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Helicobacter pylori Infection and Iron-Deficiency Anemia: Mechanisms, Clinical Evidence, and Therapeutic Implications

Alza Hamonangan Pasaribu[1]*, Wirandi Dalimunthe[2]

[1,2] **Faculty of Medicine, University Sumatera Utara, Medan, Indonesia**

Authors' contributions

The author conceived the review, conducted the literature search and evidence synthesis, revised the manuscript, and approved the final version.

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Review Article

Abstract

Iron-deficiency anemia (IDA) is common, but its persistence cannot always be explained by diet, physiological demand, blood loss, or recognized malabsorption. Helicobacter pylori infection has therefore been investigated as a potentially modifiable contributor to disturbed iron homeostasis. To critically synthesize recent evidence on the association between H. pylori infection and iron deficiency (ID)/IDA, the biological mechanisms involved, and the implications for testing and eradication. A structured narrative search of PubMed, Google Scholar, ScienceDirect, Frontiers, and publisher websites was updated through 18 June 2026. Six core human studies with direct iron or hematological outcomes were comparatively appraised, while systematic reviews, mechanistic literature, and international guidelines were used for contextual interpretation. The evidence is population-dependent rather than uniformly consistent. In non-elderly adults, infection was associated with ID (adjusted odds ratio [OR] 3.03) but not clearly with anemia or IDA. A large sex-stratified study reported an association with ID/IDA in women. Pediatric meta-analyses found increased odds of ID (OR 1.52) and IDA (OR 1.83), although one analysis found no significant association with anemia as a broad endpoint and reported substantial heterogeneity. A longitudinal birth cohort suggested a strong association between CagA-positive strains and IDA, whereas a seven-patient adolescent case series documented sustained hematological improvement after successful eradication. The most plausible pathways are impaired gastric iron handling and inflammation-mediated hepcidin upregulation; bacterial iron acquisition and microscopic blood loss remain biologically credible but less directly quantified. H. pylori should be regarded as a possible contributor, not a universal cause, of IDA. Testing is most defensible in unexplained or iron-refractory IDA after common causes have been

*Corresponding author: Email: alzapasaribu11@gmail.com

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evaluated. Pediatric testing should follow endoscopy-based guideline indications rather than routine non-invasive screening.

Keywords: *Helicobacter pylori; iron deficiency; iron-deficiency anemia; eradication therapy; narrative review*

1. INTRODUCTION

Iron-deficiency anemia (IDA) is the hematological consequence of insufficient iron availability for erythropoiesis. It is usually approached through four broad etiological domains: inadequate intake, increased physiological requirement, chronic blood loss, and impaired absorption. The clinical implications extend beyond reduced hemoglobin concentration because iron depletion may impair cognition, physical performance, pregnancy outcomes, immune function, and childhood development. Consequently, persistent IDA should not be treated as a laboratory abnormality alone; it requires an etiological diagnosis that distinguishes absolute iron deficiency from inflammation-mediated iron restriction and from anemia caused by other conditions (Lopez et al., 2016).

Helicobacter pylori is a gastric-adapted Gram-negative bacterium with a worldwide distribution. Contemporary estimates indicate that infection remains highly prevalent, although the burden varies substantially by age, geography, socioeconomic conditions, and sanitation (Li et al., 2023). Its established consequences include chronic gastritis, peptic ulcer disease, gastric adenocarcinoma, and mucosa-associated lymphoid tissue lymphoma. However, the organism has also been associated with extra-gastric disorders, including IDA, immune thrombocytopenia, and vitamin B12 deficiency (Gravina et al., 2020; Malfertheiner et al., 2022).

The possible relationship with iron metabolism is clinically plausible because the stomach influences non-heme iron bioavailability, while chronic gastric inflammation can alter acid secretion, ascorbic acid concentration, mucosal integrity, and systemic iron regulation. In addition, *H. pylori* requires iron for growth and can modify epithelial iron trafficking. These mechanisms provide biological coherence, but coherence is not equivalent to causality. Patients with infection may simultaneously experience nutritional deprivation, parasitic disease, menstrual blood loss, occult gastrointestinal bleeding, or inflammatory conditions that also reduce iron stores.

The epidemiological evidence is therefore heterogeneous. Some cross-sectional and cohort studies report lower ferritin or hemoglobin concentrations among infected participants, whereas others identify an association with iron deficiency but not with anemia or IDA. Pediatric meta-analyses also differ according to whether the endpoint is iron deficiency, strictly defined IDA, or anemia of any cause. These distinctions are important: a broad anemia endpoint includes conditions that are not iron-mediated and can dilute an association that becomes more apparent when ferritin and transferrin saturation are incorporated into the outcome definition (Hudak et al., 2017; Walle et al., 2025; Wang, Tan, & Xiao, 2025).

