Summary

*Helicobacter pylori* is found predominantly in human gastric mucosa. Transfer of the bacterium remains an open topic, but it is likely that infection is usually acquired at a young age, particularly where lower socio-economic conditions prevail. Transmission via an external source such as water supply is a possibility but, in general, infection is probably passed from person to person. Arguments for and against faecal-oral, oral-oral and gastric-oral transmission have been presented, but the dominance of one of these routes is still to be determined.

Keywords: *Helicobacter pylori*

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**H. pylori infection: key risk factors**

- Increased prevalence with:
  - age
  - lower socio-economic status
  - lower standard of living conditions in childhood

Box 1

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Transmission of *Helicobacter pylori*

Margaret A Stone

Effective public health interventions to reduce the incidence of infection with *Helicobacter pylori* are dependent on a good understanding of those most at risk of acquisition and of the sources and routes of transmission of this common infection. Although some risk factors have been convincingly demonstrated, conclusive evidence about how *H. pylori* infection is transmitted remains elusive. When a patient diagnosed with *H. pylori* infection asks where the infection was acquired, a simple answer cannot be given with confidence.

**Age of acquisition**

It is likely that infection with *H. pylori* occurs mainly in early childhood. In a study from Finland, the annual incidence of infection was calculated to be only 0.3% in a group of children followed up for sero-conversion between ages 3 to 12 years, suggesting that in this population infection is generally acquired before 3 years of age. Results of a follow-up study of Irish children with confirmed eradication of *H. pylori* suggested that those under 5 years of age were most at risk of infection and re-infection, with re-infection common before the age of 5 years (59.25% per child per year), but rare in older children. Higher than average rates of infection in some occupational groups suggest, however, that infection may also occur in adult life. In some studies, for example, gastroenterologists have been shown to be significantly more likely to be infected than age-matched controls, raising the possibility of transmission from patients to doctors during endoscopy. In general, however, adults in developed countries are likely to be at low risk of acquiring new *H. pylori* infection, with studies suggesting sero-conversion rates of only 0.5% and 0.3% per person per year.

**Social class and living conditions as risk factors for infection**

The link between *H. pylori* infection and factors such as social class and living conditions in childhood is well documented, confirming childhood as the most likely time for infection to occur. In general, however, the infection is more prevalent in older age groups, a potential anomaly which may be the result of a ‘cohort effect’. According to this theory, the higher prevalence of infection in older population groups is explained by the existence of lower socio-economic conditions during the childhood of older people, resulting in a higher incidence of infection when they were young, rather than by the additional years during which they might have acquired the infection. A birth cohort study from Japan, for example, showed that infection was most prevalent in those born in the 1940s and 1950s, a period of post-war deprivation.

**Routes of transmission**

The way in which *H. pylori* infection is acquired remains unclear. *H. pylori* has been identified in drinking water using polymerase chain reaction (PCR) amplification; infection passed through contaminated water has also been suggested by epidemiological evidence linking the municipal water supply to infection in Peru, and transmission via food such as uncooked vegetables treated with sewage has been proposed. Although *H. pylori* has been isolated in non-human mammals, in general, the bacterium is confined to the gastric mucosa of man. The bacterium has also been found in monkeys, but few populations have close contact with non-human primates. The isolation of *H. pylori* from a commercially reared species of domestic cat has also been reported, suggesting that the organism could be a zoonotic pathogen, but firm evidence to support zoonotic transmission is lacking. The authors of a study of workers at an abattoir in Bologna, Italy, suggested that the high prevalence of infection amongst workers involved in animal-related tasks compared with clerical workers would support transmission from animals to humans. Conversely, a study based on an American population found that pet owners had a lower frequency of *H. pylori* infection; the authors, however,
found that this could be explained by the association between pet ownership and higher socio-economic status.\(^5\) Findings from the Bologna study\(^2\) could also be related to differences in socio-economic status between clerical and manual workers.

Although a common external source of infection cannot be ruled out, it appears likely that transmission of *H pylori* is most commonly from person to person, as suggested by a high prevalence of infection amongst those living in institutions,\(^23\)\(^-\)\(^25\) and by intrafamilial clustering.\(^26\)\(^-\)\(^31\) Evidence both for\(^26\)\(^-\)\(^29\) and against\(^32\) transmission between spouses has been presented, suggesting that this does occur, but perhaps relatively infrequently.

