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Review article

Gastric Cancer: Detection and Prevention

Ricardo Oliveros-Wilches,[1*] Helena Facundo-Navia,[2] Raúl Pinilla-Morales,[3] Ana Bonilla,[4] Gustavo Aguirre.[5]

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[1] Gastrointestinal Surgeon. Advisor, National Cancer Institute. Bogotá, Colombia.
[2] Specialist in Gastrointestinal Surgery and Digestive Endoscopy. Specialist Physician, National Cancer Institute. Bogotá, Colombia.
[3] Physician, General Surgeon, Gastroenterologist, and Digestive Endoscopist, National Cancer Institute. Specialist Physician, Clínica Méderi and Clínica del Country. Assistant Professor of Surgery, Universidad Nacional de Colombia. Bogotá, Colombia.
[4] Physician and Surgeon, General Surgeon, Gastrointestinal Surgeon, National Cancer Institute. Bogotá, Colombia.
[5] Physician and General Surgeon, Gastrointestinal Surgeon, and Digestive Endoscopist. Specialist in Education for Clinicians, Fellow of the American College of Surgeons. Gastrointestinal Surgeon, National Cancer Institute. Bogotá, Colombia.

***Correspondence:** Ricardo Oliveros-Wilches.
roliveros4@yahoo.es

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Abstract

Models for both primary and secondary prevention of gastric cancer have been proposed. However, at least in Western countries, these strategies have not been widely implemented at a scale sufficient to achieve the anticipated public health impact. This article offers a reflective overview of the disease's natural history, primary and secondary prevention concepts, risk-stratification strategies, and screening approaches, with particular emphasis on endoscopy and eradication of *Helicobacter pylori.* The article also discusses considerations for regions with intermediate incidence rates. Several of these strategies have been successfully adopted in other parts of the world as global prevention measures aimed at reducing the current and future burden of gastric cancer.

Keywords

Gastric cancer, screening, early detection, disease prevention, *Helicobacter pylori*, risk factors.

INTRODUCTION

Gastric cancer (GC) is a public health problem because its morbidity and mortality rates remain very high and continue to rise worldwide. Prevention is the key to reducing its incidence and mortality[1]. By 2022, gastric cancer was the fifth most common cancer globally and the fifth leading cause of cancer-related death, with 968,350 new cases and 659,853 related deaths, respectively[2].

In Colombia, according to Globocan 2022, it is the fourth most common cancer in men and women combined, the third in men, the fifth in women, and the leading cause of cancer-related mortality in men. Gastric cancer risk is defined based on the age-standardized annual incidence rate. A region is considered high risk when the annual incidence exceeds 20 cases per 100,000 inhabitants, intermediate risk when incidence ranges between 10 and 20 cases per 100,000 inhabitants, and low risk when it is <10 cases per 100,000 inhabitants[2–5].

Colombia has an intermediate incidence of 17.4 per 100,000 inhabitants and a mortality rate of 13.4 per 100,000 inhabitants[4]. However, there are high-risk and low-risk areas in different regions of the country[6]. Although its incidence has gradually decreased, gastric cancer continues

to represent a major disease burden and an epidemiological challenge worldwide[3–5].

NATURAL COURSE OF GASTRIC CANCER

Gastric carcinogenesis is a progressive, long-term process resulting from the interaction of infectious, environmental, dietary, and genetic factors. Correa's model describes a well-established sequence that progresses from chronic non-atrophic gastritis to atrophic gastritis, intestinal metaplasia, dysplasia, and finally gastric adenocarcinoma[7,8]. Among environmental factors, high salt intake and consumption of salt-preserved foods such as smoked or dried meat and fish, combined with low fiber, fruit, and vegetable intake, significantly increase the risk of non-cardia GC[9]. However, the most important and widely recognized confirmed risk factor for GC is *Helicobacter pylori* infection. In recent years, a causal relationship has been firmly established between the chronic inflammatory process of the gastric mucosa triggered by this bacterium and subsequent gastric carcinogenesis[10]. Because this process can take decades to develop, we can improve early detection by identifying precancerous lesions and implementing endoscopic surveillance in high-risk patients, allowing us to diagnose malignancies at early, operable stages with better prognosis. In a large 10-year follow-up study, progression rates for patients with atrophic gastritis, intestinal metaplasia, low-grade dysplasia, or high-grade dysplasia were estimated at 0.8%, 1.8%, 4.0%, and 33.0%, respectively[9].

