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Supplement

Clinical Practice Guideline for the Diagnosis and Treatment of Helicobacter Pylori Infection in Adults: 2025 Update

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Abstract

Introduction: In 2015, the Colombian Association of Gastroenterology (ACG) published a clinical practice guideline (CPG) for the diagnosis and treatment of Helicobacter pylori infection in adults. In 2025, the ACG updated this guideline to reflect advances in diagnostic and therapeutic strategies, tailored to the local microbiological context and the clinical characteristics of the Colombian population. **Materials and Methods:** The ACG guideline committee, in collaboration with epidemiologists, updated the guidelines based on a systematic review of clinical recommendations and meta-analyses, following the GRADE methodology. This short version of the 2025 update summarizes the evidence supporting the recommendations. The full guideline is available at www.gastrocol.com. **Results:** The updated guideline provides recommendations on treatment indications for H. pylori eradication and prevention of associated diseases, invasive and non-invasive diagnostic methods, therapeutic alternatives across different lines of treatment and specific considerations such as high-resistance regions, penicillin allergy, and special populations. **Conclusion:** Accurate diagnosis and effective treatment of H. pylori infection are essential due to its role as a risk factor for chronic gastritis, peptic ulcers, gastric MALT lymphoma, and gastric adenocarcinoma.

Keywords

Helicobacter Pylori, Diagnosis, Therapy, Clinical Practice Guideline.

INTRODUCTION

Helicobacter pylori is a gram-negative bacterium that affects approximately 44% of the global population[1]. It is considered the most common chronic bacterial infection in humans and represents the main risk factor for chronic gastritis, peptic ulcers, gastric MALT lymphoma, and gastric adenocarcinoma[2]. The World Health Organization (WHO) has classified *H. pylori* as a Group I carcinogen[3]. In Colombia, managing this infection poses a major public health challenge due to three critical epidemiological factors: the high prevalence of infection, an intermediate but significant incidence of gastric cancer (11.4–14.95)[4], and high antimicrobial resistance rates[5], which have reduced the effectiveness of traditional therapeutic regimens[6]. This situation requires the continuous updating of diagnostic and therapeutic strategies adapted to the local microbiological context and the clinical characteristics of the population.

SCOPE OF THE CLINICAL PRACTICE GUIDELINE

This Clinical Practice Guideline (CPG) is intended for healthcare professionals who directly manage patients infected with *H. pylori*, and indirectly for health decision-makers in clinical settings, insurance companies, payers, and policymakers. The goal of this CPG is to promote appropriate diagnosis, treatment, and follow-up in the population described.

We focus on recommendations concerning diagnostic, therapeutic, and follow-up methods for adults over 18 years of age with *H. pylori* infection. This guideline offers specific recommendations rather than protocols. In some cases, physicians may need to deviate from the guideline, provided they have appropriate justification. This CPG does not exempt clinicians from their responsibility to perform a complete and thorough evaluation of each patient.

INTENDED USERS AND CLINICAL SETTINGS

This guideline is aimed at general practitioners, internists, family physicians, gastroenterologists, infectious disease specialists, hematologists, and surgeons working in outpatient clinics, emergency departments, general inpatient wards, or intensive care units.

PATIENT GROUPS CONSIDERED IN THE CLINICAL PRACTICE GUIDELINE

- Adults over 18 years of age with suspected *H. pylori* infection.
- Adults over 18 years of age with confirmed *H. pylori* infection.

Pediatric patients, pregnant women, and breastfeeding women are not included.

OBJECTIVES OF THE CLINICAL PRACTICE GUIDELINE

- Provide healthcare professionals treating patients with *H. pylori* infection with the most up-to-date evidence regarding diagnostic methods, verification of eradication, and the appropriate protocols to minimize false-negative results.
- Identify, using the best available evidence, which diseases can be cured, improved, or whose risk can be reduced by eradicating *H. pylori*.
- Establish, based on the best published evidence, empiric therapeutic regimens recommended for Colombia and Latin America, considering local antibiotic resistance patterns and drug availability.

METHODOLOGY

Composition of the Update Group

The update panel was led by two gastroenterologists (W. O. R., H. M. M.F.) and one clinical bacteriologist (A. A. T. R.) with clinical and scientific experience in *H. pylori*. It included the Clinical Practice Guidelines Group of the Colombian Association of Gastroenterology (ACG) and was supported by epidemiologists. All participants submitted conflict of interest declarations.

Decision on the Update

During this process, we reviewed the scope, target population, users, objectives, and clinical questions from the 2015 version of the CPG[7]. The clinically relevant outcomes from that version were maintained: eradication rate, treatment adherence, and adverse effects.

Systematic Literature Review

We conducted a systematic review of practice recommendations and systematic reviews with meta-analyses, with the last search performed on July 2, 2025. Details of the review methodology are available in the full version of the guideline hosted in the Gastrocol digital repository.

Certainty of Evidence and Strength of Recommendation

The review and update of practice recommendations were based on the assessment of certainty of evidence and strength of recommendation using the GRADE (Grading

of Recommendations, Assessment, Development, and Evaluation) Evidence-to-Decision (EtD) framework (**Table 1**)[8]. Supporting materials for these assessments are available in the full version of the guideline in the Gastrocol digital repository.

RECOMMENDATIONS

In this section, we present a summary of the evidence analysis supporting each recommendation. For a more detailed review, we recommend consulting the full version of the Clinical Practice Guideline (CPG), available in the Gastrocol digital repository.

Question 1: In adults over 18 years of age, which clinical conditions justify treating *H. pylori* infection, compared with not treating it, in terms of preventing associated complications (ulcers, anemia, gastric cancer, among others)?

We recommend offering treatment to all adult patients infected with *H. pylori*, regardless of the presence or absence of symptoms.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

Eradication of *H. pylori* significantly reduces the incidence and mortality of gastric cancer— by 36%–44% and 22%, respectively[9]. Major international and U.S. guidelines[10] recommend treating all patients with confirmed active infection, regardless of whether symptoms are present, except in cases with contraindications or limited life expectancy[11].

Question 2: In adult patients with suspected or previous *H. pylori* infection, what is the diagnostic accuracy and clinical usefulness of invasive tests (biopsy, urease) compared with noninvasive tests (urea breath test, stool antigen) to confirm diagnosis or eradication?

We recommend histopathology as the diagnostic method of choice to identify active infection in patients requiring upper gastrointestinal endoscopy.
Strength of Recommendation: Strong in favor
Quality of Evidence: Moderate

Histopathological examination of gastric biopsies obtained during upper gastrointestinal endoscopy is considered the best direct diagnostic method for *H. pylori*[12]. However, its accuracy depends on bacterial load within the biopsies[13]. Therefore, four biopsies are recommended: two from the body (greater and lesser curvatures) and two from the antrum (greater and lesser curvatures). For the rapid urease test (RUT), one biopsy should be taken from the body and one from the antrum[2]. Histopathology has a sensitivity of 80%–95% compared with 80%–90% for RUT, and specificity greater than 95% versus greater than 90%, respectively[14]. Histopathology is less affected by proton pump

Table 1. GRADE criteria for strength of recommendation and certainty of evidence

Dimension	Meaning
Strength of Recommendation	
Strong in favor	The desirable consequences clearly outweigh the undesirable ones. It is recommended to do it.
Conditional in favor	The desirable consequences probably outweigh the undesirable ones. It is suggested to do it.
Conditional against	The undesirable consequences probably outweigh the desirable ones. It is suggested not to do it.
Strong against	The undesirable consequences clearly outweigh the desirable ones. It is recommended not to do it.
Certainty of Evidence	
High	It is very unlikely that new studies will change our confidence in the estimated effect.
Moderate	It is likely that new studies will have an important impact on our confidence in the estimated effect and may change it.
Low	It is very likely that new studies will have an important impact on our confidence in the estimated effect and may change it.
Very low	Any estimated effect is very uncertain.
Good Practice Point	Recommended practice based on the clinical experience of the CPG Development Group.

CPG: Clinical Practice Guideline. Author's own research.

inhibitor (PPI) or antibiotic use than RUT, and it allows evaluation of the severity of chronic atrophic gastritis, intestinal metaplasia, and other pathologies.

Question 3: Which noninvasive test is preferred for detecting and confirming eradication of *H. pylori*: the urea breath test using carbon-13 or carbon-14, or stool antigen testing?

We recommend the urea breath test with carbon-13 as the noninvasive test of choice for diagnosing *H. pylori* infection and confirming eradication.
Strength of Recommendation: Weak in favor
Quality of Evidence: Low

A recent meta-analysis found that the C13 urea breath test has greater accuracy than the C14 test, with sensitivity and specificity of 96.60% and 96.97%, respectively, compared with 96.15% and 89.84%[15]. Detection of *H. pylori* stool antigens using monoclonal antibodies against *H. pylori* antigens such as catalase, performed in laboratory-based (not rapid) tests, can achieve sensitivity and specificity between 94% and 98%[15]. However, a Cochrane meta-analysis found that the C13 urea breath test had higher sensitivity (94% vs. 83%) and similar specificity (90%) compared with stool antigen testing.

Question 4: What is the effectiveness and safety of *H. pylori* eradication therapy guided by susceptibility testing (culture, polymerase chain reaction [PCR], or next-generation sequencing) compared with empirical therapy?

We recommend susceptibility-guided therapies for *H. pylori* infection, as they are more effective than empirical therapies.
Strength of Recommendation: Weak in favor
Quality of Evidence: Very low

A recent meta-analysis including 34 studies with a total of 9,613 patients found that only 17% of guided-therapy studies achieved ≥95% success, and 56% reported success rates below 90%[16]. No empirical therapy achieved ≥95%, and 85% reported success rates below 90%. Regarding safety, both approaches showed similar adverse event profiles, though some studies reported a lower incidence of adverse events with susceptibility-guided therapy, likely due to reduced antibiotic exposure[16]. Nonetheless, treatment discontinuation rates due to adverse effects were minimal and not statistically significant. In patients with refractory infection, susceptibility-guided therapy has not demonstrated statistical superiority over well-designed empirical regimens based on treatment history, although it may be useful in selected cases, especially when regimen selection is unclear after considering prior antibiotic exposure and allergies[17].

Question 5: In patients with *H. pylori* infection, how effective is tailoring treatment based on antibiotic susceptibility results and local resistance prevalence in achieving higher eradication rates compared with standard empirical therapy?

We should tailor clinical decisions, taking into account test availability, local resistance epidemiology, patient treatment history, probable adherence, and healthcare system resources.
Strength of Recommendation: Weak in favor
Quality of Evidence: Low

Tailoring *H. pylori* treatment according to antibiotic susceptibility testing and local resistance prevalence provides a modest advantage over standard clarithromycin-based triple therapy, particularly in regions with high resistance rates (>20%)[17]. A meta-analysis reported a relative risk (RR) for eradication of 1.14 (95% confidence interval [CI]: 1.07–1.21) in favor of guided therapy[18]. However, this advantage disappears when more effective empirical regimens, such as bismuth-based quadruple therapy or the "3-in-1" capsule, are used, as these achieve eradication rates above 85% even in the presence of clarithromycin or metronidazole resistance[17,19]. In patients with previous treatment failure, although some observational studies suggest benefits of guided therapy, randomized controlled trials have not shown statistically significant differences compared with well-designed empirical regimens[16,17].

Question 6: In patients requiring treatment for *H. pylori*, what are the diagnostic accuracy and clinical usefulness of culture with antibiogram compared with molecular tests (such as PCR or sequencing) to guide treatment and improve eradication rates?

In patients with *H. pylori* infection who require targeted therapy, both culture with antibiogram and molecular tests (PCR or sequencing) allow us to select effective regimens and achieve higher eradication rates compared with empirical therapy. Molecular tests show greater diagnostic sensitivity, better logistical performance, and broader clinical applicability, particularly for detecting resistance to clarithromycin and levofloxacin. Therefore, we suggest using molecular tests over culture with antibiogram, except in cases of multidrug resistance or when assessing susceptibility to less common antibiotics is necessary—situations in which culture remains useful.

Strength of Recommendation: Weak in favor
Quality of Evidence: Moderate

Both culture and molecular tests improve treatment effectiveness compared with empirical therapy, especially after previous failures[5,6], with eradication rates approaching 88% versus 78% for empirical treatment[20]. Guidelines recommend susceptibility testing in patients with treatment failure or prior exposure to high-risk antibiotics, and molecular methods are preferred when culture is not feasible or when a rapid result is required[17,21].

Question 7: In patients with suspected antimicrobial resistance in *H. pylori*, which technique is the most accurate and reliable for detecting resistance: phenotypic tests or genotypic tests (such as PCR or sequencing)?

We recommend using genotypic tests (PCR or next-generation sequencing [NGS]) over phenotypic tests to detect resistance to clarithromycin and levofloxacin because of their greater sensitivity, rapid turnaround time, and clinical applicability. Culture remains useful when evaluating less well-characterized resistances (e.g., metronidazole, amoxicillin) or when a complete phenotypic profile is required.
Strength of Recommendation: Weak in favor
Quality of Evidence: Moderate

In patients with suspected *H. pylori* resistance, current evidence supports the use of genotypic tests (PCR, sequencing, NGS) over phenotypic tests (culture and antibiogram) for detecting resistance to clarithromycin and levofloxacin due to their higher diagnostic sensitivity, rapidity, and clinical utility[22,23]. Genotypic tests allow us to identify specific mutations such as A2142G/C/A2143G (clarithromycin) and mutations in gyrA (levofloxacin), even in noninvasive samples like stool or gastric juice, with sensitivity ranging from 94% to 100% and specificity from 74% to 99%[24]. NGS also offers additional advantages, including detection of multiple resistance genes and high performance in fixed specimens[17].

