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Epidemiology of *H. pylori* in Western countries

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INTRODUCTION

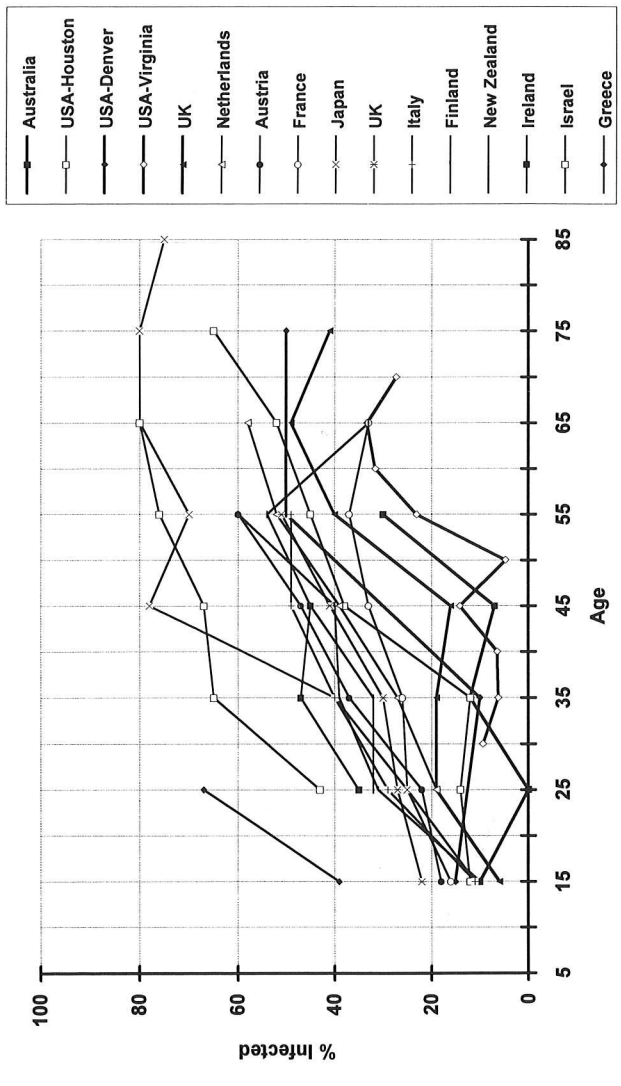
Before discussing the epidemiology of *Helicobacter pylori* infection the methodology for cited studies needs to be examined. Ideally, the detection of *H. pylori* should be by a harmless, non-invasive method which does not bias the selection of those being tested. At present the best method appears to be the [^{13}C]urea breath test, which also has the advantage of being given with food so that even small children can be easily tested. The disadvantage of the test is that it takes 60 min, so that persons tested are likely to be those with time to spare, possibly leading to an over-representation of unemployed persons, a lower socioeconomic group. The [^{13}C]urea breath test gives a sensitivity and specificity of at least 95%, and has been used in epidemiological studies of the southern USA by Graham *et al.* in Texas[1].

Serological testing can be done on large numbers of persons, with far less effort. The disadvantage is that a needlestick is involved. Serological studies therefore might select a group of persons who are willing to assist medical research, or who volunteer as blood donors. Thus serological studies might sample a higher socioeconomic group. Serological tests which detect specific IgG directed towards *H. pylori* have been shown to be highly accurate in volunteers and patients, with a sensitivity and specificity of around 95%[2].

PREVALENCE OF *H. PYLORI* IN WESTERN COUNTRIES

Figure 1 shows the seroprevalence of *H. pylori* in several Western countries, including Australia[3], the USA[2,4,5], the UK[6], Netherlands[7], Austria[8], France[9], Japan[10], the UK[11], Italy[12], Finland[13], New Zealand[14], Ireland[15], Israel[16] and Greece[17].

The most obvious feature of the prevalence curves is that *H. pylori* is more common in older persons. Note also that some of the curves run parallel to the X-axis below the age of 40 years, then rise between 40 and 60 years, but



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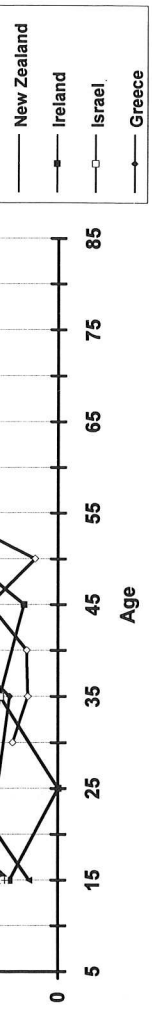

