

Prevalence of *Helicobacter pylori* infection and gastric intestinal metaplasia in Greek patients

Stergios N. Kouvaras^{a,b}, Ioannis G. Koumarios^a, Konstantinos Ekmektzoglou^b, George A. Kounis^c, Charikleia Spiliadi^d, Sotirios D. Georgopoulos^e, Theodoros Rokkas^b

Private Endoscopy Clinic; Henry Dunant Hospital, Athens; Statistical Analysis; Athens Medical Group; GI Dept Athens Medical, Paleo Faliron Hospital

Abstract

Background Knowledge of the local prevalence of *Helicobacter pylori* (*H. pylori*) infection and gastric intestinal metaplasia (GIM) is imperative in screening the population for gastric cancer. The aim of our study was to estimate the histopathological prevalence of *H. pylori* infection and GIM in Greek patients.

Methods This was a single-center retrospective study. The age, sex, endoscopic diagnosis, the presence of *H. pylori* gastritis and the presence of either complete or incomplete GIM, were extracted from the medical reports of our study group and stored in Microsoft Excel. The analysis was focused on the epidemiologic behavior of 2 histologic diagnoses: the presence of *H. pylori* gastritis and GIM.

Results *H. pylori* gastritis was recorded in 910 of the 2343 patients studied (38.8%, 95% confidence interval [CI] 36.8-40.8%). GIM was found in 601 of 2317 patients (25.9%, 95%CI 24.2-27.8%). The prevalence of incomplete GIM was 15.2%. These results are consistent with those observed in other European countries.

Conclusions This study is the first large Greek study to estimate the histopathological prevalence of *H. pylori* infection and GIM in a population from a primary care gastrointestinal unit. There was a strong association between *H. pylori* infection and the development of GIM. *H. pylori* were more prevalent in non-operated stomachs compared with operated. There was no difference in the prevalence of GIM between operated and non-operated stomachs.

Keywords *Helicobacter pylori*, gastric intestinal metaplasia, prevalence, Greek population

Ann Gastroenterol 2025; 38 (6): 1-6

^aPrivate Endoscopy Clinic (Stergios N. Kouvaras, Ioannis G. Koumarios); ^bHenry Dunant Hospital, 1st Gastroenterology Clinic (Stergios N. Kouvaras, Konstantinos Ekmektzoglou, Theodoros Rokkas); ^cStatistical Analysis (George Kounis); ^dGI Dept Athens Medical, Paleo Faliron Hospital (Sotirios D. Georgopoulos); ^eAthens Medical Group, Pathology Department (Charikleia Spiliadi)

Conflict of interest: None

Correspondence to: Stergios N. Kouvaras, PhD, First Gastroenterology Clinic, Henry Dunant Hospital, Athens and Private Endoscopy Clinic, 16 Phavierou str, Chalkida, 34100 Greece, e-mail: stergioskouvaras@gmail.com

Received 2 February 2025; accepted 28 August 2025; published online 15 October 2025

DOI: <https://doi.org/10.20524/aog.2025.1014>

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms

Introduction

Gastric cancer is the 5th most common cancer and the third leading cancer related to death worldwide [1]. In 1994, the International Agency for Research on Cancer announced *Helicobacter pylori* (*H. pylori*) as a group I human carcinogen for gastric adenocarcinoma in combination with genetic and environmental factors [2]. There is a wide variation in the prevalence of *H. pylori* infection among different populations, according to socioeconomics and living conditions [3]. The highest prevalences were reported in developing areas, such as Africa (70.1%, 95% confidence interval [CI] 62.6-77.7%) and South America (69.4%, 95% CI 63.9-74.9%). The lowest *H. pylori* prevalence was reported in developed countries, in the regions of Oceania (24.4%, 95% CI 18.5-30.4%), North America (37%, 95% CI 32.3-41.9%), and Western Europe (34.3%, 95% CI 31.3-37.7%) [4]. In this meta-analysis, the pooled *H. pylori* seroprevalence was 54.4%, while the histopathological prevalence was 51.4%.

A few old small Greek sero-epidemiological studies estimated that the seroprevalence of *H. pylori* varies from 70% in blood donors [5], 46% in healthy people, 85.5% in patients with peptic ulcer and non-ulcer dyspepsia [6], and 19.01% in navy recruits [7], to 49.2% in a small sample of adult outpatients [8]. A small retrospective Greek study, carried out between 2005 and 2008, estimated the histopathological prevalence of *H. pylori* at 34% [9].

