ORIGINAL ARTICLE

Worldwide prevalence of *Helicobacter pylori* in patients with Mucosa-Associated Lymphoid Tissue (MALT) lymphomas: A systematic review and meta-analysis

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ABSTRACT

Background and aim: Chronic inflammation resulting from infections or autoimmune disorders plays a pivotal role in the pathogenesis of mucosa-associated lymphoid tissue (MALT) lymphoma, an extranodal non-Hodgkin lymphoma. *Helicobacter pylori* (*H. pylori*) is a major contributor to gastric MALT lymphomas. Eradicating *H. pylori* can induce regression of these lymphomas. However, the global prevalence of *H. pylori* among MALT lymphoma patients remains insufficiently studied. The present study aimed to determine this global prevalence, crucial for developing effective treatments and improving outcomes.

Methods: An electronic search of five databases using appropriate keywords was performed, including PubMed, Scopus, Cochrane, ScienceDirect, and Google Scholar. The quality of the involved manuscripts was

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evaluated utilizing the Joanna Briggs Institute critical appraisal tool. The random-effects model was used to report the results as percentages with a 95%
confidence interval (CI). The protocol was registered with PROSPERO (CRD42024541706).
Results: Of the 2524 titles identified, 102 studies met the inclusion criteria. The prevalence of *H. pylori* in patients with MALT lymphomas was 73.0% (95% CI, 69.0–77.1%, n = 8725). The outcomes of sensitivity analysis were consistent, and 70.6% of studies were deemed high quality. Significant publication bias was found by Egger's test.
Conclusions: *H. pylori* was found in a significantly high proportion of patients with MALT lymphomas. This study recommends that *H. pylori* testing and eradication should be considered for prediction, targeted treatment protocols, and assessment of the clinical status of MALT lymphomas. (www.actabiomedica.it)

Key words: *Campylobacter pylori*, *Helicobacter pylori*, lymphoma of mucosa associated lymphoid tissue, MALT lymphomas, marginal zone b-cell lymphoma

Introduction

Mucosa-associated lymphoid tissue (MALT) is a group of lymphoid neoplasms with B-cell origin (1, 2). The World Health Organization classification of hematolymphoid tumors has classified MALT lymphomas as a subtype of marginal zone lymphomas (MZL), a subset of indolent lymphoid tumors accounting for 7% of mature non-Hodgkin lymphomas (NHL) (1, 2). MALT lymphomas comprise 60-70% of MZL, followed by nodal MZL (20-30%) and pediatric/primary cutaneous MZL (5-10%) (1, 2). MALT lymphoma is commonly an illness of aged people, with a median age above 60 years, and it predominantly affects males (2, 3). Depending on its origin and clinical presentation, MALT lymphoma is typically described as a low-grade type of B-neoplastic cell lymphoma (2, 3). Low-grade MALT lymphoma is characterized by an indolent clinical course and distinctive histopathological features, including small to medium-sized B-cells with minimal atypia, forming lymphoepithelial lesions within marginal zone patterns (1-3). However, the risk lies in its potential to transform into aggressive high-grade B-cell lymphoma types, such as large B-cell lymphoma (LBCL) (2, 3). High-grade is histologically identified by the presence of confluent clusters or sheets of large blast-like cells, comprising ≥20% of the cellular composition, often with increased mitotic activity and nuclear pleomorphism. This dichotomy reflects biological aggressiveness, where high-grade disease typically shows genomic instability (e.g., MYC/BCL2 alterations) and poorer treatment response (2, 3). Furthermore, MALT lymphoma is characterized by infiltration of heterogenous B-neoplastic cells in the different marginal zones of extranodal localizations of the body (2, 3). These localizations further classify MALT lymphoma into several categories: the stomach (35%), ocular adnexa (12%), skin (10%), lungs (9.0%), salivary glands (8.0%), small intestine (4.0%), and with less commonly in the kidney, bladder, thyroid, breast, thymus, and lacrimal glands (2, 4). The evolution of MALT lymphoma begins as lymphoepithelial lesions in the tissues of the extranodal anatomical body sites because of immune cells responding to chronic inflammation (2, 5). The main drivers of this inflammation are either bacterial and/or viral infections, such as *Helicobacter pylori* (*H. pylori)* causing chronic gastritis, or autoimmune illnesses like Hashimoto's thyroiditis, Sjögren's syndrome, and systematic lupus erythematosus (SLE) (1, 2, 4-6). Moreover, genetic alternations such as abnormal frequent mutations in *TNIFAIP3* and *CREBPP* and genetic translocations like t(14; 18) (q32;q21), t(1;14)(p22, q23), t(11;18)(q21;q21), and t(3;14)(p14.1;q32) are frequently linked and contribute to the pathogenesis of the MALT lymphomas (1, 2, 4). The most frequent genomic translocation in MALT lymphoma is t(11;18) (q21;q21) associated with advanced stages, therapy resistance and anatomical site (1, 2, 4). A gram-negative microaerophilic *H. pylori*,

formerly known as *Campylobacter pylori*, is a member of the *Proteobacteria* phylum, class *Camoylobacteria*, order *Campylobaterales*, and *Helicobacteracea* family (7, 8). It is distinguished by its spiral morphology and virulence factors, such as cytotoxin-associated gene A (CagA) factor, which enable it to invade and colonize deep in gastrointestinal tissues, causing severe lesions (7-9). The presence of CagA-positive *H. pylori* is substantially linked to t(11;18)(q21;q21) MALT lymphoma (10). *H. pylori* strains that are CagA-positive are significantly more effective at inducing inflammatory responses in the host, generating extremely genotoxic oxygen-reactive species (8, 10). It is anticipated that four billion individuals are infected worldwide (11). *H. pylori* infection is frequently documented in low socioeconomic and low-profile sterilization communities (9). Transmission and spreading of *H. pylori* among the individuals in these communities are via oral-oral, fecal-oral, or gastro-oral means (9). Successfully reducing the number of infected individuals depends on accurate diagnosis and effective treatments for eradicating *H. pylori* (9, 11). Currently, various diagnostic methods, such as serological, histological, endoscopy, rapid urease test, and molecular tests, are used (9, 12, 13). The elimination of *H. pylori* involves treatment using a combination of triple antibiotics, proton pump inhibitors, and H2 receptor inhibitors(9, 12, 14-16). However, 10% to 20% of gastric infections transform into adenocarcinoma or MALT lymphoma(2, 17). Various studies have shown the link between *H. pylori* and MALT, the contributing factors and the local prevalence of this microorganism in MALT patients. Nevertheless, no previous study has investigated this prevalence worldwide. Therefore, the aim of this systematic and meta-analysis study is to highlight the global prevalence of *H. pylori* in patients with MALT lymphomas.

Methods

Compliance with protocol registration

This study adhered to the guidelines of the Preferred Reporting Items for Systematic-Reviews and Meta-Analyses (PRISMA) and Meta-analysis of Observational-Studies in Epidemiology (MOOSE) (18, 19), PROSPERO registration ID: CRD42024541706.

Eligibility criteria and literature search

The prevalence of *H. pylori* in patients with marginal zone B-cell lymphoma (MALT), including both gastric and non-gastric subtypes, was assessed across published studies. Studies were inclusive of all anatomical locations to quantify the global prevalence of *H. pylori* in MALT lymphoma up to October 31, 2023. The study involved articles that were performed from all ethnicities, socioeconomic and educational backgrounds. Comprehensive searches were conducted in PubMed, Scopus, Cochrane, ScienceDirect, and Google Scholar, resulting in a total 2524 papers. Search terms included MALT Lymphomas, *H. pylori*, and related terms (Table S1).

Article selection and data extraction

Initial screening involved independent evaluation of titles and abstracts, followed by the full text assessment by two authors (A.A. and M.J.A.-K.). Included studies encompassed cross-sectional, case-control, and cohort designs reporting the prevalence of *H. pylori* in patients with gastric, non-gastric, or mixed-site MALT lymphoma. Excluded studies were non-human studies, review articles, case studies, randomized trials, *in vitro* studies, and non-research articles. Issues regarding inclusion were resolved through deliberation involving A.A, M.J.A.-K., and O.M.A, ultimately resulting in a consensus. Data extraction from selected studies was independently performed by A.A. M.J.A.-K., and O.M.A. using a predefined Excel spreadsheet. Extracted data included study details, patient demographics, number of MALT lymphoma patients, number of positive *H. pylori* cases in MALT lymphoma, follow-up period, number of positive *H. pylori* cases in MALT lymphoma after eradication, treatment types and duration, and methodological details. *H. pylori* infection was defined as a positive result from at least one of the following diagnostic methods: histological assays, serological assays, molecular assays, and/or microbiological assays.