Fig. 1 Prevalence of *H. pylori* in Western countries

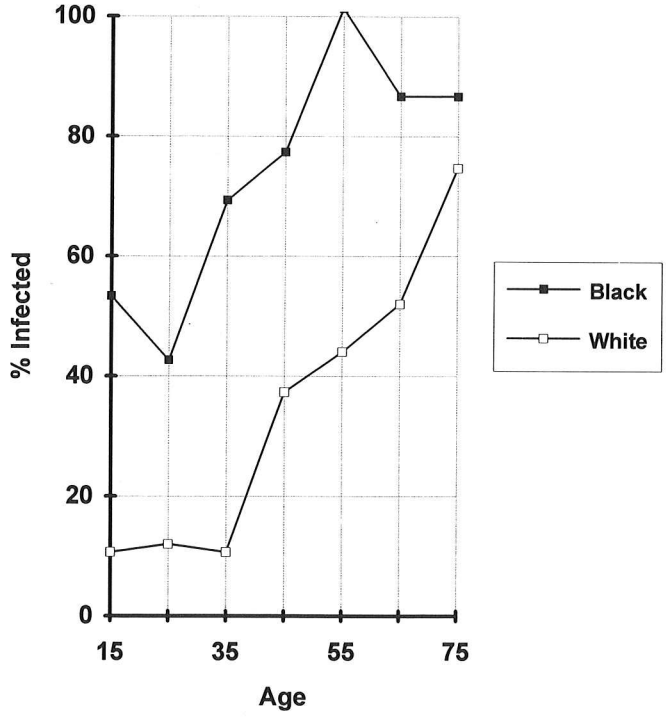

Fig. 2 Relationship between age and *H. pylori* according to race

level out again after 60. In a few studies prevalence has even decreased slightly after age 60. An apparent increasing prevalence with age led many to believe that *H. pylori* was commonly acquired in adulthood with an acquisition rate of 1–2% per annum. As discussed below, however, this may not actually be so.

The truth behind the rising prevalence of *H. pylori* with age may be seen in Figs 2 and 3. In the study descibed in Fig. 2, black volunteers in Houston were more than twice as likely to be infected with *H. pylori* than whites. This increased prevalence could have been either racial or socioeconomic. Figure 3, however, shows that even whites have a high prevalence of *H. pylori* infection when they are poor. Figure 3 can be used to predict *H. pylori* prevalence in whites throughout the USA. Referring to Fig. 1, for example, in Charlottesville, Virginia, where average family income is $40 000 per annum, only 15% of white blood donors have *H. pylori*[5]. In Houston blacks, however, no good correlation between *H. pylori* prevalence and income was seen. This may be because improved living conditions take at least one generation to affect prevalence in the population. For example, even affluent blacks in Houston may have had a poorer childhood socioeconomic status.

Further light has been thrown on this point by a study reported by Mendall *et al.*[18]. They looked at *H. pylori* serological status and tried to correlate it with various socioeconomic indicators. The most outstanding

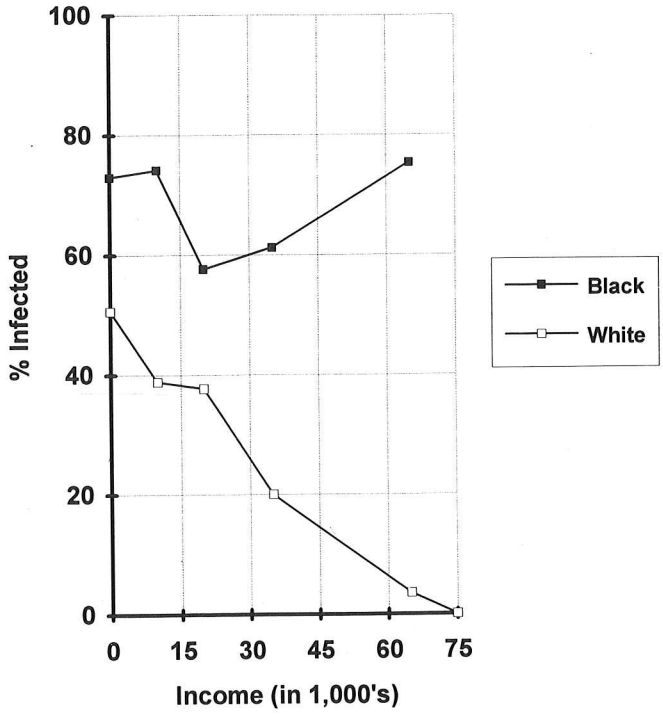

Fig. 3 Relationship between *H. pylori* and income (by race)

predictors of infection were related to childhood socioeconomic status. The presence of hot running water in the family home, and the number of persons per room in the house, were the most important predictors of *H. pylori*. *H. pylori* was particularly common in adults who had more than one person per room in their home during childhood.

