




The occupational risk of *Helicobacter pylori* infection: a systematic review

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Abstract

Purpose The aim of this systematic review was to describe the prevalence of *Helicobacter pylori* infection in specific occupational groups and to compare them with the general population.

Methods We searched PubMed® to identify original studies reporting the prevalence of *H. pylori* infection in occupational groups. The differences between occupational groups and the general population were analyzed taking into account the direction and statistical significance of the differences observed when comparing each occupational group with a reference group (either recruited in the same study or using an external comparator).

Results A total of 98 studies addressing the prevalence of *H. pylori* infection in occupational groups were included in the systematic review. Overall, health professionals showed a significantly higher prevalence of *H. pylori* infection than the general population, especially among those working at gastrointestinal units. Similar results were found in subjects involved in agricultural, forestry and fishery, as well as in sewage workers, miners, and workers at institutions for the intellectually disabled, although differences were less pronounced.

Conclusions Our results show an occupational risk of *H. pylori* infection supporting the role of oral–oral, fecal–oral, and zoonotic transmission. Studies comparing specific occupational groups with adequate comparators may contribute to better identify groups at higher risk of infection. The recognition of this infection as an occupational disease would result in early detection and treatment, as well as prevention and control of its transmission in workplaces.

Keywords *Helicobacter pylori* · Prevalence · Occupations · Systematic review

Introduction

Helicobacter pylori

Helicobacter pylori is a spiral, flagellated, Gram-negative bacterium (Marshall 2002) that is primarily found in the gastric mucosa of humans (Testerman and Morris 2014). Since its discovery in 1983 (Marshall and Warren 1984), it has been recognized as a major causal factor of histological changes leading to severe gastro-duodenal disease including gastric cancer (IARC Working Group on the Evaluation of Carcinogenic Risks to Humans 1994; Peleteiro et al. 2012). Globally, *H. pylori* infection was estimated to affect more than half of the adult population (Parkin 2006) and to have accounted for almost 80% of all gastric cancer cases in 2012 (89.0% in noncardia and 17.8% in cardia cancers) (Plummer et al. 2016).

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In the last few decades, the prevalence of *H. pylori* infection has been declining (Peleteiro et al. 2014), but there are significant differences in its prevalence both within and between countries (Lunet and Barros 2003). Improvements in socioeconomic and educational levels, namely, regarding sanitation and general living conditions, have been associated with this decrease, which also had an effect on gastric cancer trends, though with significant differences across geographical regions (Ferro et al. 2014).

Diagnosis of *H. pylori* infection

A number of methods are available for detection of *H. pylori*, which can be classified as invasive or non-invasive. Invasive methods use endoscopy to obtain gastric biopsy specimens for histopathology, microbiological culture or rapid urease tests that detect active infection (Megraud and Lehours 2007). The “gold standard” to detect active *H. pylori* infection still includes the need of a culture obtained from endoscopy, usually complemented with a biopsy urease test and/or histology (Brown 2000). Infection with *H. pylori* may also be identified by DNA amplification through polymerase chain reaction (PCR); however, this technique is not widely used as it is expensive when compared with culture, histology and rapid urease tests, and requires special laboratory conditions (Megraud and Lehours 2007).

H. pylori infection may also be assessed through non-invasive methods such as antibody-based tests, stool antigen test (SAT), and urea breath test (UBT). A variety of commercial and in-house diagnostic tests have been developed for the detection of *H. pylori*-specific antibodies in serum, saliva, and urine (Garza-Gonzalez et al. 2014). These tests are also called passive detection methods (Ricci et al. 2007) as they do not measure actual infection but the exposure to the bacteria at some time. Most of these diagnostic tests are serum-based; they are generally simple, reproducible and inexpensive, and can be done using stored samples; thus, they have been used widely in epidemiological studies (Brown 2000). Infection with *H. pylori* may also be diagnosed using SAT. The latter can detect either the bacterium or part of it (DNA or other molecules), and therefore, the infection may be assessed by culture, PCR or by the detection of antigens (Megraud and Lehours 2007), which may be particularly useful for epidemiological studies involving children (Versalovic 2003). Finally, UBT detects current infection and can be used as a screening test for *H. pylori* and to assess if an eradication treatment was successful (Brown 2000); however, the test might be unreliable when assessing patients who have undergone gastric surgery or using urease inhibiting drugs, due to great variations in pH that may lead to false negative results (Gisbert and Pajares 2004).

H. pylori transmission

Various pathways for the transmission of *H. pylori* infection have been described. Person-to-person transmission, including oral–oral, fecal–oral, and gastro–oral routes, appears to be the main mode (Allaker et al. 2002; Leung et al. 1999; Parsonnet et al. 1999). Evidence of the importance of close contacts for the acquisition of the infection comes from studies showing an increased prevalence of infection within institutionalized populations (Bohmer et al. 1997; Lambert et al. 1995) and clustering of *H. pylori* infection in families (Brenner et al. 2006; Cervantes et al. 2010; Rothenbacher and Brenner 2003), particularly between mothers and their children (Weyermann et al. 2006), and among siblings (Kivi and Tindberg 2006). The detection of *H. pylori* in body fluids, such as feces (Makristathis et al. 1998), gastric juice, urine, and saliva (Vaira and Vakil 2001), has also supported person-to-person transmission of infection (Brown 2000). Therefore, occupationally acquired infections may occur through close personal contact with infected patients or their body fluids (De Schryver et al. 2004). In fact, iatrogenic contamination following endoscopy is still the only proven mode of transmission (Brown 2000). *H. pylori* can be transmitted through upper gastrointestinal endoscopy (Langenberg et al. 1990; Shimada et al. 1996), nasogastric tubes (Graham et al. 1988), pH probes, and contaminated endoscopes (Roosendaal et al. 1993). The complex structure of medical apparatuses and difficulty in disinfecting them makes iatrogenic infection a potential risk factor for *H. pylori* infection in health-care workers (Axon 1991; Fantry et al. 1995). In addition, the impact of control measures would affect a large number of individuals, as the health-care sector represents 10% of all workers in the European Union (European Agency for Safety and Health at Work 2007).

Some studies have also suggested that infection may be acquired from the environment through water (Aziz et al. 2015), possibly due to fecal contamination, especially in areas of the world with deficiencies in water treatment (Brown 2000), or zoonotic transmission (Cover 1997; Megraud and Broutet 2000), through sheep (Dore et al. 1999; Goodman et al. 1996) or houseflies (Osato et al. 1998; Vaira and Holton 1998). Accordingly, contact with contaminated water or infected animals may pose an additional risk of infection in some occupational groups.

Aim

Identifying the main routes of transmission of *H. pylori* infection should be a priority in health research, as it may

allow the definition of preventive strategies for further reducing its morbidity and mortality burden. Since different occupations may represent distinct pathways for acquiring the infection, we aimed to describe the prevalence of *H. pylori* infection in specific occupational groups and to compare them with the general population, through a systematic review of published studies.

Methods

A study protocol was predefined by the authors and followed throughout the review.

Search strategy and selection of studies

PubMed® was searched, from inception to September 2015, using the search expression provided in the systematic review flowchart shown in Online Appendix A.

Each reference retrieved was screened independently by two reviewers (AF, ARC, and SM were involved in this step), following predefined criteria to determine eligibility for the systematic review. Original studies reporting the prevalence of *H. pylori* in occupational groups were eligible. The exclusion criteria were the following: (1) papers not written in English, Portuguese, Spanish, French, Italian, or Polish; (2) research not involving humans (e.g., *in vitro* or animal research); (3) non-eligible publication types (reviews, editorials, comments, guidelines, and case reports); (4) studies not evaluating or not providing the prevalence of *H. pylori* infection in specific occupational groups; (5) studies including only *H. pylori*-infected subjects (e.g., *H. pylori* eradication trials); (6) studies with data not related to *H. pylori* prevalence or addressing other outcomes (e.g., cost-effectiveness analyses); and (7) studies with a non-systematic assessment of *H. pylori* infection in biological samples (e.g., self-reported information, secondary data on infection status retrieved from laboratory databases).

When more than one report referred to the same study/sample, the one presenting the results with more detail (e.g., regarding the prevalence according to specific occupational groups), or providing data for the largest sample was considered, although any of the reports could be used to obtain information on the study characteristics.

The decisions taken independently by the reviewers were compared and discrepancies were resolved by consensus, or after discussion with another researcher (BP).

Data extraction

Two investigators (HK and BP) evaluated independently the selected studies to extract data regarding: year of publication; period of data collection [when this was not specified,

we assumed the publication year minus the median difference between the publication year and the mid-point of years of data collection in the studies for which that information was available (3.5 years)]; sampling procedures; sample characteristics; and methods used to determine *H. pylori* infection status.

Occupational groups recruited in each study were identified and classified according to the International Standard Classification of Occupations (International Labour Organization 2007).

The prevalence of *H. pylori* in different occupational groups was extracted, and the comparison between the occupational group and a reference group selected by criteria not related with occupation (e.g., general population, blood donors, volunteers), was retrieved whenever available. When studies did not provide data for a suitable reference group, estimates of *H. pylori* prevalence in the general population from the same country were obtained from studies included in a previous systematic review on the prevalence of *H. pylori* worldwide (Peleteiro et al. 2014), among those using the same type of assessment of infection status (past versus active infection) (Miftahussurur and Yamaoka 2016), conducted within 10 years of the corresponding study of occupational groups. If more than one eligible study presenting estimates of infection in age strata similar to the occupational group was available, the following criteria were applied consecutively until only one study was selected: (1) nationally representative or general population samples; (2) data collection closest to the one of the corresponding study of occupational groups; and (3) largest sample size.

Differences in the data extracted by the two investigators were discussed until consensus, and involving a third researcher (SM), whenever necessary.

Data analysis

Due to the heterogeneity of the occupational groups evaluated in the studies identified through systematic review and options for presenting results in each report, it was not possible to perform a quantitative synthesis of the main findings. Therefore, the differences in the prevalence of *H. pylori* between occupational groups and the general population were analyzed taking into account the direction and statistical significance of the differences observed when comparing each occupational group with a reference group (either from the same study or using an external comparator). Differences were classified as “Lower prevalence” or “Higher prevalence” in the occupational group than in the comparison group. In addition, these differences between the occupational and reference groups were classified as “statistically significant”, “not statistically significant” and “not available”, as reported in the papers.

Results were summarized in tables showing the prevalence of infection in occupational groups and the general population or comparison groups, and in harvest plots presenting the number of studies according to the statistical significance.

Results

Study characteristics

We identified 98 studies addressing the prevalence of *H. pylori* infection in 144 occupational groups (Online Appendix A and Online Appendix B). The reports were published between 1989 and 2015, and data collection took place between 1969 and 2013. Regarding the geographic coverage, 18 studies were conducted in the Americas, 33 in Asia, 43 in Europe and 4 in Oceania. The studies covered a wide age range, between 17 and 64 years, with sample sizes varying from 19 to 30,810 subjects. Most had a cross-sectional design ($n=75$), with the exception of eight case–control studies (data extracted referred to the control group) and 15 cohort studies (data extracted referred to the baseline evaluation). The assessment of *H. pylori* status was mainly done using serum samples ($n=80$), mostly measuring IgG antibodies, UBT was used in 14 studies, invasive techniques were used in two, whereas one study each evaluated urine and stool samples.