Therapeutic evidence is similarly nuanced. Several reports describe improvement in ferritin or hemoglobin after bacterial eradication, particularly when eradication is combined with iron supplementation, yet the magnitude and consistency of benefit vary by age, baseline iron status, study design, and follow-up duration. Successful eradication is necessary to remove a putative gastric contributor, but it does not establish that infection was the sole cause of the anemia. For this reason, eradication studies should be interpreted together with exclusion of other causes and confirmation that iron indices improve over time.

Guidelines reflect this evidentiary asymmetry. Adult consensus guidance recognizes unexplained IDA as a setting in which *H. pylori* evaluation may be clinically relevant, whereas pediatric guidance advises against routine non-invasive testing as the initial investigation for IDA. In children, testing may be considered when endoscopy is otherwise indicated after failed iron therapy and after more common etiologies have been excluded (Homan et al., 2024; Malfertheiner et al., 2022). A rigorous review must therefore differentiate evidence by population and avoid translating pediatric pooled estimates directly into adult practice.

This structured narrative review critically examines the association between *H. pylori* infection and ID/IDA, with three objectives: (1) to compare recent human evidence across adults, women, children, and adolescents; (2) to distinguish better-supported biological pathways from more speculative mechanisms; and (3) to define proportionate diagnostic and therapeutic implications that do not exceed the strength of the evidence.

2. METHODS

2.1 Review design and scope

This article was revised as a structured narrative review rather than an original research article or a formal systematic review. The approach was designed to provide a transparent and clinically oriented synthesis while avoiding a claim of exhaustive evidence capture or a new pooled effect estimate. The review focused on human evidence linking active or documented *H. pylori* infection with ferritin, serum iron, transferrin saturation, hemoglobin, ID, or IDA.

2.2 Information sources and search strategy

Searches were updated through June 2026 using PubMed, Google Scholar, ScienceDirect, Frontiers, and publisher websites. Citation chaining from relevant systematic reviews and international guidelines was used to verify older seminal evidence. The principal search strings were: ("*Helicobacter pylori*" OR "*H. pylori*") AND ("iron deficiency" OR "iron-deficiency anemia" OR ferritin OR hepcidin); and ("*Helicobacter pylori*" OR "*H. pylori*") AND eradication AND (hemoglobin OR ferritin OR iron). English-language publications from 2020 to 2026 were prioritized, while earlier studies were retained when they were mechanistically important, guideline-defining, or frequently cited in the contemporary evidence base.

Table 1. Search framework used for the structured narrative review

Sources: PubMed, Google Scholar, ScienceDirect, Frontiers, and publisher websites. Primary strings: ("*Helicobacter pylori*" OR "*H. pylori*") AND ("iron deficiency" OR "iron-deficiency anemia" OR ferritin OR hepcidin); and ("*Helicobacter pylori*" OR "*H. pylori*") AND eradication AND (hemoglobin OR ferritin OR iron). Coverage: updated 18 June 2026; English-language literature from 2020-2026 prioritized; citation chaining and publisher metadata used for verification.

2.3 Eligibility and evidence selection

Primary human studies were eligible when they directly reported an iron or hematological outcome in relation to *H. pylori* status or after eradication. Systematic reviews and meta-analyses were included when they quantified the relationship between infection and anemia, ID, or IDA. International consensus guidelines and mechanistic reviews were used to interpret clinical practice and biological plausibility. Single-patient case reports, non-human studies, articles without a direct iron-related outcome, and duplicate publications were not used as core comparative evidence. Six studies were retained as the core evidence set because they directly represented adult, female, pediatric, longitudinal, meta-analytical, and post-eradication perspectives. Additional sources were used for interpretation rather than counted as independent outcome studies.

2.4 Data extraction and methodological appraisal

For each core study, the following information was extracted: population and setting, design and sample size, method used to identify *H. pylori*, principal iron or hematological result, and the limitation most relevant to causal interpretation. Because this was a structured narrative review, no formal risk-of-bias score was calculated. Evidence was appraised narratively according to study design, diagnostic validity, directness of the outcome, control of confounding, precision, consistency, and applicability. This decision is reported explicitly to prevent the phrase “critical appraisal” from implying use of an unreported checklist.

2.5 Synthesis approach

Findings were synthesized by population and evidence function. Adult and female studies were interpreted separately from pediatric evidence; epidemiological association was separated from post-eradication response; and mechanisms were classified as better supported, plausible but incompletely quantified, or context-dependent. No new meta-analysis was performed.

