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REVIEW ARTICLE

Hygienic-dietary measures for the control and prevention of acid-peptic disease

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Abstract

Peptic ulcer disease is a multifactorial condition, but *Helicobacter pylori* infection and the use of nonsteroidal anti-inflammatory drugs are considered the main risk factors. Diet is considered a related factor; however, current scientific evidence remains inconclusive. Therefore, the objective of this review was to analyze the available scientific evidence on dietary habits and peptic ulcer disease, and their impact on the clinical course and symptom control. A narrative literature review was conducted in PubMed, including observational studies, clinical trials, systematic reviews, and meta-analyses published between 2015 and 2025. The findings indicate that high consumption of alcohol and ultra-processed foods is associated with a higher frequency of gastric inflammation and a greater presence of symptoms, while diets rich in fiber, fruits, and vegetables could be considered a protective factor. Foods such as coffee and chili peppers have not been shown to have a direct causal role, so their restriction should be individualized according to the patient's tolerance. Hygienic and dietary measures could be recommended as adjuncts to pharmacological treatment, especially the use of probiotics in *H. pylori* infection, which has stronger scientific support. In conclusion, all dietary recommendations should be based on healthy and individualized eating patterns, avoiding unnecessary restrictions that affect quality of life.

Keywords: Peptic ulcer. *Helicobacter pylori.* Diet. Probiotics.

Medidas higiénico-dietéticas para el control y la prevención de la enfermedad ácido-péptica

Resumen

La enfermedad ácido-péptica es de etiología multifactorial, pero la infección por *Helicobacter pylori* y el uso de antiinflamatorios no esteroideos se consideran los principales factores de riesgo. La dieta se considera uno de los factores relacionados; sin embargo, la evidencia científica actual aún no es contundente, por lo cual el objetivo de esta revisión fue analizar la evidencia disponible sobre los hábitos dietéticos y los síntomas de la enfermedad ácido-péptica, así como su impacto en la evolución clínica y el control de los síntomas. Se realizó una revisión narrativa de la literatura en PubMed, incluyendo estudios observacionales, ensayos clínicos, revisiones sistemáticas y metaanálisis publicados entre 2015 y 2025. Los hallazgos indican que el consumo elevado de alcohol y de alimentos ultraprocesados se asocia con una mayor frecuencia de inflamación gástrica y una mayor presencia de síntomas, mientras que las dietas ricas en fibra, frutas y verduras podrían considerarse factores protectores. Alimentos como el café y el chile no han demostrado un papel causal directo, por lo que su restricción

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debe individualizarse según la tolerancia del paciente. Las medidas higiénico-dietéticas podrían recomendarse como coadyuvantes al tratamiento farmacológico, en especial el uso de probióticos en la infección por *H. pylori*, ya que cuentan con un mayor respaldo científico. En conclusión, todas las recomendaciones dietéticas deben basarse en patrones alimentarios saludables e individualizados, evitando restricciones innecesarias que afecten la calidad de vida.

Palabras clave: Úlcera péptica. *Helicobacter pylori.* Dieta. Probióticos.

Introduction

Peptic ulcer disease (PUD) is a condition characterized by lesions in the gastric and duodenal mucosa, caused by an imbalance between acid secretion, pepsin, and the defense mechanisms of the mucosa. Its etiology is multifactorial, but there are some determining factors, such as *Helicobacter pylori* infection, the use of nonsteroidal anti-inflammatory drugs (NSAIDs), physiological stress, and diet. Specifically, *H. pylori* infection and NSAIDs trigger complications such as ulcer formation.[1-3]

Although diet has been considered a factor associated with the etiology, evolution, and symptoms of the disease, scientific evidence is scarce and not conclusive. Nevertheless, patients report symptom improvement with highly restrictive regimens, and recent studies show that certain specific dietary patterns influence the risk of developing PUD, symptom intensity, or treatment response. Although diet does not constitute a primary etiological factor, the intake of certain foods could modulate the manifestation of the disease and significantly affect the patient's quality of life[2-4] (Fig. 1). Therefore, the objective of this review was to evaluate the hygienic-dietary factors with the highest level of evidence related to PUD symptoms.