According to Correa's Cascade, gastric intestinal metaplasia (GIM) is the precursor of gastric cancer [10]. GIM is asymptomatic and its prevalence is evaluated among patients undergoing upper gastrointestinal endoscopy (UGIE) with histological evaluation. The true prevalence in the general population is unknown. There is a wide variation in the prevalence of GIM. The overall prevalence in European studies varies from 13.8-19% [11,12]. In those infected it was 33.9% compared to 15.2% of those not infected [13]. In high-incidence regions, such as Japan and China, the prevalence of GIM was 37% and 29.3%, respectively, in *H. pylori* infected patients [14]. In American studies there was wide variation among different racial and ethnic subgroups. The prevalence was highest among Hispanic patients, 29.5% (23.7-36.1%), followed by African Americans, 25.5% (22.4-28.9%) and non-Hispanic white patients, 13.7% (11.9-15.7%). There was a strong association with age and *H. pylori* positivity [15].

There are no Greek studies that estimate the prevalence of GIM in Greek patients undergoing UGIE. Knowledge of the local prevalence of *H. pylori* infection and GIM is imperative when screening the population for gastric cancer. The aim of our study was to estimate the histopathological prevalence of *H. pylori* infection and GIM in Greek patients undergoing UGIE.

Patients and methods

The study data were extracted from the database of our private endoscopy clinic, which is located in Chalkida, Greece, and serves as a primary gastroenterology care unit for the population of Evia island and the west part of Viotia prefecture.

We performed a retrospective analysis of all medical reports of patients who underwent UGIE between January 2008 and December 2018 in our endoscopy clinic. The main reason for UGIE was dyspeptic symptoms and/or anemia. The analysis of these reports was performed manually. During this period, 4648 gastroscopies were carried out in 3788 patients (Fig. 1). In 1162 patients a CLO test was performed without histology. Another 283 patients were excluded from the study, of whom 6 had undergone total gastrectomy at the initial endoscopy. The remaining 277 were excluded because biopsies had not been taken for different reasons (receiving anticoagulants, upper gastrointestinal bleeding), or because the histology reports were not found. Biopsies were taken for histology in 2343 patients, of whom 534 underwent multiple endoscopies for the follow up of pre-malignant gastric lesions or re-evaluation of endoscopic findings (e.g., gastric ulcer). These 2343 patients constituted our study population.

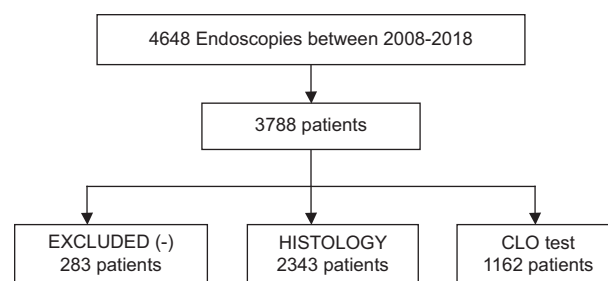


Figure 1 Flow chart of the study

The age, sex, endoscopic diagnosis, the presence of *H. pylori* gastritis and the presence of complete or incomplete GIM, or both, were extracted from the medical reports of our study group and stored in Microsoft Excel (Microsoft Redmond WA). If multiple endoscopies were performed on the same patient, the positive result for *H. pylori* gastritis or GIM was included once in the study, regardless of whether it was detected at the first or at the follow-up endoscopy. The age of a given patient was determined the first time he or she presented either negative or positive on each parameter. Positive GIM patients include either complete or incomplete, or both. The analysis was focused on the epidemiologic behavior of 2 histologic diagnoses: the presence of *H. pylori* gastritis and GIM. Patients with intestinal metaplasia in the metaplastic Barrett's epithelium were excluded from the study of GIM prevalence.

UGIEs were performed after local anesthesia with spray xylocaine and light sedation with 4 mg midazolam IV. A Gastroscope Olympus GIF H180 with narrow-band imaging was used to take target biopsies. Five biopsies were taken to each patient (2 from antrum, 1 from incisura and 2 from body). All biopsies were fixed in 10% neutral formalin and sections were stained with hematoxylin and eosin, and modified Giemsa. If intestinal metaplasia was detected, a supplementary stain with periodic acid-Schiff/Alcian blue and high iron diamine Alcian blue was performed. Subtypes of GIM (complete or incomplete) were determined according to the classification of Filipe MI and Jass JR [16]. The location of the biopsy (antrum vs. body) could not be determined in approximately 40% of the records, because GIM was an incidental diagnosis during UGIE. In these cases, the specimens were placed in a single bottle. In the case of GIM, a follow-up endoscopy and mapping biopsies were performed according to the Sydney protocol. The biopsy specimens were evaluated by the same group of pathologists, who are experts in gastrointestinal pathology. We also performed a subgroup analysis to evaluate the presence of *H. pylori* and GIM in the operated patients.