Tables 1, 2, 3, and 4 present the prevalence of *H. pylori* infection in the occupational groups and in the reference groups. The most frequently studied were health professionals, in a total of 49 studies, followed by studies on armed forces (14 studies). Agricultural, forestry, and fishery workers were evaluated in 8 studies, whereas 9 recruited factory workers and 14 assessed administrative staff. There were four studies each evaluating sewage workers and miners, and travelers. Finally, three studies each recruited teaching professionals, transportation workers, and workers at institutions for intellectually disabled people.

A total of 20 studies compared different occupational groups and in 29 studies the prevalence of infection in specific occupational groups was compared with control groups, selected in the same studies, not defined by occupation (Tables 1, 3). An additional 49 studies did not assess control groups selected based on criteria not including occupation, and therefore, an external comparator was selected for the present analysis (Tables 2, 4).

Health professionals

H. pylori prevalence in health professionals ranged from 3.8% in anesthesiologists to 82.4% in gastrointestinal endoscopists. Within gastroenterology staff,

gastroenterology nurses presented the lowest values (16.8%), whereas clinical students were the ones presenting the lowest prevalence (6.4%) and dentists the highest (70.0%) among dental staff. Figure 1 presents the differences in the prevalence of *H. pylori* infection between health professionals and the reference group. Overall, a total of 13 studies showed a statistically significant higher prevalence of *H. pylori* among health professionals when compared with a reference group, particularly among gastroenterology staff ($n=7$). However, eight studies recruiting health professionals from gastroenterology departments showed no statistically significant differences or no comparison, despite presenting a higher prevalence of *H. pylori* infection than the comparison groups. Nevertheless, there was a considerable amount of studies showing a lower prevalence of *H. pylori* infection when compared with the reference group, although this difference was only statistically significant in four studies. In addition, a high number of studies did not provide an adequate comparison between the occupational and the reference group ($n=31$), especially among other health professionals ($n=22$). For dental staff, most studies showed a lower prevalence of infection among these health professionals than controls ($n=10$); however, this result was not statistically significant ($n=5$) or not adequately compared ($n=5$).

Other occupational groups

In other occupations, most studies show a lower prevalence of *H. pylori* infection in the occupational groups, but most of them did not present a suitable reference group (Fig. 2).

For administrative staff, *H. pylori* prevalence ranged from 6.1% in abattoir clerks and painters to 72.0% in faculty staff. Most studies showed a lower prevalence of *H. pylori* infection when compared to an external comparator. Among agricultural, forestry and fishery workers, *H. pylori* prevalence was the lowest in pig carcass workers (14.8%) and the highest in farmers (77.6%). Three studies showed a higher and seven studies showed a lower prevalence of *H. pylori* infection for agricultural, forestry, and fishery occupations compared with the reference group, with only one study describing a statistically significant difference. In the armed forces, *H. pylori* prevalence was lower among navy submarine crews (10.4%) and higher in low-rank officials (82.5%). Almost all studies evaluating subjects from the armed forces showed a lower prevalence of *H. pylori* infection when compared with the general population. Even though one study used an in study comparator, no formal comparison was reported. Within the group of factory workers, daytime workers who had never done shift work were the ones presenting the lowest values of *H. pylori* prevalence (13.4%), while skilled and unskilled professional workers presented the highest value (69.7%). Among factory workers, a slightly higher number of studies showed a lower

Table 1 Prevalence of *H. pylori* infection in health professionals and in study comparators, and *p* value and/or risk of infection

Authors Year of publication	Prevalence of <i>H. pylori</i> infection (%)		<i>p</i> Value and/or risk of infection (RR/OR, 95%CI) ^a
	Occupational group	In study comparator	
(Mitchell et al. 1989)	Gastroenterologists = 51.5 Gastroenterology nurses = 19.1 General practitioners = 28.6	Blood donors = 21.5	<i>p</i> < 0.01 <i>p</i> = NS <i>p</i> = NS
(Reiff et al. 1989)	Endoscopy staff = 55.5 Dental staff = 27.6	Controls = 49.3	<i>p</i> = NS <i>p</i> = NS
(Royo et al. 1991)	Gastroenterology medical and nursing staff = 30.4	Control group 1 = 38.5	<i>p</i> = 0.620
(Wilhoite et al. 1993)	Nurses = 39.2 Resident physicians = 23.7 Nursing students = 13.6 Medical students = 19.6	Blood donors = 25.8	<i>p</i> < 0.01 <i>p</i> = NS <i>p</i> = NS <i>p</i> = NS
(Chong et al. 1994)	Gastroenterologists/endoscopy nurses = 53.3 Gastroendoscopists = 52.2 Endoscopy nurses = 54.5	Healthy blood donors = 14.1	<i>p</i> < 0.0001
(Lin et al. 1994)	Gastroenterologists = 69.2 General internists = 40.0 Gastroenterology nurses = 16.8 General nurses = 19.0	Controls = 36.9 Controls = 37.5 Controls = 27.8 Controls = 24.2	OR = 5.00 (2.00–12.00) OR = 1.00 (0.30–3.10) OR = 0.58 (0.28–1.22) OR = 0.90 (0.33–2.47)
(Pristautz et al. 1994)	Endoscopists = 47.7	Controls = 47.0	<i>p</i> = NS
(Banatvala et al. 1995)	Dental practitioners = 15.7 Clinical dental students = 6.4 Pre-clinical dental students = 9.7	Controls = 25.7 Controls = 6.4 Controls = 9.7	OR = 0.6 (0.2–1.5) OR = 1.0 (0.3–3.7) OR = 1.0 (0.2–5.4)
(De Vecchi et al. 1995)	Nursing staff = 26.3	Blood donors = 58.0	<i>p</i> = NS
(Bergenzaun et al. 1996)	Medical students = 10.0	Health-care population = 11.8	–
(Liu et al. 1996)	Medical staff = 70.0 GI endoscopists = 82.4 Endoscopy nurses = 77.7 GI doctors = 74.6 GI nurses = 74.2 Internists = 66.4 General nurses = 65.8	Control group = 44.6	OR = 2.90 (2.38–3.45)
(Su et al. 1996)	Endoscopists = 80.0	Controls = 51.6	<i>p</i> = 0.007
(Braden et al. 1997)	Medical staff = 37.1 Endoscopy staff = 37.8 Non-endoscopy medical staff = 35.9 Physicians = 37.4 Endoscopy physicians = 38.0 Non-endoscopy physicians = 36.4 Nurses = 35.3 Endoscopy nurses = 36.7 Non-endoscopy nurses = 31.8	Controls = 27.1	OR = 1.59 (1.25–2.01) OR = 1.63 (1.27–2.09) OR = 1.51 (1.14–1.98) OR = 1.61 (1.26–2.04) OR = 1.64 (1.28–2.12) OR = 1.54 (1.17–2.04) – OR = 1.80 (1.24–2.63) OR = 1.64 (0.96–1.64)
(Abbas et al. 1998)	Endoscopy personnel = 78.8 Endoscopy doctors = 68.4 Endoscopy nurses and assistants = 92.9	Non-medical subjects = 57.6	<i>p</i> = 0.06 <i>p</i> = 0.43 <i>p</i> = 0.019
(Lin et al. 1998)	Dentists = 22.8 Dental nurses = 17.5 5th year dental students = 18.2 1st year dental students = 16.7	Controls = 33.2 Controls = 30.6 Controls = 21.4	OR = 0.58 (0.30–1.10) OR = 1.00 (0.40–2.90) OR = 1.22 (0.25–5.90) OR = 1.36 (0.27–6.80)
(Nishikawa et al. 1998)	Endoscopy personnel = 29.8 Gastrointestinal endoscopists = 30.4 Endoscopy nurses = 27.6	Controls = 24.8	<i>p</i> = 0.406
(Shelley and Haddadin 1998)	Anesthesiologists = 3.8	Control groups = 19.7 (Chong et al. 1994; Wilhoite et al. 1993)	<i>p</i> < 0.01

Table 1 (continued)

Authors Year of publication	Prevalence of <i>H. pylori</i> infection (%)		<i>p</i> Value and/or risk of infection (RR/OR, 95%CI) ^a
	Occupational group	In study comparator	
(Mones et al. 1999)	Medical staff = 52.7 Gastroenterologists = 53.3 Endoscopists = 53.3 Non-endoscopists = 53.5 Non-gastroenterologists = 50.0	Healthy controls = 51.9	<i>p</i> > 0.05
(Robertson et al. 1999)	ICU nurses = 40.0	Blood donors = 19.0	<i>p</i> < 0.001
(Hildebrand et al. 2000)	Gastroenterologists = 39.1	Healthy controls = 38.1	–
(Martinez et al. 2000)	Emergency physicians = 6.8	Controls = 15.9	–
(Pronai et al. 2000)	Gastroenterologists = 35.2 With endoscopy practice = 29.6 Without endoscopy practice = 38.8 General practitioners = 36.3	Healthy controls = 54.4	<i>p</i> < 0.05 <i>p</i> < 0.05 <i>p</i> < 0.05
(Honda et al. 2001)	Dentists = 70.0	Control group = 38.3	OR = 3.80 (1.76–8.02)
(Upile et al. 2002)	Surgical personnel = 50.0	Control group = 25.7 (Banatvala et al. 1995)	<i>p</i> < 0.0001
(Kim et al. 2013)	Nurses = 29.8 Doctors = 34.5	Nonhospital controls = 52.9	–

RR relative risk, OR odds ratio, 95% CI 95% confidence interval, NS not significant, GI gastrointestinal, ICU intensive care unit

^aReference category is “in study comparator” and “occupational group” is the risk group, unless otherwise specified

prevalence of *H. pylori* infection when compared to an external control. Sewage workers presented a lower prevalence of *H. pylori* infection (16.7%) than miners (48.1%). In teaching professionals, transportation workers and travelers, *H. pylori* prevalence ranged from 24.1 to 45.1% in the first group, from 37.0 to 77.4% in the second, and from 17.0 to 91.7% in the third group. Among studies recruiting sewage workers and miners, three studies showed a higher and another, a lower prevalence of infection, with no adequate comparators. Similar results were found for travelers, with two studies showing a higher prevalence and two the opposite when compared to groups of the general population. Regarding transportation workers and teaching professionals, all studies identified presented a lower prevalence of *H. pylori* infection with no formal comparison. For workers at institutions for the intellectually disabled, *H. pylori* prevalence ranged between 14.1 and 47.1%. The three studies identified showed a higher prevalence of *H. pylori* infection among these workers, although the difference was only statistically significant in one.

Discussion

Overall, most studies addressing the prevalence of *H. pylori* infection in specific occupational groups did not provide data from control groups not defined by their occupation. Nevertheless, gastrointestinal staff showed a statistically significant higher prevalence of *H. pylori* infection than their

controls. Similar results were found for a high proportion of other health professionals and workers at institutions for the intellectually disabled. However, in the other occupational groups, differences in the prevalence of *H. pylori* when compared to the corresponding reference groups were less pronounced.

