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HELICOBACTER PYLORI

# Prevalence of *Helicobacter pylori* in a representative Anglo-Celtic population of urban Melbourne

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Abstract The aims of this study were to assess the prevalence of *Helicobacter pylori* and its relationship with different epidemiological factors in an Anglo-Celtic Australian population in the Melbourne urban area. Two hundred and seventy-three (120 men and 153 women with a mean age of 55.6 and range of 20 to 80 years) of 396 eligible subjects randomly sampled from the telephone directory were studied. An ELISA technique was used to detect *H. pylori* immunoglobulin (Ig)G antibody and self-administered questionnaires were completed. The overall seroprevalence of *H. pylori* was 38% and increased with age from 18% (20–30 years old) to 53% (over 70 years; $P < 0.0001$). The acquisition of *H. pylori* infection was 1% per year. The prevalence of *H. pylori* was 48% in men and 30% in women ($P < 0.01$). The frequency of *H. pylori* was also associated with low-income levels and current smoking, but was not associated with peptic ulcer disease history. The prevalence of *H. pylori* infection in a representative Australian population was found to be similar to other developed countries. The risk factors for *H. pylori* infection include age, male sex, low household income and a smoking habit. No correlation between *H. pylori* status and dyspepsia symptoms were observed.

Key words: Australian, epidemiology, *Helicobacter pylori*, representative population.

## INTRODUCTION

*Helicobacter pylori* is one of the world's most common bacterial infections. Several epidemiological studies of the prevalence of *H. pylori* infection have been reported from different countries.[1–3] The prevalence of *H. pylori* is different from country to country and is higher in certain countries. No data is available on the prevalence of *H. pylori* in a representative normal Australian population. This study provides information on the epidemiology of *H. pylori* infection in a representative Anglo-Celtic population within Australia.

The aims of this study were to assess: (i) the prevalence of *H. pylori* in a representative Anglo-Celtic population in the Melbourne urban area; (ii) the association between the prevalence of *H. pylori* infection and demographic variables such as sex, age, socio-economic status and family size; and (iii) the association between the prevalence of *H. pylori* infection and past and present medical history as well as family history of medical illness.

## METHODS

### Sample size

A sample size of 250 subjects was determined based on previous small blood donor studies of the seroprevalence of *H. pylori* in the urban Australian populations of Sydney and Melbourne.[4,5] Using this estimate, a sample size of 250 would be required to estimate the prevalence of *H. pylori* to within 5% of its true value with a probability of 95%.[6] The significance level was set at 5%.

### Sampling and recruitment

One thousand and forty-two subjects with Anglo-Celtic names were randomly selected from the telephone directory from the Melbourne metropolitan area in 1991 and had a socio-demographic profile similar to the overall Melbourne Statistical Division (ABS Census

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1986). An introductory letter was sent explaining the purpose of the study. The first telephone contact was to establish eligibility for entry to the study. Eligible subjects were both males and females aged 20–80 years old, were born in Australia and both parents were Caucasian.

### Response rate of subjects' participation

Seven hundred and fifty subjects were contacted and of these 396 (53%) were eligible according to the above criteria. Of the eligible study subjects, 31% (123/396) refused to be studied. The response rate of eligible subjects was thus 69% (273/396) which resulted in a total of 273 participants in the survey.

All subjects completed a self-administered questionnaire which was reviewed and clarified by a research nurse or doctor at the time of interview. Informed consent was obtained from all participants before the interview took place. Venous blood (10 mL) was obtained from a peripheral vein at the time of interview and serum were stored at −20°C until it could be analysed.

### Questionnaire

The questionnaire consisted of questions relating to demographic data, gastrointestinal symptoms (heartburn, upper abdominal pain, nausea, vomiting or bloating), smoking, alcohol consumption and use of non-steroidal anti-inflammatory drugs (NSAID). Date on family history of peptic ulcer of first degree relatives was sought. Education was recorded as the highest number of years of schooling completed and was divided into four categories. Gross annual household income was divided into five categories. Current occupation data were collected and recorded using the National Heart Foundation categorization.

### Detection of *H. pylori* infection

*Helicobacter pylori*-specific immunoglobulin (Ig)G antibodies were measured by an enzyme-linked immunosorbent assay (ELISA) technique (Hel-pTEST™; Amrad Corporation, Melbourne, Australia) as previously described.[7] The sensitivity was 92% and the specificity was 88%. *Helicobacter pylori* infection was defined as a positive ELISA test result.

### Statistical methods

The SAS program (SAS Institute, Cary, NC, USA) was used for statistical analysis of data. The Maximum Likelihood Chi-squared test and Mantel–Haenszel test were used to assess the trends in the data. Relative risk was calculated along with the 95% confidence intervals. The logistic regression model was performed to take into account the continuous effect of age and socio-demographic variables on the seroprevalence of *H. pylori*. Odds ratios were calculated to assess the strength of associations between the seropositivity of *H. pylori* and explanatory variables.