Data quality assessment and analyses

The Joanna Briggs Institute's (JBI) tools were utilized to evaluate the quality of the included studies (20, 21). If the score was less than 49%, between 50 and

69%, or greater than 70%, the studies were classified as poor-quality (high-risk of bias), moderate-quality (moderate-risk of bias), or high-quality (low-risk of bias), respectively (20, 21). Any discrepancies in the quality assessment were discussed and resolved by A.A., M.J.A.-K., O.M.A., M.F.J., F.A.H., A.M.Z., O.A., K.J., S.A.O., N.H.I., A.A.M.Y., M.A.I., A.Al., and N.P.R. A funnel plot was employed to compare the prevalence estimate against standard error to assess the publication bias. The asymmetry of the funnel plot was assessed using Egger's test. Heterogeneity among studies was evaluated utilizing a tau-squared (I^2) test, with $p < 0.05$ indicating statistically significant (22), categorizing studies as greater homogeneity (I^2 value close to zero), low heterogeneity (I^2 <50%), moderate heterogeneity (I^2 between 50% and 75%), or high heterogeneity (I^2 >75%). Additionally, Galbraith plots were employed for exploring heterogeneity sources. The sensitivity assessments included: (1) excluding studies with a small size (n < 100); (2) excluding studies with low-quality (high-risk of bias); (3) including only cross-sectional studies; (4) containing only case-control studies; (5) including only cohort studies; and (6) excluding outlier studies. Data were analyzed using the meta package (version 7.0-0) and metafor package (version 4.6-0) of R Studio software (version 2024.04.1+748) (23) (Table S2).

Subgroup analyses

Firstly, *H. pylori* prevalence trends over time intervals (1995–2002, 2003–2009, 2010–2016, and 2017–2023). Secondly, geographical locations (Southern Asia, Western Asia, Eastern Asia, Southeastern Asia, Southern Europe, Western Europe, Northern Europe, Eastern Europe, Middle Africa, Northern Africa, and Northern America) based on the standard country codes for statistical use (24). Thirdly, detection methods: (1) histological assays (Hematoxylin and Eosin (H&E) staining, modified Giemsa stain, Warthin-Starry silver stain, Genta stain, indirect immunoperoxidase, and immunohistochemical (IHC) stains); (2) serological assays (enzyme-linked immunosorbent assay (ELISA), Western blotting, immunoblot assay, and dot blot assay); (3) molecular assays (polymerase chain reaction (PCR), nested PCR, real-time PCR (qPCR), loop-mediated isothermal amplification (LAMP), DNA microarrays, and next-generation sequencing (NGS)); and/or (4) microbiological assays (tissue culture, rapid urease test, and urea breath test). Fourthly, MALT lymphoma types (gastric, non-gastric, and gastric & non-gastric). Fifthly, MALT grades (low-grade and high-grade). Sixthly, the sociodemographic index (SDI), including information on the world's educational systems, economies, and fertility rates, as follows: (1) high SDI (0.805129 to 1); (2) high–middle SDI (0.689504 to 0.805129); (3) middle SDI (0.607679 to 0.689504); (4) low–middle SDI (0.454743 to 0.607679); (5) and low SDI (0 to 0.454743) (25) (Tables S3 and S4).

Result

Selection and inclusion of studies

From the database search (PubMed, Scopus, Cochrane, ScienceDirect, and Google Scholar), 2,524 studies qualified for initial screening. Of these, 1,629 studies were excluded due to being duplicates (n = 525), review articles (n = 583), case reports (n = 359), and other types such as books, letters, conferences, editorials, and comments (n = 162). Consequently, 895 studies were further assessed for eligibility through detailed screening of the titles, abstracts, and full texts. After excluding 793 studies that did not meet the objectives of this study, 102 studies met the eligible criteria for inclusion in this systematic review and meta-analysis, as illustrated in the PRISMA flow diagram (Figure 1).

Study characteristics

Literature search yielded 102 articles published between 1995 and 2023, examining the prevalence of *H. pylori* in MALT lymphoma patients. Table 1 illustrates detailed characteristics and references of the included studies. This meta-analysis reported data from 8,725 patients with MALT lymphoma (51.7% female). The ages of these patients ranged from 10.5 ± 3.4 to 67.58 ± 16.26 years (mean ± SD; range, 15–92). The studies originated from 10 different regions, grouped according to the geographic regions

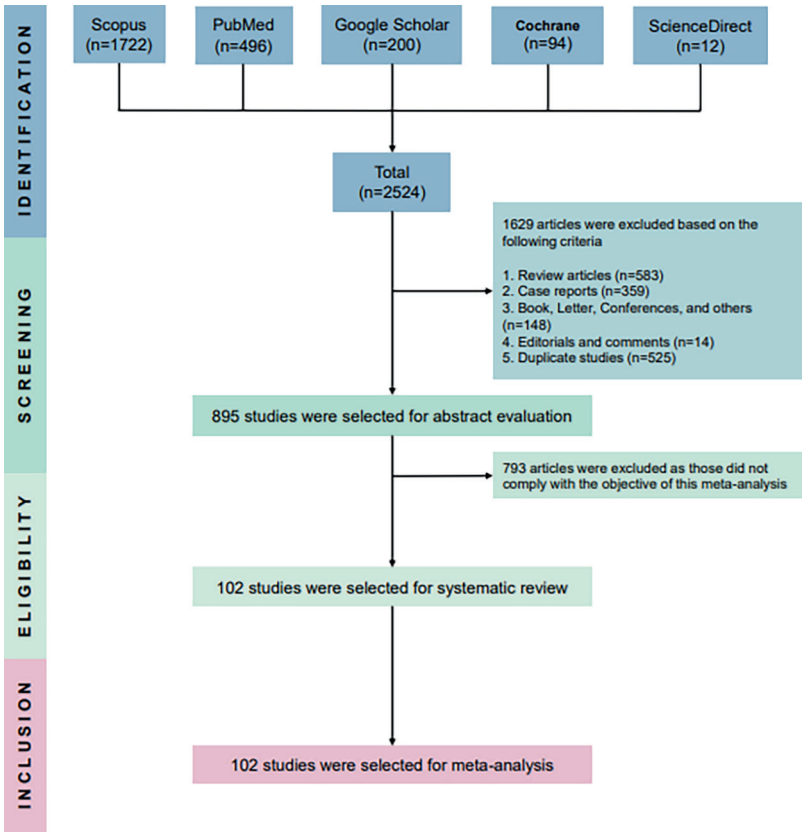

Figure 1. PRISMA flowchart of study selection.

defined under the Standard Country or Area Codes for Statistical Use (known as M49) by the United Nations Statistics Division (24): Asia / Eastern Asia (n = 5407) (26-79), Europe / Southern Europe (n = 878) (80-94), Europe / Western Europe (n = 1820) (95-114), Europe / Northern Europe (n = 7) (115), Americas / Northern America (n = 379) (116-121), Asia / Western Asia (n = 22) (122), Sub–Saharan Africa / Middle Africa (n = 10) (123), Europe / Eastern Europe (n = 30) (124, 125), Asia / Southern Asia (n = 122) (126), Africa / Northern Africa (n = 50) (127). The presence of *H. pylori* in patients with MALT lymphoma was assessed using different methods, including histological assays (26, 27, 38, 43, 52, 54, 66, 74, 75, 78, 85, 86, 88, 89, 98, 99, 106, 110, 112, 113, 115, 118, 119, 121-123, 126, 127), microbiological assays (30, 34, 37, 65, 81), serological assays (31, 73, 100, 117), molecular assays (29, 91, 97, 116), histological + microbiological assays (32, 33, 40-42, 45-49, 53, 60-62, 70, 77, 79, 80, 83, 84, 87, 92, 93, 102, 105), histological + molecular assays (95, 96, 107), histological & serological assays (57, 104, 108, 109), microbiological + serological assays (44, 124, 125), molecular + serological assays (64), and histological + microbiological + serological assays (35, 36, 39, 50, 51, 55, 56, 58, 59, 63, 68, 69, 71, 72, 76, 101). The included articles were carried out between 1995 and 2023 and were divided into four-time groups, each with an interval of approximately six years: 1995 to 2002 (27, 43, 44, 54, 61, 74, 75, 79, 81, 89, 97, 100, 108, 112, 115, 119, 121, 124, 125), 2003 to 2009 (26, 30, 31, 38, 39, 45-47, 52, 55-58, 62, 70-72, 77, 78, 83, 84, 86, 88, 90, 92, 93, 99, 101, 102, 105, 110, 111, 113, 114, 116, 122), 2010 to 2016 (28, 29, 32, 35, 36, 40, 41, 48-51, 59, 60, 63, 66, 68, 69, 76, 80, 85, 87, 94, 96, 98, 109, 117), and 2017 to 2023 (33, 34, 37, 42, 53, 64, 65, 67, 73, 82, 91, 95, 103, 104, 106, 107, 118, 120, 123, 126, 127). The socio-demographic index (SDI) was used to classify studies into five categories: high SDI (26-28, 30-33, 35, 36, 39-47, 50-74, 76, 79, 95-121), high–middle SDI (80-94, 122, 124, 125), middle SDI