Statistical analysis and ethical considerations

For contingency analysis, univariate 2x2 tables were generated for each variable and histological diagnosis of the *H. pylori* and GIM. Chi-square tests were used to evaluate associations. Effect measures, expressed as relative risks (RR) with 95%CI, were calculated for each predictor. A P-value of <0.05 was considered statistically significant. The statistical

analysis was carried out using the statistical software JMP 18 (SAS Institute Inc., Cary, NC, 1989-2023).

This study adhered to institutional ethical guidelines for research involving human subjects. All data was anonymized prior to analysis to ensure patient confidentiality.

Results

H. pylori prevalence

The percentage of patients with *H. pylori* gastritis according to age are shown in Table 1 and Fig. 2. *H. pylori* gastritis was recorded in 910 of the 2343 patients studied (38.8%, 95%CI 36.8-40.8%). The proportion of patients with *H. pylori* infection increased up to the age of 60 years and then declined (Fig. 2). Chi-square independence testing revealed a statistically significant correlation between the presence of *H. pylori* and age groups until the age of 60 years ($P=0.0039$). Supplementary Table 1 shows how positive and negative *H. pylori* infections were distributed with sex. Chi-square testing found no statistically significant differences ($P=0.73$). According to the flowchart of the study (Fig. 1) in 1162 patients we performed only a CLO test without histology. Of these patients 452 (38.9%) had positive CLO tests. Supplementary Table 2 shows the positive and negative results for *H. pylori* infections according to diagnostic test. Chi-square testing

Table 1 *Helicobacter pylori* (HP) status per decade of age

Age	Patients (n)	HP (+) n (%)	HP (-) n (%)
<30	80	25 31.25%	55 68.8%
30-40	207	64 30.9%	143 69.1%
40-50	350	139 39.7%	211 60.3%
50-60	456	203 44.5%	253 55.5%
60-70	523	219 41.9%	304 58.1%
70-80	528	193 36.6%	335 63.4%
≥80	199	67 33.7%	132 66.3%
Total	2343	910 38.8%	1433 61.2%

χ^2 (6 N=2343) =9.65; $P=0.0039$; 38.8%, 95% confidence interval 36.8-40.8%

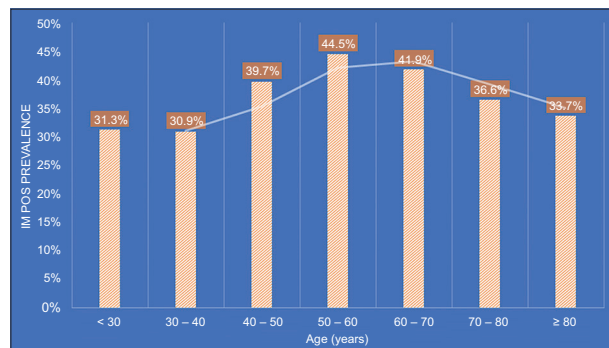


Figure 2 *Helicobacter pylori* prevalence with age

found no statistically significant differences ($P=0.97$) between the 2 tests.

GIM prevalence

Among the 2343 patients, 31 had Barrett's esophagus. In 5 of them there was concomitant intestinal metaplasia in the stomach. These patients were included in the total for GIM in the population. In the remaining 26 patients, intestinal metaplasia was limited only to metaplastic Barrett's epithelium. In accordance with the aim of the study, these patients were excluded from the total GIM in the population. However, Barrett's esophagus patients were not excluded from the study of *H. pylori* prevalence. Thus, 2343 patients were included in the calculation of *H. pylori* prevalence, whereas only 2317 were included in the calculation of GIM prevalence.