Despite recognition of the long, insidious course preceding cancer development, acceptance of the concept that gastric cancer can be prevented through detection and eradication of *H. pylori* has been slow[11]. *H. pylori* infection is the main trigger for GC regardless of histologic phenotype. Other conditions, including older age, high salt intake, diets low in fruits and vegetables, smoking, alcohol consumption, and family history, also contribute to gastric carcinogenesis[3]. Diffuse-type GC is genetically determined and less associated with environmental factors. Nevertheless, development of these tumors is also associated with *H. pylori* infection, which accounts for 90% of non-cardia GC cases and is recognized as the primary target for prevention strategies[3].

Countries with high gastric cancer incidence must recognize the immediate need to reduce mortality directly through endoscopic detection of premalignant lesions and, in addition, establish detection and treatment of *H. pylori* infection as a secondary strategy to reduce future risk[12]. Risk stratification can help identify high-risk groups and determine more intensive screening approaches and prioritization of diagnostic examinations[5].

PREVENTION

Primary cancer prevention through eradication or mitigation of modifiable risk factors represents the best opportunity to reduce the future burden of this disease. Reducing this burden will not only improve health and well-being, but will also decrease pressure on fiscal resources allocated to oncology services and the health sector as a whole[13].

Cancer prevention is not only the most effective strategy to reduce future incidence; it is also highly cost-effective. According to the WHO, every dollar invested in preventive interventions against noncommunicable diseases, including cancer, can generate an average return of seven dollars in health and economic benefits, while tobacco control programs have demonstrated savings of up to fifty dollars for every dollar invested[13,14].

Cancer prevention measures require a coordinated effort both within and beyond the health sector. These measures include targeted policies designed to reduce exposure to cancer-causing risk factors, such as tobacco and alcohol consumption, and ensuring access to vaccines that prevent cancer-related infections, such as hepatitis B and human papillomavirus (HPV)[13].

GC is associated with poor survival, with 5-year survival ranging between 20% and 40%. This unfavorable prognosis is mainly related to late-stage diagnosis. In contrast, early GC carries an excellent prognosis, with a 5-year survival rate above 90%[3,10].

Prevention strategies for GC must encompass all stages of primary and secondary prevention: maintaining a healthy lifestyle with an adequate diet, avoiding alcohol and tobacco consumption, and engaging in physical activity; screening for *H. pylori* infection and ensuring its eradication; early diagnosis of premalignant lesions, and risk stratification in patients older than 45 years through upper gastrointestinal endoscopic screening. Once high-risk individuals are identified, we must provide endoscopic surveillance and timely treatment according to disease evolution[15].

The absence of symptoms is a key feature of early-stage gastric cancer. High-definition white-light endoscopy performed by trained personnel can identify precancerous lesions as well as early-stage tumors. More advanced endoscopic techniques, such as chromoendoscopy or magnification endoscopy, are used in patients in whom a lesion has already been identified with white light, in order to achieve optical diagnosis of precancerous changes, estimate depth of invasion, and define appropriate margins for potential endoscopic resection[15].

Screening strategies must be adapted to the real conditions of each region. Although several validated tools exist for accurate GC diagnosis, identifying a universal

modality for population screening remains challenging[2]. An endoscopy-based screening program imposes a substantial socioeconomic burden due to the need for specific technical resources and highly trained personnel[3]. Such a strategy is cost-effective only in regions with high GC incidence and is focused exclusively on secondary prevention (early detection), without addressing primary prevention[3]. Despite awareness of this situation, there is still no universally accepted prevention strategy, and incidence and mortality have not been successfully reduced. Global gastric cancer prevention efforts must increase their level of effectiveness[15].

ENDOSCOPIC SCREENING AND RISK STRATIFICATION

The risk of developing gastric cancer is directly proportional to the severity of mucosal atrophy. The total gastric surface area ranges from 220 ± 27 cm^2 to 536 ± 25 cm^2. We identify the severity of gastric mucosal atrophy in biopsy samples according to the morphological visual analogue scale, based on the degree of reduction in the number of gastric glands. Through endoscopic visualization, we can estimate the surface area of atrophic stomach mucosa using the Kimura–Takemoto classification, from C1 to O3[15].