Question 8: In patients with *H. pylori* infection and prior treatment failure, what are the effectiveness and cost-effectiveness of performing antimicrobial susceptibility testing compared with not performing it, in order to improve eradication rates and reduce empirical treatment failures, considering local resistance prevalence?

We recommend performing antimicrobial susceptibility testing after treatment failure when local availability exists and when multidrug resistance is suspected. Targeted tests (culture, PCR) or next-generation approaches (NGS) increase eradication rates compared with repeated use of empirical regimens. Cost-effectiveness improves when local resistance prevalence is high (>15% for clarithromycin or fluoroquinolones).
Strength of Recommendation: Weak in favor
Quality of Evidence: Low

Population-based studies have shown that susceptibility-guided therapy can achieve eradication rates above 90%, compared with 50% when no testing is performed, while also reducing complications and the need for retreatment[25]. This strategy may yield significant cost savings per patient when local resistance prevalence and test costs are taken into account. However, in regions where empirical bismuth-based quadruple therapy maintains high effectiveness (>85%), the added benefit of susceptibility testing may be limited[17,26].

Question 9: In adults with active *H. pylori* infection, does 14-day treatment compared with seven or 10 days improve eradication rates without significantly increasing adverse effects?

Fourteen-day therapies are superior to 7-day and 10-day regimens for eradicating *H. pylori*.
Strength of Recommendation (14 vs. 7 days): Strong in favor
Quality of Evidence: High
Strength of Recommendation (14 vs. 10 days): Weak in favor
Quality of Evidence: Low

A recent meta-analysis including 15 randomized clinical trials (4,505 patients) showed that 14-day bismuth-based quadruple therapy achieved higher eradication rates than 7-day regimens (RR: 0.96; 95% CI: 0.93–0.99), while the difference between 10 and 14 days was not significant (RR: 0.98; 95% CI: 0.95–1.00)[1,2]. The incidence of adverse events and treatment adherence were similar across different treatment durations[1]. For potassium-competitive acid blocker (PCAB)–based therapies and classical triple therapy, results are similar, particularly in regions with high clarithromycin resistance, without any clinically relevant increase in adverse effects[27,28].

Question 10: In adult patients with *H. pylori* infection and no history of prior treatment, which empirical first-line regimens are most recommended?

In areas with unknown clarithromycin resistance or high clarithromycin resistance (>15%), we recommend first-

line therapy with bismuth-based quadruple regimens (using PPIs or PCABs) or dual therapy with amoxicillin (combined with a PPI or a PCAB such as tegoprazan or vonoprazan).
Strength of Recommendation: Strong in favor
Quality of Evidence: High

Bismuth-Based Quadruple Therapy

Bismuth-based quadruple therapy (a PPI or PCAB + bismuth [subsalicylate, subgallate, or subnitrate] + a nitroimidazole [metronidazole or tinidazole] + tetracycline) is the cornerstone of empirical treatment, especially where susceptibility testing is not available[29]. This regimen is highly effective and maintains eradication rates of 90%–95% regardless of metronidazole resistance[2,30]. It is also the first-line therapy recommended by Maastricht VI[2] and by the American College of Gastroenterology (ACG) guideline[17].

Dual Therapy with Amoxicillin

Dual therapy consists of high-dose amoxicillin administered 3–4 times daily together with high-dose PPIs or a PCAB[6]. Systematic reviews and meta-analyses report eradication rates of approximately 86% by intention-to-treat and 87% per protocol for amoxicillin/PPI dual therapy, and rates up to 93% per protocol for amoxicillin/vonoprazan (a PCAB). Additionally, studies from Latin America suggest that dual therapy may be an effective first-line option in regions with high clarithromycin resistance[6]. This recommendation is supported by the recent second Taipei Consensus[29].

A full analysis of concomitant bismuth-free quadruple therapy and PCAB-based treatments can be found in the complete CPG, available in the Gastrocol repository.

Question 11: In adult patients with *H. pylori* infection and no prior treatment, is optimized dual therapy with amoxicillin and a PPI or PCAB (such as tegoprazan or vonoprazan) as effective and safe as empirical quadruple therapy?

We recommend using either dual therapy or quadruple therapy as first-line options. The choice should depend on local data and patient-specific clinical characteristics.
Strength of Evidence: Strong in favor
Quality of Evidence: Moderate

Current evidence shows that vonoprazan–amoxicillin dual therapy, particularly when using high-dose amoxicillin with increased dosing frequency (for example, 750 mg four times daily or 1,000 mg three times daily), achieves eradication rates greater than 90% in per-protocol analyses and is not inferior to empirical bismuth-based quadruple therapy. It also has a significantly lower incidence of adverse events[9,17,31,32]. Vonoprazan–amoxicillin dual therapy demonstrates superior tolerability and lower discontinuation rates due to adverse effects[9,17,31,32]. Recent clinical guidelines recognize that PCAB-based regimens (such as those using vonoprazan) are superior or "noninferior" to conventional PPI-based regimens, particularly in patients infected with clarithromycin-resistant strains[2].

Question 12: When should standard triple therapy be used to treat *H. pylori*?

Standard triple therapy is indicated as first-line treatment when clarithromycin resistance prevalence is low (<15%), or when resistance is ≥15% but susceptibility testing confirms that the strain is clarithromycin-sensitive.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

Standard triple therapy consists of a PPI plus amoxicillin and clarithromycin. It should be used only in patients infected with clarithromycin-sensitive strains or in settings where local clarithromycin resistance is low (<15%) and the patient has no history of macrolide exposure[2,17]. Standard triple therapy is safe. The most common adverse events include gastrointestinal symptoms such as diarrhea, nausea, vomiting, abdominal pain, and dysgeusia. Headache may also occur, and less commonly, cutaneous allergic reactions or mild, transient elevations of liver enzymes[2].

Question 13: In adults with *H. pylori* infection living in areas with high clarithromycin and metronidazole resistance, does adding bismuth to triple therapy improve eradication rates compared with bismuth-free triple therapy?

We recommend adding bismuth to triple therapy in patients with confirmed or suspected resistance to clarithromycin or metronidazole to improve eradication rates. This approach is particularly relevant in regions with high resistance prevalence, such as Colombia.
Strength of Recommendation: Strong in favor
Quality of Evidence: Moderate

A meta-analysis found that adding bismuth to triple therapy can increase cure rates by 30%–40% in resistant infections, thereby reaching desired therapeutic thresholds (approximately 90%) even when resistance is high[33]. Integrating bismuth into clarithromycin-based triple regimens enhances efficacy even in the presence of clarithromycin resistance, resolving in many cases the initial treatment failure attributable to resistance[34]. From a safety perspective, adding bismuth does not increase adverse events. Reported

effects are similar to those seen with traditional quadruple therapy (nausea, dark stools, mild discomfort), with no evidence of increased toxicity. For these reasons, international guidelines recommend bismuth-based quadruple therapy as first-line treatment in areas with high clarithromycin resistance[2,17].

Question 14: In adults with *H. pylori* infection treated with bismuth-containing quadruple therapy, are there differences in efficacy or safety among bismuth salts (subsalicylate, subnitrate, subgallate)?

We recommend using any of the available bismuth salts (subsalicylate, subcitrate, subnitrate, or subgallate) as part of quadruple therapy, without preference based on efficacy, since differences in absorption and tolerability do not translate into clinically meaningful variations. We should base selection on local availability, cost, and adverse-effect profiles.
Strength of Recommendation: Strong
Quality of Evidence: Moderate

Current evidence indicates no clinically significant differences in *H. pylori* eradication efficacy among the various bismuth salts (bismuth subcitrate, subsalicylate, subnitrate, or pectin-bound bismuth) used in quadruple regimens, provided they are administered at appropriate doses and as part of an optimized treatment scheme[2,17,34]. Recent clinical guidelines recommend the use of any available bismuth salt in quadruple therapy, without preference for one specific formulation, as long as adequate dosing is ensured and the regimen includes the recommended companion drugs[35].

Question 15: In adults with *H. pylori* infection, does the use of PCABs in combination with antibiotics improve eradication rates and reduce adverse events compared with PPIs?

We recommend using both PCABs and PPIs (esomeprazole, rabeprazole) in *H. pylori* eradication regimens, as they provide comparable efficacy and safety. The choice should depend on local availability and cost.
Strength of Recommendation: Strong in favor
Quality of Evidence: Moderate–high

The difference between PCABs and second-generation PPIs (esomeprazole and rabeprazole) may be less pronounced than that observed with first-generation PPIs (omeprazole, lansoprazole, pantoprazole), because esomeprazole and rabeprazole depend less on CYP2C19 metabolism. This reduces interindividual variability in acid suppression and improves *H. pylori* eradication efficacy[9,17,36]. Indeed, guidelines recommend using esomeprazole or rabeprazole in patients with CYP2C19 ultrarapid or rapid metabolizer genotypes, or considering a PCAB when available[2,17,37]. However, direct data comparing PCABs specifically with esomeprazole or rabeprazole are limited. Some recent studies suggest that the difference in efficacy between PCABs and these second-generation PPIs may not be statistically significant in certain contexts and dosing regimens[38].

Question 16: In adults with active *H. pylori* infection, does using probiotics as adjuvants to antibiotic therapy improve eradication rates or reduce adverse events compared with not using them?

We suggest using probiotics—especially *Saccharomyces boulardii* and *Lactobacillus* strains such as *L. reuteri* or *L. rhamnosus GG*—as adjuvants to *H. pylori* eradication therapy to reduce gastrointestinal adverse events and, in some cases, to improve eradication rates, particularly with triple therapy.
Strength of Recommendation: Weak in favor
Quality of Evidence: Very low

Multiple meta-analyses and randomized clinical trials have shown that adding probiotics, particularly *Lactobacillus*, *Bifidobacterium*, and *Saccharomyces boulardii*, improves eradication efficacy and reduces treatment-related adverse effects such as diarrhea and nausea[39]. A meta-analysis conducted by Colombian authors[40] found that the use of probiotics as adjuvants increased the eradication rate in triple therapy regimens (from 71.1% to 79.4%; odds ratio [OR]: 1.42; 95% CI: 1.05–2.09; $p = 0.03$), without achieving a significant impact in quadruple regimens, and with a reduction in adverse effects both in triple therapy (OR = 0.50; 95% CI: 0.28–0.90) and in quadruple therapy (OR = 0.26; 95% CI: 0.09–0.74).

Question 17: When should levofloxacin-containing regimens be used?

We recommend using levofloxacin only as rescue therapy, when one or more prior regimens have failed, particularly when clarithromycin and metronidazole resistance are present or when these antibiotics cannot be used.
Strength of Recommendation: Strong in favor
Quality of Evidence: Moderate

We should reserve fluoroquinolones (e.g., levofloxacin or moxifloxacin) for specific situations, such as rescue therapy after one or more failed regimens when bacterial susceptibility information is available, or in settings with low documented resistance[19,41]. Current clinical guidelines

and recent evidence recommend considering levofloxacin-based triple therapy (PPI, amoxicillin, and levofloxacin) or quadruple regimens that include levofloxacin as second- or third-line options when local fluoroquinolone resistance rates are low (<15%) or in vitro susceptibility has been confirmed[2,17,19,41]. We do not recommend its empirical first-line use due to the risk of resistance development and the substantial reduction in efficacy when resistant strains are present[2,17,19].

Question 18: In adult patients with *H. pylori* infection refractory to prior treatment, which empirical second- or third-line regimens achieve the highest eradication rates when compared with each other?

We recommend 14-day bismuth-based quadruple therapy as the empirical second- or third-line regimen of choice. Dual regimens with a PPI or PCAB plus amoxicillin and furazolidone-containing regimens (furazolidone–tetracycline quadruple therapy or furazolidone–amoxicillin quadruple therapy for 14 days), as well as rifabutin triple therapy, are effective alternative options.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

Refractory *H. pylori* infection is defined as persistence after at least one previous failed treatment[37].

Bismuth-Based Quadruple Therapy

This regimen consists of a PPI, bismuth, tetracycline, and a nitroimidazole (metronidazole or tinidazole) administered for 10–14 days[2,17]. It consistently maintains eradication rates above 80% even in the presence of metronidazole resistance. Its effectiveness relies on antibiotic synergy and the potentiating effect of bismuth, which also helps reduce cross-resistance. This regimen stands out as the most reliable empirical option for patients with prior treatment failures and is supported by guidelines from the American Gastroenterological Association (AGA), the ACG[4], and the Maastricht VI Consensus[2,5,17,37].

High-dose Dual Therapy (HDDT)

This strategy combines a PPI or PCAB (such as vonoprazan) at high doses with high-dose amoxicillin—for example, amoxicillin 750 mg three to four times daily(2,17) or optimized at 50 mg/kg of body weight[42]—for 14 days. It is the therapy of choice following failure of bismuth-based quadruple therapy[19].

Furazolidone-Containing Regimens

In Peru, a 10-day furazolidone-containing quadruple therapy has been used successfully, consisting of furazolidone 100 mg three times daily, doxycycline 100 mg twice daily, a PPI twice daily, and bismuth subsalicylate 262 mg three times daily[19], achieving an effectiveness of 95%, which has remained stable for the last 10 years[43]. In Colombia, more than two decades ago, a quadruple therapy using omeprazole, tetracycline, amoxicillin, and furazolidone (OTAF) was used as rescue therapy in 90 patients with previous failures. The intention-to-treat (ITT) eradication rate was 81.1% (95% CI: 71.2–83.3), and the per-protocol (PP) eradication rate was 83.9% (95% CI: 73.7–90%). Adverse effects occurred in 20% of patients, and only three discontinued treatment (headache, diarrhea, and rash)[44].