Among the 2317 patients, 248 had only complete GIM (n1), 204 incomplete (n2) and 149 both (n3). So 601 patients (n1+n2+n3) had GIM giving an overall prevalence of 25.9%, while 353 (n2+n3) had incomplete GIM giving 15.2% prevalence. Table 2 shows the distribution of patients with GIM according to age. Fig. 3 shows that GIM increased with age. Chi-square independence testing found a statistically significant correlation between the presence of intestinal metaplasia and age group ($P<0.001$). Supplementary Table 3 shows how positive and negative GIM findings were distributed according to sex. Chi-square testing found significant difference odds ($P<0.001$), signifying that GIM was more prevalent in males compared to females (RR 1.32, 95%CI 1.15-1.52).

Table 3 shows that the prevalence of GIM in *H. pylori* positive patients was 30.6% (95%CI 27.7-33.7%), while in *H. pylori* negative patients it was 22.9% (95%CI 20.9-25.3%). Chi-square testing revealed significant differences ($P<0.001$), signifying that GIM was more prevalent in *H. pylori* positive patients (RR 1.33, 95%CI 1.16-1.53).

In addition, we performed a subgroup analysis in order to evaluate the prevalence of *H. pylori* and GIM in operated patients. In our study population there were 67 operated patients. Forty of them had undergone partial gastrectomy at the initial endoscopy (Group A), 8 patients underwent partial gastrectomy after the first endoscopy (Group B), 13 patients had total gastrectomy after the first endoscopy (Group C), and 6 patients had undergone total gastrectomy before the

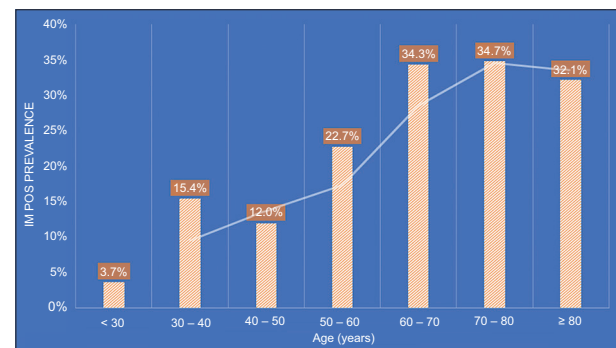


Figure 3 Gastric intestinal metaplasia (GIM) prevalence with age

Table 2 The distribution of patients with gastric intestinal metaplasia (GIM) according to age

Age	Patients (n)	GIM (+) n(%)		GIM (-) n(%)	
<30	80	3	3.75%	77	96.3%
30-40	205	32	15.6%	173	84.4%
40-50	345	42	12.2%	303	87.8%
50-60	456	104	22.8%	352	77.2%
60-70	516	177	34.3%	339	65.7%
70-80	520	180	34.6%	340	65.4%
≥80	195	63	32.3%	132	67.7%
Total	2317	601	25.9%	1716	74.1%

χ^2 (6, N=2317) =62.2, P<0.001; 25.9%, 95% confidence interval 24.2-27.8%

Table 3 The distribution of positive and negative gastric intestinal metaplasia (GIM) according to *Helicobacter pylori* (HP) status

HP status	Patients (n)	GIM positive n (%)		GIM negative n (%)	
HP (-)	1413	324	22.9%	1089	77.1%
HP (+)	904	277	30.6%	627	69.4%
Total	2317	601	25.9%	1716	74.1%

χ^2 (1, N=2317) =8.44, P<0.001; relative risk 1.33, 95% confidence interval 1.16-1.53

first endoscopy (Group D). In accordance with the aim of the study, the patients of Group D were excluded from the study. In the group of operated patients, we included the patients of Group A (n=40).

We compared the prevalence of *H. pylori* and GIM between the patients of Group A and the general population. Then we performed pairwise analysis to compare the preoperative and postoperative histological findings concerning *H. pylori* and GIM for the patients in Group B.

Supplementary Table 4 shows positive and negative *H. pylori* infections in non-operated and operated patients (n=40). *H. pylori* were significantly more prevalent in non-operated patients. Chi-square testing found significant difference odds (P=0.0016; RR 2.61, 95%CI 1.25-5.48).

Finally, we compared the presence of GIM in non-operated and operated patients. Supplementary Table 5 shows the distribution of GIM in these 2 groups. Chi-square testing found no statistically significant differences (P=0.46). GIM prevalence was 30.0% (16.3-45.1%) in operated cases and 25.9% (24.2-27.8%) in non-operated cases.