Histological risk stratification for GC involves assessing the extent and severity of atrophic gastritis and intestinal metaplasia[5]. In Asian countries, the extent of atrophy is assessed endoscopically by direct mucosal observation using the Kimura–Takemoto classification. In Western settings, histologic classification from systematic biopsies of the antrum, incisura, and corpus is preferred, and pathologists report findings using the Operative Link on Gastritis Assessment (OLGA) or Operative Link on Gastric Intestinal Metaplasia Assessment (OLGIM) systems. Both approaches require endoscopy performed in a clean stomach with sufficient observation time. One approach requires biopsies from specific sites, while the other requires expertise in endoscopic classification. We should use these examinations in combination[1,7,15].

The OLGA system is based on a combination of atrophy scores using samples taken from antral and oxyntic mucosa (Sydney protocol). Combined score values express the severity of atrophy, which parallels GC risk (OLGA stages 0, I, and II: low risk; OLGA III and IV: high GC risk). Prospective studies support the prognostic value of staging atrophic gastritis and its usefulness in clinical practice[7].

Currently, screening programs are implemented in high-risk areas. Meta-analyses indicate that endoscopic screening in high-risk Asian populations may reduce GC mortality by up to 40%[3]. In Japan, screening programs began in 1983 using indirect radiology in individuals older than 40 years, with annual frequency. When abnormalities such as stenosis, deformity, rigidity, barium retention, gastric area irregularities, fold changes, or suspected polypoid lesions are detected, endoscopy is required to verify or confirm findings. This approach significantly reduced mortality[3]. In the 2014 and 2018 Japanese guidelines, endoscopy was approved as an alternative screening method. The screening age threshold was increased to 50 years, with intervals of 2 to 3 years (**Table 1**)[3].

South Korea launched its national screening campaign in 2001 in asymptomatic adults aged 40 to 74 years, offering either barium studies or endoscopy depending on patient preference and comorbidities. There has been a marked preference for endoscopy, increasing from 31.2% in 2002 to 72.6% in 2011. In Korea, endoscopy is cost-effective,

Table 1. Screening Programs for Gastric Cancer in High-Risk Countries

	Japan	South Korea	China	China
Year of implementation	1983	1999	2008	2012
Coverage	National	National	High-risk groups, rural areas	High-risk groups, urban areas
Strategy	Indirect radiology, endoscopy	Indirect radiology, endoscopy	Endoscopy	Questionnaire, endoscopy
Starting age	>50 years	>40 years	40–69 years	40–69 years
Screening interval	Every 2–3 years	Every 2 years	Individuals with severe chronic atrophic gastritis or intestinal metaplasia: endoscopy every 3 years	Individuals with severe atrophic gastritis or intestinal metaplasia: endoscopy every 3 years Dysplasia: endoscopy within 3–6 months

Adapted from: Januszewicz W, et al. Cancers (Basel). 2023;15(3):664.[3]

and program participation increases by 4.3% annually[3]. In China, screening began in 2008 in high-risk areas, initially rural and subsequently urban (2012), targeting individuals aged 40 to 69 years. This strategy is repeated every two years and is cost-effective[3].

It is clear that these screening programs are indicated for high-risk groups. The question that follows is: what type of program should be implemented for populations at low or intermediate risk? Some authors recommend endoscopy for individuals in these groups who have additional risk factors—for example, those who have lived in high-incidence regions for more than three years, individuals older than 50 years, men, tobacco and alcohol users, those with high salt intake, *H. pylori* infection, or a family history of GC[3].

SCREENING AND TREATMENT STRATEGY FOR *HELICOBACTER PYLORI* INFECTION

According to the World Health Organization (WHO), *H. pylori* is a class I carcinogen and remains the principal global risk factor for GC. Several noninvasive tests are available to detect this pathogen, including fecal antigen testing, the urea breath test, and serologic tests (e.g., anti–*H. pylori* IgG). These tests enable straightforward access to a "test-and-treat" approach, making this strategy a preferred method for primary GC prevention in high-incidence regions[3,5]. *H. pylori* screening identifies individuals with infection, who then become targets for eradication therapy[3].

Successful eradication therapy has been shown to cure gastric mucosal inflammation and thereby prevent its progression to preneoplastic lesions. In fact, the latest Kyoto consensus recommendations for high-risk regions support active and early detection of *H. pylori* infection together with treatment, before chronic inflammation and its complications develop[3,16].

The "test-and-treat" strategy for *H. pylori* infection has already been established in several countries and has contributed to decreasing incidence in some regions. In Western countries, this strategy is currently used most frequently in patients with dyspeptic symptoms and has been shown to reduce the cost of dyspepsia evaluations in general health care. With this in mind, using a broader testing framework (that also includes asymptomatic individuals) at the population level may represent a feasible strategy within health care systems[3].