Rifabutin Triple Therapy

Current evidence supports rifabutin-based regimens as rescue therapy for *H. pylori*, particularly in patients with multiple prior failures and in settings with high resistance to clarithromycin, levofloxacin, and metronidazole[37]. The most studied regimens combine rifabutin with amoxicillin and a PPI for 10–14 days, although data also exist for vonoprazan as an alternative to PPIs[17,45]. The European *H. pylori* Registry (Hp-EuReg) reported that among more than 500 patients treated with rifabutin as second-, third-, or fourth-line therapy, modified intention-to-treat (mITT) effectiveness rates were 78%, 80%, and 66%, respectively[46]. Adherence was 89%, and at least one adverse event occurred in 26% of patients (most commonly nausea). A single patient (0.2%) developed fever, leukopenia, and thrombocytopenia requiring hospitalization[46]. Rifabutin should be reserved for third-line scenarios after prior failures.

Question 19: In adult patients with *H. pylori* infection refractory to treatment, which factors are associated with therapeutic failure compared with patients in whom eradication is achieved?

We recommend a comprehensive evaluation of factors associated with therapeutic failure in patients with refractory *H. pylori* infection before initiating a new empirical regimen. This evaluation should consider prior antibiotic exposure, adherence, body weight, the type of acid-suppressive therapy (and its relationship to CYP2C19 metabolism), prior drug selection, and local availability.
Strength of Recommendation: Strong in favor
Quality of Evidence: Moderate

To avoid errors and prevent bacterial persistence—particularly in regions with high antibiotic resistance—it is essential that we do not repeat prior regimens and that we optimize the doses of subsequent antimicrobials as well as acid-suppressive therapy[37,47]. Poor adherence is also a major cause of treatment failure. In European multicenter

studies, eradication rates with adequate adherence (≥90% of prescribed medication) were 86%–93%, whereas in non-adherent patients they dropped to 44%–71%[48]. Another important consideration is achieving both optimal acid suppression and an understanding of the patient's metabolic profile, given their impact on treatment effectiveness. Adequate acid suppression is essential to optimize the activity of antibiotics such as amoxicillin[37,47]. Enzymatic variability in hepatic metabolism of PPIs—particularly in CYP2C19 rapid metabolizers, a phenotype described in Latin American populations—represents an underrecognized cause of treatment failure in clinical practice[37,47,49].

Question 20: In adults with *H. pylori* infection in regions with high prevalence of resistance to clarithromycin, metronidazole, or levofloxacin, which empirical regimens are more effective and safer compared with conventional regimens?

We recommend 14-day bismuth-based quadruple therapy as the main empirical regimen in regions with high resistance, and as alternatives, furazolidone-based quadruple therapy (combined with amoxicillin or tetracycline), or high-dose amoxicillin dual therapy with a PPI or PCAB, depending on local availability, cost, and individual patient tolerance.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

The combination of PPI + bismuth + tetracycline + metronidazole administered for 14 days remains the regimen of choice, achieving eradication rates above 90%, even in high-resistance settings. Its success reflects the synergistic interaction between the antibiotics and bismuth, as well as the very low rates of resistance to tetracycline and bismuth[2,17]. This regimen is the most widely recommended by international guidelines, has demonstrated effectiveness even after previous treatment failures, and is the only regimen approved by the U.S. Food and Drug Administration (FDA) as rescue therapy[2,17]. A major limitation in Colombia is the limited availability of tetracycline in some regions, in which case minocycline can be used as a substitute[50].

A meta-analysis found that furazolidone-based quadruple therapy achieved a per-protocol eradication rate of 92.9%, superior to metronidazole-based regimens[51]. Choosing between amoxicillin and tetracycline as the companion antibiotic may influence both efficacy and adverse-effect profiles; combining furazolidone with tetracycline is potentially more effective in refractory patients but is associated with a higher incidence of mild adverse effects[52]. Furazolidone may replace metronidazole, and this strategy has been studied in Latin America combined with doxycycline, achieving eradication rates above 90% (furazolidone + doxycycline + PPI + bismuth)[52], or with amoxicillin instead of tetracycline and vonoprazan instead of a PPI, also with eradication rates above 90%[43].

Question 21: In adult patients with *H. pylori* infection who require tetracycline-based therapy, do minocycline or doxycycline offer efficacy and safety comparable to classical tetracycline?

We recommend using minocycline as an effective substitute for tetracycline in bismuth-based quadruple regimens for *H. pylori* eradication. Tetracycline should not routinely be replaced with doxycycline; however, doxycycline can be considered in countries where tetracycline is unavailable and where doxycycline has demonstrated effectiveness in well-structured, locally validated regimens.
Strength of Recommendation (minocycline): Strong in favor
Quality of Evidence: High
Strength of Recommendation (doxycycline): Weak in favor
Quality of Evidence: Very low

A meta-analysis including five randomized controlled trials[53] found that eradication rates with minocycline-containing quadruple therapy were identical to those achieved with tetracycline (85.5% vs. 85.5%; OR: 1.00; 95% CI: 0.67–1.47; $p = 1.00$), with no significant differences in adverse-event incidence. Systematic reviews indicate that triple regimens including doxycycline generally achieve eradication rates below 70%, which is considered suboptimal for first-line therapy. However, quadruple regimens incorporating doxycycline (e.g., PPI, bismuth, metronidazole, and doxycycline) may achieve per-protocol eradication rates close to 95%, though evidence is limited and derived from small studies with substantial heterogeneity in regimens used[54]. Internationally, doxycycline may be useful in specific contexts, but it should not replace tetracycline when the latter is available and tolerated[55].

Question 22: In adults with *H. pylori* infection, do different PPIs (such as omeprazole, esomeprazole, lansoprazole, and rabeprazole) produce significant differences in eradication rates?

We recommend using esomeprazole or rabeprazole because their pharmacokinetics are not clinically impacted by CYP2C19 rapid-metabolizer genotypes; alternatively, first-generation PPIs may be used at double doses.
Strength of Recommendation: Strong in favor
Quality of Evidence: Moderate

A meta-analysis of 25 clinical trials including 5,318 patients[56] showed that, with omeprazole or lansoprazole, eradication rates in rapid metabolizers (RM) were 77.7% (95% CI: 75.3–79.6), in intermediate metabolizers (IM) were 81.2% (95% CI: 79.3–83.0), and in poor metabolizers (PM) were 86.8% (95% CI: 83.9–88.9). In contrast, eradication rates with esomeprazole and rabeprazole were not influenced by metabolizer status, and similar eradication rates were achieved across the different metabolizer types—80%, 83%, and 89% in RM, IM, and PM, respectively, without statistically significant differences—and with rabeprazole specifically showing rates of 84%, 89%, and 92% in RM, IM, and PM, respectively. Maastricht VI[2] and the ACG Clinical Guideline[17] both recommend double-dose PPI therapy to achieve an optimal gastric environment. In countries with a high prevalence of rapid or ultrarapid CYP2C19 metabolizers (such as Colombia), esomeprazole and rabeprazole are preferred because of their lower metabolic variability; alternatively, higher doses of first-generation PPIs may be used.

Question 23: In adults with H. pylori infection and documented penicillin allergy, which alternative regimens (such as bismuth-based quadruple therapy or amoxicillin-free triple therapy) are more effective and safer when compared with each other?

We recommend bismuth-based quadruple therapy (PPI + bismuth + tetracycline + metronidazole) as the empirical regimen of choice for patients with penicillin allergy, and we also recommend furazolidone–tetracycline–bismuth–PPI quadruple therapy for 14 days as an alternative empirical regimen in patients who experience adverse effects or intolerance to metronidazole. PCAB-based regimens combined with antibiotics other than amoxicillin may also be considered.

Fluoroquinolone-based therapies (such as levofloxacin) should be reserved as a last option, only when susceptibility testing is available and with particular attention to their potential for serious adverse events (cardiac, musculoskeletal, and neuropsychiatric).

Strength of Recommendation: Strong
Quality of Evidence: High

In patients with *H. pylori* infection and confirmed penicillin allergy, the recommended first-line treatment is bismuth-based quadruple therapy administered for 14 days. This recommendation is supported by the most recent clinical guidelines and systematic reviews, which indicate that bismuth-based quadruple therapy has the strongest evidence and achieves success rates above 90%[2,17,29,37]. In cases where bismuth-based quadruple therapy is not appropriate (e.g., intolerance or contraindication to tetracycline or metronidazole), we recommend confirming penicillin allergy with specific tests, since fewer than 1% of individuals have a true type I penicillin allergy. If the allergy is not confirmed, regimens including amoxicillin may be considered[17,37]. When a true allergy exists, penicillin desensitization is an option if no effective alternatives are available[17]. A recent clinical trial demonstrated that a vonoprazan–tetracycline dual regimen in patients with penicillin allergy achieved an eradication rate above 92%, with minimal adverse effects, and was non-inferior to bismuth-based quadruple therapy, but with fewer side effects[5]. Vonoprazan has also been combined in dual therapy with minocycline, achieving 90.9% efficacy by ITT and 95.2% by PP[57]. In patients with treatment failure after two regimens, we recommend culture and antimicrobial susceptibility testing to guide therapy, and we suggest considering levofloxacin-based regimens only when susceptibility is demonstrated and local resistance prevalence is low.

Question 24: In adult populations, is *H. pylori* infection associated with lower incidence of diseases such as asthma or allergies compared with uninfected individuals?

We recommend not withholding *H. pylori* eradication for the purpose of preventing conditions such as asthma or allergies. Eradication should be indicated whenever active infection is present (confirmed by any method other than serology), regardless of its possible inverse association with immunologic diseases.

Strength of Recommendation: Strong in favor
Quality of Evidence: Moderate

Multiple epidemiological studies and meta-analyses have reported that *H. pylori* colonization is associated with lower incidence of asthma and other atopic diseases in both children and adults[58]. However, these findings represent only a negative association, and no causal relationship has been demonstrated that would define the clinical impact of eradication on the epidemiology of allergic diseases. Meanwhile, substantial evidence supports eradication in all infected individuals because of the benefits highlighted in Question 1.

Question 25: In patients with gastroesophageal reflux disease (GERD) and *H. pylori* co-infection, does eradication worsen reflux symptoms or increase the risk of Barrett's esophagus and adenocarcinoma compared with non-eradication?

We recommend eradicating *H. pylori* in all patients, including those with GERD.

Strength of Recommendation: Strong in favor
Quality of Evidence: Moderate

Although some meta-analyses report a negative association between *H. pylori* infection and erosive esophagitis (OR: 0.70; 95% CI: 0.58–0.84)[59], Barrett's esophagus (OR: 0.68; 95% CI: 0.58–0.79)[60], and esophageal adenocarcinoma (OR: 0.52; 95% CI: 0.37–0.73)[61], expert recommendations still support universal eradication[2], primarily due to the clear benefits in preventing gastric cancer highlighted in Question 1.

Question 26: In adults infected with *H. pylori*, does eradication reduce the incidence and mortality of non-cardia gastric adenocarcinoma compared with no treatment or partial treatment?

We recommend eradicating *H. pylori* in all infected adults to reduce the incidence and mortality of non-cardia gastric adenocarcinoma.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

Eradication of *H. pylori* is the most cost-effective strategy for primary prevention of non-cardia gastric cancer[2,62]. Meta-analyses of randomized clinical trials and observational studies show that eradication therapy reduces the relative risk of gastric cancer in infected individuals, with reductions of 36%–48% in incidence in general populations and up to 48% in patients with gastric neoplasia who undergo endoscopic resection[11].

Question 27: In asymptomatic adults or adults with uninvestigated dyspepsia (UD), does detecting and eradicating *H. pylori* reduce the incidence or mortality of gastric cancer (non-cardia gastric adenocarcinoma) compared with not intervening?

We recommend that in regions with high gastric cancer incidence, clinicians investigate and treat *H. pylori* infection as a primary prevention strategy for non-cardia gastric adenocarcinoma.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

Recent meta-analyses of randomized clinical trials and observational studies show that eradication therapy in infected adults without baseline gastric neoplasia reduces gastric cancer incidence by approximately 36%–46% (RR: 0.54–0.64) and gastric cancer mortality by 22%–39% (RR: 0.61–0.78) compared with not treating the infection[11]. This effect is stronger when eradication occurs before the development of premalignant lesions (atrophy, intestinal metaplasia). Most international consensus statements and clinical guidelines recommend universal eradication of *H. pylori* in infected adults as a primary gastric cancer prevention measure, particularly in high-prevalence regions and in individuals with risk factors[17,29,63].

Question 28: In patients with a history of early gastric cancer treated endoscopically and *H. pylori* infection, does eradication reduce the risk of developing a new gastric cancer compared with not eradicating the infection?

We recommend eradicating *H. pylori* in patients who undergo endoscopic resection for early gastric cancer or high-grade adenoma, as this measure significantly reduces the incidence of metachronous gastric cancers.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

A recent meta-analysis of randomized clinical trials and observational studies found a reduction in the risk of metachronous gastric cancer, with an RR of 0.52 (95% CI: 0.38–0.71) in patients treated with eradication therapy. Observational data demonstrate an even greater reduction (RR: 0.19; 95% CI: 0.06–0.61)[64]. National cohorts, such as one from Korea, confirm that eradication therapy after endoscopic resection of gastric dysplasia in more than 69,000 patients is associated with significant reductions in gastric cancer risk (adjusted HR: 0.88; 95% CI: 0.80–0.96) and metachronous neoplasia (adjusted HR: 0.76; 95% CI: 0.70–0.82), with sustained long-term protective effects[65].

Question 29: In patients with advanced gastric cancer treated with partial or total gastrectomy and *H. pylori* infection, does eradication improve clinical prognosis in terms of survival or recurrence compared with not eradicating the infection?