In general, the following statements can be made to summarize prevalence of *H. pylori* in Western countries:

1. *H. pylori* affects about 20% of persons below the age of 40 years, and 50% of those above the age of 60 years.
2. *H. pylori* is uncommon in young children.
3. Low socioeconomic status predicts *H. pylori* infection.
4. Immigration is responsible for isolated areas of high prevalence in some Western countries.

SOURCE AND MODE OF TRANSMISSION

Faecal–oral spread

H. pylori has not been detected to any extent in the environment, so transmission is probably from close contact between humans. In the Gambia,

Thomas was able to isolate *H. pylori* from the stools of nine of 23 children below the age of 2 years[19]. The high prevalence of *H. pylori* in young Gambian children suggests that faecal–oral spread is the main route of infection. In Western countries, faecal–oral spread is less common in early life because families are smaller, there is less crowding, and the background prevalence of infection is far lower.

Faecal–oral spread may be an important source of infection for nursing staff or sanitation workers exposed to faecal matter. Wilhoite *et al.*[20] found a higher serological prevalence of *H. pylori* in nurses. Age and duration of nursing career were determinants of *H. pylori* prevalence, with approximately 50% of nurses infected compared to 30% of age-matched controls.

Oral–oral spread and contaminated secretions

The importance of an *H. pylori* reservoir in the oropharynx is undecided at present. Investigators have been able to detect the *Helicobacter genus* 16S-RNA urease gene by polymerase chain reaction (PCR)[21] and some have even been able to culture *H. pylori* from dental plaque[22]. However, even with PCR, the prevalence of plaque infection in infected patients has not exceeded 40% and the viability of this *Helicobacter* genetic material is uncertain. It is known, however, that *H. pylori* can be cultured from gastric juice, so viable organisms certainly do reflux up to the oropharynx. Dentists have not been found to have a higher prevalence of *H. pylori* than controls, although some dental hygienists may have an increased prevalence[23].

Oral–oral spread of *H. pylori* may be of relevance in some occupational groups. Mitchell *et al.*[24] demonstrated that Australian gastroenterologists have a clearly increased prevalence of *H. pylori* infection consistent with the known fact that gastroenterologists did not routinely wear gloves for endoscopy prior to 1983, and the biopsy channel of endoscopes at that time was located very near the endoscopist's mouth. In Mitchell's study, 52% of senior gastroenterologists were infected; exactly twice that of the control group.

INCIDENCE OF NEW INFECTION, SPONTANEOUS CURE AND REINFECTION

The ultimate prevalence of any infectious disease in the community is related to the incidence of new infections, the duration of the infection and the rate at which the infection is cured. The incidence of new *H. pylori* infection has been studied by only a few investigators, who have been able to obtain serial serum samples from large population groups over at least a 10-year period.

Parsonnet *et al.*[25] studied serum samples from doctors recruited to the Centers for Disease Control in Atlanta. These recruits had serum stored from before 1970 with further samples taken 10 and up to 20 years later. It was thus possible to estimate the seroconversion rate over an average 12-year follow-up period. The incidence of new infection was low, less than 1%

Table 1 *H. pylori* seroconversion rate in the USA[25]

Initial status	*No. of subjects*	*No. who changed status over 12 years*	*Change per annum* (%)	*Net change per annum* (%)
H. pylori-negative	278	11 (neg. to pos.)	+0.3	
H. pylori-positive	63	6 (pos. to neg.)	−0.7	1.2

per annum, and was partially neutralized by a similar incidence of spontaneous cure. The incidence of new infection was not enough to explain the rising prevalence of *H. pylori* seen with age in most Western countries and led the authors to suggest that incidence is less today than it was before 1970. This declining incidence was reflected in the prevalence of *H. pylori* in 30-year-old CDC recruits in 1968 (42%) versus those recruited after 1974 (20%).

The net change in prevalence over time is also related to the net rate of spontaneous cure. In Parsonnet's study spontaneous cure was reported in less than 1% of persons. In an earlier longitudinal study done between 1975 and 1980, Ormiston, Gear and Codling[26] reported a 5-year follow-up of gastritis in 50 patients rebiopsied after 5 years. Of these, 37 had gastritis on initial biopsy. Gastritis healed in only two, but improved in five others. If we say that between two and seven of these had reversion from *H. pylori*-positive to *H. pylori*-negative, then the spontaneous cure rate was between 1% and 3.7% per annum. In the Ormiston *et al.* study two of 11 normal patients developed gastritis in the 5-year follow-up period, an incidence rate of 3.6% per annum. Thus it appears that the net prevalence of *H. pylori* in Western countries has been static, and may even have decreased, in recent decades.