The International Agency for Research on Cancer (IARC) has recommended developing appropriate GC prevention policies, including *H. pylori* eradication therapy, in countries with high GC incidence[5,17]. It has been estimated that if all *H. pylori* infections were eradicated, approximately 89% of noncardia GC, 29% of cardia GC, and 74% of gastric lymphoma cases could be prevented. In 2014, the IARC working group emphasized the need to allocate additional medical resources to GC control and recommended that countries explore the feasibility of implementing population-based *H. pylori* screening and eradication programs[1,5,11,17].

In Japan, eradication therapy for *H. pylori*–related chronic gastritis (*H. pylori* gastritis) was approved under the national health insurance plan in February 2013. If the incidence of *H. pylori* gastritis can be reduced through eradication therapy, the incidence of atrophic gastritis—a premalignant condition for GC—will also decline[17,18]. Consequently, in recent years we have witnessed a paradigm shift from cancer detection toward cancer prevention.

Population-based strategies for primary prevention depend on local prevalence and cost-effectiveness relationships. In high-incidence regions, *H. pylori* eradication is the most effective strategy for preventing GC. A meta-analysis including 6,497 subjects from six randomized controlled trials demonstrated reduced GC incidence in *H. pylori*–infected individuals who received eradication therapy compared with placebo or untreated controls after 4 to 14.7 years of follow-up (odds ratio [OR], 0.66; 95% confidence interval [CI], 0.46–0.95)[17,18]. Similar results were reported in another meta-analysis by Chen and colleagues, which included 10 analyses from eight randomized clinical trials, totaling 7,955 participants. The risk of cancer development was lower in the eradication group compared with controls, with a pooled relative risk (RR) of 0.64 (95% CI: 0.48–0.85). However, in subgroup analyses of patients with intestinal metaplasia or dysplasia, this difference was not observed (RR = 0.88; 95% CI: 0.59–1.31)[18,19]. Therefore, the preventive benefit of eradication is limited to *H. pylori*–infected patients without advanced precancerous conditions at baseline[10,15,19,20].

In high-incidence settings, both endoscopic screening and *H. pylori* "test-and-treat" strategies represent cost-effective models[3]. China historically has had high GC incidence and high *H. pylori* prevalence. Nearly half of all new GC cases and GC-related deaths worldwide attributable to *H. pylori* infection occurred in China[12]. With the publication of five Chinese consensus reports on *H. pylori* management and active public dissemination of *H. pylori*–related knowledge, diagnostic and therapeutic practices among clinicians have become more standardized[12]. In 2013, the Fourth Chinese National Consensus Report on the treatment of *H. pylori* infection was published, including indications, diagnosis, and treatment recommendations[12].

In 2018, the Fifth Chinese National Consensus Report on the management of *H. pylori* infection was published. Raising awareness regarding the prevention and treatment of *H. pylori* infection among the Chinese population was also essential. Most surveyed individuals (87.0%) and

physicians (82.2%) supported a national *H. pylori* screening plan to prevent GC[12]. In Changle, Fujian Province, 1,630 asymptomatic *H. pylori*–infected individuals were randomly assigned to receive either eradication therapy or placebo and were followed for 26.5 years (1994–2020). Participants who received eradication therapy had a lower incidence of GC than their placebo counterparts (hazard ratio: 0.57; 95% CI: 0.33–0.98), and the benefit was more evident in participants without premalignant gastric lesions or dyspeptic symptoms at baseline[12,20,21].

The gastric cancer incidence rate in the Matsu Islands had been the highest among all Taiwanese populations, with a standardized incidence rate exceeding 50 per 100,000 person-years. To control the gastric cancer burden, a mass eradication program for *H. pylori* infection was developed in 2004. Residents aged 30 years or older were eligible to participate, based on a cost-effectiveness analysis predicting the superiority of early *H. pylori* eradication[12,22]. Participants underwent the C13 urea breath test, and those who tested positive received one or two courses of standardized triple- or quadruple-therapy regimens, validated through a randomized trial confirming eradication[12]. The prevalence of *H. pylori* infection rapidly declined from 63% to 10%, with a reinfection rate of less than 1% per person-year. By 2016, the program resulted in a 53% reduction in gastric cancer. By 2023, fewer than 6 per 100,000 residents were being diagnosed with gastric cancer, and by 2025, incidence and mortality rates are expected to decrease by 68% and 39%, respectively (**Table 2**)[1,8,12,17].