3. RESULTS

3.1 Profile of the core evidence

The six core studies comprised two adult observational studies, one longitudinal pediatric cohort, two pediatric systematic reviews/meta-analyses, and one adolescent post-eradication case series. The studies were complementary rather than interchangeable: observational studies described association, meta-analyses assessed consistency across settings, the birth cohort examined bacterial virulence, and the case series provided a clinically suggestive but uncontrolled therapeutic signal.

Table 2. Comparative characteristics and findings of the six core studies

Study and setting	Population / design	H. pylori assessment	Principal result	Key limitation
Lee et al. (2022), Republic of Korea	281 non-elderly adults; cross-sectional health-check study	Rapid urease test during endoscopy	Infection was independently associated with ID (OR 3.03; 95% CI 1.22-7.57), but not clearly with anemia or IDA.	Cross-sectional design; selected health-check population; residual confounding.
Wang et al. (2024), China	998 adults; sex-stratified observational analysis	13C urea breath test	ID and IDA were associated with infection in women, with lower iron indices among infected participants; the association was not equivalent across sexes.	Cross-sectional data; menstrual and dietary factors may be incompletely controlled.
Queiroz et al. (2024), Brazil	Longitudinal birth cohort; 102 children analyzed after infancy	Culture/serology with CagA strain characterization	CagA-positive infection was strongly associated with IDA (OR 13.14; 95% CI 4.49-38.45).	Small cohort, wide confidence interval, virulence-specific and setting-specific finding.
Walle et al. (2025), multi-country	Systematic review/meta-analysis; 9 pediatric studies	Varied methods across included studies	Children with infection had greater odds of anemia overall (pooled OR 2.68; 95% CI 1.44-4.99); the IDA subgroup was also significant but imprecise.	Heterogeneity by design and diagnostic method; mixed anemia definitions.

Study and setting	Population / design	H. pylori assessment	Principal result	Key limitation
Wang, Tan, & Xiao (2025), multi-country	Systematic review/meta-analysis; 13,573 children from 13 countries / 16 regions	Varied methods across included studies	Higher odds of ID (OR 1.52; 95% CI 1.32-1.74) and IDA (OR 1.83; 95% CI 1.01-3.33), but no significant association with broad anemia (OR 0.94; 95% CI 0.73-1.21).	Substantial heterogeneity, especially for anemia; age and outcome definition influenced results.
Kato et al. (2022), Japan	7 adolescents with severe recurrent/refractory IDA; longitudinal case series	Clinically confirmed infection and documented eradication	Hemoglobin, serum iron, and ferritin improved after successful eradication and remained improved during follow-up.	Very small uncontrolled series; co-interventions and regression to the mean cannot be excluded.

3.2 Adult and sex-specific evidence

Adult evidence suggests that iron depletion may be more readily detected than overt anemia. In the Korean health-check study, *H. pylori* infection was independently associated with ID, yet the prevalence of anemia and strictly defined IDA did not differ significantly between infected and uninfected participants (Lee et al., 2022). This pattern is clinically plausible because ferritin depletion precedes the decline in hemoglobin. It also warns against treating ID, IDA, and anemia as interchangeable endpoints.

The study by Wang et al. (2024) provided a sex-stratified perspective. Among 998 adults assessed using the 13C urea breath test, the relationship between infection and low iron status was more apparent in women than in men. Women of reproductive age have a competing and often dominant source of iron loss through menstruation; consequently, infection may function as an additional stressor rather than an isolated cause. The cross-sectional design cannot establish temporal direction, and residual confounding from menstrual volume, diet, parity, and supplementation remains possible.

Taken together, the adult studies support a measured interpretation: *H. pylori* may be associated with diminished iron stores, but its relationship with overt IDA depends on host susceptibility and competing etiologies. The adult literature therefore does not justify universal screening of all patients with anemia.

3.3 Evidence in children and adolescents

Pediatric evidence is quantitatively stronger but remains heterogeneous. Walle et al. (2025) reported that infected children had higher pooled odds of anemia. However, subgroup effects varied by study design and diagnostic method, which indicates that the headline estimate should not be interpreted as a single, context-free causal effect.

The larger meta-analysis by Wang, Tan, and Xiao (2025) clarified why endpoint definitions matter. Infection was associated with ID and IDA, with pooled ORs of 1.52 and 1.83, respectively, but not with anemia as a broad endpoint. Heterogeneity was moderate for ID, substantial for IDA, and very high for anemia. Age was identified as an important source of variation. The discordance between this analysis and Walle et al. is not necessarily contradictory: different inclusion criteria, diagnostic methods, age structures, and definitions of anemia can produce materially different pooled results.