Method

A narrative literature review was conducted in PubMed, limited to publications between January 2015 and December 2025. Observational studies, clinical trials, meta-analyses, and systematic reviews that evaluated the relationship between diet, nutrition, and PUD symptoms were included. The level of evidence was classified according to the GRADE scale (Grading of Recommendations Assessment, Development, and Evaluation),[5] and values were considered significant with $p < 0.05$.

Results and discussion

In patients with PUD, diet could worsen symptoms after the intake of certain foods; however, current scientific evidence suggests that many of these foods do not participate directly in the etiopathogenesis of the disease, although they can modulate symptom intensity and perception of discomfort. Empirically, foods such as alcohol, chili peppers, and various seasonings have been classified as symptom triggers (irritating foods) or as protective factors[6] (Table 1).

Foods associated with PUD symptoms

Alcohol

Alcohol consumption is consistently associated with direct damage to the gastric mucosa, increased epithelial permeability, and local inflammation. Observational studies have demonstrated a significant association between high alcohol consumption and a higher prevalence of peptic ulcer, as well as poorer symptom control[7]; however, epidemiological studies and meta-analyses show an inverse association between moderate alcohol consumption and the presence of *H. pylori*, suggesting that those who consume alcohol have lower seroprevalence or active infection than non-drinkers.[8] This inverse relationship appears to be stronger with certain types of beverages, such as wine and cocktails, and in people over 40 years of age.[8] On the other hand, in a meta-analysis evaluating the dose-response effect, moderate alcohol consumption (10-30 g/day) was found to be associated with a lower risk of infection, but the effect diminishes and is less consistent with higher consumption.[9] Regarding dietary factors, the quality of available evidence is heterogeneous. Alcohol consumption has moderate-level evidence, derived mainly from observational studies and some clinical trials, demonstrating direct gastric mucosal injury, increased inflammation, and exacerbation of dyspeptic symptoms, as well as its association with a less favorable clinical course.

Coffee and caffeine

Coffee is one of the most consumed beverages in the world. It contains multiple bioactive compounds,

Table 1. Foods associated with symptoms of acid-peptic disease according to the GRADE classification

Food	Clinical effect reported	Level of evidence
Alcohol	Direct gastric mucosal injury, increased inflammation and dyspeptic symptoms; association with poorer clinical course	Moderate
Coffee/caffeine	Increased dyspeptic symptoms; variable effect on acid secretion	Low
Ultra-processed foods	Higher risk of developing peptic ulcer and chronic gastritis	Moderate
Foods rich in saturated fats/fried foods	Delayed gastric emptying and worsening of symptoms	Low
Spicy foods (chili peppers, capsaicin)	Do not worsen healing, but may cause symptoms in sensitive patients	Moderate