At the general population level, *H. pylori* screening and treatment strategies are most cost-effective in young adults in regions with high GC incidence and are preferably recommended before the development of atrophic gastritis and intestinal metaplasia[8,17].

RISK PREDICTION MODELS AND RISK FACTORS

An effective way to improve the critical situation of gastric cancer (GC) diagnosis and treatment in China was to implement screening in patients at high risk for GC. Current national GC screening guidelines in China recommend screening from age 40 onward for all members of high-risk populations (those who have lived in high-incidence regions for more than three years or who have *H. pylori* infection, a positive family history of GC, or other GC risk factors, among others). It is estimated that more than 300 million individuals were classified as high risk and required upper gastrointestinal endoscopy, which was likely unfeasible in clinical practice. Therefore, a practical risk prediction rule was needed to more accurately stratify GC risk within the Chinese high-risk population[12].

From 2015 to 2017, the China Alliance for the Prevention and Treatment of Early Gastrointestinal Cancer conducted a nationwide, multicenter cross-sectional study to identify high-risk individuals prior to endoscopy, recruiting 14,929 persons aged 40–80 years who attended hospitals for screening gastroscopy[22,23]. Seven variables—age, sex, PG I/II ratio, G-17 level, *H. pylori* infection, and consumption of pickled and fried foods—were evaluated to predict risk, with scores ranging from 0 to 25[21,22]. Risk categories were defined as low, intermediate, and high when the scores ranged from 0–11, 12–16, and 17–25, respectively. A total of 66.7% were classified as low risk, 27.6% as intermediate risk, and 5.7% as high risk. The prevalence of GC was 1.2%, 4.4%, and 12.3%, respectively (p <0.001). Notably, 70.8% of patients with advanced gastric cancer (AGC) and 70.3% of patients with early gastric cancer (EGC) were diagnosed when endoscopy was performed in the intermediate- and high-risk groups identified by the scoring system.

Table 2. *Helicobacter pylori* Screening Programs Worldwide

Country	Starting Age (years)	Program Start Year	Screening Interval	Strategy	Outcome
Japan	20	2013	Once	Diagnosis, *H. pylori* detection, and endoscopic screening	6% reduction in mortality in 2016
Republic of Korea	40-65	2014	Once	Breath test	Reduced GC incidence through *H. pylori* eradication
China	18	2022	Once	Family-based breath testing	Prevention of intrafamilial *H. pylori* transmission and consequent reduction in GC incidence
Taiwan	30	2004	Every 2 years	Breath test	53% reduction in GC incidence and 25% reduction in mortality

GC: gastric cancer. Adapted from: Conti CB, et al. Int J Environ Res Public Health. 2023;20(3):2149.[1]

Endoscopy requirements could be reduced by up to 66.7%, corresponding to the low-risk proportion. This study was successfully developed and validated nationwide[12,24].

SCREENING PROGRAM AND *HELICOBACTER PYLORI*

To implement a screening program of this scale, we need a structure in which collaboration among health policy makers, frontline general practitioners, and the population is guided and monitored through multiple quality indicators[11].

Screening everyone simultaneously is unrealistic for several reasons; in particular, it would impose a substantial burden on subsequent treatment and follow-up. Therefore, the program should be defined as a continuous process rather than a "one-time" project, with balanced costs relative to potential health care expenditures[11]. In regions where gastric cancer is a major public health concern, the proportion of individuals infected with *H. pylori* tends to be high[24,25]. Combining *H. pylori* eradication with endoscopic screening has shown promise in reducing GC incidence in high-incidence populations[24].

The Maastricht IV European *Helicobacter* Study Group (EHSG) report supported Asian expert opinions with the following statements:

- *H. pylori* infection is the most consistent risk factor for GC; therefore, eradication is the most promising strategy to reduce GC incidence (level of evidence: 1a).
- GC risk can be reduced most effectively by providing eradication therapy before preneoplastic conditions develop (level of evidence: 1a).
- *H. pylori* eradication to prevent GC should be implemented in high-risk populations (level of evidence: 1c)[24,25].

There is also evidence on combining *H. pylori* infection screening with fecal occult blood testing to identify patients eligible for eradication therapy. This approach has been shown to increase individual participation, and although a direct association between infection and increased endoscopic findings of precancerous lesions has not yet been demonstrated, adding risk factor identification will likely help us select patients for endoscopic assessment (**Table 3**)[25,26].

