

***Helicobacter pylori* Infection: Epidemiology and Occupational Risk for Health Care Workers**

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Abstract

Introduction: *Helicobacter pylori* has been recognised as a major cause of gastroduodenal diseases, including gastric and duodenal ulcers with faeco-oral, oro-oral and gastro-oral transmission occurring. With the close personal contact inherent in patient care, health care workers may be at an increased risk of acquiring *H. pylori* and subsequent development of associated conditions. The objective of this review was to review the transmission and the occupational risk for health care workers. **Methods:** A literature search was performed using Pubmed (January 1990 to May 2001). Relevant key words were used and additional manual searches were made using the reference lists from the selected articles to retrieve other papers relevant to the topic. **Results:** Current knowledge implies various pathways of agent transmission, favouring person-to-person mode of transmission early in life. Faeco-oral, oro-oral and gastro-oral transmissions are proposed and may be of different relevance among various populations. As for health care workers, after elimination of the methodological weak studies, the risk seems to be increased in gastroenterologists, endoscopy staff and intensive care nurses. Results in other groups are conflicting. **Conclusions:** *H. pylori* infection is an occupational risk in some groups of health care workers. Studies are needed to elucidate the risk in other occupational groups.

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Introduction

Since Warren and Marshall successfully cultured *Helicobacter pylori* in 1983, it has been recognised as a major cause of several gastroduodenal diseases, including gastric and duodenal ulcers,¹⁻³ although most infections remain asymptomatic. Infection with *H. pylori* has been associated with an increased risk of gastric cancer and gastric lymphoma.⁴⁻⁶ *H. pylori* infection has also been associated with growth retardation in children⁷ and recent studies suggest that it increases the risk of adult coronary heart disease. It is not clear whether *H. pylori* infection is a cause of non-ulcer dyspepsia.⁸⁻¹¹

However, the exact source of infection and its mode of transmission are still under investigation. There is mounting evidence in favour of a person-to-person transmission mechanism as one of the most likely modes and primary route of spread.¹²

With the close contact of health care workers (HCW) with patients, it seems possible that person-to-person transmission occurs from an infected patient to the HCW.

Since a complete summary of the literature on *H. pylori* is beyond the scope of this review, we will briefly discuss the microbiologic characteristics of *H. pylori*, its epidemiology and transmission route in adults and the hypothesis of an occupational risk in HCW.

We performed a literature search using Pubmed (January 1990 up to May 2001) using relevant key words and additional manual searches using the reference lists from the selected articles to retrieve other papers relevant to the topic.

Microbiology of *Helicobacter pylori*

H. pylori is one of a large family of related bacteria that are well adapted to persist in the stomachs of vertebrates for the life-span of their hosts. The organisms are oxidase-positive, catalase-positive and strongly urease-positive gram-negative curved or spiral rods that are motile by means of multiple flagellae. The benefit of urease to *H. pylori* is still much debated but its usefulness in diagnosis was quickly recognised and, in 1985, the

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biopsy urease test was first described in a modified form by McNulty.¹³

Epidemiology

Acquisition of *H. pylori* infection seems to occur mostly in early childhood and persists throughout life.¹⁴ Data on seroconversion show a low incidence of infection in adults that ranges from 0% to 0.6% per year, whereas a higher rate of acquisition is reported in children.^{15,16} Infection with *H. pylori* occurs in all parts of the world. Prevalence in childhood ranges from 5% to 10% in industrialised countries to 80% to 90% in developing countries.¹⁷ Variation in prevalence within populations seems to be largely associated with socio-economic factors. Low socio-economic status and poor living and housing conditions are clearly related to infection status.¹⁸⁻²⁰ Differences between age groups in adulthood within a population seem to reflect a birth cohort effect, indicating a higher rate of infection in earlier birth cohorts.²¹

Natural History

The natural history of the infection is still not completely understood. Although *H. pylori* infection is thought to be lifelong, recent prospective studies in children have reported reversion of a previous positive to negative serology for *H. pylori*. Although this may be related to the low sensitivity of serological assays in young children, other studies have shown a reversion of a previous positive to negative breath test and endoscopically proved spontaneous clearance of *H. pylori* colonisation.²²⁻²⁵ This points to transient colonisation before established long-term colonisation ensues. Studies in adults have also reported seroreversion, which suggest that spontaneous clearance of infection can occur.²⁶