We recommend eradicating *H. pylori* in patients with gastric cancer who have undergone surgery—whether partial or total gastrectomy—as eradication has a positive impact on survival and on reducing recurrence of metachronous gastric cancer in the gastric remnant.
Strength of Recommendation: Strong in favor
Quality of Evidence: Very low

A recent meta-analysis found that *H. pylori* eradication is associated with better overall survival (HR ≈ 2.4; p <0.001) and lower gastric cancer recurrence (HR ≈ 1.8; p = 0.001) in patients who underwent gastrectomy, although heterogeneity among studies is acknowledged and further

randomized trials are needed[66]. Some meta-analyses and systematic reviews note that the impact of infection and eradication may vary by age subgroup and tumor characteristics, and that the association is not universally significant across all post-gastrectomy scenarios. *H. pylori* eradication may also help improve the microbiota, which could enhance systemic immunity and tolerance to oncologic treatments.

Question 30: In patients with low-grade gastric MALT lymphoma, does *H. pylori* eradication induce remission compared with other therapeutic strategies or compared with not treating the infection?

We recommend eradicating *H. pylori* in all patients with gastric MALT lymphoma, particularly low-grade and early-stage disease, before initiating other therapies.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

In patients with low-grade gastric MALT lymphoma and *H. pylori* infection, bacterial eradication is the recommended initial therapeutic strategy and achieves long-term complete remission in approximately 75% of cases according to cohort studies and meta-analyses[67]. In patients with localized gastric MALT lymphoma who are *H. pylori* negative, remission rates with eradication therapy are low, although attempting eradication may be reasonable in selected cases because of the possibility of false-negative results in detecting the bacterium[68]. High-grade gastric lymphomas (such as diffuse large B-cell lymphoma) do not respond to *H. pylori* eradication and require systemic immunochemotherapy[69].

Question 31: In patients with uninvestigated dyspepsia and *H. pylori* positivity, does bacterial eradication improve or cure symptoms compared with placebo or symptomatic management?

We recommend *H. pylori* eradication in patients with dyspepsia.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

A systematic review and meta-analysis of 29 randomized clinical trials (n = 6,781)[2,70] found that *H. pylori* eradication significantly reduced the proportion of patients with persistent symptoms compared with placebo (RR: 0.91; 95% CI: 0.88–0.94). The number needed to treat (NNT) was 14 to achieve a symptom benefit attributable to eradication therapy. This benefit is greater in patients in whom eradication is successful, with an RR of persistent symptoms of 0.74 and an NNT of approximately nine. The ACG guideline also supports this intervention[17]. Regarding safety, eradication regimens may be associated with mild adverse effects such as diarrhea, dysgeusia, and abdominal discomfort, but the incidence of serious events was similar to placebo. The RR of adverse effects was 2.2 (95% CI: 1.8–2.7), and all events were self-limited[70].

Question 32: In adults with uninvestigated dyspepsia in Colombia, is a "test and treat *H. pylori*" strategy cost-effective compared with empirical therapy or early endoscopy?

We recommend performing early endoscopy in adults ≥35 years with uninvestigated dyspepsia in Colombia, followed by treatment if *H. pylori* is detected, and subsequently confirming eradication.
Strength of Recommendation: Strong in favor
Quality of Evidence: Very low

In Colombia, upper gastrointestinal endoscopy is a low-cost technology that is available even at the first level of care through the national health insurance system (Plan Obligatorio de Salud), covered by the health-promoting entities (EPS), and not limited by financial barriers. This has led the 2015 national clinical guidelines to recommend endoscopy for all patients older than 35 years with persistent dyspepsia, regardless of alarm symptoms[71,72]. From the standpoint of certainty, applicability, and overall benefit–risk balance, an early-endoscopy strategy followed by specific treatment—including *H. pylori* eradication when detected—may be superior to empirical strategies in the Colombian context[71–73], although no local studies have evaluated this directly. While a test-and-treat approach may be reasonable in younger patients without risk factors, its usefulness has not been studied locally, and only national guideline recommendations are available[71–73].

Question 33: In adults with active peptic ulcer or a history of ulcer and *H. pylori* infection, does bacterial eradication reduce the risk of recurrence or complications compared with not treating the infection or using antisecretory therapy alone?

We recommend *H. pylori* eradication in all adults with active peptic ulcer disease or a history of ulcer, in addition to antisecretory therapy.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

In adults with active peptic ulcer or a history of ulcer and *H. pylori* infection, bacterial eradication significantly reduces

the risk of recurrence and complications compared with not treating the infection or using antisecretory agents alone[74,75]. Without eradication therapy, annual recurrence rates of duodenal and gastric ulcers can reach 64% and 52%, respectively; after eradication, these rates fall to <2% annually in follow-up periods of up to 10 years[74,75]. The rebleeding rate after a peptic ulcer bleed is 2.9% with eradication therapy versus 20% with antisecretory treatment without eradication, and 1.6% versus 5.6% compared with maintenance antisecretory therapy[7,76]. Confirming eradication is essential, as persistence or reinfection is associated with recurrence and complications[74].

Question 34: In adults beginning long-term treatment with non-steroidal anti-inflammatory drugs (NSAIDs) or aspirin who are infected with *H. pylori*, does prior eradication reduce the risk of peptic ulcer compared with not eradicating the infection?

We recommend *H. pylori* eradication in infected patients who are about to start long-term NSAID or aspirin therapy to reduce the risk of peptic ulcers.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

International guidelines and systematic reviews confirm that *H. pylori* eradication reduces ulcer risk by approximately half in NSAID users, with a clear additive interaction between infection and NSAID exposure. The relative risk of ulcer bleeding increases substantially when both risk factors are present, and prior eradication is superior to placebo for primary prevention of ulcers in these patients (RR ≈ 0.35; 95% CI: 0.20–0.61)[77]. A meta-analysis by Braun et al. concluded that NSAID-naive patients benefit significantly from a "screen-and-treat" strategy, whereas in chronic NSAID users without prior ulcer history, the benefit is smaller and additional antisecretory therapy is required after eradication[78].

Question 35: In adults with idiopathic iron-deficiency anemia and *H. pylori* positivity, does bacterial eradication improve hemoglobin or ferritin levels compared with not eradicating the infection?

We recommend investigating and eradicating *H. pylori* in adults with idiopathic iron-deficiency anemia to improve iron metabolism parameters (hemoglobin, serum iron, ferritin).
Strength of Recommendation: Strong in favor
Quality of Evidence: Low

Meta-analyses of randomized clinical trials have shown that combined *H. pylori* eradication therapy plus oral iron leads to greater increases in hemoglobin (standardized mean difference [SMD]: 1.48; 95% CI: 0.96–2.00) and ferritin (SMD: 1.84; 95% CI: 1.20–2.48) compared with oral iron alone[79]. These benefits are more evident in patients with moderate or severe anemia and in those with anemia refractory to oral iron therapy, with improvements in hematologic parameters typically observed within 2–6 months after eradication.

Question 36: In adults with *H. pylori* infection and gastric atrophy or intestinal metaplasia, does bacterial eradication reverse or halt lesion progression compared with not eradicating the infection?

We recommend eradicating *H. pylori* in adults with atrophy or intestinal metaplasia, as eradication halts atrophic progression and may slow the progression of metaplasia, with a potential protective effect on gastric cancer risk.
Strength of Recommendation: Strong
Quality of Evidence: Moderate–high

Recent meta-analyses and prospective studies show that *H. pylori* eradication significantly reduces progression of precancerous lesions and promotes regression of both atrophy and intestinal metaplasia, particularly in early stages[80,81]. Histologic improvement is more pronounced in atrophy than in intestinal metaplasia, and the effect is greater when eradication occurs before lesions become extensive or advanced. In patients with advanced intestinal metaplasia, reversal is less likely, but eradication remains useful to prevent progression to dysplasia and gastric cancer[80,81]. Eradication also reduces the risk of metachronous gastric neoplasia and improves gastric mucosal immune function[80].

Question 37: In adults with advanced chronic gastritis (OLGA III–IV or OLGIM III–IV) and *H. pylori* infection, does eradication—compared with not eradicating the infection—reduce the risk of developing gastric cancer?

We recommend eradicating H. pylori in patients with OLGA III–IV or OLGIM III–IV stages, as eradication reduces the risk of developing gastric cancer compared with not treating the infection.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

Patients with advanced chronic gastritis—defined as stage III–IV according to the Operative Link on Gastritis Assessment (OLGA) or the Operative Link on Gastric Intestinal Metaplasia Assessment (OLGIM)—have a

higher risk of gastric cancer[82]. Even though a "point of no return" may exist in these advanced stages, evidence shows that *H. pylori* eradication reduces gastric cancer risk compared with non-eradication, although the absolute benefit may be smaller than in earlier steps of the Correa's cascade[17,63]. A recent meta-analysis of randomized clinical trials and observational studies reported a significant reduction in gastric cancer risk after eradication (RR: 0.64 in healthy individuals and RR: 0.52 in patients with gastric neoplasia treated by endoscopic resection)[11].

Question 38: In adults older than 65 years with *H. pylori* infection, does eradication reduce the risk of developing gastric cancer compared with not performing eradication therapy?

We recommend eradicating *H. pylori* in adults older than 65 years—unless a specific contraindication exists—because eradication reduces gastric cancer risk.
Strength of Recommendation: Strong in favor
Quality of Evidence: High

A Korean population-based study of more than 900,000 individuals found that both gastric cancer incidence and mortality were significantly lower in those treated for *H. pylori* across all age groups, including those older than 70 years. In the groups aged 70–74, 75–79, and ≥80 years, standardized incidence rates (SIR) for gastric cancer were 44%–64% lower (SIR: 0.56, 0.48, and 0.36, respectively). Gastric cancer mortality also decreased significantly (standardized mortality ratios [SMR]: 0.30, 0.38, and 0.43, respectively) compared with the general population[2]. Individual risk–benefit assessment is essential in this older population, considering the potential reduction in gastric cancer risk, treatment tolerability, and overall impact on quality of life[83].

Question 39: In patients with *H. pylori* infection, does bacterial eradication reduce the risk of developing colorectal cancer (CRC) compared with non-eradication?

We do not recommend *H. pylori* eradication solely to prevent CRC; however, given the consistency of epidemiologic findings and the Bradford Hill causality criteria, eradication may be considered good practice in individual contexts—particularly in patients with additional risk factors or a family history of CRC.
Strength of Recommendation: Strong in favor
Quality of Evidence: Low

A recent retrospective study[84] including more than 800,000 individuals found that successful *H. pylori* treatment compared with ineffective treatment was associated with lower incidence and mortality from CRC after 15 years of follow-up (absolute risk reduction: 0.23%–0.25%). Patients who were *H. pylori* positive had an 18% higher CRC risk than *H. pylori*–negative individuals (adjusted HR: 1.18; 95% CI: 1.12–1.24), and a 12% higher mortality (HR: 1.12; 95% CI: 1.13–1.34). Compared with treated individuals, untreated *H. pylori*–positive patients had 23% higher CRC incidence (adjusted HR: 1.23; 95% CI: 1.13–1.34) and 40% higher mortality (adjusted HR: 1.40; 95% CI: 1.24–1.58). Based on these results, the authors concluded that *H. pylori*–positive patients may have higher CRC incidence and mortality[84].

Question 40: In patients with primary immune thrombocytopenia (ITP) and *H. pylori* infection, does eradication therapy improve platelet count or induce hematologic remission compared with non-eradication?

We suggest eradicating *H. pylori* in adults with ITP and documented infection as part of initial management, to improve platelet counts and achieve remission in a meaningful proportion of cases.
Strength of Recommendation: Strong in favor
Quality of Evidence: Moderate

In adults with ITP and *H. pylori* infection, eradication therapy improves platelet counts and can induce hematologic remission in a substantial proportion of patients, especially in settings of moderate thrombocytopenia and high infection prevalence. *H. pylori* eradication is associated with platelet responses in approximately 40%–50% of infected patients, with complete remission (platelet count ≥100 × 10^9/L) and overall response (platelet count ≥30 × 10^9/L and at least doubling of baseline) rates higher than those in untreated patients or in patients without infection[85,86].

Question 41: In patients with *H. pylori* infection, does eradication—compared with non-eradication—increase the risk of developing eosinophilic esophagitis?

We do not contraindicate *H. pylori* eradication due to concern about developing eosinophilic esophagitis.
Strength of Recommendation: Strong in favor
Quality of Evidence: Very moderate

A recent meta-analysis[5] found an inverse association between *H. pylori* infection and eosinophilic esophagitis in retrospective studies with a high risk of bias, but not in the subgroup of studies with moderate-to-low risk of bias (OR:

0.91; 95% CI: 0.69–1.21)[87]. No studies with robust data demonstrate that eradication increases the risk of developing eosinophilic esophagitis.

Question 42: In pregnant or breastfeeding women with active *H. pylori* infection, is eradication recommended during pregnancy or lactation—using regimens safe for the mother and fetus/infant—compared with deferring treatment until after delivery or temporary cessation of breastfeeding, in order to improve maternal and neonatal outcomes and prevent gastric complications?

We suggest deferring treatment in pregnant or breastfeeding women with active *H. pylori* infection until the end of pregnancy or the breastfeeding period, except in cases of severe complications (e.g., bleeding peptic ulcer), because no significant benefit has been demonstrated for routine treatment during these stages and there are potential risks for the fetus or infant.
Strength of Evidence: Strong in favor
Quality of Evidence: Low

Eradication therapy during pregnancy presents major safety challenges due to the antibiotics involved. Although *H. pylori* infection during pregnancy is associated with worse maternal and neonatal outcomes, eradication therapy should generally be deferred until after delivery—except in clinically severe situations—because no eradication regimen is considered safe for use during pregnancy or breastfeeding according to current medical literature[88–90]. Evidence supports screening and treatment before pregnancy to avoid complications. Clinical decisions must be tailored to each case and based on a careful risk–benefit assessment, following current clinical practice guideline recommendations[91,92].