Table 1. Main characteristics of the studies involved.

Study ID Country		Type of Study	Age (mean ± SD / range)	Number of MALT patients (% female)	Prevalence of HP in MALT (HP positivity) # (%)	HP positivity after Eradication # (%)	Sample type	Detection method	MALT types		Socio-demographic Index (SDI)
									Type	# (%)	
1	Akamatsu 2006 (26) Japan	Cohort	62.7 (27-84)	57 (49.1)	41 (71.9)	0/38 (0)	FFPE	Histology assays	Gastric	57 (100)	High SDI
2	Albano 2016 (80) Italy	Cross-sectional	59 (22-83)	69 (38)	29 (42)	NR	Tumor biopsy	Histology and Microbiological assays	Gastric	69 (100)	High–middle SDI
3	Amiot 2014 (96) France	Cohort	59.4± 12.5 (24.9-83.9)	107 (51)	58 (55)	NR	Tumor biopsy	Histology and Molecular assays	Gastric	107 (100)	High SDI
4	Amiot 2017 (95) France	Cohort	57.3 ± 14.4 (42.9-71.7)	175 (48.6)	131 (74.9)	51/131 (38.9)	Tumor biopsy	Histology and Molecular assays	Gastric	175 (100)	High SDI
5	An 2001 (27) Korea	Cross-sectional	52 (31-80)	26 (57.1)	20 (77)	NR	Tumor biopsy	Histology	Gastric Low-Grade	20 (77)	High SDI
6	Asano 2012 (28) Japan	Cohort	56.8 ± 11 (36-73)	158	140 (89)	NR	Tumor biopsy	NR	Gastric	158 (100)	High SDI
7	Ashton-Key 1996 (115) UK	Cross-sectional	NR	7 (NR)	7 (100)	NR	FFPE	Histology assays	Gastric Low-Grade	7 (100)	High SDI
8	Begueret 2002 (97)	Cross-sectional	65.2 (46 - 78)	14 (37.5)	0 (0)	NR	FFPE	Molecular assays	Non-Gastric (lung)	14/16 (87.5)	High SDI
9	Bessede 2012 (98) France	Case-control	Median 57.5 (24-80)	39 (41)	19 (48.7)	NR	Tumor biopsy	Histology assays	Gastric	39 (100)	High SDI
10	Cai 2012 (29) China	Cross-sectional	55±16.76 (14-83)	14 (35.7)	2 (14.2)	NR	Tumor biopsy	Molecular assays	Non-Gastric (Conjunctival)	14 (100)	Middle SDI
11	Cammarota 1995 (81) Italy	Case-control	56 (18-74)	200 (35)	151 (75.5)	NR	Tumor biopsy	Microbiological assays	Gastric Low-Grade	200 (100)	High–middle SDI
12	Chan 2004 (116) USA	Case-control	56±27.1 (10-81)	5 (NR)	4 (80)	NR	Tumor biopsy	Molecular assays	Non-Gastric (Conjun-ctival)	5 (100)	High SDI
13	Chen 2005 (30) Taiwan	Cohort	60 (30 – 84)	34 (55.9)	32 (94.1)	6/30 (20)	Tumor biopsy	Microbiological assays	Gastric High-Grade	34 (100)	High SDI
14	Cheng 2006 (31) Taiwan	Case-control	56.2±13.4 (10-81)	62 (48.4)	56 (90.3)	NR	Blood	Serological assays	Gastric	36 (58.1)	High SDI
15	Choi 2011 (32) Korea	Cross-sectional	52.23±10.29 (30-79)	39 (44)	30 (77)	NR	FFPE	Histology and Microbiological assays	Gastric Low-Grade	39 (100)	High SDI
16	Choi 2018 (33) Korea	Cohort	Median 52 (17–81)	132 (64.4)	82 (62.1)	0/2 (0)	Tumor biopsy	Histology and Microbiological assays	Gastric	132 (100)	High SDI
17	Claviez 2006 (99) Germany	Cross-sectional	10.5±3.4 (7-15)	4 (25)	2 (50)	NR	FFPE	Histology assays	Gastric	1 (25)	High SDI

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18	Couto 2021 (82) Portugal	Cohort	Median 61 (18–87)	60 (53.3)	41 (68.3)	NR	Tumor biopsy	NR	Gastric	60 (100)	High–middle SDI
19	Delchier 2001 (100) France	Case-control	52.6±14.6	37 (59.5)	29 (78)	NR	Blood	Serological assays	Gastric Low-Grade	37 (100)	High SDI
20	Diraimondo 2006 (83) Italy	Cohort	54.6±16.6 (24-78)	16 (50)	15 (93.8)	NR	Tumor biopsy	Histology and Microbiological assays	Gastric	16 (100)	High–middle SDI
21	El-zahabi 2007 (122) Lebanon	Cohort	52±14 (6-55)	22 (31.8)	20 (90.9)	1/19 (5.3)	Tumor biopsy	Histology assays	Gastric	22 (100)	High–middle SDI
22	Faujo Nintewoue 2023 (123) Cameroon	Cross-sectional	47.13± 19.81	10 (20)	5 (50)	NR	FFPE	Histology assays	Gastric	10 (100)	Low–middle SDI
23	Feng 2023 (34) China	Case-control	NR	216	182 (84.3)	NR	Tumor biopsy	Microbiological assays	Gastric	216 (100)	Middle SDI
24	Gisbert 2006 (84) Spain	Cross-sectional	61.62± 14	37 (37.8)	17 (45.9)	NR	FFPE	Histology and Microbiological assays	Gastric	37 (100)	High–middle SDI
25	Gong 2016 (35) Korea	Cohort	Median 53 (46–62)	468 (58.8)	392 (84.5)	57/403 (14.1)	Tumor biopsy	Histology, Microbiological, and Serological assays	Gastric	468 (100)	High SDI
26	Gong 2016a (36) Korea	Cohort	Median 53 (45–60)	345 (56.5)	317 (91.9)	43/317 (13.6)	Tumor biopsy	Histology, Microbiological, and Serological assays	Gastric	345 (100)	High SDI
27	Gruenberger 2008 (101) Austria	Cross-sectional	Median 64 (18–94)	45 (48.9)	15/39 (38)	NR	FFPE	Histology, Microbiological, and Serological assays	Non-Gastric (Ocular adnexal)	45 (100)	High SDI
28	Guitart 2014 (117) USA	Case-control	55± 18 (37–73)	80 (52.5)	16 (20)	NR	Skin biopsy	Serological assays	Non-Gastric (Skin)	80 (100)	High SDI
29	Hajder 2014 (85) Serbia	Cross-sectional	Median 63.5 (35–77)	35 (40)	20/30 (57.4)	NR	Tumor biopsy	Histology assays	Gastric	35 (100)	High–middle SDI
30	He 2017 (37) China	Cross-sectional	Median 57 (13-80)	25 (32)	10/23 (43.5)	NR	Tumor biopsy	Microbiological assays	Gastric	25 (100)	Middle SDI
31	Hellmig 2005 (102) Germany	Case-control	Median 57 (31 – 73)	87 (47)	83 (95)	NR	Tumor biopsy	Histology and Microbiological assays	Gastric Low-Grade	87 (100)	High SDI
32	Hu 2006 (38) China	Cross-sectional	45 (22-73)	31 (64.1)	27 (87.1)	NR	FFPE	Histology assays	Gastric Low-Grade	31 (100)	Middle SDI
33	Inagaki 2004 (39) Japan	Cohort	59 (16-87)	115 (43.5)	99 (86)	15/70 (21.4)	Tumor biopsy	Histology, Microbiological, and Serological assays	Gastric	115 (100)	High SDI

Table 1 continues

Table 1. Main characteristics of the studies involved.