Also, the issue of time sequence between prior *H. pylori* infection and the subsequent development of peptic disease has only recently been addressed. It has not been established whether the association of *H. pylori* with peptic ulcer disease is present in all populations, in particular since the virulence of the organism may vary from place to place.^{27,28} Two studies have addressed this problem—a prospective study from Japan²⁹ and a nested case-control study from Israël.³⁰

In the first study, an odds ratio (OR) of 4 was found for the presence of anti-*H. pylori* antibodies among 50- to 80-year-old Japanese men with duodenal ulcer and their controls. In the second study, a similar OR of 3.8 (95% CI, 1.4-10.2) was found for the presence of anti-*H. pylori* antibodies among young Israelis with peptic ulcer diseases compared to matched controls. However, in the second study, the OR for Israelis from Western origin was 10.5 (95% CI, 1.72-63.8) compared to 2.3 (95% CI, 0.6-8.6) for

Israelis from Eastern origin. Although the power of the study was insufficient to detect even strong ethnic interactions, these data point to an unidentified co-factor or constellation of factors (including genetic determinants) associated with increased risk.

Transmission

H. pylori is a highly adaptable organism confined predominantly to the gastric mucosa in humans but is also found in other primates.³¹ Other animal reservoirs remain hypothetical. A number of investigators have suggested that animals such as pigs and cats could harbour and transmit *H. pylori* to humans. Although *H. pylori* has been isolated from a population of research cats,³² the significance of this finding is still controversial.

Volunteers infected by oral ingestion develop acute transient dyspeptic symptoms a few days after ingestion but in the majority of subjects, symptoms can be absent when primary infection occurs.³³ There is little knowledge of the nature of symptoms at acquisition. This likely accounts for the difficulty in studying the mode of naturally occurring transmission.³⁴

Current knowledge implies various pathways of agent transmission, favouring person-to-person mode of transmission early in life. Both faeco-oral as well as oro-oral transmission³⁴ and, more recently, gastro-oral have been proposed.³⁵ It is possible that all three routes, together with water and food contamination, may occur and may be of different relevance among various populations.^{31,36}

Evidence for person-to-person transmission comes from studies on patients living in institutions, where close contact is the rule and where the same *H. pylori* strain is isolated from children living in the same room.^{37,38} Clustering of infection in families has been reported in several countries and the same strain has been isolated within the same family.³⁹⁻⁴¹ Transmission seems to be most likely from mother to child,⁴² although other studies contradict this.⁴³ High density living and bed sharing in childhood are risk factors for acquiring the infection.^{7,20} This confirms that close contact favours spread through the person-to-person route, but a common source of infection cannot be entirely excluded.

Evidence for the oro-oral route comes from studies showing an increased prevalence of infection in spouses of infected patients with *H. pylori* duodenal ulcers, where the increase was associated with the duration of cohabitation.^{44,45} Moreover, about half of the infected spouses harbour the same ribopattern of their partner.⁴⁶

The oro-oral route of transmission is also supported by isolated reports confirming transmission of *H. pylori* through sharing of nasogastric tubes,⁴⁷ pH probes and contaminated endoscopes⁴⁸ and by a higher prevalence of *H. pylori* in

Chinese Australians who use chopsticks, which are sometimes used to share food.⁴⁹ Furthermore, there have been reports confirming the recovery of viable *H. pylori* organisms from dental plaque. However, if the oropharynx and dental plaque were reservoirs for *H. pylori*, an increased prevalence of *H. pylori* would be expected in dental workers. Studies of the prevalence of *H. pylori* infection in this group of HCW have not confirmed that they are at increased risk for infection. Also, the finding that the agreement between *H. pylori* and Epstein-Barr virus seropositivity was no better than chance is an argument against the oro-oral route.⁵⁰⁻⁵³

Arguments for a faeco-oral route of transmission are based on studies showing that viable *H. pylori* can be cultured from human faeces.⁵⁴ Besides this, infections transmitted by the faeco-oral route spread more readily among young children, in whom *H. pylori* is most commonly acquired. The prevalence of *H. pylori* also appears to parallel the prevalence of hepatitis A virus infection, again suggesting a faeco-oral route of transmission.^{55,56}