## RESULTS

A total of 273 subjects were studied. For personal reasons, 10% (30/273) of subjects did not answer the questions relating to household income and education from the questionnaire.

The clinical and demographic data profile is shown in Table 1. The male-to-female ratio was 1:1.3, with the mean age not significantly different between men and women. Subjects with a family history of peptic ulcer disease represented 6% (17/273) of the study population.

The study population was categorized into six age groups as shown in Table 2. Subjects in the age group 61–70 years accounted for 34.8% of the overall study population. The overall seroprevalence of *H. pylori* in the study population was 38%. The prevalence of *H. pylori* infection increased with age from 18% (20–30 years old) to 53% (over 70 years; $P < 0.0001$). The yearly rate of acquisition of *H. pylori* infection was calculated[8] to be 1% per year (Fig. 1) assuming that no cohort effect existed.

Table 1 Demographic data of the study population

| | Total population | Male | Female | *P* value* |
|---|---|---|---|---|
| No. of subjects | 273 | 120 | 153 | |
| Mean age (years) (±SD) | 55.6 (±14.8) | 57.2 (±15.1) | 54.4 (±14.9) | 0.13 |
| Range (years) | 20–80 | 20–80 | 20–79 | |
| Duodenal ulcer history | 24 | 15 | 9 | 0.055 |
| Gastric ulcer history | 6 | 4 | 2 | 0.42 |

*Comparison between male and female.

Table 2 The age structure of the study population

| Age groups (years) | Total population No.* | %† | Men No. | % | Women No. | % |
|---|---|---|---|---|---|---|
| 20–30 | 17 | 6.2 | 5 | 4.2 | 12 | 7.8 |
| 31–40 | 38 | 13.9 | 18 | 15 | 20 | 13.1 |
| 41–50 | 41 | 15 | 15 | 12.5 | 26 | 17 |
| 51–60 | 37 | 13.6 | 14 | 11.6 | 23 | 15 |
| 61–70 | 95 | 34.8 | 45 | 37.5 | 50 | 32.7 |
| >70 | 45 | 16.5 | 23 | 19.2 | 22 | 14.4 |

*Number and †percentage of subjects.

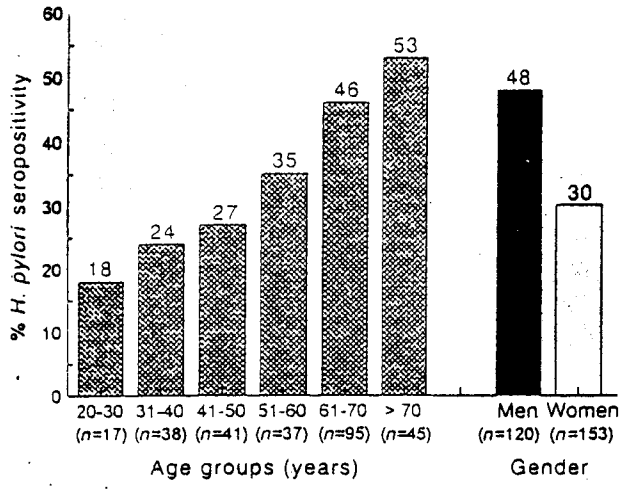


Figure 1 The prevalence of *Helicobacter pylori* infection increased with age ($P < 0.0001$) at a rate of approximately 1% per year. *Helicobacter pylori* infection was more common in men than in women (relative risk, 1.6; 95% confidence interval, 1.2–2.2; $P = 0.002$).

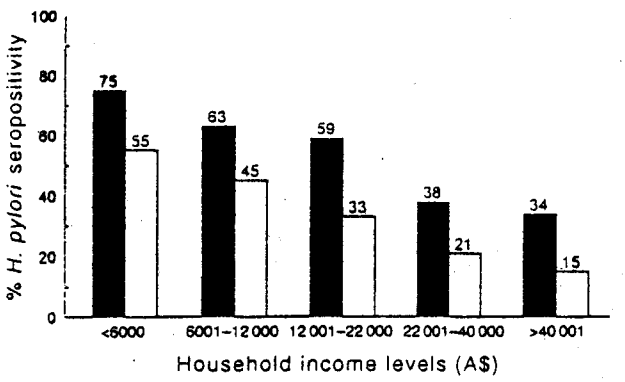


Figure 2 The prevalence of *Helicobacter pylori* infection in relation to household income levels in all subjects ($P < 0.0001$) as well as in males (■, $P = 0.01$) and females (□, $P = 0.001$).