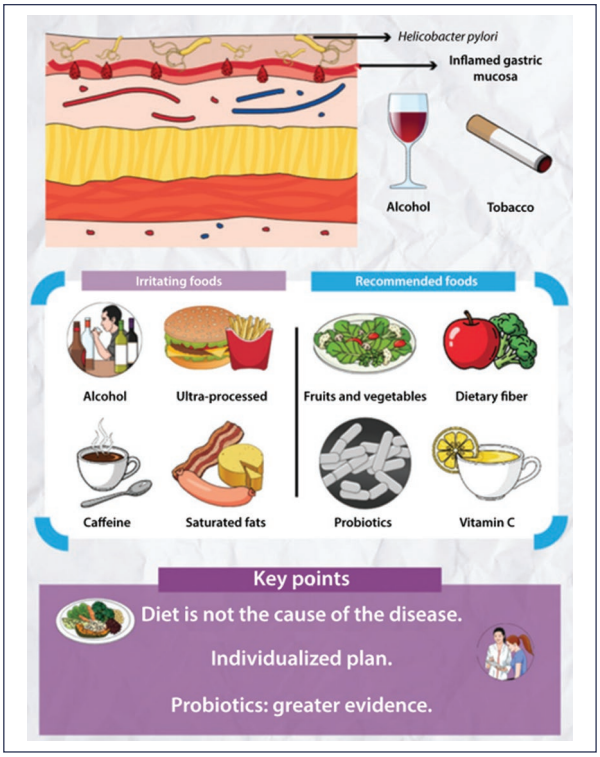

Figure 1. Graphic summary.

including caffeine, polyphenols (mainly chlorogenic acids), and diterpenes such as cafestol and kahweol, with antioxidant, anti-inflammatory, antifibrotic, and microbiome-modulating effects.[10]

Regarding PUD symptoms, studies have not demonstrated a significant association between coffee consumption and the development of gastric ulcer, duodenal ulcer, or PUD, which questions its classification as a non-recommended food.[10,11] On the other hand, although recent evidence does not demonstrate a direct relationship between coffee and PUD symptoms, it stimulates gastric acid secretion and some studies report exacerbation of symptoms, possibly associated with sugar in the preparation, so its restriction is recommended empirically and in an individualized manner.[12,13] The effect of coffee and caffeine is supported by low-level evidence, with inconsistent results regarding its impact on acid secretion and exacerbation of dyspeptic symptoms, without conclusive demonstration of sustained structural damage.

Ultra-processed foods

The term "ultra-processed foods" (UPF) refers to industrialized products with multiple ingredients and additives, typically high in calories, saturated fats, sugar, and salt, and low in fiber, micronutrients, and bioactive compounds. This type of diet has been associated with various metabolic and inflammatory damage. Some recent studies have explored whether UPF consumption is associated with a higher incidence of *H. pylori* infection and with PUD symptoms.[14,15] In a case-control study conducted by Ebrahimi et al.[14] in 2024, it was reported that individuals in the highest quartile of UPF consumption presented a significant increase in the probability of *H. pylori* infection (adjusted odds ratio approximately 1.5-2.0, depending on the statistical model), even after adjusting for body mass index, age, sex, energy consumption, physical activity, and smoking. Consistently, in the prospective cohort of the SUN project,[15] higher UPF consumption was associated with an increased risk of peptic ulcer disease, with a relative risk close to 1.3-1.5 at the highest intake levels compared to the lowest.

UPF are foods that present moderate evidence suggesting an association with higher risk of peptic ulcer and chronic gastritis, although most data come from epidemiological studies, which limits direct causal inference. Similarly, foods rich in saturated fats or fried foods present in UPF show low evidence, mainly based

on physiological studies and clinical reports, suggesting delayed gastric emptying and possible symptomatic worsening, without robust evidence of mucosal lesion progression.

Chili peppers and capsaicin

Chili pepper is a common ingredient in the diet of numerous cultures, especially in Mexico. Its main active compound is capsaicin, which acts on the vanilloid type 1 receptor, involved in the perception of painful, thermal, and mechanical stimuli. Acute exposure to capsaicin can induce symptoms such as retrosternal burning, abdominal pain, nausea, and sensation of heat; however, repeated exposure destabilizes the receptor and improves tolerance. It has not been shown to affect gastric mucosal healing, which justifies a recommendation based on individual tolerance.[16]

On the other hand, in addition to its sensory effects, capsaicin possesses multiple potentially beneficial properties. As systemic effects, it is attributed antioxidant, anti-inflammatory, lipid-lowering, anti-obesity, and cardioprotective properties. In the digestive tract, a gastroprotective effect, increased mucosal blood flow, regulation of visceral nociception, and improved micronutrient absorption have been described. Clinical studies have shown that, in patients with functional dyspepsia and irritable bowel syndrome, regular chili pepper consumption decreases symptoms and increases the threshold of visceral sensitivity.[17,18]