Question 43: In patients with autoimmune gastritis who require *H. pylori* eradication, do therapeutic regimens without PPIs or PCABs achieve similar eradication rates and reduce the risk of worsening atrophic gastritis or developing dysplasia compared with standard regimens that include them?

In patients with autoimmune gastritis who require *H. pylori* eradication, we suggest using therapeutic regimens without PPIs or PCABs, particularly bismuth-based regimens, because they provide eradication rates similar to standard regimens and may prevent worsening of gastric atrophy and progression to precancerous lesions.
Strength of Evidence: Weak in favor
Quality of Evidence: Very low

In patients with autoimmune gastritis and achlorhydria, gastric pH is already elevated, so adding acid-suppressive therapy would not alter the gastric environment or improve antibiotic efficacy[93]. For this reason, in patients with chronic autoimmune gastritis and *H. pylori* positivity, acid suppressants are likely unnecessary during eradication therapy. A recent Italian real-world study[94] including 76 patients with severe corporal gastritis and *H. pylori* infection found that the bacterium was eradicated in 90% of cases using antibiotics without PPIs (amoxicillin, clarithromycin, and metronidazole).

Question 44: In patients with morbid obesity who are candidates for gastric bypass surgery, does active screening and eradication of *H. pylori* before surgical exclusion of the stomach—compared with not performing screening or treatment—lead to better postoperative outcomes, lower risk of gastric complications (including cancer in the excluded stomach), and improved long-term symptom control?

In candidates for gastric bypass with morbid obesity, we suggest actively screening for *H. pylori* and providing eradication therapy prior to surgery, as this strategy is associated with fewer postoperative complications in the excluded stomach, better control of digestive symptoms, and a potential reduction in long-term gastric cancer risk.
Strength of Evidence: Strong in favor
Quality of Evidence: Low

A recent systematic review of 19 observational studies evaluating the association between preoperative eradication and postoperative bariatric surgery complications estimated, in *H. pylori*–infected patients without preoperative eradication, the following odds ratios: bleeding (OR: 1.48; 95% CI: 0.80–2.73), ulcer (OR: 6.88; 95% CI: 5.60–8.45), leak (OR: 1.73; 95% CI: 0.81–3.68), stenosis (OR: 1.13; 95% CI: 0.30–4.21), and abscess (OR: 3.01; 95% CI: 0.85–10.65). Associations such as the American Society for Gastrointestinal Endoscopy and the Society of American Gastrointestinal and Endoscopic Surgeons recommend individualizing decisions regarding screening and treatment before bariatric surgery, given the lack of conclusive evidence and variability of outcomes[95].

Question 45: What is the evidence regarding the need to eradicate *H. pylori* in all family members in order to increase treatment effectiveness and prevent reinfection?

We recommend eradicating *H. pylori* in all family members to improve treatment effectiveness and reduce the risk of reinfection.

Strength of Evidence: Strong in favor
Quality of Evidence: Low

A meta-analysis of randomized clinical trials demonstrated that treating all infected family members (whole family–based eradication) results in higher eradication rates and lower recurrence compared with treating a single individual, especially in children. However, no benefit was observed in eradication rates among adult couples[96].

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Declaration of Editorial Independence

The funding and executing entities did not influence the content of this document. All work was conducted independently by the authors.

ANNEX SUMMARY OF RECOMMENDATIONS

Question 1: In adults over 18 years of age, which clinical conditions justify treating *H. pylori* infection, compared with not treating it, in terms of preventing associated complications (ulcers, anemia, gastric cancer, among others)?

Recommendation
We recommend offering treatment to all adult patients infected with *H. pylori*, regardless of the presence or absence of symptoms.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 2: In adult patients with suspected or previous *H. pylori* infection, what is the diagnostic accuracy and clinical usefulness of invasive tests (biopsy, urease) compared with noninvasive tests (urea breath test, stool antigen) to confirm diagnosis or eradication?

Recommendation
We recommend histopathology as the diagnostic method of choice to identify active infection in patients requiring upper gastrointestinal endoscopy.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Moderate

Question 3: Which noninvasive test is preferred for detecting and confirming eradication of *H. pylori*: the urea breath test using carbon-13 or carbon-14, or stool antigen testing?

Recommendation
We recommend the urea breath test with carbon-13 as the noninvasive test of choice for diagnosing *H. pylori* infection and confirming eradication.
Strength of Recommendation: Weak in favor **Quality of Evidence:** Low

Question 4: What is the effectiveness and safety of *H. pylori* eradication therapy guided by susceptibility testing (culture, polymerase chain reaction [PCR], or next-generation sequencing) compared with empirical therapy?

Recommendation
We recommend susceptibility-guided therapies for *H. pylori* infection, as they are more effective than empirical therapies.
Strength of Recommendation: Weak in favor **Quality of Evidence:** Very low

Question 5: In patients with *H. pylori* infection, how effective is tailoring treatment based on antibiotic susceptibility results and local resistance prevalence in achieving higher eradication rates compared with standard empirical therapy?

Recommendation
We should tailor clinical decisions, taking into account test availability, local resistance epidemiology, patient treatment history, probable adherence, and healthcare system resources.
Strength of Recommendation: Weak in favor **Quality of Evidence:** Low

Question 6: In patients requiring treatment for *H. pylori*, what are the diagnostic accuracy and clinical usefulness of culture with antibiogram compared with molecular tests (such as PCR or sequencing) to guide treatment and improve eradication rates?

Recommendation
In patients with *H. pylori* infection who require targeted therapy, both culture with antibiogram and molecular tests (PCR or sequencing) allow us to select effective regimens and achieve higher eradication rates compared with empirical therapy. Molecular tests show greater diagnostic sensitivity, better logistical performance, and broader clinical applicability, particularly for detecting resistance to clarithromycin and levofloxacin. Therefore, we suggest using molecular tests over culture with antibiogram, except in cases of multidrug resistance or when assessing susceptibility to less common antibiotics is necessary—situations in which culture remains useful.
Strength of Recommendation: Weak in favor **Quality of Evidence:** Moderate

Question 7: In patients requiring treatment for *H. pylori*, what are the diagnostic accuracy and clinical usefulness of culture with antibiogram compared with molecular tests (such as PCR or sequencing) to guide treatment and improve eradication rates?

Recommendation
In patients with *H. pylori* infection who require targeted therapy, both culture with antibiogram and molecular tests (PCR or sequencing) allow us to select effective regimens and achieve higher eradication rates compared with empirical therapy. However, molecular tests show greater diagnostic sensitivity, better logistical performance, and broader clinical applicability, particularly for detecting resistance to clarithromycin and levofloxacin. Therefore, we conditionally recommend molecular testing over culture with susceptibility testing, except in cases of multidrug resistance or when assessing susceptibility to less commonly used antibiotics, where culture remains useful.
Strength of Recommendation: Weak in favor **Quality of Evidence:** Low

Question 8: In patients with suspected antimicrobial resistance in *H. pylori*, which technique is the most accurate and reliable for detecting resistance: phenotypic tests or genotypic tests (such as PCR or sequencing)?

Recommendation
We recommend using genotypic tests (PCR or next-generation sequencing [NGS]) over phenotypic tests to detect resistance to clarithromycin and levofloxacin because of their greater sensitivity, rapid turnaround time, and clinical applicability. Culture remains useful when evaluating less well-characterized resistances (e.g., metronidazole, amoxicillin) or when a complete phenotypic profile is required.
Strength of Recommendation: Weak in favor **Quality of Evidence:** Moderate

Question 9: In patients with *H. pylori* infection and prior treatment failure, what are the effectiveness and cost-effectiveness of performing antimicrobial susceptibility testing compared with not performing it, in order to improve eradication rates and reduce empirical treatment failures, considering local resistance prevalence?

Recommendation
We recommend performing antimicrobial susceptibility testing after treatment failure when local availability exists and when multidrug resistance is suspected. Targeted tests (culture, PCR) or next-generation approaches (NGS) increase eradication rates compared with repeated use of empirical regimens. Cost-effectiveness improves when local resistance prevalence is high (>15% for clarithromycin or fluoroquinolones).
Strength of Recommendation: Weak in favor **Quality of Evidence:** Low

Question 10: In adults with active *H. pylori* infection, does 14-day treatment compared with seven or 10 days improve eradication rates without significantly increasing adverse effects?

Recommendation
Fourteen-day therapies are superior to 7-day and 10-day regimens for eradicating *H. pylori*.
Strength of Recommendation (7 days): Strong in favor **Quality of Evidence:** High
Strength of Evidence (10 days): Weak in favor **Quality of Evidence:** Low

Question 11: In adult patients with *H. pylori* infection and no history of prior treatment, which empirical first-line regimens are most recommended?

Recommendation
In areas with unknown clarithromycin resistance or high resistance (>15%), we recommend as first-line therapy bismuth-containing quadruple regimens (using proton pump inhibitors [PPIs] or potassium-competitive acid blockers [PCABs]) or dual therapy with amoxicillin (with PPIs or PCABs such as tegoprazan or vonoprazan).
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 12: In adult patients with *H. pylori* infection and no prior treatment, is optimized dual therapy with amoxicillin and a PPI or PCAB (such as tegoprazan or vonoprazan) as effective and safe as empirical quadruple therapy?

Recommendation
We recommend using either dual therapy or quadruple therapy as first-line options. The choice should depend on local data and patient-specific clinical characteristics.
Strength of Evidence: Strong in favor **Quality of Evidence:** Moderate

Question 13: When should standard triple therapy be used to treat *H. pylori*?

Recommendation
Standard triple therapy (STT) for *H. pylori* (PPI + amoxicillin + clarithromycin) was traditionally used as first-line treatment, but its effectiveness has declined globally due to increasing clarithromycin resistance.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 14: In adults with *H. pylori* infection living in areas with high clarithromycin and metronidazole resistance, does adding bismuth to triple therapy improve eradication rates compared with bismuth-free triple therapy?

Recommendation
We recommend adding bismuth to triple therapy in patients with confirmed or suspected resistance to clarithromycin or metronidazole to improve eradication rates. This approach is particularly relevant in regions with high resistance prevalence, such as Colombia.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Moderate

Question 15: In adults with *H. pylori* infection treated with bismuth-containing quadruple therapy, are there differences in efficacy or safety among bismuth salts (subsalicylate, subnitrate, subgallate)?

Recommendation
We recommend using any of the available bismuth salts (subsalicylate, subcitrate, subnitrate, or subgallate) as part of quadruple therapy, without preference based on efficacy, since differences in absorption and tolerability do not translate into clinically meaningful variations. We should base selection on local availability, cost, and adverse-effect profiles.
Strength of Recommendation: Strong **Quality of Evidence:** Moderate

Question 16: In adults with *H. pylori* infection, does the use of PCABs in combination with antibiotics improve eradication rates and reduce adverse events compared with PPIs?

Recommendation
We recommend using both PCABs and PPIs (esomeprazole, rabeprazole) in *H. pylori* eradication regimens, as they provide comparable efficacy and safety. The choice should depend on local availability and cost.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Moderate–high

Question 17: In adults with active *H. pylori* infection, does using probiotics as adjuvants to antibiotic therapy improve eradication rates or reduce adverse events compared with not using them?

Recommendation
We suggest using probiotics—especially *Saccharomyces boulardii* and *Lactobacillus* strains such as *L. reuteri* or *L. rhamnosus GG*—as adjuvants to *H. pylori* eradication therapy to reduce gastrointestinal adverse events and, in some cases, to improve eradication rates, particularly with triple therapy.
Strength of Recommendation: Weak in favor **Quality of Evidence:** Very low

Question 18: When should levofloxacin-containing regimens be used?

Recommendation
We recommend using levofloxacin only as rescue therapy, when one or more prior regimens have failed, particularly when clarithromycin and metronidazole resistance are present or when these antibiotics cannot be used.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Moderate

Question 19: In adult patients with *H. pylori* infection refractory to prior treatment, which empirical second- or third-line regimens achieve the highest eradication rates when compared with each other?

Recommendation
We recommend 14-day bismuth-based quadruple therapy as the empirical second- or third-line regimen of choice. Dual regimens with PPIs or PCABs plus amoxicillin and furazolidone-based regimens (bismuth–furazolidone–tetracycline quadruple therapy, or bismuth–furazolidone–amoxicillin quadruple therapy for 14 days), as well as rifabutin-based triple therapy, are also recommended as effective alternatives.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 20: In adult patients with *H. pylori* infection refractory to treatment, which factors are associated with therapeutic failure compared with patients in whom eradication is achieved?

Recommendation
We recommend a comprehensive evaluation of factors associated with therapeutic failure in patients with refractory *H. pylori* infection before initiating a new empirical regimen. This evaluation should consider prior antibiotic exposure, adherence, body weight, the type of acid-suppressive therapy (and its relationship to CYP2C19 metabolism), prior drug selection, and local availability.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Moderate

Question 21: In adults with *H. pylori* infection in regions with high prevalence of resistance to clarithromycin, metronidazole, or levofloxacin, which empirical regimens are more effective and safer compared with conventional regimens?

Recommendation
We recommend 14-day bismuth-based quadruple therapy as the main empirical regimen in regions with high resistance, and as alternatives, furazolidone-based quadruple therapy (combined with amoxicillin or tetracycline), or high-dose amoxicillin dual therapy with a PPI or PCAB, depending on local availability, cost, and individual patient tolerance.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 22: In adult patients with *H. pylori* infection who require tetracycline-based therapy, do minocycline or doxycycline offer efficacy and safety comparable to classical tetracycline?