Health professionals

Among occupational groups with a high risk of infection, health professionals are a particularly susceptible group, as was observed during the outbreaks of severe acute respiratory syndrome (SARS) (Moore et al. 2005), Ebola (Kilmarx et al. 2014) and tuberculosis (Nasreen et al. 2016), particularly in professionals that have direct contact with body fluids or personal contact with patients, such as nurses, medical doctors and their respective assistants. Nevertheless, most of the hazardous exposures are preventable if professionals follow the international guidelines for infection control, regarding contact with infected patients, management of surgical and examination material and blood and body fluids, sanitation measures and good hygiene practices, such as hand washing with disinfectants and soap, use of gloves and personal protective equipment (Noone et al. 2006). These practices are of greater importance when there is some evidence of person-to-person transmission (Noone et al. 2006), even if the only proven transmission mode for *H. pylori* infection is iatrogenic contamination following endoscopy (Brown 2000). Therefore, personnel working directly with

Table 2 Prevalence of *H. pylori* infection in health professionals and external comparators

Authors Year of publication	Prevalence of <i>H. pylori</i> infection (%)	
	Occupational group	External comparator
(Malaty et al. 1992)	Dentists = 17.0 Dental hygienists = 18.0 Dental assistants = 34.0 Dental students = 25.0	32.7 ^a (Everhart et al. 2000)
(Goh et al. 1996)	Endoscopy personnel = 32.9 Endoscopists = 41.2 Endoscopy nurse assistants = 27.1 Non-endoscopy medical personnel = 12.0	35.9 (Goh and Parasakthi 2001)
(Potts et al. 1997)	Gastroenterologists = 50.0 Respiratory physicians = 10.0	27.7 (Moayyedi et al. 2000)
(Potasman and Yitzhak 1998)	Medical students = 39.5	46.5 (Gdalevich et al. 2000)
(Gasbarrini et al. 2001)	Health-care workers = 40.0	67.9 (Bazzoli et al. 2001)
(Sachdev et al. 2001)	Conference delegates = 57.3	54.0 (Nisha et al. 2016)
(van der Voort et al. 2001)	ICU nurses with SDD unit = 18.0 Other ICU nurses = 28.8 Health-care workers = 22.9	35.4 (Wagtman et al. 1997)
(Angtuaco et al. 2002)	Gastrointestinal endoscopy personnel = 24.3	32.0 ^a (Cardenas and Graham 2005)
(Matsuda et al. 2002)	Dentists = 42.3	75.0 (Sasazuki et al. 2006)
(Triantafyllidis et al. 2002)	Nurses 1994 = 48.6 1999 = 61.0 Medical staff 1994 = 41.9 1999 = 51.6 Paramedical staff 1994 = 30.0 1999 = 46.7	49.1 (Triantafyllidis et al. 2003)
(Hoffmann et al. 2003)	Medical students = 9.5	33.1 (Bode et al. 2001)
(Melo et al. 2003)	Basic level medical students = 23.4 Professional level medical students = 32.0 Medical residents = 38.6	84.7 (Souto et al. 1998)
(Birkenfeld et al. 2004)	Gastroenterology units staff members = 72.6 Primary care clinic staff members = 71.4	–
(Mastromarino et al. 2005)	Gastrointestinal endoscopy personnel = 37.0 General medical staff = 35.2	67.9 (Bazzoli et al. 2001)
(Garza Yado Mde et al. 2006)	Resident physicians = 24.6	75.4 (Ornelas et al. 2007)
(Noone et al. 2006)	Gastroscopy nurses = 32.4 Non-gastroscopy nurses = 33.1	66.0 (Woodward et al. 2000)
(Almadi et al. 2007)	Medical students = 35.0	0.0 (Al-Refai et al. 2002)
(Campuzano-Maya et al. 2007)	Doctors = 77.2	75.4 (Bravo et al. 2002)
(Velasco Elizalde et al. 2007)	Endoscopy workers = 39.5 Non-gastroenterology workers = 7.9	–
(Monno et al. 2008)	Health-care workers = 77.9	75.0 (Resuli et al. 1999)
(Eshraghian et al. 2009)	Health-care workers = 60.6	69.0 (Nouraie et al. 2009)
(Loster et al. 2009)	Dentists = 70.0	84.2 (Laszewicz et al. 2014)
(Castro-Fernandez et al. 2012)	Medicine/nursing students = 32.7	55.7 (Lopez-Saez et al. 2010)
(Takashima et al. 2012)	Medical students = 12.4	24.4 (Fujimoto et al. 2007)

ICU intensive care unit, SDD selective decontamination of the digestive tract

^aAge-standardized *H. pylori* prevalence

Table 3 Prevalence of *H. pylori* infection in occupational groups and in study comparators, and *p* value and/or risk of infection

Authors Year of publication	Prevalence of <i>H. pylori</i> infection (%)		<i>p</i> Value and/or risk of infection (RR/OR, 95%CI) ^a
	Occupational group	In study comparator	
Administrative staff			
Kim et al. (2013)	Office, administration, technical and pharmacy workers = 30.7	Nonhospital controls = 52.9	–
Agricultural, forestry and fishery workers			
Rocha et al. (1992)	Abattoir workers = 65.6 Abattoir workers with no direct contact with the animals = 65.0	Blood donors = 61.8	<i>p</i> = 0.56
Ullah et al. (2010)	Fish handlers = 77.3	Non-fish handlers = 37.5	<i>p</i> < 0.001
Armed forces			
Pateraki et al. (1990)	Recruits = 67.0	Blood donors = 70.0	–
Workers at institutions for intellectually disabled people			
Bohmer et al. (1997)	Employees at in institutes for the intellectually disabled = 27.2 Group 1 = 31.6 Groups 2/3 = 14.1	Control group = 25.0 (Loffeld et al. 1990)	–

RR relative risk, OR odds ratio, 95% CI 95% confidence interval

^aReference category is “in study comparator” and “occupational group” is the risk group, unless otherwise specified

endoscopic material and with infected patients represent a potential risk group (De Schryver et al. 2004).

The risk of *H. pylori* infection among gastroenterologists and their assistants was previously evaluated (Peters et al. 2011) and an increased risk, as observed from our results, was described. However, the statistical significance of the increased risk shown before depended on the type of controls used in each study, with only comparisons with non-medical controls leading to statistically significant results. The main conclusion of Peters et al. was that further studies involving suitable control groups should be conducted for a valid assessment of occupational exposure risks. Our results support those of the previous review and add to that list of studies those recruiting dental staff as well as other health professionals. The latter have been widely used as controls for gastroenterology staff, contributing to the absence of statistically significant differences in the prevalence of *H. pylori* between the two groups.

To have an extensive overview of *H. pylori* infection in occupational groups, we have included in our systematic review reports recruiting occupational groups as controls. In that case, we selected an external comparator included in a systematic review on the prevalence of *H. pylori* worldwide (Peleteiro et al. 2014), from which the present systematic review was also derived. However, this option has compromised the formal comparison between occupational groups and the general population. Nevertheless, even when comparators were recruited in the same study, a statistical comparison was not always available, resulting in reporting bias. Furthermore, studies recruiting other occupational groups or unsuitable controls, such as symptomatic patients, as the reference group to be compared with the health professionals had to be excluded if no other adequate comparator could

be found. This strategy, however, has minimized publication bias as these studies would have been excluded when performing a quantitative synthesis of the results.

Other occupations

Most studies focusing on subjects within the armed forces show a lower prevalence of infection than the general population of the same country. These results may be explained by the fact that most of these studies evaluated recruits at the beginning of their military training. Therefore, this occupational group is mainly composed of young males that are typically healthier than the general population (Carreira et al. 2012) and who have not yet been exposed to the expected risk factors in a military environment, such as poor sanitary and hygienic conditions during missions abroad. When that occurs, some studies show a higher prevalence of *H. pylori* infection when compared with the general population (Shinchi et al. 1997; Taylor et al. 1997).

Other occupational groups that were expected not to be at an increased risk of acquiring *H. pylori* infection were administrative staff, and transportation and factory workers. The lower prevalence found in our systematic review confirms this assumption. Conversely, the other occupational groups were expected to have an increased risk of *H. pylori* infection. Overall, our results support this hypothesis for sewage workers and miners, and for workers at institutions for the intellectually disabled, but not for those involved in agriculture, forestry and fishery, teaching professionals, and travelers. This may be because the latter is a more heterogeneous group, with missionaries moving from high- to low-income countries, whereas migrant workers have an opposite pattern of migration (De Schryver et al. 2004). Regarding

Table 4 Prevalence of *H. pylori* infection in occupational groups and external comparators

Authors Year of publication	Prevalence of <i>H. pylori</i> infection (%)	
	Occupational group	External comparator
<i>Administrative staff</i>		
Husson et al. (1991)	Abattoir clerks and painters = 6.1	32.9 (Bergey et al. 2005)
Parsonnet et al. (1992)	Epidemiologists = 19.9	32.7 ^a (Everhart et al. 2000)
Webb et al. (1994)	Non-manual occupation = 21.5	13.4 (Vyse et al. 2002)
Friis et al. (1996)	Laborers = 29.0	18.0 (Sorberg et al. 2003)
Goh et al. (1996)	Sales personnel from pharmaceutical companies = 10.7	35.9 (Goh and Parasakthi 2001)
Kikuchi et al. (1998)	Public service workers = 30.5	75.0 (Sasazuki et al. 2006)
(Chiloiro et al. 2001)	Administrative staff = 50.0	45.0 (Palli et al. 1993)
Matsuda et al. (2002)	College employees = 40.0	75.0 (Sasazuki et al. 2006)
Triantafyllidis et al. (2002)	Administrative and technical staff 1994 = 44.1 1999 = 55.1	49.1 (Triantafyllidis et al. 2003)
Chimienti et al. (2003)	Faculty staff = 72.0	57.7 (Zagari et al. 2010)
Mastromarino et al. (2005)	Health-care personnel with no patient contact = 19.2	67.9 (Bazzoli et al. 2001)
De Schryver et al. (2008)	Administrative workers = 29.2	14.6 (De Hert et al. 1997)
Satoh et al. (2010)	Local government employees = 45.4	75.0 (Sasazuki et al. 2006)
<i>Agricultural, forestry and fishery workers</i>		
Husson et al. (1991)	Poultry internal organ workers = 24.3 Poultry carcass workers = 19.5 Pig carcass workers = 14.8 Polyvalent slaughterers = 14.8	32.9 (Bergey et al. 2005)
Laurila et al. (1999)	Reindeer herders = 65.1	59.0 (Salomaa-Rasanen et al. 2006)
Babazono et al. (2004)	Agricultural workers = 52.7	75.0 (Sasazuki et al. 2006)
Bener et al. (2006)	Farmers IgG = 77.6 IgA = 55.3	–
Liu et al. (2007)	Farmers and herders = 35.4	56.4 (Chen et al. 2007)
Hu et al. (2013)	Maritime workers = 44.9	56.4 (Chen et al. 2007)
<i>Armed forces</i>		
Hammermeister et al. (1992)	German submarine crews = 31.7 German air force = 16.2 French infantry regular officers = 13.7 French infantry recruits = 18.5	40.7 (Kuepper-Nybelen et al. 2005) 32.9 (Bergey et al. 2005)
Basso et al. (1994)	Soldiers and officers = 31.5 Private, trooper, signalman, gunner and corporal = 30.0 Sergeant, company, sergeant major = 53.8 Lieutenant, captain, commandant = 14.3	43.0 (Buckley et al. 1998)
Smoak et al. (1994)	Recruits = 26.3	32.7 ^a (Everhart et al. 2000)
Hyams et al. (1995)	Recruits = 24.0 Shipboard personnel = 26.0 Desert Storm troops = 22.0	32.7 ^a (Everhart et al. 2000)
Shinchi et al. (1997)	Self-defense officials = 79.3 According to rank Low = 82.5 Middle = 75.2 High = 75.0	70.0 (Kikuchi et al. 2005)
Stroffolini et al. (1998)	Air force students = 17.5	45.0 (Palli et al. 1993)
Taylor et al. (1997)	Soldiers = 37.2	32.7 ^a (Everhart et al. 2000)
Biselli et al. (1999)	Air force students = 17.2	45.0 (Palli et al. 1993)
Kyriazanos et al. (2001)	Navy recruits = 19.0	49.1 (Triantafyllidis et al. 2003)
Kyriazanos et al. (2002)	Navy recruits = 27.2	49.1 (Triantafyllidis et al. 2003)