Transfer of *H pylori* from stomach to stomach has been shown to occur via inadequately disinfected endoscopy equipment.\(^33\) Similarly, the epidemic gastritis reported in 17 of 37 healthy volunteers taking part in an acid secretion study was probably due to cross-infection with *H pylori* via pH probes.\(^34\) Clearly, however, direct gastric–gastric transfer is not the normal route. Three methods by which the organism is passed from the human stomach of an infected subject to the stomach of a previously uninfected person without the involvement of an external source have been suggested: by faeces, saliva and vomitus.\(^35\)\(^-\)\(^37\)

### THE FAECAL–ORAL ROUTE

Evidence that transmission occurs mainly in early childhood concurs with the suggested faecal–oral pathway, but arguments for this mode of transfer are far from conclusive. Although PCR technology has been used to show *H pylori* DNA in faeces,\(^38\) the viability of these bacteria has not been proven and culture of *H pylori* from stools, though reported,\(^39\)\(^40\) has proved difficult. It has been suggested that transmission of *H pylori* via faeces may be restricted to young children with acute infection and adults with reduced acid secretion.\(^41\) If the faecal–oral route characterises the spread of *H pylori*, it would be expected that patterns of prevalence similar to those for hepatitis A would be found. Evidence of this type is unclear; although similar sero-prevalence curves have been found in Thailand\(^41\) and South Africa,\(^42\) results from the UK were not supportive.\(^43\)

### THE ORAL–ORAL ROUTE

The oral cavity as a possible reservoir of *H pylori* has been suggested, with gastric juice transporting viable organisms to the mouth during regurgitation. Attempts to demonstrate the presence of *H pylori* in the mouth have, however, yielded inconclusive results in much the same way as attempts to isolate the bacteria in faeces. In one study, the bacteria were found in dental plaque from only one of 29 patients with positive stomach biopsies, with negative results in all saliva samples,\(^44\) whilst another group failed to isolate *H pylori* from any samples of saliva or plaque.\(^45\) In both these studies, samples from the mouth were collected prior to endoscopy to exclude the possibility of contamination during this procedure. Contamination during withdrawal of the endoscope may have been the case where *H pylori* was successfully isolated from gingival crevices and dental plaque,\(^46\) a limitation subsequently acknowledged by the research group.\(^47\)

Use of chopsticks as a risk factor for infection, after adjustments for other risk factors, has been used to argue the case for oral–oral transmission,\(^47\) although it has been suggested\(^48\) that this could be the result of greater contact with recent immigrants in Chinese families who used chopsticks compared with those who did not. Contrasting results in two animal studies have been used in support of the dominance of the oral–oral route over the faecal–oral pathway. A study in beagles showed transmission of *H pylori* and similar bacteria from infected to uninfected puppies and it was noted that these animals had a tendency to lick one another extensively; by contrast, rats and mice, who are coprophagic but have little oral–oral contact, did not transmit bacteria of this type.\(^49\) Other evidence has, however, failed to support the predominance of the oral–oral route, for example, the lack of increased risk of infection found in dentists,\(^50\)\(^-\)\(^52\) and those with higher numbers of sexual partners.\(^53\)

### THE GASTRIC–ORAL ROUTE

It has been suggested that *H pylori* may be passed more directly from stomach to mouth, without the need for an oral reservoir. Transmission via vomitus has been suggested as a possibility\(^54\) and has more recently been proposed as the main route of transfer, specifically during epidemic acute *H pylori* infection in childhood.\(^54\)\(^55\) The animal studies mentioned above\(^46\) have also been used in support of the gastric–oral route,\(^48\) since rats and mice do not vomit. In suggesting this route of transmission, it has also been argued that mucus-rich vomiting caused by acute *H pylori* infection may be the bacterium’s mechanism for promoting survival, by providing a vehicle for transmission to new hosts.\(^56\)\(^-\)\(^59\)

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**Proposed external sources of transmission of *H pylori***

- contaminated drinking water
- sewage via uncooked vegetables
- infected animals

**Proposed person-to-person transmission routes of *H pylori***

- faecal–oral
- oral–oral
- gastric–oral
For the gastric–oral route to be feasible, *H pylori* would need to be able to survive in vomitus, but survival of the bacteria in an acidic environment without the presence of urea is limited. To counter this argument, it has been suggested that the period of hypochlorhydria likely to accompany infection may aid survival of the bacterium outside the stomach by creating an environment in which vomitus lacking in acid is produced. Some studies have shown an association between *H pylori* infection in children and their mothers but not children and their fathers. It has been suggested that although this may be due to infection passed from mother to child, an alternative explanation could be that infection is passed from a vomiting child to the mother, the parent most likely to clean up vomit.

Conclusions

Transmission of *H pylori* remains an open topic. From the evidence to date, it seems probable that transmission may occur through multiple pathways, both from person to person and via external sources, with the dominant route perhaps varying between different populations.

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