Study ID Country		Type of Study	Age (mean ± SD / range)	Number of MALT patients (% female)	Prevalence of HP in MALT (HP positivity) # (%)	HP positivity after Eradication # (%)	Sample type	Detection method	MALT types		Socio-demographic Index (SDI)
									Type	# (%)	
34	Insabato 2003 (86) Italy	Cross-sectional	57 (22-81)	95 (45.3)	13/26 (50)	NR	FFPE	Histology assays	Gastric	95 (100)	High–middle SDI
35	Isomoto 2013 (40) Japan	Cross-sectional	56± 18.2 (31–79)	7 (28.6)	6 (85.7)	NR	Tumor biopsy	Histology and Microbiological assays	Gastric	7 (100)	High SDI
36	Kiesewetter 2020 (103) Austria	Cohort	Median 63 (22–85)	137 (49)	93 (68)	39/93 (41.9)	Tumor biopsy	Histology and Serological assays	Gastric	137 (100)	High SDI
37	Kiesewetter 2020a (104) Austria	Cohort	Median 65 (25–88)	159 (57)	127 (80)	NR	NR	NR	Gastric, non-gastric	32 (20), 127 (80)	High SDI
38	Kim 2016 (41) Korea	Cohort	58.8±9.8 (38–82)	54 (54.2)	42 (78)	6/41 (14.6)	Tumor biopsy	Histology and Microbiological assays	Gastric	54 (100)	High SDI
39	Kim 2021 (42) Korea	Cohort	56±12	1163 (57.6)	873 (75.2)	NR	Tumor biopsy	Histology and Microbiological assays	Gastric	1163 (100)	High SDI
40	Kobayashi 1998 (43) Japan	Cross-sectional	NR	24	22 (91.67)	NR	FFPE + Fresh frozen	Histology assays	Gastric Low-Grade	24 (100)	High SDI
41	Kodera 2000 (44) Japan	Cohort	NR	7	6 (85.7)	0/6 (0)	Tumor biopsy	Microbiological and Serological assays	Gastric Low-Grade	7 (100)	High SDI
42	Konturek 2000 (124) Poland	Case-control	Median 54 (38–65)	20 (25)	18 (90)	NR	Sera	Microbiological and Serological assays	Gastric Low-Grade	20 (100)	High–middle SDI
43	Konturek 2002 (125) Poland	Case-control	Median 56 (38–67)	10 (50)	10 (100)	NR	Sera	Microbiological and Serological assays	Gastric Low-Grade	10 (100)	High–middle SDI
44	Kuo 2004 (47) Taiwan	Cohort	55.8±15.2 (21–83)	22 (72.7)	14 (63.6)	0/14 (0)	Tumor biopsy	Histology and Microbiological assays	Gastric High-Grade	22 (100)	High SDI
45	Kuo 2005 (45) Taiwan	Cohort	58.3±15.8 (21–84)	26 (73.1)	16 (61.5)	0/16 (16)	Tumor biopsy	Histology and Microbiological assays	Gastric High-Grade	26 (100)	High SDI
46	Kuo 2008 (46) Taiwan	Cohort	Median 62.5 (38–71)	14 (64.3)	11 (78.6)	NR	Tumor biopsy	Histology and Microbiological assays	Gastric	14 (100)	High SDI

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47	Lamarque 2006 (105) France	Case-control	45±2.1	45 (56)	39 (86.7)	NR	FFPE	Histology and Microbiological assays	Gastric	45 (100)	High SDI
48	Li 2013 (49) China	Cohort	56 (19-81)	62 (42)	38/51 (74.5)	NR	FFPE	Histology and Microbiological assays	Gastric	62 (100)	Middle SDI
49	Li 2016 (48) China	Cohort	Median 53 (19-85)	103 (50)	97 (94)	0/94(0)	FFPE	Histology and Microbiological assays	Gastric	103 (100)	Middle SDI
50	Lim 2016 (50) Korea	Cohort	Median 51 (17–74)	33 (48.5)	16 (48.5)	NR	NR	Histology, Microbiological, and Serological assays	Gastric	33 (100)	High SDI
51	Matysiak-Budnik 2019 (106) France	Cohort	Median 67 (23□97)	416 (49.8)	212/394 (54)	200/339 (59)	Tumor biopsy	Histology assays	Gastric	416 (100)	High SDI
52	Min 2006 (52) Korea	Case-control	56.3 (22-80)	35 (40)	30 (85.7)	NR	FFPE	Histology assays	Gastric	35 (100)	High SDI
53	Min 2014 (51) Korea	Cohort	53.3±11.4 (30-78)	194 (54.6)	154 (79.4)	NR	NR	Histology, Microbiological, and Serological assays	Gastric	194 (100)	High SDI
54	Moleiro 2016 (87) Portugal	Cohort	56±13 (13–83)	144 (47)	97/127 (76.4)	2/84 (2.4)	Tumor biopsy	Histology and Microbiological assays	Gastric	144 (100)	High–middle SDI
55	Na 2021 (53) Korea	Cohort	Range 20–74	143 (46.9)	124 (86.7)	NR	Tumor biopsy	Histology and Microbiological assays	Gastric	143 (100)	High SDI
56	Nakamura 1998 (54) Japan	Case-control	54 ± 13	28 (61)	24(86)	NR	FFPE	Histology assays	Gastric	28 (100)	High SDI
57	Nakamura 2003 (55) Japan	Cohort	59 (29-81)	47 (44.7)	36/46 (78.3)	NR	Fresh or frozen tissue	Histology, Microbiological, and Serological assays	Gastric	47 (100)	High SDI
58	Nakamura 2005 (56) Japan	Cohort	61.7 (16-84)	96 (53.1)	89 (92.7)	0/89 (0)	Tumor biopsy	Histology, Microbiological, and Serological assays	Gastric	96 (100)	High SDI
59	Nakamura 2005a (58) Japan	Cross-sectional	61.8 (16-84)	65 (47.7)	58 (89.2)	NR	Fresh or frozen tumor biopsy	Histology, Microbiological, and Serological assays	Gastric	65 (100)	High SDI
60	Nakamura 2006 (57) Japan	Cross-sectional	62 (16-84)	137 (54.7)	125 (91)	NR	Fresh or frozen tumor biopsy	Histology and Serological assays	Gastric	137 (100)	High SDI

Table 1 continues

Table 1. Main characteristics of the studies involved.