In the pairwise analysis of 8 patients in Group B (patients operated after the first endoscopy), there was a trend for lower prevalence of *H. pylori* postoperatively, while there was no trend for GIM. Given the small number of operated patients, the statistical power is low (Supplementary Tables 6 and 7). More studies with a larger number of operated patients are needed to confirm these results.

Discussion

The wide variation in the prevalence of *H. pylori* infection in different countries has an important implication for the prevalence of diseases associated with *H. pylori*, including peptic ulcer disease and gastric cancer. In a large meta-analysis carried out by Hooi *et al* [4], the serological prevalence of *H. pylori* infection in southern Europe ranged from 54.9% in Spain to 52.7% in Croatia, while in northern Europe it was much lower (Norway 30.7%, Sweden 26.2%, and UK 35.5%). In our population the estimated histopathological prevalence of *H. pylori* was 38.8%. Our results are slightly larger than those observed in a small Greek histo-epidemiological study, carried out between 2005-2008, which found the prevalence of *H. pylori* gastritis to be 34% [9]. The prevalence of *H. pylori* increases up to the age of 50-60 years and then it declines. This could be attributed to the phenomenon that chronic *H. pylori* infection leads to intestinal metaplasia, which is no longer hospitable to *H. pylori*. This phenomenon occurs between 50-60 years of age (Fig. 2). However, in a large epidemiological study in the USA this rise was restricted to 40 years of age [17]. In our study there were no statistically significant differences (P=0.73) between male and female patients as far as *H. pylori* prevalence was concerned (Supplementary Table 1). The 2 invasive diagnostic tests for *H. pylori* (histology and CLO) had equal diagnostic accuracy (P=0.97) (Supplementary Table 2). In the subgroup analysis we found that *H. pylori* infection was more prevalent in non-operated compared to operated patients (P=0.0016; RR 2.61, 95%CI 1.25-5.48; Supplementary Table 4). Partial gastrectomy due to peptic ulcer disease or gastric cancer increases the occurrence of biliary enterogastric reflux. Billroth II and Billroth I procedures had higher mean pH and n-nitrosamine concentrations, which create a non-hospitable microenvironment for *H. pylori* [18]. The Billroth II procedure is a major risk factor for these changes compared with Billroth I. Several factors contribute to changes in the microenvironment for *H. pylori* colonization after partial gastrectomy, and thus influence the survival of *H. pylori*: (1) the hypochlorhydric environment due to antrectomy [19]; (2) biliary enterogastric reflux increases the pH of gastric juice and inhibits *H. pylori* growth [20]; and (3) the substitution of intestinal type epithelium in the gastric remnant makes the mucosa more resistant to *H. pylori* infection [21,22]. These findings explain our results, which show that *H. pylori* prevalence was lower in operated compared to non-operated patients.

The American Society of Gastrointestinal Endoscopy, the European Society of Gastrointestinal Endoscopy, and other European academic societies have developed guidelines for the management of patients with GIM [23]. However, local estimation of the prevalence of GIM in different countries is essential in managing patients with intestinal metaplasia. The overall prevalence of GIM (complete, incomplete or both) in our population was 25.9% (95%CI 24.2-27.7%) and increased significantly with age. In *H. pylori*-positive patients it was 30.6% and in *H. pylori*-negative ones 22.9% (Table 3). Our results are consistent with those reported in other European studies. In a Dutch study, GIM was present in 25.3% of patients undergoing

endoscopy for dyspepsia [13]. A multicenter European study found the prevalence of GIM to be 31.4% in *H. pylori* positive patients [24]. In our population GIM was more prevalent in males than in females ($P < 0.001$). However, there was equal *H. pylori* prevalence among males and females ($P = 0.73$). From these findings we conclude that, after *H. pylori* infection, males are more prone to develop GIM than females (RR 1.32, 95%CI 1.15-1.52). In our population GIM was significantly ($P < 0.001$) more prevalent in *H. pylori*-positive patients (RR 1.33, 95%CI 1.16-1.53). These findings confirm the causal relationship between *H. pylori* infection and the development of GIM.

In subgroup analysis there was no statistically significant difference ($P = 0.46$) in the prevalence of GIM between non-operated and operated patients (Supplementary Table 5). Bile acids in the gastric lumen (induced by entero gastric reflux in the Billroth II and Billroth I procedures) act as signaling molecules involved in the development of GIM by regulating several signaling pathways, including regulation of the expression of caudal-related homeobox transcription-factor 2 [25], activation of bile acid receptors FXR and TGR5, secretion of exosomes of macrophages, epigenetics and miRNA involvement [26]. Thus, according to the results of our study, despite the elimination of *H. pylori*, the entero gastric reflux is a risk factor for the persistence of GIM in the operated stomach.