Recommendation
We recommend using minocycline as an effective substitute for tetracycline in bismuth-based quadruple regimens for *H. pylori* eradication. Tetracycline should not routinely be replaced with doxycycline; however, doxycycline can be considered in countries where tetracycline is unavailable and where doxycycline has demonstrated effectiveness in well-structured, locally validated regimens.
Strength of Recommendation (minocycline): Strong in favor **Quality of Evidence:** High
Strength of Recommendation (doxycycline): Weak in favor **Quality of Evidence:** Very low

Question 23: In adults with *H. pylori* infection, do different PPIs (such as omeprazole, esomeprazole, lansoprazole, and rabeprazole) produce significant differences in eradication rates?

Recommendation
We recommend using esomeprazole or rabeprazole because their clinical effectiveness is not significantly affected by the CYP2C19 rapid-metabolizer genotype, or alternatively using first-generation PPIs with a 100% dose increase.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Moderate

Question 24: In adults with *H. pylori* infection and documented penicillin allergy, which alternative regimens (such as bismuth-based quadruple therapy or amoxicillin-free triple therapy) are more effective and safer when compared with each other?

Recommendation
We recommend bismuth-based quadruple therapy (PPI + bismuth + tetracycline + metronidazole) as the empirical regimen of choice for patients with penicillin allergy, and we also recommend furazolidone–tetracycline–bismuth–PPI quadruple therapy for 14 days as an alternative empirical regimen in patients who experience adverse effects or intolerance to metronidazole. PCAB-based regimens combined with antibiotics other than amoxicillin may also be considered. Fluoroquinolone-based therapies (such as levofloxacin) should be reserved as a last option, only when susceptibility testing is available and with particular attention to their potential for serious adverse events (cardiac, musculoskeletal, and neuropsychiatric).
Strength of Recommendation: Strong **Quality of Evidence:** High

Question 25: In adult populations, is *H. pylori* infection associated with lower incidence of diseases such as asthma or allergies compared with uninfected individuals?

Recommendation
We recommend not withholding *H. pylori* eradication for the purpose of preventing conditions such as asthma or allergies. Eradication should be indicated whenever active infection is present (confirmed by any method other than serology), regardless of its possible inverse association with immunologic diseases.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Moderate

Question 26: In patients with gastroesophageal reflux disease (GERD) and *H. pylori* co-infection, does eradication worsen reflux symptoms or increase the risk of Barrett's esophagus and adenocarcinoma compared with non-eradication?

Recommendation
We recommend eradicating *H. pylori* in all patients, including those with GERD.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Moderate

Question 27: In adults infected with *H. pylori*, does eradication reduce the incidence and mortality of non-cardia gastric adenocarcinoma compared with no treatment or partial treatment?

Recommendation
We recommend eradicating *H. pylori* in all infected adults to reduce the incidence and mortality of non-cardia gastric adenocarcinoma.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 28: In asymptomatic adults or adults with uninvestigated dyspepsia (UD), does detecting and eradicating *H. pylori* reduce the incidence or mortality of gastric cancer (non-cardia gastric adenocarcinoma) compared with not intervening?

Recommendation
We recommend that in regions with high gastric cancer incidence, clinicians investigate and treat *H. pylori* infection as a primary prevention strategy for non-cardia gastric adenocarcinoma.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 29: In patients with a history of early gastric cancer treated endoscopically and *H. pylori* infection, does eradication reduce the risk of developing a new gastric cancer compared with not eradicating the infection?

Recommendation
We recommend eradicating *H. pylori* in patients who undergo endoscopic resection for early gastric cancer or high-grade adenoma, as this measure significantly reduces the incidence of metachronous gastric cancers.
Strength of Recommendation: Strong **Quality of Evidence:** High

Question 30: In patients with advanced gastric cancer treated with partial or total gastrectomy and *H. pylori* infection, does eradication improve clinical prognosis in terms of survival or recurrence compared with not eradicating the infection?

Recommendation
We recommend eradicating *H. pylori* in patients with gastric cancer who have undergone surgery—whether partial or total gastrectomy—as eradication has a positive impact on survival and on reducing recurrence of metachronous gastric cancer in the gastric remnant.
Strength of Recommendation: Strong **Quality of Evidence:** Very low

Question 31: In patients with low-grade gastric MALT lymphoma, does *H. pylori* eradication induce remission compared with other therapeutic strategies or compared with not treating the infection?

Recommendation
We recommend eradicating *H. pylori* in all patients with gastric MALT lymphoma, particularly low-grade and early-stage disease, before initiating other therapies.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 32: In patients with uninvestigated dyspepsia and *H. pylori* positivity, does bacterial eradication improve or cure symptoms compared with placebo or symptomatic management?

Recommendation
We recommend *H. pylori* eradication in patients with dyspepsia.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 33: In adults with uninvestigated dyspepsia in Colombia, is a "test and treat *H. pylori*" strategy cost-effective compared with empirical therapy or early endoscopy?

Recommendation
We recommend performing early endoscopy in adults ≥35 years with uninvestigated dyspepsia in Colombia, followed by treatment if *H. pylori* is detected, and subsequently confirming eradication.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Very low

Question 34: In adults with active peptic ulcer or a history of ulcer and *H. pylori* infection, does bacterial eradication reduce the risk of recurrence or complications compared with not treating the infection or using antisecretory therapy alone?

Recommendation
We recommend *H. pylori* eradication in all adults with active peptic ulcer disease or a history of ulcer, in addition to antisecretory therapy.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 35: In adults beginning long-term treatment with non-steroidal anti-inflammatory drugs (NSAIDs) or aspirin who are infected with *H. pylori*, does prior eradication reduce the risk of peptic ulcer compared with not eradicating the infection?

Recommendation
We recommend *H. pylori* eradication in infected patients who are about to start long-term NSAID or aspirin therapy to reduce the risk of peptic ulcers.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 36: In adults with idiopathic iron-deficiency anemia and *H. pylori* positivity, does bacterial eradication improve hemoglobin or ferritin levels compared with not eradicating the infection?

Recommendation
We recommend investigating and eradicating *H. pylori* in adults with idiopathic iron-deficiency anemia to improve iron metabolism parameters (hemoglobin, serum iron, ferritin).
Strength of Recommendation: Strong **Quality of Evidence:** Low

Question 37: In adults with *H. pylori* infection and gastric atrophy or intestinal metaplasia, does bacterial eradication reverse or halt lesion progression compared with not eradicating the infection?

Recommendation
We recommend eradicating *H. pylori* in adults with atrophy or intestinal metaplasia, as eradication halts atrophic progression and may slow the progression of metaplasia, with a potential protective effect on gastric cancer risk.
Strength of Recommendation: Strong **Quality of Evidence:** Moderate–high

Question 38: In adults with advanced chronic gastritis (OLGA III–IV or OLGIM III–IV) and *H. pylori* infection, does eradication—compared with not eradicating the infection—reduce the risk of developing gastric cancer?

Recommendation
We recommend eradicating H. pylori in patients with OLGA III–IV or OLGIM III–IV stages, as eradication reduces the risk of developing gastric cancer compared with not treating the infection.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 39: In adults older than 65 years with *H. pylori* infection, does eradication reduce the risk of developing gastric cancer compared with not performing eradication therapy?

Recommendation
We recommend eradicating *H. pylori* in adults older than 65 years—unless a specific contraindication exists—because eradication reduces gastric cancer risk.
Strength of Recommendation: Strong in favor **Quality of Evidence:** High

Question 40: In patients with *H. pylori* infection, does bacterial eradication reduce the risk of developing colorectal cancer (CRC) compared with non-eradication?

Recommendation
H. pylori eradication should not be recommended systematically for the purpose of colorectal cancer prevention; however, given the consistency of epidemiological findings and the Bradford Hill causality criteria, eradication may be considered good practice in selected individuals, especially in those with additional risk factors or a family history of colorectal cancer.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Low

Question 41: In patients with primary immune thrombocytopenia (ITP) and *H. pylori* infection, does eradication therapy improve platelet count or induce hematologic remission compared with non-eradication?

Recommendation
We suggest eradicating *H. pylori* in adults with ITP and documented infection as part of initial management, to improve platelet counts and achieve remission in a meaningful proportion of cases.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Moderate

Question 42: In patients with *H. pylori* infection, does eradication—compared with non-eradication—increase the risk of developing eosinophilic esophagitis?

Recommendation
We do not contraindicate *H. pylori* eradication due to concern about developing eosinophilic esophagitis.
Strength of Recommendation: Strong in favor **Quality of Evidence:** Very moderate

Question 43: In pregnant or breastfeeding women with active *H. pylori* infection, is eradication recommended during pregnancy or lactation—using regimens safe for the mother and fetus/infant—compared with deferring treatment until after delivery or temporary cessation of breastfeeding, in order to improve maternal and neonatal outcomes and prevent gastric complications?

Recommendation
We suggest deferring treatment in pregnant or breastfeeding women with active *H. pylori* infection until the end of pregnancy or the breastfeeding period, except in cases of severe complications (e.g., bleeding peptic ulcer), because no significant benefit has been demonstrated for routine treatment during these stages and there are potential risks for the fetus or infant.
Strength of Evidence: Strong in favor **Quality of Evidence:** Low

Question 44: In patients with autoimmune gastritis who require *H. pylori* eradication, do therapeutic regimens without PPIs or PCABs achieve similar eradication rates and reduce the risk of worsening atrophic gastritis or developing dysplasia compared with standard regimens that include them?

Recommendation
In patients with autoimmune gastritis who require *H. pylori* eradication, we suggest using therapeutic regimens without PPIs or PCABs, particularly bismuth-based regimens, because they provide eradication rates similar to standard regimens and may prevent worsening of gastric atrophy and progression to precancerous lesions.
Strength of Evidence: Weak in favor **Quality of Evidence:** Very low

Question 45: In patients with morbid obesity who are candidates for gastric bypass surgery, does active screening and eradication of *H. pylori* before surgical exclusion of the stomach—compared with not performing screening or treatment—lead to better postoperative outcomes, lower risk of gastric complications (including cancer in the excluded stomach), and improved long-term symptom control?

Recommendation
In candidates for gastric bypass with morbid obesity, we suggest actively screening for *H. pylori* and providing eradication therapy prior to surgery, as this strategy is associated with fewer postoperative complications in the excluded stomach, better control of digestive symptoms, and a potential reduction in long-term gastric cancer risk.
Strength of Evidence: Strong in favor **Quality of Evidence:** Low

REFERENCES

1. Chen YC, Malfertheiner P, Yu HT, Kuo CL, Chang YY, Meng FT, et al. Global Prevalence of Helicobacter pylori Infection and Incidence of Gastric Cancer Between 1980 and 2022. Gastroenterology. 2024;166(4):605-19. https://doi.org/10.1053/j.gastro.2023.12.022
2. Malfertheiner P, Megraud F, Rokkas T, Gisbert JP, Liou JM, Schulz C, et al. Management of Helicobacter pylori infection: the Maastricht VI/Florence consensus report. Gut. 2022;71(9):1724-62. https://doi.org/10.1136/gutjnl-2022-327745
3. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Biological agents. IARC Monogr Eval Carcinog Risks Hum. 2012;100(Pt B):1-441.
4. Ferlay J, Ervik M, Lam F, Laversanne M, Colombet M, Mery L, et al. Global Cancer Observatory: Cancer Today (version 1.1) [Internet]. Lyon, France: International Agency for Research on Cancer; 2024 [consultado el 30 de septiembre de 2025]. Disponible en: https://gco.iarc.who.int/today
5. Arévalo A, Otero WA, Trespalacios AA. Helicobacter pylori: resistencia múltiple en pacientes de Bogotá, Colombia. Biomed. 2019;39(Sp. 1):125-34. https://doi.org/10.7705/biomedica.v39i3.4437
6. Otero R. W, Riquelme A, Remes-Troche JM, Laudanno O, Piscoya A, Marulanda H, et al. Actualización en el tratamiento de Helicobacter pylori: revisión del LATAMGCHMSG. Rev Gastroenterol Peru. 2024;44(4):359-73. https://doi.org/10.47892/rgp.2024.444.1797
7. Otero W, Trespalacio AA, Otero L, Vallejo MT, Torres M, Pardo R, et al. Guía de práctica clínica para el diagnóstico y tratamiento de la infección por Helicobacter pylori en adultos. Rev Col Gastroenterol. 2015;30(Supl 1):17-33.
8. Schünemann H, Brożek J, Guyatt G, Oxman A. GRADE handbook for grading quality of evidence and strength of recommendations using the GRADE approach. The GRADE Working Group; 2013. Disponible en: guideline-development.org/handbook
9. Patel A, Laine L, Moayyedi P, Wu J. AGA Clinical Practice Update on Integrating Potassium-Competitive Acid Blockers Into Clinical Practice: Expert Review. Gastroenterology. 2024;167(6):1228-38. https://doi.org/10.1053/j.gastro.2024.06.038
10. Sun M, Liu E, Yang L, Cao H, Han M. A scoping review of worldwide guidelines for diagnosis and treatment of Helicobacter pylori infection. Syst Rev. 2025;14(1):107. https://doi.org/10.1186/s13643-025-02816-0
11. Ford AC, Yuan Y, Park JY, Forman D, Moayyedi P. Eradication Therapy to Prevent Gastric Cancer in Helicobacter pylori-Positive Individuals: Systematic Review and Meta-Analysis of Randomized Controlled Trials and Observational Studies. Gastroenterology. 2025;169(2):261-76. https://doi.org/10.1053/j.gastro.2024.12.033
12. Godbole G, Mégraud F, Bessède E. Review: Diagnosis of Helicobacter pylori infection. Helicobacter. 2020;25(S1):e12735. https://doi.org/10.1111/hel.12735
13. Cardos AI, Maghiar A, Zaha DC, Pop O, Fritea L, Miere Groza F, et al. Evolution of Diagnostic Methods for *Helicobacter pylori* Infections: From Traditional Tests to High Technology, Advanced Sensitivity and Discrimination Tools. Diagnostics (Basel). 2022;12(2):508. https://doi.org/10.3390/diagnostics12020508
14. Ansari S, Yamaoka Y. Helicobacter pylori Infection, Its Laboratory Diagnosis, and Antimicrobial Resistance: a