Study ID Country		Type of Study	Age (mean ± SD / range)	Number of MALT patients (% female)	Prevalence of HP in MALT (HP positivity) # (%)	HP positivity after Eradication # (%)	Sample type	Detection method	MALT types		Socio-demographic Index (SDI)
									Type	# (%)	
61	Nakamura 2012 (59) Japan	Cohort	Median 61 (16-87)	420 (56)	376 (89.5)	53/376 (14.1)	Tumor biopsy	Histology, Microbiological, and Serological assays	Gastric	420 (100)	High SDI
62	Nonaka 2014 (60) Japan	Cohort	60.1 (22-79)	16 (37.5)	12 (75)	0/12 (0)	Tumor biopsy	Histology and Microbiological assays	Gastric	16 (100)	High SDI
63	Ohashi 2000 (61) Japan	Cohort	59+13.7 (29-81)	23 (52.2)	13 (56.5)	3/11 (27.3)	Tumor biopsy	Histology and Microbiological assays	Gastric	23 (100)	High SDI
64	Ono 2008 (62) Japan	Cohort	61.3+8.1 (45-72)	11 (63.6)	10 (90.9)	0/10 (0)	Tumor biopsy	Histology and Microbiological assays	Gastric	11 (100)	High SDI
65	Ono 2011 (63) Japan	Cohort	NR	21	16 (76.2)	0/16 (0)	Tumor biopsy	Histology, Microbiological, and Serological assays	Gastric	17 (100)	High SDI
66	Overby 2017 (64) Japan	Case-control	NR	40	21 (52.5)	NR	Tumor biopsy	Molecular and Serological assays	Gastric	40 (100)	High SDI
67	Park 2010 (66) Korea	Cohort	Median 53 (16-81)	292 (56.2)	143/198 (72.2)	10/143 (7)	Tumor biopsy	Histology assays	Gastric, non-gastric	198 (67.8), 94 (32.2)	High SDI
68	Park 2019 (65) Korea	Cohort	55.4±11.7	50 (64)	42 (84)	2/41 (4.9)	Tumor biopsy	Microbiological assays	Gastric	50 (100)	High SDI
69	Park 2021 (67) Korea	Cohort	53 ± 11 (23–77)	115 (53.9)	96 (83.5)	NR	Tumor biopsy	NR	Gastric Low-Grade	115 (100)	High SDI
70	Parween 2020 (126) Pakistan	Cross-sectional	44.39 ± 12.29 (15–65)	122 (48.36)	81 (66.4)	NR	Tumor biopsy	Histology assays	Gastric	122 (100)	Low SDI
71	Rentien 2018 (107) France	Cohort	57.4±13.7	179 (47.5)	119 (66.5)	NR	Tumor biopsy	Histology and Molecular assays	Gastric	179 (100)	High SDI
72	Ruskone-fourmestraux 2001 (108) France	Cohort	56 (21-76)	44 (34.1)	34 (77)	5/34 (9)	Tumor biopsy	Histology and Serological assays	Gastric Low-Grade	44 (100)	High SDI
73	Ruskone-fourmestraux 2015 (109) France	Cohort	Median 60 (30–78)	53 (45.3)	38 (71.7)	1/38 (3)	Tumor biopsy	Histology and Serological assays	Gastric	53 (100)	High SDI

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74	Ryu 2014 (68) Korea	Cohort	57	57 (57.9)	43 (75)	4/39 (9.3)	Tumor biopsy	Histology, Microbiological, and Serological assays	Gastric	57 (100)	High SDI
75	Saifi 2021 (118) USA	Cohort	Median 71 (25-88)	42 (52.4)	11 (26.2)	6/26 (23.1)	Tumor biopsy	Histology assays	Gastric	42 (100)	High SDI
76	Saito 2012 (69) Japan	Cohort	62.5+10 (40-77)	20 (65)	15 (75)	0/12 (0)	FFPE	Histology, Microbiological, and Serological assays	Gastric Low-Grade	20 (100)	High SDI
77	Sakuma 2007 (70) Japan	Cohort	59.5 ± 12.9 (16–82)	45 (46.7)	36 (80)	13/35 (37.1)	FFPE	Histology and Microbiological assays	Gastric	45 (100)	High SDI
78	Siakantaris 2009 (88) Greece	Cohort	Median 57 (33–86)	62 (41.9)	51 (82)	0/50 (0)	FFPE	Histology assays	Gastric	62 (100)	High–middle SDI
79	Sorrentino 1997 (89) Italy	Cohort	57 ± 14 (29–80)	16 (68.8)	12 (75)	3/12 (25)	FFPE	Histology assays	Gastric	16 (100)	High–middle SDI
80	Sretenovic 2009 (90) Serbia	Cohort	Median 59 (29–78)	51 (47)	12/42 (28.6)	NR	Tumor biopsy	NR	Gastric, non-gastric	4 (7.8), 47 (92.2)	High–middle SDI
81	Stathis 2009 (110) Switzerland	Cohort	Median 64 (20–94)	105 (51)	85 (81)	0/85 (0)	FFPE	Histology assays	Gastric Low-Grade	104 (99)	High SDI
82	Sumida 2009 (71) Japan	Cohort	59.8 (20-89)	66 (56.1)	57 (86.4)	10/57 (17.5)	Tumor biopsy + Serum	Histology, Microbiological, and Serological assays	Gastric Low-Grade	66 (100)	High SDI
83	Taji 2005 (72) Japan	Cohort	55 (49 to 72)	13 (46.2)	12 (92.3)	5/12 (45.7)	Tumor biopsy	Histology, Microbiological, and Serological assays	Gastric Low-Grade	13 (100)	High SDI
84	Takigawa 2021 (73) Japan	Cohort	Median 62 (20–89)	182 (43)	148 (81)	0/148 (100)	Tumor biopsy	Serological assays	Gastric	182 (100)	High SDI
85	Teresa 2019 (91) Italy	Case-control	67.58±16.26 (57-80)	24 (66.67)	11 (45.83)	NR	Tumor biopsy	Molecular assays	Gastric	24 (100)	High–middle SDI
86	Todorovic 2008 (92) Serbia	Cohort	Median 62 (34-82)	34 (47.1)	25 (73.5)	NR	Tumor biopsy	Histology and Microbiological assays	Gastric	34 (100)	High–middle SDI
87	Troch 2008 (111) Austria	Cohort	57.2 ± 13.6 (31–80)	13 (76.9)	7 (53.8)	0/7 (0)	Tumor biopsy	NR	Gastric, non-gastric	5 (38.5), 8 (61.5)	High SDI
88	Türe-Özdemir 2008 (93) Greece	Case-control	Median 61 (29-75)	56 (46.4)	56 (100)	NR	Tumor biopsy	Histology and Microbiological assays	Gastric	56 (100)	High–middle SDI
89	Ullrich 2002 (112) Germany	Cross-sectional	63.6 (30-89)	38	36 (95)	NR	Tumor biopsy	Histology assays	Gastric	38 (100)	High SDI

Table 1 continues

Table 1. Main characteristics of the studies involved.

Study ID Country		Type of Study	Age (mean ± SD / range)	Number of MALT patients (% female)	Prevalence of HP in MALT (HP positivity) # (%)	HP positivity after Eradication # (%)	Sample type	Detection method	MALT types		Socio-demographic Index (SDI)
									Type	# (%)	
90	Virijevic 2014 (94) Serbia	Cross-sectional	58 (28–82)	87 (52.9)	27/79 (34.2)	NR	Tumor biopsy	NR	Gastric, non-gastric	37 (42.5), 50 (57.5)	High–middle SDI
91	Vrieling 2008 (113) Netherlands	Cohort	Median 60 (22–86)	115 (46)	86/99 (86.9)	NR	Tumor biopsy	Histology assays	Gastric	115 (100)	High SDI
92	Watanobe 2002 (74) Japan	Case-control	NR	13	8 (61.5)	NR	Tumor biopsy	Histology assays	Gastric Low-Grade	5 (38.5)	High SDI
93	Weston 1999 (119) USA	Cohort	63.1±12.6 (33.7-80.1)	68 (1.5)	65 (95.6)	8/65 (12.3)	FFPE	Histology assays	Gastric Low-Grade	Gastric Low-Grade	High SDI
94	Wohrer 2004 (114) Austria	Cross-sectional	Median 57 (22–85)	52	30 (57.7)	NR	NR	NR	Gastric	52 (100)	High SDI
95	Xu 1997 (75) Hong Kong	Cross-sectional	51.4 (29-92)	53 (56.6)	29 (54.7)	NR	FFPE	Histology assays	Gastric	53 (100)	Middle SDI
96	Yahalom 2021 (120) USA	Cohort	Median 63 (25-89)	178 (56)	35 (20)	0/35 (0)	Tumor biopsy	NR	Gastric Low-Grade	178 (100)	High SDI
97	Yang 2016 (76) Korea	Cohort	Median 54.2 (23–79)	51 (65.4)	47 (90.4)	4/50 (8)	Tumor biopsy	Histology, Microbiological, and Serological assays	Gastric	51 (100)	High SDI
98	Yeh 2003 (77) China	Case-control	Median 61 (29–80)	20 (100)	20 (45)	17 (85)	Tumor biopsy	Histology and Microbiological assays	Gastric Low-Grade	20 (100)	Middle SDI
99	Yin 2004 (78) China	Cross-sectional	NR	NR	15 (100)	10 (66.7)	FFPE	Histology assays	Gastric	15 (100)	Middle SDI
100	Yokoi 1999 (79) Japan	Cohort	60.1±14.3 (40 -78)	8 (37.5)	3 (37.5)	3 (37.5)	Serum + Tumor biopsy	Histology and Microbiological assays	Gastric Low-Grade	8 (100)	High SDI
101	Yoo 1998 (121) USA	Cohort	66 (61-76)	6 (100)	5 (83.3)	5 (83.3)	Tumor biopsy	Histology assays	Gastric	6 (100)	High SDI
102	Zenzri 2020 (127) Tunisia	Cohort	51	50 (46)	44 (88)	7/25 (28)	Tumor biopsy	Histology assays	Gastric	50 (100)	Middle SDI