The overall frequency of *H. pylori* infection was more common in men (48%) than in women (30%; ($P < 0.002$; Fig. 1). The prevalence of *H. pylori* infection increased with age in both males and females ($P < 0.01$).

The Chi-squared test was used to investigate the relationship between the prevalence of *H. pylori* infection and each study variable. The results were analysed on the overall population and also according to gender (Table 3).

*Helicobacter pylori* infection was more common in low-income level subjects (Fig. 2).

The overall education levels were inversely related to *H. pylori* seropositivity. The prevalence of *H. pylori* was not associated with education level in men, but was significantly associated with education in women. Women with higher education had a lower seroprevalence of *H. pylori* infection ($P < 0.016$).

The prevalence of *H. pylori* infection was not associated with occupation ($P = 0.82$).

An association between prevalence of *H. pylori* infection and smoking habits was found in the overall population studied ($P = 0.04$). However, with separate analysis by gender, an association was found only in women ($P = 0.032$), with an odds ratio of 2.8 for current smokers compared with subjects who had never smoked. There was no association between *H. pylori* infection and the age at which subjects started smoking or the number of cigarettes smoked per day.

The prevalence of *H. pylori* infection was not associated with: alcohol consumption in both men and women; peptic (gastric or duodenal) ulcer disease history; or NSAID use. No correlation was observed between *H. pylori* status and gastrointestinal symptoms.

### Logistic regression adjustment for confounding variables

As there were interrelationships between variables, the prevalence of *H. pylori* was adjusted for each variable by logistic regression analysis and the results are shown in Table 4.

The prevalence of *H. pylori* infection was associated with age, gender, household income and smoking habits; however, there was no significant association with education levels, occupation status, alcohol consumption, NSAID use, or family size. There was a significant association between *H. pylori* infection and current smoking habits, with subjects currently smoking having a higher rate of infection than those who had never smoked ($P = 0.03$) after adjustment for age, sex and socio-economic status (Table 5). No significant difference was found between ex-smokers and subjects who had never smoked.

## DISCUSSION

### Prevalence of *H. pylori*

*Helicobacter pylori* infection occurs worldwide. The prevalence of *H. pylori* infection is similar across Western countries and lower than in developing countries where the infection is acquired at an earlier age. Study from USA has shown that the prevalence of *H. pylori* infection in the white population was 15% at age 30 and 40% at age 50.[3] Our study population was selected from a sample considered representative of the Anglo-Celtic population in urban Melbourne. Our data also indicates that *H. pylori* infection increases with age at a rate of about 1% per year. Eighteen per cent of subjects were infected by age 30 and 46% were infected by age 60 years. This was largely similar to that reported in the literature from developed countries[1–3] and the Australian blood donor population.[4,5] The frequency of *H. pylori* infection differs in populations, both between and within countries. This may be due to ethnic, cultural and geographical differences. The prevalence of *H.*

Table 3 The association between different variables and *Helicobacter pylori* status

