Helicobacter pylori* infection. Helicobacter. 2014;19(Suppl 1):1–5. doi:10.1111/hel.12165.
3. Laszewicz W, Iwańczak F, Iwańczak B. Seroprevalence of *Helicobacter pylori* infection depending on socioeconomic status and living conditions in Poland. Adv Med Sci. 2014;59(1):147–150. doi:10.1016/j.advms.2014.01.003.
4. Biernat MM, Iwańczak B, Bińkowska A, et al. Prevalence of *Helicobacter pylori* infection in symptomatic children: a 13-year Lower Silesia study. Adv Clin Exp Med. 2016;25(2):303–308. doi:10.17219/acem/44372.
5. Dyrla P, Wesołowska M, Kaczor MP. Zakażenie *Helicobacter pylori* u dzieci — aktualny problem kliniczny. Pediatr Med Rodz. 2015;11(1):80–85.
6. Savoldi A, Carrara E, Graham DY, et al. Prevalence of antibiotic resistance in *Helicobacter pylori* : a systematic review and meta-analysis. Gastroenterology. 2018;155(5):1372–1382. doi:10.1053/j.gastro.2018.07.007.
7. Thung I, Aramin H, Vavinskaya V, et al. The global emergence of *Helicobacter pylori* antibiotic resistance. Aliment Pharmacol Ther. 2016;43(4):514–533. doi:10.1111/apt.13497.
8. Dang BN, Graham DY. *Helicobacter pylori* infection and antibiotic resistance: a WHO high priority? Nat Rev Gastroenterol Hepatol. 2017;14(7):383–384. doi:10.1038/nrgastro.2017.57.
9. Korona-Głowniak I, Cichoż-Lach H, Siwiec R, et al. Antibiotic resistance and genotypes of *Helicobacter pylori* strains in patients with gastroduodenal diseases in southeast Poland. J Clin Med. 2019;8(7):1071. doi:10.3390/jcm8071071.
10. Krzyżek P, Pawełka D, Gościniak G, et al. High primary antibiotic resistance of *Helicobacter pylori* strains isolated from pediatric and adult patients in Poland during 2016–2018. Antibiotics. 2020;9(5):228. doi:10.3390/antibiotics9050228.
11. Malfertheiner P, Megraud F, Rokkas T, et al. Management of *Helicobacter pylori* infection — the Maastricht VI/Florence consensus report. Gut. 2022;71(9):1724–1762. doi:10.1136/gutjnl-2022-327745.

12. Bartnik W, Celińska-Cedro D, Dzieniszewski J, et al. Wytyczne Polskiego Towarzystwa Gastroenterologii dotyczące diagnostyki i leczenia zakażenia *Helicobacter pylori* . Gastroenterol Klin. 2014;6(2):41–49. doi:10.5603/gek.38379.
13. Hu Y, Wen S, Wang M, Zheng X. Advances in understanding *Helicobacter pylori* pathogenesis. Front Cell Infect Microbiol. 2017;7:168. doi:10.3389/fcimb.2017.00168.
14. Chey WD, Mégraud F, Laine L, et al. Vonoprazan triple and dual therapy for *Helicobacter pylori* infection. Gastroenterology. 2022;163(3):608–619. doi:10.1053/j.gastro.2022.05.055.
15. Kalfus IN, Graham DY, Riff DS, Panas RM. Rifabutin-containing triple therapy (RHB-105) for eradication of *Helicobacter pylori* . Antibiotics. 2020;9(10):685. doi:10.3390/antibiotics9100685.
16. Graham DY, Lee YC, Wu MS. Rational *Helicobacter pylori* therapy: evidence-based medicine rather than medicine-based evidence. Clin Gastroenterol Hepatol. 2014;12(2):177-e13. doi:10.1016/j.cgh.2013.05.028.
17. Rokkas T, Gisbert JP, Malfertheiner P, et al. Comparative effectiveness of multiple therapies for *Helicobacter pylori* . Gut. 2021;70(9):1711–1720. doi: 10.1053/j.gastro.2021.04.012.
18. Homan M, Orel R. Are probiotics useful in *Helicobacter pylori* eradication? World J Gastroenterol. 2015;21(37):10644–10653. doi:10.3748/wjg.v21.i37.10644.
19. Ruggiero P. Use of probiotics in the fight against *Helicobacter pylori* . World J Gastrointest Pathophysiol. 2014;5(4):384–391. doi:10.4291/wjgp.v5.i4.384.
20. Huang TT, Zhang Y, Zhang L, et al. Novel therapeutic regimens against *Helicobacter pylori* : an updated systematic review. Front Microbiol. 2024;15:1418129. doi:10.3389/fmicb.2024.1418129.
21. Moss SF, Dang LP, Chua D, et al. Evolving concepts in *Helicobacter pylori* management: a contemporary review. Gastroenterology. 2024;166:267–283. doi:10.1053/j.gastro.2023.09.047.
22. Duan Y, Du X, Liu M, et al. *Helicobacter pylori* and gastric cancer: mechanisms and new perspectives. J Hematol Oncol. 2025;18:14. doi:10.1186/s13045-024-01654-2.
23. World Health Organization. *Helicobacter pylori* and gastric cancer: global burden analysis. WHO Technical Report; 2023.
24. Talebi Bezmin Abadi A. Vaccine against *Helicobacter pylori* : inevitable approach. World J Gastroenterol. 2016;22(11):3150–3157. doi:10.3748/wjg.v22.i11.3150.

25. Czinn S, Blanchard T. Vaccinating against *Helicobacter pylori* infection. Nat Rev Gastroenterol Hepatol. 2011;8:133–140. doi:10.1038/nrgastro.2011.1.
26. Waldum H, Fossmark R. Eradication of *Helicobacter pylori* by a potassium-competitive acid blocker alone? Scand J Gastroenterol. 2025;60(1):10–12. doi:10.1080/00365521.2024.2444477.
27. Antequera CM, Orleck K, Jacob R, et al. Potassium-competitive acid blockers: rethinking acid suppression for gastroesophageal reflux disease and *Helicobacter pylori* . Postgrad Med. 2024;136(2):131–140. doi:10.1080/00325481.2024.2320081.