APPLICABILITY IN INTERMEDIATE-RISK REGIONS

Gastric cancer (GC) screening programs should not be implemented in countries with low or intermediate risk[3]. Colombia is considered an intermediate-incidence country overall, but certain municipalities in departments such as Boyacá and Nariño have very high incidence rates[4,26,27]. In most countries, incidence varies geographically and correlates with the prevalence of *H. pylori* infection[11]. A test-and-treat strategy for *H. pylori* reduces future GC incidence, but it does not help to reduce current GC mortality[2,3]. This tool provides the greatest benefit in individuals at higher risk and has been considered cost-effective based on a willingness-to-pay threshold for gains in life-years[5].

H. pylori infection is the main cause of GC, but it is not an adequate standalone predictor of cancer development. Approximately 90% of noncardia GC cases are related to *H. pylori* infection[28]; however, among 100 infected patients, only one will develop GC. Since only a very small proportion of infected individuals will develop cancer in the future, not all infected subjects will require treatment. Treating everyone after detection would lead to unnecessary therapies and related adverse effects, such as antibiotic resistance, obesity, and increased risk of other diseases[2].

When we encounter a patient from an intermediate-incidence GC region with a positive *H. pylori* test, should we always treat them? The answer is not always. The magnitude of the protective effect of *H. pylori* eradication also depends on baseline risk[5].

In the initial patient evaluation, we must define individual risk. If the patient is young (symptomatic or asymptomatic), we should treat them. Younger individuals benefit most from *H. pylori* eradication because it cures *H. pylori*–related gastritis, reduces GC risk, and decreases transmis-

Table 3. Intervention Strategies for Gastric Cancer Screening

High-Risk Areas	Low- or Intermediate-Risk Areas
Endoscopic screening; assess cost-effectiveness	Targeted endoscopic screening in at-risk individuals
Diagnosis and treatment of *H. pylori*	*H. pylori* "test-and-treat" strategy for at-risk individuals (e.g., family history of GC, precancerous gastric lesions, *H. pylori* infection)
Population: above 40 or 50 years	*H. pylori* stool antigen testing combined with a colorectal cancer screening program using fecal occult blood testing (FOBT)

CRC: colorectal cancer; GC: gastric cancer; FOBT: fecal occult blood test. Adapted from: Januszewicz W, et al. Cancers (Basel). 2023;15(3):664.[3]

sion to their children[5,28]. If the subject is a first-degree relative of a patient diagnosed with GC, we should treat them. It is estimated that 20% of GC patients have a family history of GC, which confers a 2- to 10-fold increased risk[9]. If the patient comes from a high-risk region, we should treat them. If the patient is otherwise low risk but has lived in a high-risk area for at least three years, we should treat them. At the individual level, *H. pylori* eradication reduces the risk of metachronous gastric cancer in patients with early GC after curative endoscopic resection[5,6,9,10].

H. pylori eradication does not increase the risk of gastroesophageal reflux disease, and there is no evidence that it increases the risk of metabolic syndrome, autoimmune disease, or inflammatory bowel disease (IBD)[5]. A retrospective cohort study showed an association between *H. pylori* eradication and a significantly higher risk of IBD and certain autoimmune diseases[29]. However, large-scale prospective studies are needed to clarify the impact of eradication on the incidence of these diseases in adults[5]. Gut microbiota diversity is lower in infected individuals compared with uninfected individuals, and gradual recovery is achieved within two to three months after treatment[5,28].

CONCLUSIONS

Prevention and screening strategies for GC must be adapted to the epidemiological conditions of each region. In high-incidence countries, implementing population-based endoscopic screening programs is a priority to enable earlier diagnoses and reduce mortality. Furthermore, when *H. pylori* prevalence is high, screening and eradication strategies emerge as key complementary measures to reduce future GC incidence. Conversely, in intermediate- or low-risk regions, it is more efficient to focus screening endoscopy on individuals with risk factors—for example, those older than 50 years, men, smokers, or individuals with a family history of gastric cancer—rather than implementing universal screening.

Ultimately, primary prevention (dietary improvements, *H. pylori* eradication, tobacco control, among others) and secondary prevention (early detection through endoscopy supported by risk-stratification models) are not mutually exclusive; instead, they must be integrated in a complementary manner.

GC has received very limited attention in terms of preventive action policies. An integrated and sustained approach, adapted to the characteristics and resources of each community, is essential to effectively reduce the global burden of GC. It is imperative that health authorities and health systems develop policies and programs that combine detection and prevention in a cost-effective manner, ensuring long-term implementation in accordance with local epidemiological needs.

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