The importance of our study is that it is the first large Greek study to estimate the histopathological prevalence of *H. pylori* infection and GIM in a population examined in a primary care gastrointestinal unit. However, some limitations of the study should be stressed. First of all, it was a single-center study, rather than a multicenter study from different areas of Greece with different population compositions. Furthermore, the extent of GIM is lacking, and according to the OLGIM classification, the extent of GIM is an important premalignant factor—along with the presence of incomplete GIM [27], which was calculated in our study and found to be 15.2%. However, such limitations were mentioned in other similar retrospective studies, the main reason being that GIM was diagnosed incidentally in the first endoscopy, without separation of biopsy specimens in different bottles [28]. Another limitation of this study is that ongoing proton pump inhibitor (PPI) therapy was not considered, since the assessment of *H. pylori* infection is affected by such therapy (nearly 20% lower than controls). This limitation is also present in many retrospective studies.

Regarding this issue, we would like to emphasize 3 points: (1) according to a Greek study [29], 32.5% of patients had received PPIs prior to hospitalization. Assuming a similar proportion in our study population, a potential 20% reduction in detection rate in patients with ongoing PPI therapy would not have substantially influenced the overall prevalence, nor would it have altered the classification of our population, as a high-prevalence for *Helicobacter pylori* infection; (2) histological alterations induced by PPI therapy in *H. pylori*-infected patients have been described by Kuipers *et al* and Graham *et al* [30,31]. In such cases, the antrum often appears nearly normal, whereas the corpus may exhibit chronic active gastritis. Consequently, when infection persists predominantly in the corpus, sampling only from the antrum may further reduce the sensitivity of

histology in PPI users. In the present study, 5 gastric biopsies were obtained in accordance with the Sydney System protocol. As reported by Calvet [32], this approach, in combination with 2 stains (hematoxylin and eosin, and Giemsa that were used in our study), minimizes the risk of false-negative results in patients receiving PPIs; and (3) excluding these patients from the analysis would have introduced selection bias.

Finally, we estimated the prevalence of *H. pylori* infection in symptomatic patients undergoing upper GI endoscopy and this does not reflect the prevalence in the general population. Therefore, more multicenter, prospective studies are needed for accurate estimation of the prevalence of *H. pylori* infection and GIM in the Greek population.

In conclusion, the histopathological prevalence of *H. pylori* infection in our population was 38.8% and the prevalence of GIM (complete, incomplete or both) was 25.9%, while the prevalence of incomplete GIM was 15.2%. There was a strong association between *H. pylori* infection and the development of GIM. *H. pylori* is more prevalent in non-operated compared with operated stomachs. There is no difference in the prevalence of GIM between operated and non-operated stomachs.

Summary Box

What is already known:

- There is a great variation in the prevalence of *Helicobacter pylori* (*H. pylori*) infection in several old small Greek studies
- There are no Greek studies that estimated the prevalence of gastric intestinal metaplasia (GIM) in Greek patients
- Knowledge of the local prevalence of *H. pylori* and GIM is imperative in screening the population for gastric cancer

What the new findings are:

- The histopathological prevalence of *H. pylori* in our population was 38.8%
- The prevalence of GIM (complete, incomplete or both) was 25.9%, while the prevalence of incomplete GIM was 15.2%

References

1. Morgan E, Arnold M, Camargo MC, et al. The current and future incidence and mortality of gastric cancer in 185 countries, 2020-40: a population-based modeling study. *EClinicalMedicine* 2022;47:101404.
2. IARC Working Group on the evaluation of carcinogenic risks to humans, schistosomes, liver flukes and *Helicobacter pylori*. Vol 61 of IARC monographs on the evaluation of carcinogenic risk to humans. International Agency for Research on Cancer, Lyon, 1994.
3. Everhart JE, Kruszon-Moran D, Perez-Perez GI, ralka TS,