Table 4 (continued)

Authors Year of publication	Prevalence of <i>H. pylori</i> infection (%)	
	Occupational group	External comparator
Furesz et al. (2004)	First screening = 23.0 Second screening = 33.0	59.0 (Kalabay et al. 2002)
Jackman et al. (2006)	Navy submarine crews = 10.4	32.0 ^a (Cardenas and Graham 2005)
Monno et al. (2008)	Conscripts = 53.8	75.0 (Resuli et al. 1999)
<i>Factory workers</i>		
Webb et al. (1994)	Manual occupation = 39.9	13.4 (Vyse et al. 2002)
Furuta et al. (1997)	Factory workers = 40.0	70.0 (Kikuchi et al. 2005)
Yang et al. (1999)	Employees working in a manufacturing plant = 39.6	75.0 (Sasazuki et al. 2006)
Ogihara et al. (2000)	Workers in textile companies = 48.3	70.0 (Kikuchi et al. 2005)
Bener et al. (2006)	Skilled and unskilled professional workers IgG = 69.7 IgA = 46.7	–
Pfefferle and Kramer (2008)	Employees = 42.8	40.7 (Kuepper-Nybelen et al. 2005)
Van Hooste et al. (2010)	Laboratory personnel, operators and maintenance workers = 13.6	14.6 (De Hert et al. 1997)
van Mark et al. (2010)	All workers = 27.6 Shift workers = 34.6 Shift workers with night shifts = 34.7 Shift workers without night shifts = 34.2 Daytime workers = 16.0 Daytime workers, never shift work = 13.4 Daytime workers, former shift workers = 21.6	40.7 (Kuepper-Nybelen et al. 2005)
Han et al. (2016)	Retirees = 49.6	46.8 (Cheng et al. 2009)
<i>Sewage workers and miners</i>		
Friis et al. (1996)	Sewage workers = 28.5	18.0 (Sorberg et al. 2003)
Jeggli et al. (2004)	Sewage exposed workers = 33.6	57.1 (Ammann et al. 2000)
Van Hooste et al. (2010)	Sewage workers = 16.7	14.6 (De Hert et al. 1997)
Siva et al. (2013)	Miners = 48.1	13.4 (Vyse et al. 2002)
<i>Teaching professionals</i>		
Lin et al. v2000)	Teachers = 30.8	71.4 (Wang et al. 2008)
Lin et al. (2007)	Teachers = 45.1	71.4 (Wang et al. 2008)
Lynn et al. (2007)	Educators = 24.1	32.0 ^a (Cardenas and Graham 2005)
<i>Transportation workers</i>		
Vare et al. (2001)	Railway staff = 37.0	59.0 ^a (Salomaa-Rasanen et al. 2006)
Kim et al. (2005)	Subway workers = 77.4	80.8 (Shin et al. 2005)
Saijo et al. (2007)	Transit company employees = 57.8	75.0 (Sasazuki et al. 2006)
<i>Travelers</i>		
Katellaris et al. v1992)	Monks = 77.2	61.5 (Wang et al. 2002)
Ahmad et al. (1997)	Migrant workers = 91.7	73.6 (Kibria et al. 2015)
Becker et al. (1999)	Missionaries = 17.0	32.7 ^a (Everhart et al. 2000)
(Xia et al. 2012)	Migrant workers = 41.0	46.8 (Cheng et al. 2009)
<i>Workers at institutions for intellectually disabled people</i>		
Angtuaco et al. (2002)	HDC workers = 47.1	32.0 ^a (Cardenas and Graham 2005)
De Schryver et al. (2008)	Workers in institutions for people with intellectual disability = 40.6	14.6 (De Hert et al. 1997)

IgG immunoglobulin G, IgA immunoglobulin A, HDC human development center

^aAge-standardized *H. pylori* prevalence

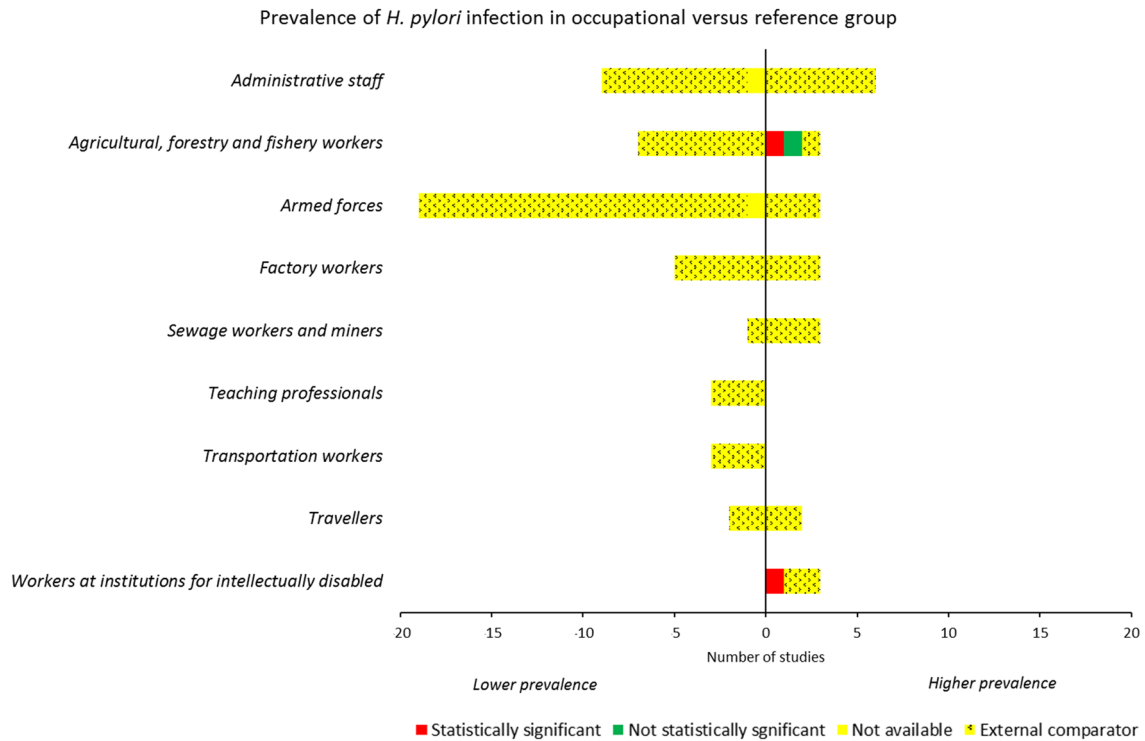


Fig. 2 Differences in the prevalence of *Helicobacter pylori* between the occupational group and the reference group—farmers (agricultural, forestry, and fishery workers), and skilled and unskilled pro-

fessional workers (Factory workers) from Bener et al. (2006) are not shown as no external comparator was available

prevalence in specific occupational groups, because they recruited a mixture of different occupations or mixed occupational groups with other population types. In addition, comparisons made between the occupational and the reference group using prevalences obtained from papers other than the ones describing occupational groups may have influenced our results. Although this selection was based on a previous detailed assessment of each eligible study to be selected as possible reference (carried out independently of the specific objective of this systematic review), it should be noted that the studies are not entirely concordant regarding the period of data collection; therefore, comparisons should be made cautiously as the prevalence of infection has decreased over time, at an international level (Peleteiro et al. 2014). To minimize potential bias, we attempted to choose nationally representative or general population and age-specific estimates with a similar period of data collection. However, some studies presented blood donors as surrogate for the general population which may have introduced bias to the comparisons performed, since blood donors are generally considered healthier than the general population (Golding et al. 2013).

Even when adequate control groups and formal comparisons were presented in the studies, most of the results were not adjusted for covariates, resulting in lack of control

for potential confounding, namely, by socioeconomic status (Eusebi et al. 2014). The evidence generated by the present systematic review is naturally limited by the quality and heterogeneity of the original reports regarding the methods used and the presentation of results. In addition, temporality between exposure and outcome, i.e., risk occupation and infection by *H. pylori*, cannot be assured, since most included studies had a cross-sectional design. Taken together, these factors preclude the establishment of a causal relation.

Conclusion

Although there is some debate about the occupational risk of acquiring *H. pylori* infection, we showed that health professionals are at higher risk of acquiring the infection, especially gastroenterology staff, favoring the iatrogenic route of transmission. However, other occupational groups, such as those working at institutions for the intellectually disabled, are also at higher risk of infection, supporting other routes of transmission, namely, person-to-person.

Further studies recruiting specific occupational groups should be conducted using adequate comparators and study designs to ascertain the main sources of *H. pylori*

infection as well as to identify groups at higher risk of infection and ways to prevent it.

Nevertheless, our results suggest an evaluation of whether *H. pylori* infection can be included as an occupational disease, at least for some risk groups such as health professionals. This infection is not yet recognized by the several lists of occupational diseases that exist (European Commission 2003; International Labour Organization 2010), its inclusion would result in an early detection and treatment, as well as the prevention and control of its transmission at these workplaces.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