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(29, 34, 37, 38, 48, 49, 75, 77, 78, 127), low–middle SDI (123), and low SDI (126). The MALT patients in the included studies were classified into three groups based on the type of MALT lymphoma: gastric (26-28, 30-65, 67-89, 91-93, 95, 96, 98-100, 102, 103, 105-110, 112-115, 118-127), non-gastric (29, 97, 101, 116, 117), and both gastric and non-gastric (66, 90, 94, 104, 111). Furthermore, gastric MALT lymphoma was graded into two types: low–grade lymphoma (27, 32, 38, 43, 44, 67, 69, 71, 72, 74, 77, 79, 81, 100, 102, 108, 110, 115, 119, 120, 124, 125) and high–grade lymphoma (30, 45, 47).

Outcomes

The pooled prevalence of *H. pylori* in patients with MALT lymphoma was 73.0% (95% CI, 69.0–77.1%, n = 8725), as illustrated in Figure 2.

Subgroup analyses

Based on the subgroup analyses of the prevalence of *H. pylori* in patients with MALT lymphoma over four-time intervals, there was a fluctuation in the prevalence: 75.3% (95% CI: 63.9 to 86.7; $p < 0.01$) from 1995 to 2002, which increased to 78.6% (95% CI: 72.9 to 84.2; $p < 0.01$) from 2003 to 2009, then decreased to 69.0% (95% CI: 60.6 to 77.4; $p < 0.01$) from 2010 to 2016, and finally further decreased to 67.8% (95% CI: 59.3 to 76.2; $p < 0.01$) from 2017 to 2023, which was lower than the pooled prevalence for the entire period (Table 2 and Figure S1). The subgroup analysis according to different geographical locations discovered a significantly increased prevalence in Asia/Eastern Asia at 79.5% (95% CI: 75.9 to 83.1; $p < 0.01$) compared to the pooled prevalence. In contrast, significantly decreased prevalence rates were observed in Europe/Western Europe at 66.7% (95% CI: 56.6 to 76.9; $p < 0.01$), Europe/Southern Europe at 64.4% (95% CI: 53.2 to 75.6; $p < 0.01$), and the Americas/Northern America at 52.6% (95% CI: 23.5 to 81.8; $p < 0.01$) (Table 2 and Figure S2). Furthermore, subgroup analyses according to the approaches of *H. pylori* detection showed a significantly increased prevalence when compared to the pooled prevalence, with microbiological methods at 85.5% (95% CI: 78.7 to 92.2; $p < 0.01$); histological +

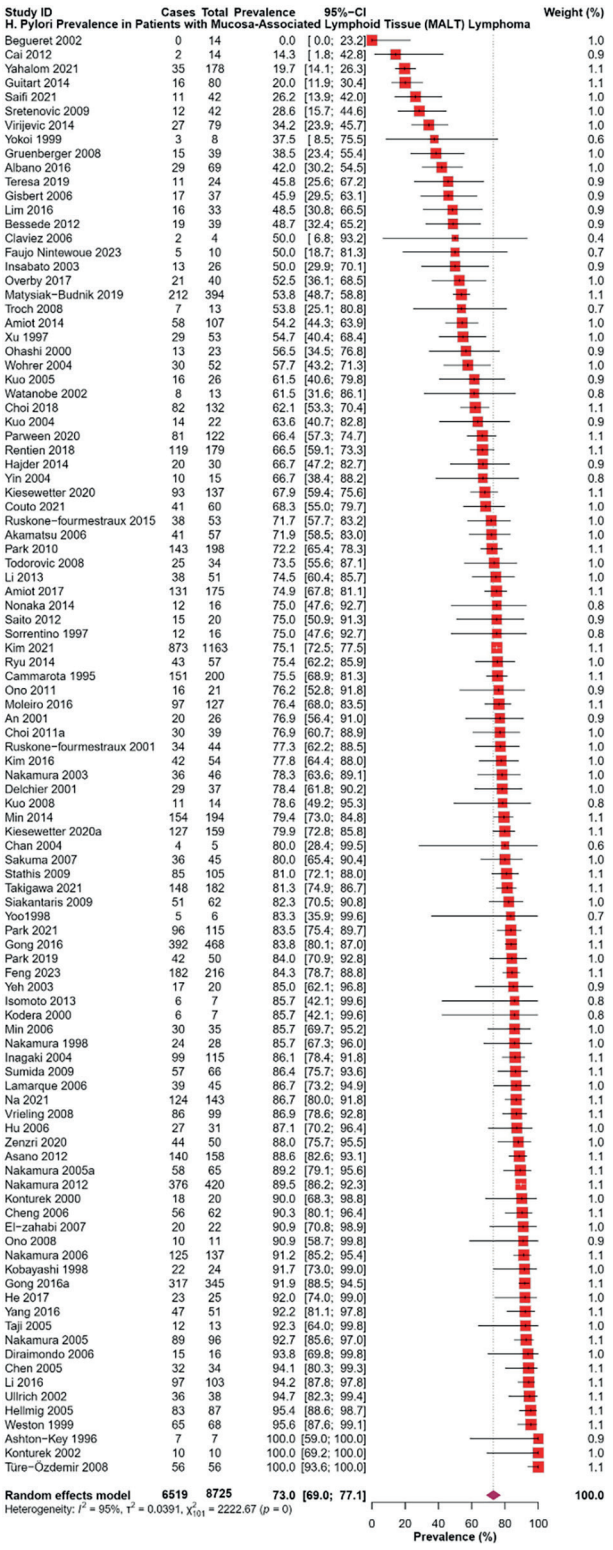

Figure 2. Forest plots presenting the prevalence of *H. pylori* in patients with MALT lymphoma (26-127).

Table 2. Subgroup analysis of prevalence of H. pylori in patients with MALT lymphoma.

Subgroups	Prevalence of *H. pylori* [95% CI]	Studies Number	*H. pylori* Positivity cases	Heterogeneity	
				I^2, %	*p* Value
Time interval trend					
From 1995 to 2002	75.3 [63.9, 86.7]	19	492	96	< 0.01
From 2003 to 2009	78.6 [72.9; 84.2]	36	1336	89	< 0.01
From 2010 to 2016	69.0 [60.6; 77.4]	26	2190	96	< 0.01
From 2017 to 2023	67.8 [59.3; 76.2]	21	2501	96	< 0.01
Geographical location					
Asia / Eastern Asia	79.5 [75.9; 83.1]	54	4378	85	< 0.01
Europe / Western Europe	66.7 [56.6; 76.9]	20	1243	96	< 0.01
Europe / Southern Europe	64.4 [53.2; 75.6]	15	577	97	< 0.01
Americas / Northern America	52.6 [23.5; 81.8]	6	136	99	< 0.01
Europe / Eastern Europe	95.3 [85.5; 100.0]	2	28	16	0.28
Methods of *H. pylori* detection					
Histological	74.4 [67.6; 81.1]	28	1128	91	< 0.01
Histological + Microbiological	76.5 [70.2; 82.8]	25	1785	94	< 0.01
Histological + Microbiological + Serological	81.0 [74.2; 87.8]	16	1742	85	< 0.01
Microbiological	85.5 [78.7; 92.2]	5	430	75	< 0.01
Serological	67.5 [36.0; 99.0]	4	249	98	< 0.01
Molecular	32.4 [0.0; 65.7]	4	17	91	< 0.01
Histological + Serological	77.6 [66.5; 88.7]	4	290	90	< 0.01
Histological + Molecular	65.6 [54.1; 77.1]	3	308	84	< 0.01
Microbiological + Serological	94.3 [85.6; 100.0]	3	34	0	0.44
MALT Lymphoma types					
Gastric Lymphoma	76.7 [73.3; 80.1]	92	6166	94	< 0.01
Non-Gastric Lymphoma	27.4 [3.9; 50.8]	5	37	88	< 0.01
Gastric and non-Gastric Lymphomas	54.2 [33.5; 75.0]	5	361	95	< 0.01
Gastric Lymphoma types					
Low-Grade Lymphoma	80.5 [72.5; 88.4]	22	830	96	< 0.01
High-Grade Lymphoma	74.7 [52.6; 96.7]	3	62	87	< 0.01
Socio-demographic Index (SDI)					
High SDI	74.0 [69.3; 78.7]	72	5339	96	< 0.01
High–middle SDI	69.3 [58.7; 79.9]	18	625	96	< 0.01
Middle SDI	75.0 [60.5; 89.4]	10	469	91	< 0.01