An intriguing hypothesis is that the infection may be transmitted through a gastro-oral route via refluxed gastric content or vomitus.⁵⁷ Indeed, attempts to culture *H. pylori* from faeces or saliva have met with difficulties, the infection is easily transmitted by gastric intubation and acute infection is characterised by achlorhydric vomiting. Mucus may serve as a vehicle for transmission in refluxate or vomitus. A high prevalence of infection with the same strain has been found in mentally retarded cohabiting children with frequent gastro-oesophageal reflux^{37,58,59} and also in children with a history of vomiting in siblings.⁶⁰ In an experimental study, it was possible to culture *H. pylori* from vomit samples in all infected volunteers who accepted to receive Ipecac⁶¹ and isolation of *H. pylori* from the vomitus of a 6-year-old child has been reported.⁶²

Some studies suggest that *H. pylori* infection may also be acquired from the environment, either from water or from animals. Transmission via water may occur; coccoid forms of the microorganisms have been found to survive for up to one year in river water.⁶³ However, viable *H. pylori* have never been cultured from water.⁶⁴ Data on zoonotic transmission are still highly controversial.⁶⁵

Occupational Risk for Health Care Workers

Biological agents have always been considered an occupational health risk of HCW. With the close personal contact inherent in patient care, including handling of faecal and oral materials, HCW may be at an increased risk of acquiring *H. pylori* and subsequent development of associated conditions.

As *H. pylori* can be recovered from gastric secretions, saliva and dental plaque of patients infected with the

organism,⁶⁵ the likelihood of person-to-person transmission from infected patients to HCW exposed to secretions containing *H. pylori*, particularly those involved in upper gastrointestinal (GI) endoscopy procedures, seems high.

There have been case reports of *H. pylori* infection after mouth-to-mouth resuscitation;⁶⁶ several studies have shown that *H. pylori* can be transmitted through upper GI endoscopy^{67,68} and sharing of nasogastric tubes,⁴⁷ pH probes and contaminated endoscopes.⁴⁸ However, detailed studies about occupational risk in HCW are scarce and their results remain controversial. There are several reasons to explain this. First, as the mode of transmission is still uncertain, it is not clear which groups of HCW are at potential high risk. Second, most studies have been hampered by methodological problems such as convenience sampling, small sample sizes and lack of exposure data. This has probably lead to misclassification; most studies have included a mixture of occupational groups with varied exposure to *H. pylori* and consequently, varied risk of infection.

The list of studies on this subject is given in Table I. These studies, however, have come up with rather conflicting results, mostly due to methodological problems mentioned above. Therefore, for the purpose of this review and to avoid selection bias, we only retained studies with a minimal sample size of 50 subjects, a participation rate of at least 60% and adjusted for at least age in their analysis. The list of studies retained is given in Table II.

In an American study of 122 gastroenterologists and endoscopy nurses attending an advanced course and a control group of 510 blood donors, the prevalence of *H. pylori* positivity was 53% in the study group compared to 14% in the control group. This difference was statistically significant.⁷¹ In an Australian study in an adult intensive care unit of a university teaching hospital, 100 nurses were compared to 500 blood donors as community controls. In intensive care nurses, *H. pylori* seropositivity (40%) was significantly higher than in age-matched controls (19%).⁷³ In another study from Australia, the prevalence of *H. pylori* seropositivity was studied in 39 gastroenterologists, 107 gastroenterology nurses, 25 internists and 42 general nurses. The control group was obtained by random sampling of an urban population area. As only the number of gastroenterology nurses met the criteria mentioned above, we will only report the results in that group. Prevalence of *H. pylori* seropositivity was 17% compared to 32% in the controls.⁸⁰ In a Chinese study, prevalence of *H. pylori* seropositivity was studied in 1050 medical staff including 125 GI endoscopists, 45 endoscopy nurses, 71 gastroenterologists not doing endoscopy, 105 nurses working in gastroenterology wards, 417 internists and 287 general nurses. This group was compared with an age-