Currently, there is insufficient evidence to recommend capsaicin as therapy; however, it is suggested not to restrict its consumption in patients with stable *H. pylori*, except in cases of individual intolerance.[5] Spicy foods, including chili peppers and capsaicin, have moderate evidence indicating that they do not interfere with mucosal healing; however, they can induce symptoms in patients with visceral hypersensitivity, which suggests a predominantly functional rather than structural effect.

Foods associated with improvement of PUD symptoms

In patients with PUD, international guidelines have recommended easily digestible foods, low in fat, of soft consistency and with low acid content, such as rice, oatmeal, banana, pear, lean meats, and low-fat dairy products, which can contribute to symptom improvement.

Specifically, lean proteins that are easily digestible contribute to maintaining an adequate nutritional status without excessively stimulating acid secretion, and are well tolerated by most patients. Fruits, vegetables, and dietary fiber have shown, in various observational studies and systematic reviews, that they are associated with a lower risk of PUD due to their anti-inflammatory and microbiome-modulating effects.[19] A diet rich in non-acidic fruits and vegetables has moderate evidence suggesting a lower association with gastric inflammation and better digestive tolerance. However, these findings derive mainly from observational studies and some clinical trials with methodological limitations, although they have demonstrated consistency in symptom improvement[20] (Table 2). Similarly, adequate intake of dietary fiber presents moderate evidence, being associated with lower risk of peptic ulcer disease and better gastrointestinal function. Most data come from prospective epidemiological studies, with biological plausibility related to modulating effects on the microbiota and inflammation.

Probiotics

Standard treatment in patients with *H. pylori* infection includes antibiotics and proton pump inhibitors, which usually trigger bacterial resistance and side effects. This has led to investigating whether probiotics could improve therapeutic outcomes.[20,21]

Scientific evidence from recent meta-analyses indicates that the use of probiotics as adjuvants in the treatment of *H. pylori* infection improves the eradication rate and decreases the incidence of gastrointestinal adverse effects, such as diarrhea, nausea, and abdominal pain, through mechanisms such as modulation of the gastrointestinal microbiota, production of lactic acid and bacteriocins that inhibit *H. pylori* growth, and interference with bacterial adhesion to the gastric epithelium and reduction of adverse effects of antibiotic therapy. Most meta-analyses show that probiotics added to standard therapy modestly increase the probability of *H. pylori* eradication (relative risk: 1.10-1.60) compared to standard therapy alone. However, the results are not significant when compared strictly to placebo, suggesting methodological differences between both. Nevertheless, to date, probiotic consumption is the therapeutic strategy with the highest evidence as an adjuvant to decrease adverse effects and improve tolerability of standard antibiotic treatment, with an additional benefit in the eradication rate of *H. pylori*[22-26] (Table 3), particularly those present in yogurt and kefir, supported by numerous randomized clinical trials and meta-analyses.

Table 2. Recommended foods for symptom improvement of PUD according to the GRADE classification

Food	Clinical effect reported	Level of evidence (Oxford)
Fruits and vegetables (non-acidic)	Lower gastric inflammation and better digestive tolerance	Moderate
Dietary fiber	Association with lower risk of peptic ulcer and better gastrointestinal function	Moderate
Lean proteins (chicken, fish, legumes)	Maintain nutritional status without stimulating excessive acid secretion	Low
Probiotics (yogurt, kefir)	Reduction of adverse effects and improvement of *H. pylori* eradication	High
Vitamin C and dietary antioxidants	Possible support in healing and *H. pylori* control	Moderate
Olive oil and unsaturated fats	Better gastric tolerance compared to saturated fats	Low