- McQuillan G. Seroprevalence and ethnic differences in *Helicobacter pylori* infection among adults in the United States. *J Infect Dis* 2000;**181**:1359-1363.
4. Hooi JKY, Lai WY, Ng WK, et al. Global prevalence of *Helicobacter pylori* infection: systematic review and meta-analysis. *Gastroenterology* 2017;**153**:420-429.
 5. Pateraki E, Mentis A, Spiliadis C, et al. Sero epidemiology of HP infection in Greece. *FEMS Microbiology Immunology* 1990;**2**:129-136.
 6. Archimandritis A, Bitsikas J, Tjivras M, et al. *Helicobacter pylori* infection in Greece in healthy people and in patients with peptic ulcer and with dyspepsia without ulcer. *J Clin Gastroenterol* 1993;**16**:257-258.
 7. Kyriazanos I, Ilias I, Gizaris V, et al. Sero epidemiology of *Helicobacter pylori* infection in Hellenic Navy recruits. *Eur J Epidemiol* 2001;**17**:501-504.
 8. Apostolopoulos P, Vafiadis-Zouboulis I, Tjivras M, et al. *Helicobacter pylori* (*H pylori*) infection in Greece: the changing prevalence during a ten-year period and its antigenic profile. *BMC Gastroenterol* 2002;**2**:11.
 9. Katsanos KH, Tatsioni A, Tsakiris V, Christodoulou D, Tsianos EV. *Helicobacter pylori* is a major public health priority in western Balkans: an endoscopy referral center experience. *Eur J Intern Med* 2010;**21**:306-309.
 10. Correa P, Piazuelo MB, Wilson KT. Pathology of gastric intestinal metaplasia: clinical implications. *Am J Gastroenterol* 2010;**105**:493-498.
 11. Eriksson NK, Kärkkäinen PA, Färkkilä MA, Arkkila PE. Prevalence and distribution of gastric intestinal metaplasia and its subtypes. *Dig Liver Dis* 2008;**40**:355-360.
 12. Olmez S, Aslan M, Erten R, Sayar S, Bayram I. The prevalence of gastric intestinal metaplasia and distribution of *Helicobacter* infection, atrophy, dysplasia and cancer in its subtypes. *Gastroenterol Res Pract* 2015;**2015**:434039.
 13. Eidt S, Stolte M. Prevalence of intestinal metaplasia in *Helicobacter pylori* gastritis. *Scand J Gastroenterol* 1994;**29**:607-610.
 14. Uemura N, Okamoto S, et al. *Helicobacter pylori* infection and the development of gastric cancer. *N Engl J Med* 2001;**345**:784-789.
 15. Nguyen TH, Tan MC, Liu Y, Rugge M, Thrift AP, El-Serag HB. Prevalence of gastric intestinal metaplasia in a multiethnic US veterans' population. *Clin Gastroenterol Hepatol* 2021;**19**:269-276.
 16. Filipe MI, Jass JR. Current problems in tumor pathology. Gastric carcinoma: Intestinal metaplasia subtypes and cancer risk. 1st edition, Churchill Livingstone, New York, 1986:87-115.
 17. Sonnenberg A, Lash RH, Genta RM. A national study of *Helicobacter pylori* infection in gastric biopsy specimens. *Gastroenterology* 2010;**139**:1894-1901.
 18. Guadagni S, Walters CL, Smith PL, Verzaro R, Valenti M, Reed PI. N-nitroso compounds in the gastric juice of normal controls, patients with partial gastrectomies, and gastric cancer patients. *J Surg Oncol* 1996;**63**:226-233.
 19. Danesh J, Appleby P, Peto R. How often does surgery for peptic ulceration eradicate *Helicobacter pylori*? Systematic review of 36 studies. *BMJ* 1998;**316**:746-747.
 20. O'Connor HJ, Dixon MF, Wyatt JJ, et al. Effect of duodenal ulcer surgery and enterogastric reflux on *Campylobacter pyloridis*. *Lancet* 1986;**2**:1178-1181.
 21. Onoda N, Maeda K, Sawada T, Wakasa K, Arakawa T, Chung KH. Prevalence of *Helicobacter pylori* infection in gastric remnant after distal gastrectomy for primary gastric cancer. *Gastric Cancer* 2001;**4**:87-92.
 22. Yoon K, Kim N, Kim J, et al. Dynamic changes in *Helicobacter pylori* status following gastric cancer surgery. *Gut Liver* 2017;**11**:209-215.
 23. Banks M, Graham D, Jansen M, et al. British Society of Gastroenterology guidelines on the diagnosis and management of patients at risk of gastric adenocarcinoma. *Gut* 2019;**68**:1545-1575.
 24. Tulassay Z, Stolte M, Engstrand L, et al; HELIX Study Investigators. Twelve-month endoscopic and histological analysis following proton-pump inhibitor-based triple therapy in *Helicobacter pylori*-positive patients with gastric ulcers. *Scand J Gastroenterol* 2010;**45**:1048-1058.
 25. Wang M, Lou E, Xue Z. The role of bile acid in intestinal metaplasia. *Front Physiol* 2023;**14**:1115250.
 26. He Q, Liu L, Wei J, et al. Roles and action mechanisms of bile acid-induced gastric intestinal metaplasia: a review. *Cell Death Discov* 2022;**8**:158.
 27. Bogdanova I, Polaka I, Aleksandraviča I, et al. Role of pre-existing incomplete intestinal metaplasia in gastric adenocarcinoma: a retrospective case series analysis. *World J Clin Cases* 2023;**11**:2708-2715.
 28. Huang R, Ende A, Singla A, et al. Prevalence, risk factors and surveillance patterns for GIM among patients undergoing upper gastrointestinal endoscopy with biopsy. *Gastrointest Endosc* 2020;**91**:70-77.
 29. Lazaridis LD, Rizos E, Bounou L, et al. An educational intervention to optimize use of proton pump inhibitors in a Greek university hospital. *Ann Gastroenterol* 2021;**34**:781-787.
 30. Kuipers E, Uterlined A, Pena A, et al. Increase of *Helicobacter pylori* associated corpus gastritis during acid suppressive treatment: Implications for long term safety. *Am J Gastroenterol* 1995;**90**:1401-1406.
 31. Graham DY, Genta RM. Long-term proton pump inhibitor use and gastrointestinal cancer. *Curr Gastroenterol Rep* 2008;**10**:543-547.
 32. Calvet X. Diagnosis of *Helicobacter pylori* infection in the proton pump inhibitor era. *Gastroenterol Clin North Am* 2015;**44**:507-518.