Perspective of Clinical Relevance. Clin Microbiol Rev. 2022;35(3):e00258-21. https://doi.org/10.1128/cmr.00258-21
15. Lemos FFB, Castro CTD, Silva Luz M, Rocha GR, Correa Santos GL, De Oliveira Silva LG, et al. Urea breath test for Helicobacter pylori infection in adult dyspeptic patients: A meta-analysis of diagnostic test accuracy. World J Gastroenterol. 2024;30(6):579-98. https://doi.org/10.3748/wjg.v30.i6.579
16. Liou JM, Chen PY, Luo JC, Lee JY, Chen CC, Fang YJ, et al. Efficacies of Genotypic Resistance-Guided vs Empirical Therapy for Refractory Helicobacter pylori Infection. Gastroenterology. 2018;155(4):1109-19. https://doi.org/10.1053/j.gastro.2018.06.047
17. Chey WD, Howden CW, Moss SF, Morgan DR, Greer KB, Grover S, et al. ACG Clinical Guideline: Treatment of Helicobacter pylori Infection. Am J Gastroenterol. 2024;119(9):1730-53. https://doi.org/10.14309/ajg.0000000000002968
18. Gingold-Belfer R, Niv Y, Schmilovitz-Weiss H, Levi Z, Boltin D. Susceptibility-guided versus empirical treatment for Helicobacter pylori infection: A systematic review and meta-analysis. J Gastroenterol Hepatol. 2021;36(10):2649-58. https://doi.org/10.1111/jgh.15575
19. Bujanda L, Nyssen OP, Ramos J, Bordin DS, Tepes B, Perez-Aisa A, et al. Effectiveness of Helicobacter pylori Treatments According to Antibiotic Resistance. Am J Gastroenterol. 2024;119(4):646-54. https://doi.org/10.14309/ajg.0000000000002600
20. Dong F, Ji D, Huang R, Zhang F, Huang Y, Xiang P, et al. Multiple Genetic Analysis System-Based Antibiotic Susceptibility Testing in Helicobacter pylori and High Eradication Rate With Phenotypic Resistance-Guided Quadruple Therapy. Medicine (Baltimore). 2015;94(47):e2056. https://doi.org/10.1097/MD.0000000000002056
21. Jones NL, Koletzko S, Goodman K, Bontems P, Cadranel S, Casswall T, et al. Joint ESPGHAN/NASPGHAN Guidelines for the Management of Helicobacter pylori in Children and Adolescents (Update 2016). J Pediatr Gastroenterol Nutr. 2017;64(6):991-1003. https://doi.org/10.1097/MPG.0000000000001594
22. Vasapolli R, Ailloud F, Spießberger B, Malfertheiner P, Suerbaum S, Schulz C. Real-Time Assessment of H. pylori Infection to Guide Molecular Antibiotic Resistance Testing: A Combined Endoscopy-Gastric Juice Analysis Approach. Aliment Pharmacol Ther. 2025;61(3):465-71. https://doi.org/10.1111/apt.18378
23. Egli K, Wagner K, Keller PM, Risch L, Risch M, Bodmer T. Comparison of the Diagnostic Performance of qPCR, Sanger Sequencing, and Whole-Genome Sequencing in Determining Clarithromycin and Levofloxacin Resistance in Helicobacter pylori. Front Cell Infect Microbiol. 2020;10:596371. https://doi.org/10.3389/fcimb.2020.596371
24. Zhou Y, Zhong Z, Hu S, Wang J, Deng Y, Li X, et al. A Survey of Helicobacter pylori Antibiotic-Resistant Genotypes and Strain Lineages by Whole-Genome Sequencing in China. Antimicrob Agents Chemother. 2022;66(6):e02188-21. https://doi.org/10.1128/aac.02188-21
25. Aumpan N, Issariyakulkarn N, Mahachai V, Graham D, Yamaoka Y, Vilaichone RK. Management of Helicobacter pylori treatment failures: A large population-based study (HP treatment failures trial). PLoS One. 2023;18(11):e0294403. https://doi.org/10.1371/journal.pone.0294403
26. Chang YW, Shin GY, Kim JW, Moon JC, Chang EJ, Oh CH, et al. Cost-Effectiveness of Empirical Bismuth-Based Quadruple Therapy and Tailored Therapy After Clarithromycin Resistance Tests for Helicobacter pylori Eradication. Dig Dis Sci. 2022;67(4):1222-30. https://doi.org/10.1007/s10620-021-06938-y
27. Nan X, Zhao H, Guo L, Zheng R, Wang X, Wang Y, et al. Seven-day versus 14-day tegoprazan and tetracycline-containing quadruple therapy for first-line eradication of Helicobacter pylori infection: a randomized, open-label, noninferiority trial. Helicobacter. 2025;30(2):e70036. https://doi.org/10.1111/hel.70036
28. Jung YS, Kim S, Kim H, Noh SJ, Park JH, Park CH. 7-day versus 14-day tegoprazan-based triple therapy to treat HELICOBACTER PYLORI infection: Real-world evidence. J Gastroenterol Hepatol. 2022;37(10):1911-8. https://doi.org/10.1111/jgh.15939
29. Liou JM, Malfertheiner P, Hong TC, Cheng HC, Sugano K, Shah S, et al. Screening and eradication of Helicobacter pylori f or gastric cancer prevention: Taipei Global Consensus II. Gut. 2025;74(11):1767-1791. https://doi.org/10.1136/gutjnl-2025-336027
30. Nyssen OP, Bordin D, Tepes B, Pérez-Aisa Á, Vaira D, Caldas M, et al. European Registry on Helicobacter pylori management (Hp-EuReg): patterns and trends in first-line empirical eradication prescription and outcomes of 5 years and 21 533 patients. Gut. 2021;70(1):40-54. https://doi.org/10.1136/gutjnl-2020-321372
31. Qian HS, Li WJ, Dang YN, Li LR, Xu XB, Yuan L, et al. Ten-Day Vonoprazan-Amoxicillin Dual Therapy as a First-Line Treatment of Helicobacter pylori Infection Compared With Bismuth-Containing Quadruple Therapy. Am J Gastroenterol. 2023;118(4):627-34. https://doi.org/10.14309/ajg.0000000000002086
32. Ouyang M, Zou S, Cheng Q, Shi X, Sun M. Comparative Efficacy and Safety of Potassium-Competitive Acid Blockers and Proton Pump Inhibitors for First-Line Helicobacter pylori Eradication Therapy: A Systematic Review and Network Meta-Analysis. Helicobacter. 2024;29(6):e13150. https://doi.org/10.1111/hel.13150
33. Han Z, Li Y, Kong Q, Liu J, Wang J, Wan M, et al. Efficacy of bismuth for antibiotic-resistant Helicobacter pylori strains eradication: A systematic review and meta-analysis.

Helicobacter. 2022;27(6):e12930. https://doi.org/10.1111/hel.12930
34. Reum Choe A, Tae CH, Choi M, Shim K, Jung H. Systematic Review and Meta-Analysis: Bismuth Enhances the Efficacy for Eradication of Helicobacter pylori. Helicobacter. 2024;29(5):e13141. https://doi.org/10.1111/hel.13141
35. Wang J, Cao Y, He W, Li X. Efficacy and safety of bismuth quadruple regimens containing tetracycline or furazolidone for initial eradication of Helicobacter pylori. Medicine (Baltimore). 2021;100(51):e28323. https://doi.org/10.1097/MD.0000000000028323
36. Urrego JA, Otero W, Cedrón H, Marulanda H, Piscoya A, Frías-Ordoñez JS, et al. [Potassium-competitive acid blockers: state of the art]. Rev Gastroenterol Peru Organo Of Soc Gastroenterol Peru. 2025;45(1):38-55. https://doi.org/10.47892/rgp.2025.451.1873
37. Shah SC, Iyer PG, Moss SF. AGA Clinical Practice Update on the Management of Refractory Helicobacter pylori Infection: Expert Review. Gastroenterology. 2021;160(5):1831-41. https://doi.org/10.1053/j.gastro.2020.11.059
38. Taufiqqurrachman I, Syam AF, Maulahela H, Abdullah M, Miftahussurur M, Yamaoka Y. Comparative Efficacy and Safety of Potassium-Competitive Acid Blocker- and Proton Pump Inhibitor-Based Bismuth Quadruple Therapy for Helicobacter pylori Eradication: A Network Meta-Analysis. Gastro Hep Adv. 2025;4(9):100705. https://doi.org/10.1016/j.gastha.2025.100705
39. Liu YH, Zhang J, Li DH, Zhang YP, Li J, Guo QQ, et al. The impact of probiotics on Helicobacter pylori eradication with bismuth quadruple therapy: A systematic review and meta-analysis. Int J Antimicrob Agents. 2025;66(6):107600. https://doi.org/10.1016/j.ijantimicag.2025.107600
40. Jaramillo-Trujillo G, Otero-Regino WA, Estrada-Orozco KP. Efectividad y seguridad del uso de probióticos como adyuvantes en la erradicación de Helicobacter pylori. Revisión sistemática y metaanálisis. Rev Fac Med. 2022;71(2):e98018. https://doi.org/10.15446/revfacmed.v71n2.98018
41. Gisbert JP. Optimization Strategies Aimed to Increase the Efficacy of Helicobacter pylori Eradication Therapies with Quinolones. Molecules. 2020;25(21):5084. https://doi.org/10.3390/molecules25215084
42. Laudano O, Riquelme A, Ahumaran G, Thome M, Gollo P, Gonzalez P. efficacy and safety of a tailored helicobacter pylori eradicacion therapy based on high-dose amoxicillin in obese patients. a post-hoc analysis of two multicenter prospective cohort studies. Gastroenterology. 2023;164(6):S-47. https://doi.org/10.1016/S0016-5085(23)01069-7
43. Barreda-Costa CS, Piccini-Larco JR, Chu-Revollar LD, Salazar-Muente F, Barriga-Briceño JA, Herrera-Alzamora MA. Tratamiento cuádruple con doxiciclina, furazolidona, bismuto e inhibidor de bomba de protones sigue siendo efectivo frente al Helicobacter pylori en nuestra población. Rev Gastroenterol Peru. 2023;43(2):e1458. https://doi.org/10.47892/rgp.2023.432.1458
44. Gutiérrez O, Otero W, Cardona H, Quintero F, Orozco C, Sánchez L. Terapia cuádruple con furazolidona como tratamiento de rescate para la infección por Helicobacter pylori. Rev Col Gastroenterol. 2003;18(4):222-227.
45. Borraccino AV, Celiberto F, Pricci M, Girardi B, Iannone A, Rendina M, et al. Rifabutin as salvage therapy for Helicobacter pylori eradication: Cornerstones and novelties. World J Gastroenterol. 2022;28(45):6356-62. https://doi.org/10.3748/wjg.v28.i45.6356
46. Nyssen OP, Vaira D, Saracino IM, Fiorini G, Caldas M, Bujanda L, et al. Experience with Rifabutin-Containing Therapy in 500 Patients from the European Registry on Helicobacter pylori Management (Hp-EuReg). J Clin Med. 2022;11(6):1658. https://doi.org/10.3390/jcm11061658
47. Valladales-Restrepo LF, Correa-Sánchez Y, Aristizábal-Carmona BS, Machado-Alba JE. Treatment regimens used in the management of Helicobacter pylori in Colombia. Braz J Infect Dis. 2022;26(1):102331. https://doi.org/10.1016/j.bjid.2022.102331
48. Kotilea K, Mekhael J, Salame A, Mahler T, Miendje-Deyi VY, Cadranel S, et al. Eradication rate of Helicobacter Pylori infection is directly influenced by adherence to therapy in children. Helicobacter. 2017;22(4):e12383. https://doi.org/10.1111/hel.12383
49. Eto H, Suzuki S, Kusano C, Ikehara H, Ichijima R, Ito H, et al. Impact of body size on first-line Helicobacter pylori eradication success using vonoprazan and amoxicillin dual therapy. Helicobacter. 2021;26(2):e12788. https://doi.org/10.1111/hel.12788
50. Huang Y, Qiu S, Guo Y, Chen J, Li M, Ding Z, et al. Optimization of Minocycline-Containing Bismuth Quadruple Therapy for Helicobacter pylori Rescue Treatment: A Real-World Evidence Study. Helicobacter. 2024;29(5):e13138. https://doi.org/10.1111/hel.13138
51. Zhuge L, Wang Y, Wu S, Zhao R, Li Z, Xie Y. Furazolidone treatment for Helicobacter Pylori infection: A systematic review and meta-analysis. Helicobacter. 2018;23(2):e12468. https://doi.org/10.1111/hel.12468
52. Xie J, Peng J, Wu S, Yang K, Liu D, Shen L, et al. Efficacy and safety of tetracycline vs. amoxicillin in furazolidone-based rescue therapy for Helicobacter pylori: a real-world analysis. Ann Med. 2025;57(1):2464938. https://doi.org/10.1080/07853890.2025.2464938
53. Gao W, Zhu M, Yin Y, Zhang X, Wang L. Efficacy and safety of minocycline quadruple therapy for Helicobacter pylori eradication: A meta-analysis of RCTS. Helicobacter. 2023;28(6):e13022. https://doi.org/10.1111/hel.13022
54. Zhao J, Zou Y, Li K, Huang X, Niu C, Wang Z, et al. Doxycycline and minocycline in Helicobacter pylori