Reinfection is uncommon in Western countries; usually less than 1% per annum. Data to support this come from a study by Forbes *et al.*[27], in which patients in Perth, Australia, were followed up 7 years after *H. pylori* eradication. Reinfection rate was less than 1% per annum, similar to that reported earlier by Borody *et al.*[28]. Since spouses are often infected with *H. pylori*, one might ask whether or not infected spouses need to be treated to prevent reinfection of the patient. Cutler and Schubert[29] noted that the presence of an infected spouse does not affect cure rate. Thus early reinfection (before 1 month) seems to be rare.

In Virginia we have followed 300 patients at least 3 months and have seen only three reinfections after proven cure. These three persons all had infected spouses. Allowing for an average infection prevalence of 50% in spouses, it appears that the reinfection rate for a person with an infected spouse is only 2–5% per annum. Therefore, at this time it is not essential routinely to screen for *H. pylori* in asymptomatic spouses of infected patients.

Reinfection in children may be more common according to data reported by Oderda *et al.*[30]. In that report the reinfection rate was 18% after 1 year. Thus eradication therapy may not provide a permanent cure in children where infected siblings provide an easy source of reinfection[31]. It might be useful, therefore, to screen for, and treat, all *H. pylori* cases in the family when the index case is a child.

CLINICAL CURE

Change per annum (%)	*Net change per annum* (%)
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−0.7	1.2

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COMPUTER MODELLING OF PREVALENCE AND INCIDENCE

Data on infection and spontaneous cure rates can be used to predict the epidemiology of *H. pylori* in various populations. An attempt to generate known prevalence curves using constant infection rates is shown in Fig. 4a. The spontaneous remission rate of 1% per annum is used in all the shown curves. It can be seen that an infection rate of 8% per annum approximates the curve seen for some Third World countries. In contrast, however, a 1% infection rate does not completely explain the low prevalence of *H. pylori* in young whites in the USA, or the far greater proportion of infected persons seen after the age of 50 years.

If a changing incidence is allowed, however, the curve shown in Fig. 4b can be developed. This shows a constant (Third World) infection rate of 8% per annum for persons born before 1943, with a rate which halved in each of the subsequent 5 years until 1963 when a stable incidence rate of 0.5% per annum was achieved. This curve can be seen to approximate that of the epidemiology of *H. pylori* in the USA and other Western countries where the current incidence of *H. pylori* is low.

One other observation which has puzzled some authors is an apparent decline of *H. pylori* in persons above the age of 60. In a study of New Zealand endoscopy patients the prevalence was 64% at 65 years, but declined to less than 40% by age 90[32]. This can also be explained by the computer model. In any population with an initial prevalence of 65% an incidence of 0.5% or less per annum and spontaneous cure rate of at least 1% per annum will cause the prevalence to decrease towards 33%. Thus elderly patients will, on average, have a declining prevalence of *H. pylori* over time.

CONCLUSION: A DECLINING DISEASE IN WESTERN COUNTRIES

The epidemiological data indicate that *H. pylori* epidemiology is compatible with a cohort effect, in that cohorts born before 1940 have a far greater incidence of infection than subsequent cohorts. It appears that *H. pylori* was a declining disease in Western countries, even before it was rediscovered in 1981. The high prevalence of *H. pylori* in persons older than 60 years of age reflects the conditions present when they were children. Before 1940, average socioeconomic status and public health standards were poor, even in the USA. In European countries, social dislocation after the Second World War (1940–1955) probably maintained these poor conditions so that an observed decrease in *H. pylori* prevalence was delayed. Where living standards have improved rapidly, such as Japan and emerging Asian nations, we may expect a precipitous decline in the prevalence of *H. pylori* in younger persons.