Table 3. Probiotics and their relationship with acid-peptic disease symptoms

Study	Design	Population	Probiotic intervention	Results
Tanashat et al.[21]	91 RCTs	n ≈ 13,680	Probiotics plus standard therapy	Higher eradication (OR: ≈ 1.60), fewer side effects (diarrhea, pain) with multiple strains compared to controls
Felley and Michetti[22]	Review	Variable	Conceptual	Probiotics can modulate adhesion and inflammation
Lu et al.[23]	Meta-analysis	21 RCTs	Probiotics plus standard therapy vs. placebo	No statistically significant improvement was found in some placebo vs. control analyses
Penumetcha et al.[24]	11 studies	Variable	Probiotics vs. standard therapy	Probiotics alone are not very effective for eradication by themselves; in combination, there is slight improvement and reduction of effects
Yang et al.[25]	28 meta-analyses	534 combined RCTs	Probiotics plus standard therapy	Higher eradication (RR: 1.10; 95% CI: 1.06-1.14), lower risk of effects (RR: 0.54)
Zhang et al.[26]	12 RCTs	2,144	Probiotics before therapy	Higher eradication (≈ 80% vs. 70%) and fewer adverse effects

RCTs: randomized controlled trials; 95% CI: 95% confidence interval; OR: odds ratio; RR: relative risk.

Vitamin C supplementation has also shown potential benefits in reducing *H. pylori* bacterial load and in mucosal healing, although the evidence is still limited.[27] Specifically, vitamin C and other dietary antioxidants show moderate evidence, although limitations persist regarding sample size and heterogeneity of interventions.

Other dietary strategies, such as lean proteins, olive oil, and unsaturated fats, show a favorable physiological profile and adequate gastric tolerance, but the evidence supporting their direct clinical benefit on structural outcomes (inflammation, healing, or prevention of ulcer disease) is limited and is mainly based on observational studies, physiological studies, and expert recommendations. Therefore, their inclusion in the diet can be considered reasonable within a comprehensive nutritional approach, but a specific recommendation based on robust high-level evidence cannot be established with high certainty.

Hygienic-dietary measures in PUD

It has been shown that some foods have a certain relationship with the disease, so it is important to consider avoiding or moderating their consumption to improve symptoms. However, changes must also be made in eating habits that could help regulate acid secretion, decrease symptoms, and improve quality of life. Changes such as having fractional and regular meals, avoiding prolonged fasting, chewing slowly, following a fiber-rich diet, increasing the supply of high

biological value proteins, performing physical activity to promote proper protein absorption, and considering the incorporation of probiotics through fermented foods, are general strategies that favor patients' symptoms and treatment response.[4]

Scientific evidence confirms that diet and a healthy lifestyle do not replace medical treatment for PUD symptoms, but play an important role in symptom control and secondary prevention. Avoiding universal restrictive recommendations and treating patients' diet individually, based on tolerance and healthy dietary patterns, could improve symptoms and treatment response.[6]

Conclusions

Diet can influence the clinical manifestations of PUD symptoms and patients' quality of life. High consumption of alcohol and UPF is associated with a worse prognosis, while diets rich in fiber, fruits, and vegetables are safe and potentially protective. Foods such as coffee and chili peppers do not have a direct causal role, so their restriction should be individualized according to tolerance. Hygienic-dietary measures complement pharmacological treatment, highlighting probiotics as adjuvants with greater scientific support, especially in the presence of *H. pylori*. More clinical trials are required to define specific dietary recommendations.

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Conflicts of interest

The author declares no conflicts of interest.

Ethical considerations

Protection of people and animals. The author declares that no experiments were performed on humans or animals for this research.

Confidentiality, informed consent, and ethical approval. The study does not involve personal data, medical records, or human biological samples, so it does not require ethical approval. The SAGER guidelines do not apply.

Declaration on the use of artificial intelligence. The author declares that no type of generative artificial intelligence was used for the writing or content creation of this manuscript.

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