microbiological + serological methods at 81.0% (95% CI: 74.2 to 87.8; $p < 0.01$); histological + serological methods at 77.6% (95% CI: 66.5 to 88.7; $p < 0.01$); histological + microbiological methods at 76.5% (95% CI: 70.2 to 82.8; $p < 0.01$); and histological methods alone at 74.4% (95% CI: 67.6 to 81.1; $p < 0.01$). Conversely, there was a significantly decreased prevalence in serological assays at 67.5% (95% CI: 36.0 to 99.0; $p < 0.01$); histological + molecular assays at 65.6% (95% CI: 54.1 to 77.1; $p < 0.01$); and molecular assays alone

at 32.4% (95% CI: 0.0 to 65.7; $p < 0.01$) (Table 2 and Figure S3). Moreover, the subgroup analysis based on MALT lymphoma types revealed a significant increase in prevalence of gastric lymphoma at 76.7% (95% CI: 73.3 to 80.1; $p < 0.01$) compared to the pooled prevalence. In contrast, both non-gastric lymphomas and combined gastric and non-gastric lymphomas showed significantly decreased prevalence at 27.4% (95% CI: 3.9 to 50.8; $p < 0.01$) and 54.2% (95% CI: 33.5 to 75.0; $p < 0.01$), respectively (Table 2 and Figure S4). Interestingly, both low-grade and high-grade gastric MALT lymphomas revealed a significantly increased prevalence compared to the pooled prevalence, with low-grade at 80.5% (95% CI: 72.5 to 88.4; $p < 0.01$) and high-grade at 74.7% (95% CI: 52.6 to 96.7; $p < 0.01$) (Table 2 and Figure S5). The socio-demographic index (SDI) analysis showed a significantly increased prevalence compared to the pooled prevalence in both high SDI at 74.0% (95% CI: 69.3 to 78.7; $p < 0.01$) and middle SDI at 75.0% (95% CI: 60.5 to 89.4; $p < 0.01$). In contrast, the high–middle SDI group showed a significantly decreased prevalence at 69.3% (95% CI: 58.7 to 79.9; $p < 0.01$) (Table 2 and Figure S6).

Quality assessment

Tables S2–S4 present a detailed quality assessment of the entered articles. Overall, there were 70.6% high-quality studies, 25.5% moderate-quality studies, and 3.9% low-quality studies. The funnel plot and Egger's test showed evidence of publication bias for the prevalence of *H. pylori* in patients with MALT lymphoma ($p = 0.0029$) (Figure 3).

Heterogeneity and sensitivity analysis

In sensitivity analyses (Table 3 and Figure S7-S10), the highest *H. pylori* prevalence in patients with MALT lymphoma was observed when excluding outlier studies (77.7%; 95% CI: 74.8 to 80.6) (26-28, 30-89, 91-93, 95, 96, 98-116, 119, 121-127), followed by considering only case-control studies (76.4%; 95% CI: 66.2 to 86.7) (31, 34, 52, 54, 64, 74, 77, 81, 91, 93, 98, 100, 102, 105, 116, 117, 124, 125), excluding small studies with less than 100 participants (75.7%; 95% CI: 69.7 to 81.8) (28, 33-36, 39, 42, 48, 51, 53, 57, 59, 66, 67, 73, 81, 87, 95, 96, 103, 104, 106, 107, 110, 120, 126), and considering only cohort studies (75.6%; 95% CI: 71.3 to 79.9) (26, 28, 30, 33, 35, 36, 39, 41, 44-51, 53, 55, 56, 59, 61-63, 65-68, 70-73, 76, 79, 82, 83, 87, 88, 90, 92, 95, 96, 103, 104, 106-111, 113, 118-122, 127). Seven studies were identified as outliers in estimating the prevalence of *H. pylori* in patients with MALT lymphoma, as shown in the Galbraith plot (Figure 4). In contrast, the lowest *H. pylori* prevalence in patients with MALT lymphoma was observed when considering only cross-sectional studies (63.9%; 95% CI: 52.9 to 74.9) (27, 29, 32, 37, 38, 40, 43, 57, 58, 75, 78, 80, 84-86, 94, 97, 99, 101, 112, 114, 115, 123, 126), followed by excluding low- and moderate-quality studies (72.0%; 95% CI: 67.2 to 76.8) (26, 29-38, 41, 45, 47-54, 56-61, 63-69, 71, 76, 80-82, 84-88, 90, 91, 93-96, 98, 100-110, 113, 117-120, 122-126).

Discussion

Helicobacter pylori is the primary cause of chronic gastritis and can lead to severe gastroduodenal conditions in some patients, such as gastric and duodenal peptic ulcer disease (PUD), gastric cancer, and gastric mucosa-associated lymphoid tissue (MALT) lymphoma (7, 128). MALT lymphoma is characterized by lymphoepithelial lesions and tumor cell aggregates that damage the stomach glands (4, 17). Although lymphoid tissue is not ordinarily present in the gastric

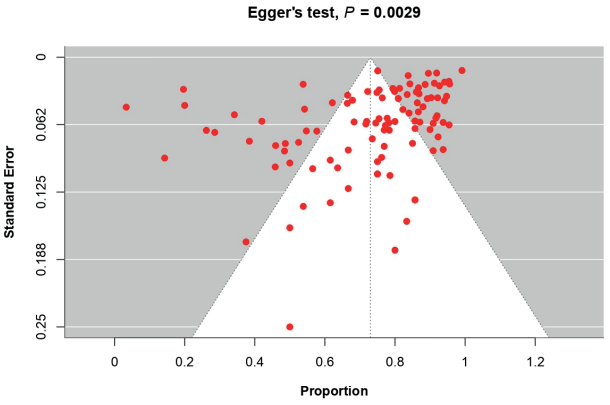

Figure 3. Funnel plots estimating the prevalence of *H. pylori* in patients with MALT lymphoma revealed significant publication bias (26-127).

Table 3. Sensitivity analyses.

Strategies of sensitivity analyses	Prevalence [95% CIs]	Difference of pooled prevalence compared to the main result	Number of studies analyzed	Total number of subjects	Heterogeneity	
					I^2	*p*-value
Excluding small studies (>100)	75.7 [69.7; 81.8]	2.7% higher	26	5972	97	< 0.01
Excluding low- and moderate-quality studies	72.0 [67.2; 76.8]	1% lower	72	6550	96	0
Considering only cross-sectional studies	63.9 [52.9; 74.9]	9.1% lower	24	963	96	< 0.01
Considering only case-control studies	76.4 [66.2; 86.7]	3.4% higher	18	1017	96	< 0.01
Considering only cohort studies	75.6 [71.3; 79.9]	2.6% higher	60	6745	94	< 0.01
Excluding outlier studies	77.7 [74.8; 80.6]	4.7% higher	95	8276	91	< 0.01

Abbreviations: CIs: Confidence intervals.