TABLE I: RESULTS OF STUDIES OF *H. PYLORI* RISK IN HEALTH CARE WORKERS

Prevalence in the general population (as measured in control group)	Study group	Increased risk	Reference
Low (not given)	Anaesthesiologists	No	(69)
11.3%	Endoscopy personnel	Yes	(70)
14.1%	Gastroenterologists/endoscopists	Yes	(71)
14.1%	Endoscopy nurses	Yes	(71)
15.9%	Emergency medicine residents	No	(72)
19.0%	Intensive care nurses	Yes	(73)
19.0%	Anaesthesiologists,	No	(74)
21.0%	Gastroenterologists	Yes	(75)
21.0%	Gastroenterology nurses	No	(75)
*21.7%	Physicians	Yes	(76)
*21.7%	Nurses	Yes	(76)
*21.7%	Endoscopy staff	Yes	(76)
*21.7%	Non-endoscopy staff	Yes	(76)
23%	Gastroenterologists/endoscopists	Yes	(77)
24.8%	All health care workers	Yes	(78)
26%	Nurses	Yes	(79)
32.0%	Gastroenterologists	Yes	(80)
32.0%	Internists	No	(80)
32.0%	Gastroenterology nurses	No	(80)
32.0%	General nurses	No	(80)
35.0%	Medical and nursing staff	No	(81)
35.0%	Gastroenterology and endoscopy staff	No	(81)
37.0%	All health care workers	No	(82)
44.6%	All medical staff	Yes	(83)
47.0%	Endoscopists	No	(84)
51.6%	Endoscopists	Yes	(85).
52.7%	Gastroenterologists	No	(86)
52.7%	Medical professionals/ non-gastroenterologists	No	(86)
52.7%	Gastroenterologists/endoscopists	No	(86)
54.4%	GPs	No	(87)
54.4%	Gastroenterologist/no endoscopy	No	(87)
54.4%	Gastroenterologists/endoscopists	No	(87)
64.8%	Endoscopy staff	Yes	(88)

All studies used serology except those marked with “*” used urea breath test.

matched population obtained from the same urban area of the city. Prevalence of *H. pylori* seropositivity was 82.4% in GI endoscopists, 74.2% in GI nurses, 66.4% in internists and 65.8% in general nurses compared to 44.6% in the general population.⁸³

From these studies that fulfilled these criteria, it appears that the occupational risk for infection was increased in most groups (both medical and nurses).^{71,73,80,83} It is not directly clear why one group of gastroenterology nurses showed no increased risk but this could be due to the specific duties assigned to them in that particular situation.⁸⁰

As already mentioned, studies in dental workers have consistently shown no increased risk for infection.^{50-53,88}

TABLE II: RESULTS OF SELECTED STUDIES OF *H. PYLORI* RISK IN HEALTH CARE WORKERS

Prevalence in the the general population (as measured in control group)	Study group	Increased risk	Reference
14.1%	Gastroenterologists/endoscopists	Yes	(71)
14.1%	Endoscopy nurses	Yes	(71)
19.0%	Intensive care nurses	Yes	(73)
32.0%	Gastroenterology nurses	No	(80)
44.6%	Gastroenterologists/endoscopists	Yes	(83)
44.6%	Gastroenterology nurses/general nurses	Yes	(83)
44.6%	Internists	Yes	(83)

All studies used serology except those marked with “*” used urea breath test.

Institutions for intellectually and physically disabled persons have a special position, as the prevalence of *H. pylori* is high among inhabitants;^{37,38,58,89} up to now only one study has been published which shows an increased risk in employees with prolonged close physical contact with the inhabitants.⁵⁹ Preliminary results of a study in progress also showed the same results.⁹⁰

Conclusion

What is the conclusion of these different studies? As already mentioned, most studies suffered from a number of weaknesses such as low numbers, cross-sectional design and no specific classification of exposure between different groups of nursing personnel.

When only studies that fulfill some methodological criteria are considered, the risk is increased in almost all studies.

To clarify the issue, studies are needed that:

- 1) are prospective with an appropriate control group,
- 2) have made a very precise assessment of the exposure to all possible transmission routes of *H. pylori* (faeco-oral as well as oro-oral and gastro-oral), and
- 3) have large enough samples to have enough statistical power to detect meaningful differences.

A comparison should be made between personnel working in high-risk departments where oro-oral, faeco-oral as well as gastro-oral transmission is possible (intensive care, paediatrics, gastroenterology, gastrosurgery, endoscopy and head and neck surgery), departments where only faeco-oral transmission is present (intermediate risk) (neurosurgery, revalidation, geriatrics but also nursing homes with a large proportion of faecal incontinent patients) and low-risk departments (and with no patient contact).

If these studies find an increased occupational risk in (some groups of) HCW, appropriate preventive action should be taken to protect workers.

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