The longitudinal Brazilian cohort adds a virulence dimension. Queiroz et al. (2024) reported a strong association between CagA-positive infection and IDA, suggesting that bacterial

phenotype may influence iron disturbance. Nevertheless, the estimate was derived from a small cohort and had a wide confidence interval. It should be interpreted as biologically informative rather than as a universally transferable magnitude of risk.

Therapeutic evidence in adolescents is clinically suggestive but less methodologically secure. Kato et al. (2022) followed seven teenagers with severe recurrent or refractory IDA and documented sustained improvement in hemoglobin, serum iron, and ferritin after successful eradication. The temporal sequence supports a contributory role for infection, but the absence of a control group and the small sample preclude a precise estimate of treatment effect.

3.4 Biological mechanisms: strength and uncertainty

The proposed mechanisms should not be presented as equally established. They can be organized into three evidentiary tiers.

Better-supported mechanism 1 - impaired gastric iron handling. Chronic gastritis can reduce acid secretion and gastric ascorbic acid, impairing the conversion and solubilization of non-heme ferric iron into a form more readily absorbed in the proximal intestine. Experimental and clinical evidence also indicates that *H. pylori* can suppress gastric proton-pump expression and alter the gastric environment (Saha et al., 2010; Kato, Gold, & Kato, 2022).

Better-supported mechanism 2 - inflammation-hepcidin-ferroportin regulation. Infection-induced inflammatory signaling can increase hepatic hepcidin. Hepcidin promotes internalization and degradation of ferroportin, reducing intestinal iron export and iron release from macrophages. This produces functional iron restriction and may coexist with absolute iron depletion (Navidifar et al., 2025; Pu et al., 2025).

Plausible but less directly quantified mechanism - bacterial iron acquisition and altered epithelial trafficking. *H. pylori* depends on iron and can perturb epithelial iron trafficking. CagA-positive strains may exert a stronger effect, consistent with the Brazilian cohort, but the contribution of bacterial consumption to whole-body iron balance has not been quantified adequately in routine clinical populations (Tan et al., 2011; Queiroz et al., 2024).

Context-dependent mechanism - chronic microscopic blood loss. Erosive gastritis or peptic ulcer disease can cause occult gastrointestinal bleeding. This mechanism is clinically important when mucosal lesions are present, but it cannot explain IDA in infected patients without bleeding and should not be assumed solely from a positive infection test.

3.5 Eradication response and the limits of therapeutic inference

Improvement after eradication strengthens biological plausibility because it introduces temporality and reversibility. Earlier adult evidence showed reversal of otherwise unexplained IDA after eradication in patients with asymptomatic gastritis, and later pediatric reports have documented similar trajectories (Annibale et al., 1999; Kato et al., 2022). Meta-analytical evidence suggests that eradication combined with iron therapy can improve ferritin more consistently than iron therapy alone, whereas the hemoglobin effect is smaller and more heterogeneous (Hudak et al., 2017; Wang, Tan, & Xiao, 2025).

Eradication studies nevertheless require careful interpretation. Iron supplementation, improved adherence, dietary change, treatment of gastric lesions, and the natural course of disease may contribute to the observed improvement. Moreover, a randomized trial comparing eradication regimens, such as the HEPYSE trial, establishes the effectiveness of bacterial elimination strategies but does not directly establish hematological benefit when hemoglobin and ferritin are not primary outcomes. For that reason, eradication-regimen trials were not treated as core direct evidence in this review.

4. DISCUSSION

4.1 Overall interpretation

The revised evidence synthesis supports an association between *H. pylori* infection and disturbed iron homeostasis, but the association is conditional rather than universal. The

most reproducible signal is lower iron storage or increased odds of ID/IDA in susceptible populations. The signal is weaker when the outcome is anemia of any cause, because anemia is etiologically heterogeneous. This distinction explains why studies can agree that infection is related to iron depletion while disagreeing on the prevalence of anemia.

The causal case is strengthened by biological coherence, longitudinal observations, strain-specific findings, and post-eradication improvement. However, each component has limitations. Biological mechanisms can coexist without determining the magnitude of clinical effect; longitudinal cohorts may remain confounded; CagA findings may not generalize to other strains; and therapeutic response can be influenced by iron supplementation and other co-interventions. The most defensible conclusion is therefore that *H. pylori* can be a modifiable contributing factor in a subset of patients.