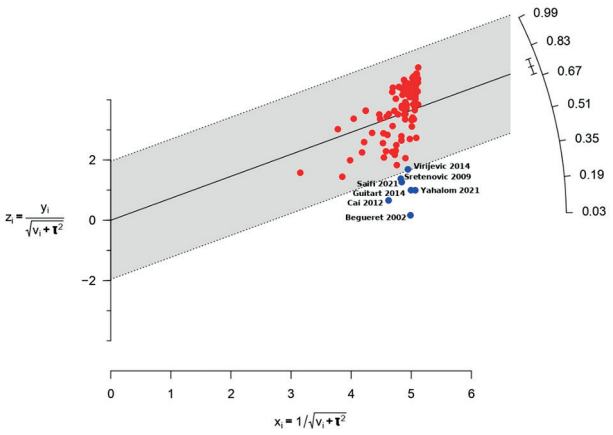

Figure 4. Galbraith plots show seven outlier studies in estimating prevalence of *H. pylori* in patients with MALT lymphoma (26-28, 30-89, 91-93, 95, 96, 98-116, 119, 121-127).

mucosa, MALT lymphomas almost develop in response to *H. pylori* colonization (4, 17). Thus, there is a substantial correlation between *H. pylori* infection and the development of gastric MALT lymphomas. *H. pylori* infection causes the stomach to become colonized with lymphoid tissue in response to antigen stimulation, leading to the recruitment of B and T lymphocytes into the gastric mucosa, which in turn causes gastritis, which eventually progresses to low-grade MALT lymphoma and later high-grade MALT lymphoma (7, 17). *H. pylori* is the most prevalent infectious agent associated with cancers globally, accounting for 5.5% of all cancer cases (129). The prevalence of *H. pylori* infection differs based on geographic region, age, socio-economic status, education level, living environment, and occupation. Most *H. pylori* infections are found in developing countries, where up to 80% of middle-aged adults may be affected (128, 130). This systematic review and meta-analysis of 102 studies evaluating the prevalence of *H. pylori* infection in patients with MALT lymphomas showed that the overall prevalence was 73% of MALT lymphoma cases worldwide. However, our study demonstrated that the prevalence of *H. pylori* in MALT lymphoma patients varies by region. Specifically, Asia/Eastern Asia showed a significantly higher prevalence (79.5%), whereas Northern Africa, Western Europe, Southern Europe and Northern America had notably lower rates compared to the pooled prevalence, with rates of 69.3%, 66.7%, 64.4%, and 52.6%, respectively. The varying prevalence of *H. pylori* across countries can be attributed to differences in sanitation, levels of hygiene, eradication methods, and genetic predispositions, as over half of the global population is affected. Another potential explanation could be related to the difference in host immune response and diet habits, where high salt and rich-fat diets can lead to higher susceptibility to *H. pylori* infection (131, 132). Interestingly, our findings closely align with a systematic review from 2007, which reported a 79% prevalence in Asia/Eastern Asia, supporting the consistency of our results (133). A possible explanation for these results may be the geographical differences in the expression of the Cytotoxin-associated gene

(CagA), the most researched aspect of *H. pylori's* pathogenicity. Although CagA is present in 70% of *H. pylori* strains worldwide, there are geographical variations. For example, in East Asian nations like South Korea, China, and Japan, the percentage of CagA-positive strains is 90–95%, whereas in other countries like England, Australia, and the United States, the proportion is only 40% (134). Mechanistically, the virulence factor CagA inhibits the accumulation of p53 via reduction of p53 transcription and prevention of apoptosis in B-lymphocytes, which both influence the development of MALT lymphomas (134, 135). Our results revealed that the incidence of *H. pylori* among males was slightly different from that in females (51.7%), which aligns with some studies indicating that *H. pylori* infection rates show few variations between males and females (136, 137). However, this result is inconsistent with a study revealed where the predominance of males is higher than females (male to female ratio is approximately 2.43 to 1) (138), and in other studies where the predominance of females is higher than males (139, 140). The gender disparity could be related to the differences in metabolic factors like lipid profile and their related disorders such as cardiovascular diseases and diabetes (141, 142). This study revealed a significant decrease in *H. pylori* prevalence among patients with MALT lymphoma across four-time intervals, dropping rates from 75.3% between 1995 and 2002 to 67.8% between 2017 and 2023. This reduction in prevalence could be attributed to improved *H. pylori control* among infected individuals, changes in sanitary conditions, enhancements in socioeconomic status, living standards, and hygiene, as well as the increased use of antibiotics and eradication therapies (4, 12, 143). Furthermore, the study examined the prevalence of *H. pylori* across regions with different Socio-demographic Index (SDI) levels: High SDI, High–middle SDI, and Middle SDI. The findings revealed significant patterns related to socio-economic development and its impact on *H. pylori* prevalence. The high prevalence of *H. pylori* in MALT patients in high SDI regions (74%) may be due to the large population size and the persistence of previous infections despite medical advancements (144). High–middle SDI regions showed a slightly lower prevalence of 69.3%, which may indicate improvements in public health and hygiene practices characteristic of developing areas. However, Middle SDI regions exhibited a prevalence of 75.0%, highlighting the ongoing challenges related to healthcare infrastructure, sanitation, and socio-economic conditions in these areas. As a result, our findings are consistent with several studies that revealed the overall prevalence of *H. pylori* infection in developed countries (High SDI) is lower than that in developing countries (High–middle SDI and/or Middle SDI) (25, 145, 146). This study identified a notable publication bias concerning *H. pylori* prevalence in patients with MALT lymphoma, consistent with findings from other studies examining *H. pylori* prevalence in various diseases, including gastric cancer ($p \leq 0.05$) (147), biliary tract cancers (148), and pancreatic cancer (149). However, our findings differ from studies related to esophageal neoplasia ($p > 0.05$) (150, 151). MALT lymphomas are considered slow-growing neoplasms, indolent tumors frequently localized at the time of diagnosis. *H. pylori* infection-induced antigen stimulation results in somatic hypermutation during T-cell-dependent B-cell response and the emergence of different antigen receptors with modified binding affinities (7, 17). Since high-grade gastric lymphoma generates a monoclonal B-cell clone that is not responsive to antigens, it is considered an *H. pylori*-independent developing tumor (7, 17). As mentioned in the literature, our study showed a significant increase in *H. pylori* prevalence among patients with low-grade MALT lymphoma (80.5%) compared to high-grade MALT lymphoma (74.7%). Additionally, given that most MALT lymphomas occur in the gastrointestinal tract (7, 17), our data showed that the prevalence of *H. pylori* in gastric lymphomas was higher than in non-gastric lymphomas, with rates of 76.7 and 27.4, respectively. In stage I IIE Lugano classification system, where tumor extension is restricted to the GI tract or extended into the abdomen, the first line treatment for all patients with MALT lymphomas should be *H. pylori* eradication therapy (triple therapy), including proton pump inhibitors (PPI), clarithromycin, amoxicillin and/or metronidazole (9, 12, 14). The second line of treatment is bismuth quadruple, including PPI, bismuth subcitrate or subsalicylate, tetracycline, and metronidazole, if triple therapy is unsuccessful (9, 12,

14). Cultures with susceptibility testing or molecular genotyping should be carried out if second-line treatment is unsuccessful. On the other hand, in the stage IV Lugano classification system, where tumor extension is disseminated into extranodal involvement, *H. pylori* eradication therapy and systemic therapy are used, including antibiotics, radiotherapy, chemotherapy, immunotherapy, and/or surgery (9, 12, 14). Our analysis showed that the most effective therapy for reduced prevalence of *H. pylori* was a combination of *H. pylori* eradication therapy and systemic therapy (risk ratio of 0.04), including PPIs, antibiotics, chemotherapy, radiotherapy, and immunotherapy. A possible explanation for this resistance to antibiotic therapy might be a point mutation in the RDXA gene of the bacterial 23S rDNA (152). Another possible explanation is that t(11;18)(q21;q21) is commonly associated with infections of helicobacter species (10). Further studies are required to find new therapeutic strategies, even though MALT lymphoma is considered a rare pathological disease.

Conclusion

The prevalence of *Helicobacter pylori* infection is linked to the development of gastric mucosa-associated lymphoid tissue (MALT) lymphomas, a subset of indolent tumors with implications ranging from gastritis to potentially severe gastric carcinoma. Our comprehensive study underscores the global prevalence of *H. pylori* in MALT lymphoma patients (73%), revealing significant regional differences influenced by socio-economic factors and genetic variations such as the distribution of CagA-positive strains and the presence of t(11;18)(q21;q21). The reduction in *H. pylori* prevalence over time reflects advancements in eradication therapies and improved socio-economic conditions. However, challenges persist, particularly in middle and lower socio-demographic index regions, necessitating healthcare and therapeutic innovation. Understanding these mechanisms is crucial for optimizing treatment strategies, emphasizing the efficacy of *H. pylori* eradication alongside systemic therapies in managing MALT lymphomas and improving patient outcomes.

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