SUMMARY

The epidemiology of *H. pylori* in Western countries shows a consistent pattern, with the disease being uncommon in persons below the age of 40 years but rising in prevalence after that to reach approximately 50% in most

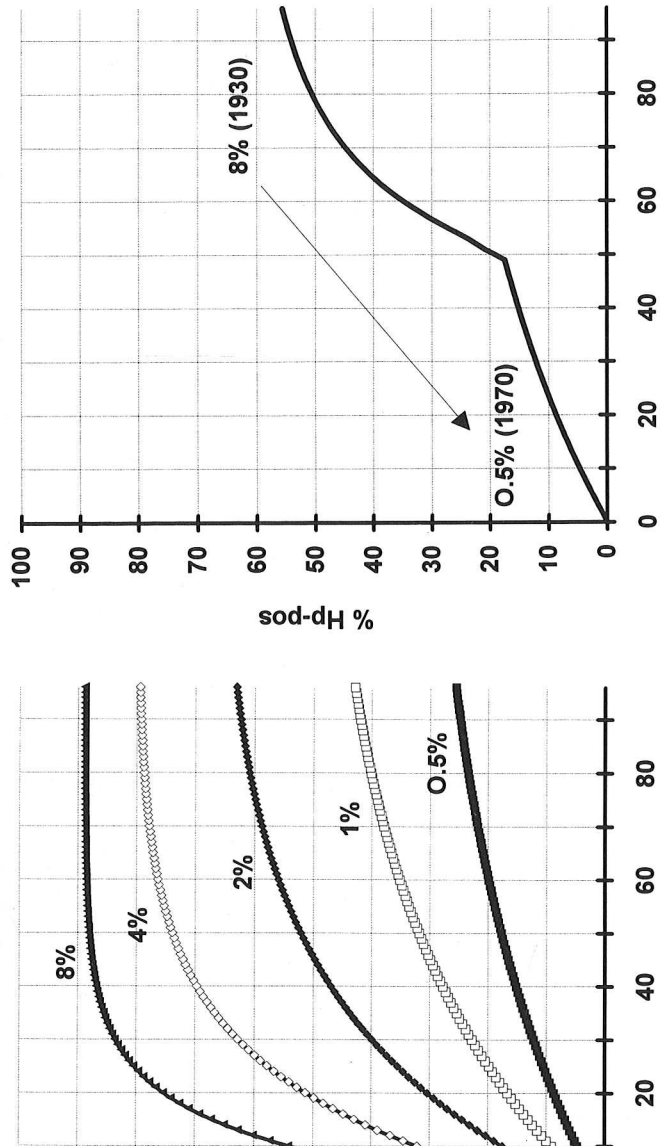

Fig. 4 (**a**) Computer-generated prevalence curves for various incidence rates, spontaneous cure rate of 1% per annum. (**b**) Computer-generated prevalence curve for changing incidence (see text)

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groups of elderly persons. Since there is no evidence for increasing infection rates during life, the apparent 1% increase per annum must be explained in other ways. Data from several studies show that the prevalence of *H. pylori* changes very little over the lifetime of any studied group, and that *H. pylori* has been decreasing in prevalence with a 50% reduction in the past 25 years. This can all be explained by the concept of birth cohorts. *H. pylori* was far more common in the environment of persons born before the Second World War (1940–45) but has decreased greatly since then. In computer models the epidemiological data can best be reproduced by a cohort effect with a rapid decline in incidence between 1940 and 1960.

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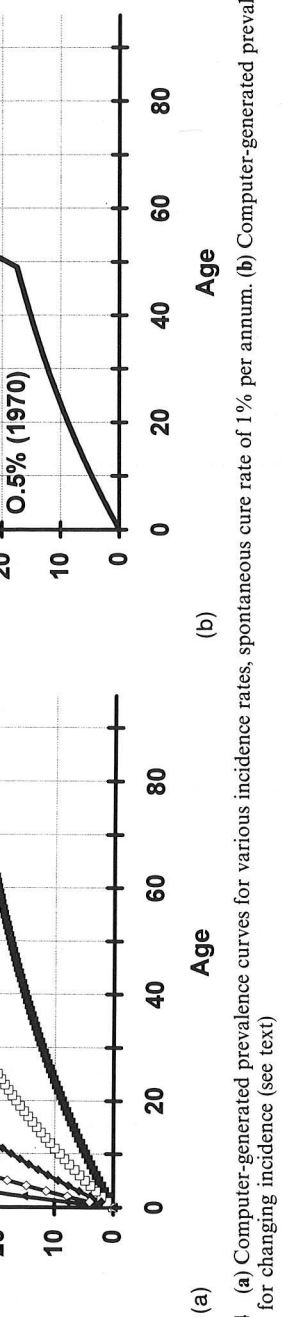

Fig. 4 (**a**) Computer-generated prevalence curves for various incidence rates, spontaneous cure rate of 1% per annum. (**b**) Computer-generated prevalence curve for changing incidence (see text)

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H. pylori in

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Peru[4]	1
Southern China[5]	1
Northern China*	

*Unpublished data