- Abbas Z, Jafri W, Khan AH, Shah MA (1998) Prevalence of *Helicobacter pylori* antibodies in endoscopy personnel and non-medical volunteers of Karachi. *J Pak Med Assoc* 48:201–203
- Ahmad MM et al (1997) Prevalence of *Helicobacter pylori* in asymptomatic population—a pilot serological study in Bangladesh. *J Epidemiol* 7:251–254
- Allaker RP, Young KA, Hardie JM, Domizio P, Meadows NJ (2002) Prevalence of *Helicobacter pylori* at oral and gastrointestinal sites in children: evidence for possible oral-to-oral transmission. *J Med Microbiol* 51:312–317. <https://doi.org/10.1099/0022-1317-51-4-312>
- Almadi MA, Aljebreen AM, Tounesi FA, Abdo AA (2007) *Helicobacter pylori* prevalence among medical students in a high endemic area. *Saudi Med J* 28:896–898
- Al-Refai AN, Fathalla SE, Nagamani R, Al-Momen S (2002) Incidence of *Helicobacter pylori* in dental plaque of Saudi gastritis patients. *J Family Community Med* 9:27–36
- Ammann P, Marschall S, Kraus M, Schmid L, Angehrn W, Krapf R, Rickli H (2000) Characteristics and prognosis of myocardial infarction in patients with normal coronary arteries. *Chest* 117:333–338
- Anguaco TL, Sharma VK, Corder FA, Raufman JP, Howden CW (2002) Seroprevalence of *H. pylori* infection and symptoms of upper gastrointestinal tract disease in two groups of health-care workers. *Dig Dis Sci* 47:292–297
- Axon AT (1991) Disinfection of endoscopic equipment. *Baillieres Clin Gastroenterol* 5:61–77
- Aziz RK, Khalifa MM, Sharaf RR (2015) Contaminated water as a source of *Helicobacter pylori* infection: a review. *J Adv Res* 6:539–547. <https://doi.org/10.1016/j.jare.2013.07.007>
- Babazono A, Miyazaki M, Une H, Yamamoto E, Tsuda T, Mino Y, Hillman AL (2004) Does seropositivity for *Helicobacter pylori* antibodies increase outpatient costs for gastric and duodenal ulcer or inflammation? *Pharmacoeconomics* 22:975–983
- Banatvala N et al (1995) *Helicobacter pylori* infection in dentists—a case-control study. *Scand J Infect Dis* 27:149–151
- Basso L, Beattie S, Lawlor S, Clune J, O’Morain C (1994) A descriptive follow-up study on *Helicobacter pylori* infection before and after exposition to a war area. *Eur J Epidemiol* 10:109–111
- Bastos J, Carreira H, La Vecchia C, Lunet N (2013) Childcare attendance and *Helicobacter pylori* infection: systematic review and meta-analysis. *Eur J Cancer Prev* 22:311–319. <https://doi.org/10.1097/CEJ.0b013e32835b69aa>
- Bazzoli F et al (2001) The Loiano-Monghidoro population-based study of *Helicobacter pylori* infection: prevalence by 13C-urea breath test and associated factors. *Aliment Pharmacol Ther* 15:1001–1007
- Becker SI, Smalligan RD, Frame JD, Kleanthous H, Tibbitts TJ, Monath TP, Hyams KC (1999) Risk of *Helicobacter pylori* infection among long-term residents in developing countries. *Am J Trop Med Hyg* 60:267–270
- Bener A, Adeyemi EO, Almehti AM, Ameen A, Beshwari M, Benedict S, Derballa MF (2006) *Helicobacter pylori* profile in asymptomatic farmers and non-farmers. *Int J Environ Health Res* 16:449–454. <https://doi.org/10.1080/09603120601093428>
- Bergenzaun P, Kristinsson KG, Thjodleifsson B, Sigvaldadottir E, Molstad S, Held M, Wadstrom T (1996) Seroprevalence of *Helicobacter pylori* in south Sweden and Iceland. *Scand J Gastroenterol* 31:1157–1161
- Bergey B, Gallian P, Stuart M, Bolla JM, De Micco P, Megraud F (2005) Prevalence of *Helicobacter pylori* infection in Corsica. *Gastroenterol Clin Biol* 29:611–612
- Birkenfeld S, Keter D, Dikman R, Shevah O, Shirin H, Niv Y (2004) Prevalence of *Helicobacter pylori* infection in health-care personnel of primary care and gastroenterology clinics. *J Clin Gastroenterol* 38:19–23
- Biselli R, Fortini M, Matricardi PM, Stroffolini T, D’Amelio R (1999) Incidence of *Helicobacter pylori* infection in a cohort of Italian military students. *Infection* 27:187–191
- Bode G, Hoffmeister A, Koenig W, Brenner H, Rothenbacher D (2001) Characteristics of differences in *Helicobacter pylori* serology and 13C-urea breath-testing in an asymptomatic sample of blood donors. *Scand J Clin Lab Invest* 61:603–608
- Bohmer CJ, Klinkenberg-Knol EC, Kuipers EJ, Niezen-de Boer MC, Schreuder H, Schuckink-Kool F, Meuwissen SG (1997) The prevalence of *Helicobacter pylori* infection among inhabitants and healthy employees of institutes for the intellectually disabled. *Am J Gastroenterol* 92:1000–1004
- Braden B, Duan LP, Caspary WF, Lembcke B (1997) Endoscopy is not a risk factor for *Helicobacter pylori* infection—but medical practice is. *Gastrointest Endosc* 46:305–310
- Bravo LE, van Doorn LJ, Realpe JL, Correa P (2002) Virulence-associated genotypes of *Helicobacter pylori*: do they explain the African enigma? *Am J Gastroenterol* 97:2839–2842. <https://doi.org/10.1111/j.1572-0241.2002.07031.x>
- Brenner H, Weyermann M, Rothenbacher D (2006) Clustering of *Helicobacter pylori* infection in couples: differences between high- and low-prevalence population groups. *Ann Epidemiol* 16:516–520. <https://doi.org/10.1016/j.annepidem.2005.09.009>
- Brown LM (2000) *Helicobacter pylori*: epidemiology and routes of transmission. *Epidemiol Rev* 22:283–297
- Buckley MJ, O’Shea J, Grace A, English L, Keane C, Hourihan D, O’Morain CA (1998) A community-based study of the

- epidemiology of *Helicobacter pylori* infection and associated asymptomatic gastroduodenal pathology. *Eur J Gastroenterol Hepatol* 10:375–379
- Campuzano-Maya G, Hoyos-Castano D, Calvo-Betancur VD, Suarez-Ramirez OA, Lizcano-Cardona D, Rojas-Arbelaes CA (2007) Prevalence of *Helicobacter pylori* infection in physicians in Medellin, Colombia. *Acta Gastroenterol Latinoam* 37:99–103
- Cardenas VM, Graham DY (2005) Smoking and *Helicobacter pylori* infection in a sample of U.S. adults. *Epidemiology* 16:586–590
- Carreira H, Pereira M, Azevedo A, Lunet N (2012) Effect of the type of population on estimates of mean body mass index and prevalence of overweight and obesity: a systematic review of studies of Portuguese adults. *Ann Hum Biol* 39:223–238. <https://doi.org/10.3109/03014460.2012.681804>
- Castro-Fernandez M, Vargas-Romero J, Hoyas E, Lamas E, Millan R, Romero-Gomez M (2012) Relationship between *H. pylori* and hepatitis A virus infection. *Rev Esp Enferm Dig* 104:220–221
- Cervantes DT, Fischbach LA, Goodman KJ, Phillips CV, Chen S, Broussard CS (2010) Exposure to *Helicobacter pylori*-positive siblings and persistence of *Helicobacter pylori* infection in early childhood. *J Pediatr Gastroenterol Nutr* 50:481–485. <https://doi.org/10.1097/MPG.0b013e3181bab2ee>
- Chen J, Bu XL, Wang QY, Hu PJ, Chen MH (2007) Decreasing seroprevalence of *Helicobacter pylori* infection during 1993–2003 in Guangzhou, southern China. *Helicobacter* 12:164–169. <https://doi.org/10.1111/j.1523-5378.2007.00487.x>
- Cheng H et al (2009) Prevalence of *Helicobacter pylori* infection and identification of risk factors in rural and urban Beijing, China. *Helicobacter* 14:128–133. <https://doi.org/10.1111/j.1523-5378.2009.00668.x>
- Chiloiro M, Russo F, Riezzo G, Leoci C, Clemente C, Messa C, Di Leo A (2001) Effect of *Helicobacter pylori* infection on gastric emptying and gastrointestinal hormones in dyspeptic and healthy subjects. *Dig Dis Sci* 46:46–53
- Chimienti G et al (2003) *Helicobacter pylori* is associated with modified lipid profile: impact on Lipoprotein(a). *Clin Biochem* 36:359–365
- Chong J, Marshall BJ, Barkin JS, McCallum RW, Reiner DK, Hoffman SR, O'Phelan C (1994) Occupational exposure to *Helicobacter pylori* for the endoscopy professional: a sera epidemiological study. *Am J Gastroenterol* 89:1987–1992
- Cover TL (1997) Commentary: *Helicobacter pylori* transmission, host factors, and bacterial factors. *Gastroenterology* 113:S29–30
- Cupella F, Alessio I, Intropido L, Pozzi V, Einaudi A, Pozzi U (1991) Dyspepsia and *Helicobacter pylori* infection. A study of a population of workers. *G Ital Med Lav* 13:81–84
- De Vecchi AF et al (1995) Epidemiology of *Helicobacter pylori* in dialysis patients. *Perit Dial Int* 15:178–179
- De Hert M, Hautekeete M, De Wilde D, Peuskens J (1997) High prevalence of *Helicobacter pylori* in institutionalized schizophrenic patients. *Schizophr Res* 26:243–244
- De Schryver AA, Van Hooste WL, Van Winckel MA, Van Sprundel MP (2004) *Helicobacter pylori* infection: a global occupational risk for healthcare workers? *Int J Occup Environ Health* 10:428–432. <https://doi.org/10.1179/oe.2004.10.4.428>
- De Schryver A, Cornelis K, Van Winckel M, Moens G, Devlies G, Derthoo D, van Sprundel M (2008) The occupational risk of *Helicobacter pylori* infection among workers in institutions for people with intellectual disability. *Occup Environ Med* 65:587–591. <https://doi.org/10.1136/oem.2007.035022>
- Dore MP et al (1999) High prevalence of *Helicobacter pylori* infection in shepherds. *Dig Dis Sci* 44:1161–1164
- Eshraghian A et al (2009) *Helicobacter pylori* infection as a risk factor for insulin resistance. *Dig Dis Sci* 54:1966–1970. <https://doi.org/10.1007/s10620-008-0557-7>
- European Agency for Safety and Health at Work (2007) Risk assessment in health care. E-fact 18. Available at: <http://osha.europa.eu>
- European Commission (2003) European list of occupational diseases. Official Journal of the European Union. Available at: <http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=CELEX:32003H0670:EN:NOT>
- Eusebi LH, Zagari RM, Bazzoli F (2014) Epidemiology of *Helicobacter pylori* infection. *Helicobacter* 19(Suppl 1):1–5. <https://doi.org/10.1111/hel.12165>
- Everhart JE, Kruszon-Moran D, Perez-Perez GI, Tralka TS, McQuillan G (2000) Seroprevalence and ethnic differences in *Helicobacter pylori* infection among adults in the United States. *J Infect Dis* 181:1359–1363. <https://doi.org/10.1086/315384>
- Fantry GT, Zheng QX, James SP (1995) Conventional cleaning and disinfection techniques eliminate the risk of endoscopic transmission of *Helicobacter pylori*. *Am J Gastroenterol* 90:227–232
- Ferro A et al (2014) Worldwide trends in gastric cancer mortality (1980–2011), with predictions to 2015, and incidence by subtype. *Eur J Cancer* 50:1330–1344. <https://doi.org/10.1016/j.ejca.2014.01.029>
- Friis L, Engstrand L, Edling C (1996) Prevalence of *Helicobacter pylori* infection among sewage workers. *Scand J Work Environ Health* 22:364–368
- Fujimoto Y, Furusyo N, Toyoda K, Takeoka H, Sawayama Y, Hayashi J (2007) Intrafamilial transmission of *Helicobacter pylori* among the population of endemic areas in Japan. *Helicobacter* 12:170–176. <https://doi.org/10.1111/j.1523-5378.2007.00488.x>
- Furesz J, Lakatos S, Nemeth K, Fritz P, Simon L, Kacszerka K (2004) The prevalence and incidence of *Helicobacter pylori* infections among young recruits during service in the Hungarian Army. *Helicobacter* 9:77–80
- Furuta T, Kamata T, Takashima M, Futami H, Arai H, Hanai H, Kaneko E (1997) Study of transmission routes of *Helicobacter pylori* in relation to seroprevalence of hepatitis A virus. *J Clin Microbiol* 35:1891–1893
- Garza Yado Mde L et al (2006) Prevalence of seropositividad to antibodies IgG and IgM against *Helicobacter pylori* in the medical residents of the University Hospital of Puebla. *Rev Alerg Mex* 53:69–72
- Garza-Gonzalez E, Perez-Perez GI, Maldonado-Garza HJ, Bosques-Padilla FJ (2014) A review of *Helicobacter pylori* diagnosis, treatment, and methods to detect eradication. *World J Gastroenterol* 20:1438–1449. <https://doi.org/10.3748/wjg.v20.i6.1438>
- Gasbarrini A et al (2001) Prevalence of and risk factors for *Helicobacter pylori* infection among healthcare workers at a teaching hospital in Rome: the Catholic University Epidemiological Study. *Eur J Gastroenterol Hepatol* 13:185–189
- Gdalevich M, Cohen D, Ashkenazi I, Mimouni D, Shpilberg O, Kark JD (2000) *Helicobacter pylori* infection and subsequent peptic duodenal disease among young adults. *Int J Epidemiol* 29:592–595
- Gisbert JP, Pajares JM (2004) Review article: 13C-urea breath test in the diagnosis of *Helicobacter pylori* infection -- a critical review. *Aliment Pharmacol Ther* 20:1001–1017. <https://doi.org/10.1111/j.1365-2036.2004.02203.x>
- Glasbrenner B, Malfertheiner P, Nilius M, Steinbruck C, Bruckel J, Wiesneth M, Adler G (1996) Prevalence of *Helicobacter pylori* infection and dyspepsia in young adults in Germany. *Z Gastroenterol* 34:478–482
- Goh KL, Parasakthi N (2001) The racial cohort phenomenon: seroepidemiology of *Helicobacter pylori* infection in a multi-racial South-East Asian country. *Eur J Gastroenterol Hepatol* 13:177–183
- Goh KL, Parasakthi N, Ong KK (1996) Prevalence of *Helicobacter pylori* infection in endoscopy and non-endoscopy personnel:

- results of field survey with serology and 14C-urea breath test. *Am J Gastroenterol* 91:268–270
- Golding J, Northstone K, Miller LL, Davey Smith G, Pembrey M (2013) Differences between blood donors and a population sample: implications for case-control studies. *Int J Epidemiol* 42:1145–1156. <https://doi.org/10.1093/ije/dyt095>
- Goodman KJ et al (1996) *Helicobacter pylori* infection in the Colombian Andes: a population-based study of transmission pathways. *Am J Epidemiol* 144:290–299
- Graham DY, Alpert LC, Smith JL, Yoshimura HH (1988) Iatrogenic *Campylobacter pylori* infection is a cause of epidemic achlorhydria. *Am J Gastroenterol* 83:974–980
- Hammermeister I, Janus G, Schamarowski F, Rudolf M, Jacobs E, Kist M (1992) Elevated risk of *Helicobacter pylori* infection in submarine crews. *Eur J Clin Microbiol Infect Dis* 11:9–14
- Han X et al (2016) *Helicobacter pylori* infection is associated with type 2 diabetes among a middle- and old-age Chinese population. *Diabetes Metab Res Rev* 32:95–101. <https://doi.org/10.1002/dmrr.2677>
- Hildebrand P, Meyer-Wyss BM, Mossi S, Beglinger C (2000) Risk among gastroenterologists of acquiring *Helicobacter pylori* infection: case-control study. *Bmj* 321:149
- Hoffmann KM, Eherer AJ, Krejs GJ (2003) Are dyspeptic symptoms linked to *Helicobacter pylori*? A prospective cohort study among medical students. *Wien Klin Wochenschr* 115:175–178
- Honda K, Ohkusa T, Takashimizu I, Watanabe M, Amagasa M (2001) High risk of *Helicobacter pylori* infection in young Japanese dentists. *J Gastroenterol Hepatol* 16:862–865
- Hu D, Shao J, Wang L, Zheng H, Xu Y, Song G, Liu Q (2013) Prevalence and risk factors of *Helicobacter pylori* infection in Chinese maritime workers. *Ann Hum Biol* 40:472–476. <https://doi.org/10.3109/03014460.2013.804121>
- Husson MO, Vincent P, Grabiard MH, Furon D, Leclerc H (1991) Anti-*Helicobacter pylori* IgG levels in abattoir workers. *Gastroenterol Clin Biol* 15:723–726
- Hyams KC, Taylor DN, Gray GC, Knowles JB, Hawkins R, Malone JD (1995) The risk of *Helicobacter pylori* infection among U.S. military personnel deployed outside the United States. *Am J Trop Med Hyg* 52:109–112
- IARC Working Group on the Evaluation of Carcinogenic Risks to Humans (1994) Schistosomes, liver flukes and *Helicobacter pylori*. vol 61, 1994/01/01 edn. IARC, Lyon
- International Labour Organization (2007) Resolution concerning updating the International Standard Classification of Occupations. ILO, Geneva
- International Labour Organization (2010) ILO List of Occupational Diseases (revised 2010). Programme on Safety and Health at Work and the Environment (SafeWork). Available at: http://www.ilo.org/wcmsp5/groups/public/---ed_protect/---protrav/---safework/documents/publication/wcms_125137.pdf
- Jackman RP, Schlichting C, Carr W, Dubois A (2006) Prevalence of *Helicobacter pylori* in United States Navy submarine crews. *Epidemiol Infect* 134:460–464. <https://doi.org/10.1017/s0950268805005169>
- Jeggli S, Steiner D, Joller H, Tschopp A, Steffen R, Hotz P (2004) Hepatitis E, *Helicobacter pylori*, and gastrointestinal symptoms in workers exposed to waste water. *Occup Environ Med* 61:622–627
- Kalabay L et al (2002) *Helicobacter pylori* infection in connective tissue disorders is associated with high levels of antibodies to mycobacterial hsp65 but not to human hsp60. *Helicobacter* 7:250–256
- Kamat AH, Mehta PR, Bhatia SJ, Koppikar GV (1999) Seroprevalence of IgG antibodies to *Helicobacter pylori* among gastrointestinal endoscopists, radiologists and paramedical personnel. *J Assoc Physicians India* 47:866–868
- Katellaris PH, Tippet GH, Norbu P, Lowe DG, Brennan R, Farthing MJ (1992) Dyspepsia, *Helicobacter pylori*, and peptic ulcer in a randomly selected population in India. *Gut* 33:1462–1466
- Kibria KM, Hossain ME, Sultana J, Sarker SA, Bardhan PK, Rahman M, Nahar S (2015) The prevalence of mixed *Helicobacter pylori* infections in symptomatic and asymptomatic subjects in Dhaka, Bangladesh. *Helicobacter* 20:397–404. <https://doi.org/10.1111/hel.12213>
- Kikuchi S, Kurosawa M, Sakiyama T (1998) *Helicobacter pylori* risk associated with sibship size and family history of gastric diseases in Japanese adults. *Jpn J Cancer Res* 89:1109–1112
- Kikuchi S et al (2005) Serum pepsinogen values and *Helicobacter pylori* status among control subjects of a nested case-control study in the JACC study. *J Epidemiol* 15(Suppl 2):S126–133
- Kilmarx PH et al (2014) Ebola virus disease in health care workers—Sierra Leone, 2014. *MMWR Morb Mortal Wkly Rep* 63:1168–1171
- Kim N, Lim SH, Lee KH, Kim JM, Cho SI, Jung HC, Song IS (2005) Seroconversion of *Helicobacter pylori* in Korean male employees. *Scand J Gastroenterol* 40:1021–1027
- Kim HY, Kim N, Kim SM, Seo JH, Park EH, Lee DH (2013) Seroprevalence of *Helicobacter pylori* Infection in Korean Health Personnel. *Gut Liver* 7:648–654. <https://doi.org/10.5009/gnl.2013.7.6.648>
- Kivi M, Tindberg Y (2006) *Helicobacter pylori* occurrence and transmission: a family affair? *Scand J Infect Dis* 38:407–417. <https://doi.org/10.1080/00365540600585131>
- Kopanski Z, Schlegel-Zawadzka M, Golec E, Witkowska B, Micherdzinski J, Cienciala A, Kustra Z (1997) The significance of selected epidemiologico-clinical factors in the prevalence of the *Helicobacter pylori* infection in young males. *Eur J Med Res* 2:358–360
- Kuepper-Nybelen J, Thefeld W, Rothenbacher D, Brenner H (2005) Patterns of alcohol consumption and *Helicobacter pylori* infection: results of a population-based study from Germany among 6545 adults. *Aliment Pharmacol Ther* 21:57–64. <https://doi.org/10.1111/j.1365-2036.2004.02276.x>
- Kyriazanos I, Ilias I, Lazaris G, Hountis P, Deros I, Dafnopoulou A, Datsakis K (2001) A cohort study on *Helicobacter pylori* serology before and after induction in the Hellenic Navy. *Mil Med* 166:411–415
- Kyriazanos ID, Sfiniadakis I, Gizaris V, Hountis P, Hatziveis K, Dafnopoulou A, Datsakis K (2002) The incidence of *Helicobacter pylori* infection is not increased among obese young individuals in Greece. *J Clin Gastroenterol* 34:541–546
- Lambert JR, Lin SK, Sievert W, Nicholson L, Schembri M, Guest C (1995) High prevalence of *Helicobacter pylori* antibodies in an institutionalized population: evidence for person-to-person transmission. *Am J Gastroenterol* 90:2167–2171
- Langenberg W, Rauws EA, Oudbier JH, Tytgat GN (1990) Patient-to-patient transmission of *Campylobacter pylori* infection by fiberoptic gastroduodenoscopy and biopsy. *J Infect Dis* 161:507–511
- Laszewicz W, Iwanczak F, Iwanczak B (2014) Seroprevalence of *Helicobacter pylori* infection in Polish children and adults depending on socioeconomic status and living conditions. *Adv Med Sci* 59:147–150. <https://doi.org/10.1016/j.advms.2014.01.003>
- Laurila A, Bloigu A, Nayha S, Hassi J, Leinonen M, Saikku P (1999) Association of *Helicobacter pylori* infection with elevated serum lipids. *Atherosclerosis* 142:207–210
- Leung WK, Siu KL, Kwok CK, Chan SY, Sung R, Sung JJ (1999) Isolation of *Helicobacter pylori* from vomitus in children and its implication in gastro-oral transmission. *Am J Gastroenterol* 94:2881–2884. <https://doi.org/10.1111/j.1572-0241.1999.01431.x>
- Lin SK, Lambert JR, Schembri MA, Nicholson L, Korman MG (1994) *Helicobacter pylori* prevalence in endoscopy and medical staff. *J Gastroenterol Hepatol* 9:319–324