4.2 Population-specific meaning

In adults, infection appears more consistently associated with depleted iron stores than with overt IDA. Clinical interpretation must therefore account for menstrual loss, gastrointestinal bleeding, dietary intake, inflammatory disease, renal disease, medication exposure, and malabsorption. A positive *H. pylori* test should not end the etiological investigation when another plausible source of iron loss remains.

In children and adolescents, the effect may be more clinically visible because iron requirements are high during growth and baseline reserves may be limited. Nevertheless, pediatric pooled estimates should not be transferred directly to adults. Nor should they be used to justify indiscriminate test-and-treat strategies, because invasive testing, antibiotic exposure, and resistance have different risk-benefit implications in children.

4.3 Heterogeneity and conflicting findings

Differences across studies likely arise from at least six sources: (1) variable definitions of anemia, ID, and IDA; (2) differing infection tests, including serology, breath testing, stool antigen, histology, and culture; (3) age and sex distributions; (4) background nutritional and infectious burdens; (5) incomplete adjustment for menstrual or gastrointestinal blood loss; and (6) bacterial strain heterogeneity. These factors can change both exposure classification and the probability that iron depletion progresses to low hemoglobin. The conflicting broad-anemia results in the two 2025 pediatric meta-analyses should therefore be presented as an important indicator of outcome-definition sensitivity rather than concealed by a generalized statement of consistency.

4.4 Clinical implications

Adults: Consider testing for *H. pylori* in unexplained or iron-refractory IDA after adequate assessment of dietary insufficiency, menstrual loss, occult gastrointestinal bleeding, medication-related bleeding, celiac disease or other malabsorption, and relevant chronic disease. This position is consistent with adult consensus guidance that recognizes unexplained IDA as an extra-gastric indication in which eradication may be beneficial (Malfertheiner et al., 2022).

Children and adolescents: Do not use non-invasive *H. pylori* testing as the routine initial investigation for IDA. When IDA persists despite appropriate iron therapy, common causes have been excluded, and upper endoscopy is otherwise clinically indicated, guideline-based testing may be considered; confirmed infection should then be treated with an antibiotic-susceptibility-informed regimen where possible (Homan et al., 2024).

After treatment: Confirm successful eradication according to guideline timing and reassess hemoglobin, ferritin, and the response to iron replacement. Persistent or recurrent IDA after eradication requires renewed investigation rather than automatic retreatment.

Avoid overreach: The current evidence does not support population-wide screening for *H. pylori* solely to prevent IDA, empiric eradication without documented infection, or the assumption that a positive test establishes the cause of anemia.

4.5 Strengths and limitations of this review

The principal strength of this review is its explicit separation of ID, IDA, and broad anemia, together with population-specific interpretation and correction of bibliographic and evidentiary misclassification in the original manuscript. The core table also distinguishes direct iron-outcome studies from eradication-regimen research that does not measure hematological benefit.

Several limitations remain. This was a structured narrative review rather than a registered systematic review; therefore, the search may not have captured every eligible study, no formal risk-of-bias score was calculated, and publication bias was not quantified. The core synthesis included only six studies with mixed designs. Diagnostic methods and outcome definitions varied, and much of the quantitative evidence came from pediatric populations or women, limiting generalizability. The review did not perform an independent meta-analysis and cannot estimate a causal treatment effect. Finally, clinical recommendations depend partly on consensus guidelines and should be adapted to local prevalence, endoscopy availability, antimicrobial resistance, and patient-specific risk.

5. CONCLUSION

Helicobacter pylori infection is associated with impaired iron status in several populations, but the strength of the relationship depends on age, sex, bacterial virulence, diagnostic method, and the outcome used. The evidence is more consistent for ID and strictly defined IDA than for anemia as a broad endpoint. Impaired gastric iron handling and inflammation-mediated hepcidin regulation are the most credible pathways, while bacterial iron acquisition and microscopic blood loss are plausible but context-dependent. Successful eradication can improve iron indices in selected patients, although treatment evidence remains heterogeneous and does not establish benefit for every infected individual. Accordingly, *H. pylori* testing is most appropriate in unexplained or iron-refractory IDA after common causes have been evaluated. In children, testing should follow endoscopy-based guideline indications rather than routine non-invasive screening. Future prospective studies should use standardized IDA definitions, validated infection tests, prespecified confounder control, and hematological outcomes measured after confirmed eradication.

Competing interests

The author declares no known financial or non-financial competing interests that could have influenced the work reported in this article.

Ethical approval

Ethical approval was not required because this study reviewed previously published literature and did not involve new human participants, identifiable data, or animal experimentation.

Data availability

All evidence discussed in this review is available in the cited publications.

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