Supplementary material

Supplementary Table 1 *Helicobacter pylori* (HP) status according to sex

Sex	Patients (n)	HP positive n (%)		HP negative n (%)	
Male	1127	434	38.5%	693	61.5%
Female	1216	476	39.1%	740	60.9%
Total	2343	910	38.8%	1433	61.2%

P=0.73

Supplementary Table 2 *Helicobacter pylori* (HP) status according to diagnostic test

Diagnostic test	Patients (n)	HP positive n (%)		HP negative n (%)	
CLO-test	1162	452	38.9%	710	61.1%
Histology	2343	910	38.8%	1433	61.2%

P=0.97

Supplementary Table 3 The distribution of patients positive and negative for gastric intestinal metaplasia (GIM) according to sex

Sex	Patients (n)	GIM positive n (%)		GIM negative n (%)	
Male	1107	331	29.9%	776	70.1%
Female	1210	270	22.3%	940	77.7%
Total	2317	601	25.9%	1716	74.1%

χ^2 (1, N=2317) =8.66, P<0.001; relative risk 1.32, 95% confidence interval 1.15-1.52

Supplementary Table 4 The distribution of positive and negative findings for *Helicobacter pylori* (HP) in operated and non-operated patients

Operation	Patients (n)	HP positive n (%)		HP negative n (%)	
Non-operated	2303	904	39.3%	1399	60.7%
Operated	40	6	15.0%	34	85.0%
Total	2343	910		1433	

χ^2 (1, N=2343)=5.56, P=0.0016; relative risk 2.61, 95% confidence interval 1.25-5.48

Supplementary Table 5 The distribution of positive and negative gastric intestinal metaplasia (GIM) in operated and non-operated patients

Operation	Patients (n)	GIM positive n (%)		GIM negative n (%)	
Non-operated	2277	589	25.9%	1688	74.1%
Operated	40	12	30.0%	28	70.0%
Total	2317	601		1716	

P=0.46

Supplementary Table 6 *Helicobacter pylori* (HP) status in pre- and post-operated patients

Operation time	Patients (n)	HP positive n (%)		HP negative n (%)	
Pre-op	8	3	37.5%	5	62.5%
Post-op	8	1	12.5%	7	87.5%
Total	16	4		12	

P=0.24

Supplementary Table 7 The distribution of positive and negative gastric intestinal metaplasia (GIM) in pre- and post-operated patients

Operation time	Patients (n)	GIM positive n (%)		GIM negative n (%)	
Pre-op	8	7	87.5%	1	12.5%
Post-op	8	7	87.5%	1	12.5%
Total	16	14		2	

P>0.99