treatment: A systematic review and meta-analysis. Helicobacter. 2021;26(5):e12839. https://doi.org/10.1111/hel.12839
55. Niv Y. Doxycycline in Eradication Therapy of Helicobacter pylori - A Systematic Review and Meta-Analysis. Digestion. 2016;93(2):167-73. https://doi.org/10.1159/000443683
56. Morino Y, Sugimoto M, Nagata N, Niikiura R, Iwata E, Hamada M, et al. Influence of Cytochrome P450 2C19 Genotype on Helicobacter pylori Proton Pump Inhibitor-Amoxicillin-Clarithromycin Eradication Therapy: A Meta-Analysis. Front Pharmacol. 2021;12:759249. https://doi.org/10.3389/fphar.2021.759249
57. Gao W, Liu J, Wang X, Li J, Zhang X, Ye H, et al. Simplified Helicobacter pylori therapy for patients with penicillin allergy: a randomised controlled trial of vonoprazan-tetracycline dual therapy. Gut. 2024;73(9):1414-20. https://doi.org/10.1136/gutjnl-2024-332640
58. Liu M, Wang Y, Du B. Update on the association between Helicobacter pylori infection and asthma in terms of microbiota and immunity. Allergy Asthma Clin Immunol. 2024;20(1):4. https://doi.org/10.1186/s13223-024-00870-2
59. Zamani M, Alizadeh-Tabari S, Hasanpour AH, Eusebi LH, Ford AC. Systematic review with meta-analysis: association of Helicobacter pylori infection with gastro-oesophageal reflux and its complications. Aliment Pharmacol Ther. 2021;54(8):988-98. https://doi.org/10.1111/apt.16585
60. Erőss B, Farkas N, Vincze Á, Tinusz B, Szapáry L, Garami A, et al. Helicobacter pylori infection reduces the risk of Barrett's esophagus: A meta-analysis and systematic review. Helicobacter. 2018;23(4):e12504. https://doi.org/10.1111/hel.12504
61. Rokkas T, Pistiolas D, Sechopoulos P, Robotis I, Margantinis G. Relationship Between Helicobacter pylori Infection and Esophageal Neoplasia: A Meta-analysis. Clin Gastroenterol Hepatol. 2007;5(12):1413-1417.e2. https://doi.org/10.1016/j.cgh.2007.08.010
62. Bae S, Kim JS, Lee MW, Kim GH, Kim YI, Jung WT, et al. The Cost-Effectiveness of Adding Helicobacter Pylori Screening to the National Gastric Cancer Screening Program in Korea: Protocol for a Cost-Effectiveness Modeling Study. JMIR Res Protoc. 2025;14:e72228. https://doi.org/10.2196/72228
63. Shah SC, Wang AY, Wallace MB, Hwang JH. AGA Clinical Practice Update on Screening and Surveillance in Individuals at Increased Risk for Gastric Cancer in the United States: Expert Review. Gastroenterology. 2025;168(2):405-416.e1. https://doi.org/10.1053/j.gastro.2024.11.001
64. Yoo HW, Hong SJ, Kim SH. Helicobacter pylori Treatment and Gastric Cancer Risk After Endoscopic Resection of Dysplasia: A Nationwide Cohort Study. Gastroenterology. 2024;166(2):313-322.e3. https://doi.org/10.1053/j.gastro.2023.10.013
65. Ono A, Tanaka S, Sawada N, Goto A, Tsugane S, Muraki I, et al. Helicobacter pylori eradication and gastric cancer prevention in a pooled analysis of large-scale cohort studies in Japan. Sci Rep. 2025;15(1):21307. https://doi.org/10.1038/s41598-025-00713-z
66. Rocha Soares Menegat BL, Rocha Soares Menegat AL, Tadeu Freitas Uchôa Matheus G, Ribeiro MG, Braz L, Ayoub Silva L, et al. Effect of helicobacter pylori eradication on prognosis after gastrectomy for gastric cancer: A systematic review and meta-analysis. J Clin Oncol. 2025;43(16_suppl):e16104. https://doi.org/10.1200/JCO.2025.43.16_suppl.e16104
67. Lemos FFB, Castro CTD, Calmon MS, Silva Luz M, Pinheiro SLR, Faria Souza Mendes Dos Santos C, et al. Effectiveness of Helicobacter pylori eradication in the treatment of early-stage gastric mucosa-associated lymphoid tissue lymphoma: An up-to-date meta-analysis. World J Gastroenterol. 2023;29(14):2202-21. https://doi.org/10.3748/wjg.v29.i14.2202
68. Laoruangroj C, Habermann TM, Wang Y, King RL, Lester SC, Thompson CA, et al. Should All Patients With Stage IE Gastric Mucosa-Associated Lymphoid Tissue Lymphoma Receive Antibiotic Eradication Therapy for Helicobacter pylori ? JCO Oncol Pract. 2024;20(8):1103-8. https://doi.org/10.1200/OP.23.00624
69. Nakamura S, Hojo M. Diagnosis and Treatment for Gastric Mucosa-Associated Lymphoid Tissue (MALT) Lymphoma. J Clin Med. 2022;12(1):120. https://doi.org/10.3390/jcm12010120
70. Ford AC, Forman D, Bailey AG, Goodman KJ, Axon AT, Moayyedi P. Eradication therapy for Helicobacter pylori in non ulcer dyspepsia: systematic review and meta-analysis of randomized controlled trials. BMJ. 2020;371:m3103. https://doi.org/10.1136/bmj.m3103
71. Pineda LF, Rosas MC, Amaya M, Rodriguez A, Luque A, Agudelo F, et al. Guía de Práctica Clínica para el diagnóstico y tratamiento de la dispepsia en adultos. Rev Col Gastroenterol 2015;30(Suppl. 1):9-16.
72. Gómez M, Riveros J, Ruiz O, Concha A, Ángel D, Torres M, et al. Guía práctica para la prevención, Diagnóstico y tratamiento del cáncer gástrico temprano. Rev Col Gastroenterol 2015; 30(Suppl. 1):34- 42.
73. Oliveros-Wilches R, Grillo-Ardila CF, Vallejo-Ortega M, Gil-Parada F, Cardona-Tobón M, Páramo-Hernández D, et al. Guía de práctica clínica para la prevención primaria y secundaria y diagnóstico temprano de cáncer gástrico. Rev colomb cancerol. 2022;26(1):39-96. https://doi.org/10.35509/01239015.754
74. Ford AC, Gurusamy KS, Delaney B, Forman D, Moayyedi P. Eradication therapy for peptic ulcer disease in Helicobacter pylori-positive people. Cochrane Database Syst Rev. 2016;4(4):CD003840. https://doi.org/10.1002/14651858.CD003840.pub5
75. Vakil N. Peptic Ulcer Disease: A Review. JAMA. 2024;332(21):1832-1842. https://doi.org/10.1001/jama.2024.19094

76. Gisbert JP, Khorrami S, Carballo F, Calvet X, Gené E, Dominguez-Muñoz JE. H. pylori eradication therapy vs. antisecretory non-eradication therapy (with or without long-term maintenance antisecretory therapy) for the prevention of recurrent bleeding from peptic ulcer. Cochrane Database Syst Rev. 2004;(2):CD004062. https://doi.org/10.1002/14651858.CD004062.pub2
77. Lanza FL, Chan FK, Quigley EM; Practice Parameters Committee of the American College of Gastroenterology. Guidelines for prevention of NSAID-related ulcer complications. Am J Gastroenterol. 2009;104(3):728-38. https://doi.org/10.1038/ajg.2009.115
78. Chen MJ, Tseng HM, Huang YL, Hsu WN, Yeh KW, Wu TL, et al. Long-term outcome and short-term survival of patients with systemic lupus erythematosus after bacteraemia episodes: 6-yr follow-up. Rheumatology. 2008;47(9):1352-7. https://doi.org/10.1093/rheumatology/ken196
79. Wenzhen Y, Yumin L, Kehu Y, Bin M, Quanlin G, Donghai W, et al. Iron deficiency anemia in Helicobacter pylori infection: meta-analysis of randomized controlled trials. Scand J Gastroenterol. 2010;45(6):665-76. https://doi.org/10.3109/00365521003663670
80. Zhu F, Zhang X, Li P, Zhu Y. Effect of Helicobacter pylori eradication on gastric precancerous lesions: A systematic review and meta-analysis. Helicobacter. 2023;28(6):e13013. https://doi.org/10.1111/hel.13013
81. Liang Y, Yang Y, Nong R, Huang H, Chen X, Deng Y, et al. Do atrophic gastritis and intestinal metaplasia reverse after Helicobacter pylori eradication? Helicobacter. 2024;29(1):e13042. https://doi.org/10.1111/hel.13042
82. Benites-Goñi H, Cabrera-Hinojosa D, Latorre G, Hernandez AV, Uchima H, Riquelme A. OLGA and OLGIM staging systems on the risk assessment of gastric cancer: a systematic review and meta-analysis of prospective cohorts. Ther Adv Gastroenterol. 2025;18:17562848251325461. https://doi.org/10.1177/17562848251325461
83. Sitkin S, Lazebnik L, Avalueva E, Kononova S, Vakhitov T. Gastrointestinal microbiome and Helicobacter pylori: Eradicate, leave it as it is, or take a personalized benefit-risk approach? World J Gastroenterol. 2022;28(7):766-74. https://doi.org/10.3748/wjg.v28.i7.766
84. Shah SC, Camargo MC, Lamm M, Bustamante R, Roumie CL, Wilson O, et al. Impact of Helicobacter pylori Infection and Treatment on Colorectal Cancer in a Large, Nationwide Cohort. J Clin Oncol. 2024;42(16):1881-9. https://doi.org/10.1200/JCO.23.00703
85. Han B, Kim HJ, Yhim HY, Oh D, Bae SH, Shin HJ, et al. Sequential eradication of Helicobacter pylori as a treatment for immune thrombocytopenia in patients with moderate thrombocytopenia: a multicenter prospective randomized phase 3 study. Ann Hematol. 2022;101(7):1435-45. https://doi.org/10.1007/s00277-022-04782-2
86. Lee A, Hong J, Chung H, Koh Y, Cho SJ, Byun JM, et al. Helicobacter pylori eradication affects platelet count recovery in immune thrombocytopenia. Sci Rep. 2020;10(1):9370. https://doi.org/10.1038/s41598-020-66460-5
87. Gutiérrez-Ramírez L, Garcia-Dionisio SL, Feo-Ortega S, González-Cervera J, Tejera-Muñoz A, Lucendo AJ, et al. The Association Between HELICOBACTER PYLORI Infection and Eosinophilic Esophagitis: Systematic Review and Meta-Analysis. Helicobacter. 2025;30(2):e70038. https://doi.org/10.1111/hel.70038
88. Tang Y, Yang Y, Lv Z. Adverse pregnancy outcomes and Helicobacter pylori infection: A meta-analysis. Int J Clin Pract. 2021;75(10):e14588. https://doi.org/10.1111/ijcp.14588
89. Zhan Y, Si M, Li M, Jiang Y. The risk of Helicobacter pylori infection for adverse pregnancy outcomes: A systematic review and meta-analysis. Helicobacter. 2019;24(2):e12562. https://doi.org/10.1111/hel.12562
90. Nguyen CT, Davis KA, Nisly SA, Li J. Treatment of Helicobacter pylori in Special Patient Populations. Pharmacother J Hum Pharmacol Drug Ther. 2019;39(10):1012-22. https://doi.org/10.1002/phar.2318
91. Kothari S, Afshar Y, Friedman LS, Ahn J. AGA Clinical Practice Update on Pregnancy-Related Gastrointestinal and Liver Disease: Expert Review. Gastroenterology. 2024;167(5):1033-45. https://doi.org/10.1053/j.gastro.2024.06.014
92. ACOG Committee Opinion No. 776: Immune Modulating Therapies in Pregnancy and Lactation. Obstet Gynecol. 2019;133(4):e287-95. https://doi.org/10.1097/AOG.0000000000003176
93. Gomez Cifuentes JD, Sparkman J, Graham DY. Management of upper gastrointestinal symptoms in patients with autoimmune gastritis. Curr Opin Gastroenterol. 2022;38(6):600-6. https://doi.org/10.1097/MOG.0000000000000878
94. Dilaghi E, Mosciatti L, Dottori L, Ligato I, Esposito G, Pilozzi E, et al. Therapeutic regimens against Helicobacter pylori infection without proton pump inhibitors in patients with corpus atrophic gastritis: a real-life single-centre longitudinal observational study. Ther Adv Gastroenterol. 2025;18:17562848241308035. https://doi.org/10.1177/17562848241308035
95. Evans JA, Muthusamy VR, Acosta RD, Bruining DH, Chandrasekhara V, Chathadi KV, et al. The role of endoscopy in the bariatric surgery patient. Gastrointest Endosc. 2015;81(5):1063-72. https://doi.org/10.1016/j.gie.2014.09.044
96. Zhao J, Yuan L, Yu X, Shao Q, Ma J, Yu M, et al. Whole family—based Helicobacter pylori eradication is a superior strategy to single-infected patient treatment approach: A systematic review and meta-analysis. Helicobacter. 2021;26(3):e12793. https://doi.org/10.1111/hel.12793