- Lin SK, Lambert JR, Schembri MA, Nicholson L, Johnson IH (1998) The prevalence of *Helicobacter pylori* in practising dental staff and dental students. *Aust Dent J* 43:35–39
- Lin DB et al (2000) Association between seropositivity of antibodies against hepatitis A virus and *Helicobacter pylori*. *Am J Trop Med Hyg* 63:189–191
- Lin DB, Lin JB, Chen CY, Chen SC, Chen WK (2007) Seroprevalence of *Helicobacter pylori* infection among schoolchildren and teachers in Taiwan. *Helicobacter* 12:258–264. <https://doi.org/10.1111/j.1523-5378.2007.00496.x>
- Liu WZ, Xiao SD, Jiang SJ, Li RR, Pang ZJ (1996) Seroprevalence of *Helicobacter pylori* infection in medical staff in Shanghai. *Scand J Gastroenterol* 31:749–752
- Liu L, Liu Y, Tong W, Ye H, Zhang X, Cao W, Zhang Y (2007) Pathogen burden in essential hypertension. *Circ J* 71:1761–1764
- Loffeld RJ, Stobberingh E, van Spreeuwel JP, Flendrig JA, Arends JW (1990) The prevalence of anti-*Helicobacter* (Campylobacter) *pylori* antibodies in patients and healthy blood donors. *J Med Microbiol* 32:105–109. <https://doi.org/10.1099/00222615-32-2-105>
- Lopez-Saez JB, Gomez-Biondi V, Santamaria-Rodriguez G, Dominguez-Villar M, Amaya-Vidal A, Lorenzo-Penuelas A, Senra-Varela A (2010) Concurrent overexpression of serum p53 mutation related with *Helicobacter pylori* infection. *J Exp Clin Cancer Res* 29:65. <https://doi.org/10.1186/1756-9966-29-65>
- Loster BW et al (2009) Prevalence and characterization of *Helicobacter pylori* (*H. pylori*) infection and colonization in dentists. *J Physiol Pharmacol* 60(Suppl 8):13–18
- Lunet N, Barros H (2003) *Helicobacter pylori* infection and gastric cancer: facing the enigmas. *Int J Cancer* 106:953–960. <https://doi.org/10.1002/ijc.11306>
- Lynn TV, Bruce MG, Landen M, Beller M, Bulkow L, Gold B, Parkinson A (2007) *Helicobacter pylori* infection among non-Native educators in Alaska. *Int J Circumpolar Health* 66:135–143
- Makrithathis A, Pasching E, Schutze K, Wimmer M, Rotter ML, Hirschl AM (1998) Detection of *Helicobacter pylori* in stool specimens by PCR and antigen enzyme immunoassay. *J Clin Microbiol* 36:2772–2774
- Malaty HM, Evans DJ Jr, Abramovitch K, Evans DG, Graham DY (1992) *Helicobacter pylori* infection in dental workers: a seroepidemiology study. *Am J Gastroenterol* 87:1728–1731
- Marshall B (2002) *Helicobacter pylori*: 20 years on. *Clin Med (Lond)* 2:147–152
- Marshall BJ, Warren JR (1984) Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. *Lancet* 1:1311–1315
- Martinez M, Kordisch F, Lesser S (2000) The prevalence of *Helicobacter pylori*: are emergency medicine residents at risk? *J Emerg Med* 19:213–215
- Massarrat S, Saberi-Firoozi M, Soleimani A, Himmelmann GW, Hitzges M, Keshavarz H (1995) Peptic ulcer disease, irritable bowel syndrome and constipation in two populations in Iran. *Eur J Gastroenterol Hepatol* 7:427–433
- Mastromarino P, Conti C, Donato K, Strappini PM, Cattaruzza MS, Orsi GB (2005) Does hospital work constitute a risk factor for *Helicobacter pylori* infection? *J Hosp Infect* 60:261–268. <https://doi.org/10.1016/j.jhin.2004.12.019>
- Matsuda R, Morizane T, Tsunematsu S, Kawana I, Tomiyama M (2002) *Helicobacter pylori* prevalence in dentists in Japan: a seroepidemiological study. *J Gastroenterol* 37:255–259
- Megraud F, Broutet N (2000) Review article: have we found the source of *Helicobacter pylori*? *Aliment Pharmacol Ther* 14(Suppl 3):7–12
- Megraud F, Lehours P (2007) *Helicobacter pylori* detection and antimicrobial susceptibility testing. *Clin Microbiol Rev* 20:280–322. <https://doi.org/10.1128/cmr.00033-06>
- Melo ET, Lopes EP, Almeida JR, Albuquerque MF, Moura IM (2003) Seroprevalence of *Helicobacter pylori* antibodies in medical students and residents in Recife, Brazil. *J Clin Gastroenterol* 36:134–138
- Miftahussurur M, Yamaoka Y (2016) Diagnostic methods of *Helicobacter pylori* infection for epidemiological studies: critical importance of indirect test validation. *Biomed Res Int* 2016:4819423. <https://doi.org/10.1155/2016/4819423>
- Mitchell HM, Lee A, Carrick J (1989) Increased incidence of *Campylobacter pylori* infection in gastroenterologists: further evidence to support person-to-person transmission of *C. pylori*. *Scand J Gastroenterol* 24:396–400
- Moayyedi P, Forman D, Braunholtz D, Feltbower R, Crocombe W, Liptrott M, Axon A (2000) The proportion of upper gastrointestinal symptoms in the community associated with *Helicobacter pylori*, lifestyle factors, and nonsteroidal anti-inflammatory drugs. Leeds HELP Study Group. *Am J Gastroenterol* 95:1448–1455. https://doi.org/10.1111/j.1572-0241.2000.2126_1.x
- Mones J, Martin-de-Argila C, Samitier RS, Gisbert JP, Sainz S, Boixeda D (1999) Prevalence of *Helicobacter pylori* infection in medical professionals in Spain. *Eur J Gastroenterol Hepatol* 11:239–242
- Monno R et al (2008) *Helicobacter pylori* seroprevalence in selected groups of Albanian volunteers. *Infection* 36:345–350. <https://doi.org/10.1007/s15010-008-6338-6>
- Moore D, Gamage B, Bryce E, Copes R, Yassi A (2005) Protecting health care workers from SARS and other respiratory pathogens: organizational and individual factors that affect adherence to infection control guidelines. *Am J Infect Control* 33:88–96. <https://doi.org/10.1016/j.ajic.2004.11.003>
- Nasreen S, Shokoohi M, Malvankar-Mehta MS (2016) Prevalence of latent tuberculosis among health care workers in high burden countries: a systematic review and meta-analysis. *PLoS One* 11:e0164034. <https://doi.org/10.1371/journal.pone.0164034>
- Nisha KJ, Nandakumar K, Shenoy KT, Janam P (2016) Periodontal disease and *Helicobacter pylori* infection: a community-based study using serology and rapid urease test. *J Investig Clin Dent* 7:37–45. <https://doi.org/10.1111/jicd.12122>
- Nishikawa J, Kawai H, Takahashi A, Seki T, Yoshikawa N, Akita Y, Mitamura K (1998) Seroprevalence of immunoglobulin G antibodies against *Helicobacter pylori* among endoscopy personnel in Japan. *Gastrointest Endosc* 48:237–243
- Noone PA, Waclawski ER, Watt AD (2006) Are endoscopy nurses at risk of infection with *Helicobacter pylori* from their work? *Occup Med (Lond)* 56:122–128. <https://doi.org/10.1093/occmed/kqj013>
- Nouraei M et al (2009) Childhood hygienic practice and family education status determine the prevalence of *Helicobacter pylori* infection in Iran. *Helicobacter* 14:40–46. <https://doi.org/10.1111/j.1523-5378.2009.00657.x>
- Ogihara A, Kikuchi S, Hasegawa A, Kurosawa M, Miki K, Kaneko E, Mizukoshi H (2000) Relationship between *Helicobacter pylori* infection and smoking and drinking habits. *J Gastroenterol Hepatol* 15:271–276
- Ornelas IJ, Galvan-Potrillo M, Lopez-Carrillo L (2007) Protective effect of yoghurt consumption on *Helicobacter pylori* seropositivity in a Mexican population. *Public Health Nutr* 10:1283–1287. <https://doi.org/10.1017/s1368980007696372>
- Osato MS, Ayub K, Le HH, Reddy R, Graham DY (1998) Houseflies are an unlikely reservoir or vector for *Helicobacter pylori*. *J Clin Microbiol* 36:2786–2788
- Palli D et al (1993) *Helicobacter pylori* antibodies in areas of Italy at varying gastric cancer risk. *Cancer Epidemiol Biomarkers Prev* 2:37–40

- Parkin DM (2006) The global health burden of infection-associated cancers in the year 2002. *Int J Cancer* 118:3030–3044. <https://doi.org/10.1002/ijc.21731>
- Parsonnet J, Blaser MJ, Perez-Perez GI, Hargrett-Bean N, Tauxe RV (1992) Symptoms and risk factors of *Helicobacter pylori* infection in a cohort of epidemiologists. *Gastroenterology* 102:41–46
- Parsonnet J, Shmueli H, Haggerty T (1999) Fecal and oral shedding of *Helicobacter pylori* from healthy infected adults. *JAMA* 282:2240–2245
- Pateraki E, Mentis A, Spiliadis C, Sophianos D, Stergiatou I, Skandalis N, Weir DM (1990) Seroepidemiology of *Helicobacter pylori* infection in Greece. *FEMS Microbiol Immunol* 2:129–136
- Peleteiro B, La Vecchia C, Lunet N (2012) The role of *Helicobacter pylori* infection in the web of gastric cancer causation. *Eur J Cancer Prev* 21:118–125. <https://doi.org/10.1097/CEJ.0b013e32834a7f66>
- Peleteiro B, Bastos A, Ferro A, Lunet N (2014) Prevalence of *Helicobacter pylori* infection worldwide: a systematic review of studies with national coverage. *Dig Dis Sci* 59:1698–1709. <https://doi.org/10.1007/s10620-014-3063-0>
- Peters C, Schablon A, Harling M, Wohler C, Costa JT, Nienhaus A (2011) The occupational risk of *Helicobacter pylori* infection among gastroenterologists and their assistants. *BMC Infect Dis* 11:154. <https://doi.org/10.1186/1471-2334-11-154>
- Pfefferle PI, Kramer A (2008) *Helicobacter pylori*-infection status and childhood living conditions are associated with signs of allergic diseases in an occupational population. *Eur J Epidemiol* 23:635–640. <https://doi.org/10.1007/s10654-008-9276-9>
- Plummer M, de Martel C, Vignat J, Ferlay J, Bray F, Franceschi S (2016) Global burden of cancers attributable to infections in 2012: a synthetic analysis. *Lancet Glob Health* 4:e609–616. [https://doi.org/10.1016/s2214-109X\(16\)30143-7](https://doi.org/10.1016/s2214-109X(16)30143-7)
- Potasman I, Yitzhak A (1998) *Helicobacter pylori* serostatus in backpackers following travel to tropical countries. *Am J Trop Med Hyg* 58:305–308
- Potts LF, Lewis SJ, Mountford RA (1997) Prevalence of *Helicobacter pylori* in respiratory physicians performing bronchoscopy: a comparison with gastroenterologists using the carbon 13 urea breath test. *Helicobacter* 2:152–154
- Pristautz H et al (1994) Prevalence of *Helicobacter pylori* antibodies in the serum of gastroenterologists in Austria. *Endoscopy* 26:690–696. <https://doi.org/10.1055/s-2007-1009067>
- Pronai L, Schandl L, Tulassay Z (2000) Prevalence of *Helicobacter pylori* infection in Hungarian gastroenterologists and general practitioners. *Eur J Gastroenterol Hepatol* 12:473–474
- Reiff A, Jacobs E, Kist M (1989) Seroepidemiological study of the immune response to *Campylobacter pylori* in potential risk groups. *Eur J Clin Microbiol Infect Dis* 8:592–596
- Resuli B, Prifti S, Guma S, Stone M (1999) Prevalence of *Helicobacter pylori* infection in an Albanian population. *Postgrad Med J* 75:318
- Ricci C, Holton J, Vaira D (2007) Diagnosis of *Helicobacter pylori*: invasive and non-invasive tests. *Best Pract Res Clin Gastroenterol* 21:299–313. <https://doi.org/10.1016/j.bpg.2006.11.002>
- Robertson MS, Cade JF, Clancy RL (1999) *Helicobacter pylori* infection in intensive care: increased prevalence and a new nosocomial infection. *Crit Care Med* 27:1276–1280
- Rocha GA, Queiroz DM, Mendes EN, Oliveira AM, Moura SB, Silva RJ (1992) Source of *Helicobacter pylori* infection: studies in abattoir workers and pigs. *Am J Gastroenterol* 87:1525
- Roosendaal R, Kuipers EJ, van den Brule AJ, Pena AS, Meuwissen SG, Walboomers JM, de Graaff J (1993) Detection of *Helicobacter pylori* DNA by PCR in gastrointestinal equipment. *Lancet* 341:900
- Rothenbacher D, Brenner H (2003) Burden of *Helicobacter pylori* and *H. pylori*-related diseases in developed countries: recent developments and future implications. *Microbes Infect* 5:693–703
- Royo G, Ferez A, Esteban A, Martin C, Perez-Mateo M (1991) [Antibodies against *Helicobacter pylori* in gastroenterology personnel, patients and the healthy population]. *Rev Esp Enferm Dig* 80:233–236
- Rudi J, Toppe H, Marx N, Zuna I, Theilmann L, Stremmel W, Raedtsch R (1997) Risk of infection with *Helicobacter pylori* and hepatitis A virus in different groups of hospital workers. *Am J Gastroenterol* 92:258–262
- Sachdev V, Ahuja V, Kumar D, Sharma MP (2001) Prevalence of *Helicobacter pylori* infection by 13C-urea breath test in conference delegates. *Indian J Gastroenterol* 20:78
- Saijo Y, Yoshioka E, Fukui T, Kawaharada M, Sata F, Sato H, Kishi R (2007) *H. pylori* seropositivity and cytokine gene polymorphisms. *World J Gastroenterol* 13:4445–4451
- Salem OE, Thabet TM, El-Deeb SK, Youssri AH (1993) Study of the prevalence of specific human IgG against *H. pylori* among medical personnel. *J Egypt Public Health Assoc* 68:309–331
- Salomaa-Rasanen A, Kosunen TU, Karjalainen J, Aromaa A, Knekt P, Sarna S, Rautelin H (2006) IgA antibodies in persisting *Helicobacter pylori* infection in Finnish adults. *Clin Microbiol Infect* 12:236–240. <https://doi.org/10.1111/j.1469-0691.2005.01357.x>
- Sasazuki S et al (2006) Effect of *Helicobacter pylori* infection combined with CagA and pepsinogen status on gastric cancer development among Japanese men and women: a nested case-control study. *Cancer Epidemiol Biomarkers Prev* 15:1341–1347. <https://doi.org/10.1158/1055-9965.epi-05-0901>
- Satoh H, Saijo Y, Yoshioka E, Tsutsui H (2010) *Helicobacter pylori* infection is a significant risk for modified lipid profile in Japanese male subjects. *J Atheroscler Thromb* 17:1041–1048
- Shelley KH, Haddadin AS (1998) Is *Helicobacter pylori* infection an occupational hazard for anesthesiologists? *Anesth Analg* 87:973–974
- Shimada T, Terano A, Ota S, Takikawa H, Sumino S (1996) Risk of iatrogenic transmission of *Helicobacter pylori* by gastroscopes. *Lancet* 347:1342–1343
- Shin A, Shin HR, Kang D, Park SK, Kim CS, Yoo KY (2005) A nested case-control study of the association of *Helicobacter pylori* infection with gastric adenocarcinoma in Korea. *Br J Cancer* 92:1273–1275. <https://doi.org/10.1038/sj.bjc.6602467>
- Shinchi K, Ishii H, Imanishi K, Kono S (1997) Relationship of cigarette smoking, alcohol use, and dietary habits with *Helicobacter pylori* infection in Japanese men. *Scand J Gastroenterol* 32:651–655
- Siva R, Birring SS, Berry M, Rowbottom A, Pavord ID (2013) Peptic ulceration, *Helicobacter pylori* seropositivity and chronic obstructive pulmonary disease. *Respirology* 18:728–731. <https://doi.org/10.1111/resp.12075>
- Smoak BL, Kelley PW, Taylor DN (1994) Seroprevalence of *Helicobacter pylori* infections in a cohort of US Army recruits. *Am J Epidemiol* 139:513–519
- Sorberg M, Nyren O, Granstrom M (2003) Unexpected decrease with age of *Helicobacter pylori* seroprevalence among Swedish blood donors. *J Clin Microbiol* 41:4038–4042
- Souto FJ, Fontes CJ, Rocha GA, de Oliveira AM, Mendes EN, Queiroz DM (1998) Prevalence of *Helicobacter pylori* infection in a rural area of the state of Mato Grosso, Brazil. *Mem Inst Oswaldo Cruz* 93:171–174
- Sporea I, Popescu A, van Blankenstein M, Sirli R, Focsea M, Danila M (2003) The prevalence of *Helicobacter pylori* infection in western Romania. *Rom J Gastroenterol* 12:15–18
- Stroffolini T, Rosmini F, Ferrigno L, Fortini M, D'Amelio R, Matricardi PM (1998) Prevalence of *Helicobacter pylori* infection in a cohort of Italian military students. *Epidemiol Infect* 120:151–155

- Su YC, Wang WM, Chen LT, Chiang W, Chen CY, Lu SN, Jan CM (1996) High seroprevalence of IgG against *Helicobacter pylori* among endoscopists in Taiwan. *Dig Dis Sci* 41:1571–1576
- Takashima T et al (2012) Endoscopic reflux esophagitis and *Helicobacter pylori* infection in young healthy Japanese volunteers. *Digestion* 86:55–58. <https://doi.org/10.1159/000338849>
- Taylor DN, Sanchez JL, Smoak BL, DeFraitres R (1997) *Helicobacter pylori* infection in Desert Storm troops. *Clin Infect Dis* 25:979–982
- Testerman TL, Morris J (2014) Beyond the stomach: an updated view of *Helicobacter pylori* pathogenesis, diagnosis, and treatment. *World J Gastroenterol* 20:12781–12808. <https://doi.org/10.3748/wjg.v20.i36.12781>
- Triantafyllidis JK et al (2002) *Helicobacter pylori* infection in hospital workers over a 5-year period: correlation with demographic and clinical parameters. *J Gastroenterol* 37:1005–1013. <https://doi.org/10.1007/s005350200170>
- Triantafyllidis JK et al. (2003) Relation between *Helicobacter pylori* infection, thyroid hormone levels and cardiovascular risk factors on blood donors. *Hepatogastroenterology* 50 Suppl 2:cccxxviii–cccxxx
- Ullah SS et al (2010) Seropositivity of *Helicobacter pylori* among the fish handlers. *Mymensingh Med J* 19:219–224
- Upile T, East C, Paun S, Patel N, Battacharyya A (2002) *Helicobacter pylori* infection in surgical personnel. *Clin Otolaryngol Allied Sci* 27:310–313
- Vaira D, Holton J (1998) Vector potential of houseflies (*Musca domestica*) for *Helicobacter pylori*. *Helicobacter* 3:65–66
- Vaira D, Vakil N (2001) Blood, urine, stool, breath, money, and *Helicobacter pylori*. *Gut* 48:287–289
- Van Hooste W, Charlier AM, Rotsaert P, Bulterys S, Moens G, van Sprundel M, De Schryver A (2010) Work-related *Helicobacter pylori* infection among sewage workers in municipal wastewater treatment plants in Belgium. *Occup Environ Med* 67:91–97. <https://doi.org/10.1136/oem.2008.040436>
- van Mark A, Spallek M, Groneberg DA, Kessel R, Weiler SW (2010) Correlates shift work with increased risk of gastrointestinal complaints or frequency of gastritis or peptic ulcer in H. pylori-infected shift workers? *Int Arch Occup Environ Health* 83:423–431. <https://doi.org/10.1007/s00420-009-0495-5>
- van der Voort PH, van der Hulst RW, Zandstra DF, van der Ende A, Kesecioglu J, Geraedts AA, Tytgat GN (2001) Gut decontamination of critically ill patients reduces *Helicobacter pylori* acquisition by intensive care nurses. *J Hosp Infect* 47:41–45. <https://doi.org/10.1053/jhin.2000.0861>
- Vare PO, Heikius B, Silvennoinen JA, Karttunen R, Niemela SE, Lehtola JK, Karttunen TJ (2001) Seroprevalence of *Helicobacter pylori* infection in inflammatory bowel disease: is *Helicobacter pylori* infection a protective factor? *Scand J Gastroenterol* 36:1295–1300
- Velasco Elizalde C, Fernandez Ferrer MA, Rodriguez Muniz N (2007) Serologic diagnosis of *Helicobacter pylori* in endoscopy personnel. Serology in endoscopists. *Rev Esp Enferm Dig* 99:88–93
- Versalovic J (2003) *Helicobacter pylori*. pathology and diagnostic strategies. *Am J Clin Pathol* 119:403–412
- Vyse AJ et al (2002) The burden of *Helicobacter pylori* infection in England and Wales. *Epidemiol Infect* 128:411–417
- Wagtman MJ et al (1997) Low seroprevalence of *Helicobacter pylori* antibodies in historical sera of patients with Crohn's disease. *Scand J Gastroenterol* 32:712–718
- Wang RT et al (2002) *Helicobacter pylori* infection and gastric cancer: evidence from a retrospective cohort study and nested case-control study in China. *World J Gastroenterol* 8:1103–1107
- Wang X, Terry P, Yan H (2008) Stomach cancer in 67 Chinese counties: evidence of interaction between salt consumption and *Helicobacter pylori* infection. *Asia Pac J Clin Nutr* 17:644–650
- Webb PM, Knight T, Greaves S, Wilson A, Newell DG, Elder J, Forman D (1994) Relation between infection with *Helicobacter pylori* and living conditions in childhood: evidence for person to person transmission in early life. *Bmj* 308:750–753
- Weyermann M, Adler G, Brenner H, Rothenbacher D (2006) The mother as source of *Helicobacter pylori* infection. *Epidemiology* 17:332–334. <https://doi.org/10.1097/01.ede.0000201257.31155.a0>
- Wilhoite SL, Ferguson DA Jr, Soike DR, Kalbfleisch JH, Thomas E (1993) Increased prevalence of *Helicobacter pylori* antibodies among nurses. *Arch Intern Med* 153:708–712
- Woodward M, Morrison C, McColl K (2000) An investigation into factors associated with *Helicobacter pylori* infection. *J Clin Epidemiol* 53:175–181
- Xia P, Ma MF, Wang W (2012) Status of *Helicobacter pylori* infection among migrant workers in Shijiazhuang, China. *Asian Pac J Cancer Prev* 13:1167–1170
- Yang X, Nishibayashi H, Takeshita T, Morimoto K (1999) Prevalence of *Helicobacter pylori* infection in Japan: Relation to educational levels and hygienic conditions. *Environ Health Prev Med* 3:202–206. <https://doi.org/10.1007/bf02932259>
- Zagari RM, Law GR, Fuccio L, Pozzato P, Forman D, Bazzoli F (2010) Dyspeptic symptoms and endoscopic findings in the community: the Loiano-Monghidoro study. *Am J Gastroenterol* 105:565–571. <https://doi.org/10.1038/ajg.2009.706>