| | Total | | | Men | | | Women | | |
|---|---|---|---|---|---|---|---|---|---|
| | No.* | %** | RR† | No. | % | RR | No. | % | RR |
| Household income (A$) | | | | | | | | | |
| <6000 | 15 | 60 | 2.4 | 4 | 75 | 2.1 | 11 | 55 | 3.7 |
| 6001–12 000 | 38 | 52 | 2.1 | 16 | 63 | 1.8 | 22 | 45 | 3.1 |
| 12 001–22 000 | 65 | 44 | 1.8 | 29 | 59 | 1.7 | 36 | 33 | 2.3 |
| 22 001–40 000 | 68 | 27 | 1.1 | 29 | 38 | 1.1 | 39 | 21 | 1.4 |
| >40 001# | 56 | 25 | $P=0.0001$ | 29 | 34 | $P=0.01$ | 27 | 15 | $P=0.001$ |
| Education level (years) | | | | | | | | | |
| <6 | 30 | 46 | 1.3 | 9 | 66 | 1.2 | 21 | 38 | 2.2 |
| 7–9 | 81 | 45 | 1.3 | 39 | 48 | 0.9 | 42 | 42 | 2.5 |
| 10–12 | 74 | 27 | 0.7 | 31 | 35 | 0.6 | 43 | 20 | 1.2 |
| >12# | 57 | 35 | $P=0.07$ | 28 | 53 | $P=0.6$ | 29 | 17 | $P=0.02$ |
| Occupation | | | | | | | | | |
| Retired | 81 | 37 | 1.1 | 15 | 40 | 0.9 | 66 | 36.4 | 1.7 |
| Domestic service | 18 | 33 | 1.0 | 13 | 38.5 | 0.9 | 5 | 20 | 0.9 |
| Manual | 37 | 43 | 1.3 | 27 | 55.6 | 1.3 | 10 | 10 | 0.5 |
| Clerical worker | 57 | 40.4 | 1.2 | 18 | 55.6 | 1.3 | 39 | 33.3 | 1.6 |
| Professional# | 70 | 32.9 | $P=0.82$ | 41 | 41.5 | $P=0.65$ | 29 | 20.7 | $P=0.26$ |
| Alcohol consumption | | | | | | | | | |
| >once week | 141 | 36 | 1.0 | 75 | 44 | 1.1 | 66 | 29 | 0.6 |
| <once week | 53 | 39 | 1.1 | 22 | 63 | 2.3 | 31 | 23 | 0.5 |
| No use# | 72 | 37 | $P=0.9$ | 19 | 42 | $P=0.7$ | 53 | 36 | $P=0.4$ |
| Smoking habit | | | | | | | | | |
| Current smoking | 51 | 45 | 1.5 | 22 | 40 | 1.0 | 29 | 48 | 1.9 |
| Ex-smoked | 88 | 44 | 1.4 | 48 | 58 | 1.4 | 40 | 27 | 1.1 |
| Never smoked | 134 | 31 | $P=0.04$ | 50 | 42 | $P=0.7$ | 84 | 25 | $P=0.03$ |
| DU history | | | | | | | | | |
| Yes | 24 | 50 | 1.4 | 15 | 60 | 1.3 | 9 | 33 | 1.2 |
| No# | 249 | 36 | $P=0.2$ | 105 | 46 | $P=0.34$ | 144 | 30 | $P=0.82$ |
| GU history | | | | | | | | | |
| Yes | 6 | 50 | 1.3 | 4 | 50 | 1.0 | 2 | 50 | 2.4 |
| No# | 267 | 37 | $P=0.5$ | 116 | 48 | $P=0.95$ | 151 | 29 | $P=0.54$ |
| NSAID use | | | | | | | | | |
| Yes | 97 | 41 | 1.1 | 37 | 48 | 1.0 | 60 | 37 | 1.7 |
| No# | 176 | 36 | $P=0.4$ | 83 | 48 | $P=0.9$ | 93 | 25 | $P=0.15$ |
| GI symptoms | | | | | | | | | |
| Yes | 57 | 43 | 1.1 | 30 | 43 | 1.1 | 27 | 44 | 1.1 |
| No# | 216 | 36 | $P=0.34$ | 90 | 35 | $P=0.4$ | 126 | 37 | $P=0.48$ |

# Reference group; * number of subjects; ** percentage of *H. pylori* seropositive; † relative risk; DU, duodenal ulcer; GU, gastric ulcer; NSAID, non-steroidal anti-inflammatory drug; GI, gastrointestinal.

*pylori* infection was significantly higher in the Melbourne Chinese population compared with this study population.[9]

Current data suggests that once *H. pylori* infection is acquired, it persists until old age. The progressive increase in seroprevalence might thus be considered a surrogate for incidence data. Age has previously been found to be a risk factor for *H. pylori* infection, as shown in this study. The apparent age risk may be attributable to the year of birth or cohort effect. The decrease in infection prevalence over the years may reflect improvement in socio-economic status and hygienic conditions in Australia. The long-term follow up of a cohort of subjects from birth should help our understanding of the epidemiology and the pathogenicity of *H. pylori* and, in particular, its host response.

## Risk factors for *H. pylori* infection

### *Sex*

No specific correlations with chronic gastritis have been observed as regards sex. Previous studies have indicated that the prevalence of *H. pylori* infection is similar in males and females.[1,3] However, in our data, the fre-

Table 4 The results of logistic regression analysis, comparing the prevalence of *Helicobacter pylori* with simultaneous adjustment for all variables shown

| Variables value | β | OR* | 95% CI** | *P* |
|---|---|---|---|---|
| Age | 0.0354 | 1.04 | 1.01–1.1 | 0.01 |
| Sex | –0.8783 | 2.41 | 1.3–4.5 | 0.01 |
| Household income | 0.3886 | 1.47 | 1.1–2.4 | 0.02 |
| Education levels | 0.1915 | 1.21 | 0.9–1.7 | 0.27 |
| Occupation | –0.0103 | 0.99 | 0.9–1.1 | 0.80 |
| Smoking habit | 0.7111 | 2.04 | 1.4–3.1 | 0.001 |
| Alcohol consumption | –0.2919 | 0.75 | 0.5–1.1 | 0.11 |
| Family size | 0.1793 | 1.2 | 0.7–2.2 | 0.56 |
| NSAID use | –0.3382 | 0.71 | 0.4–1.3 | 0.28 |

*Odds ratio; **95% confidence interval.

Table 5 The relationship between prevalence of *Helicobacter pylori* infection and smoking habit after adjustment for age, sex and socio-economic status

| Smoking habit | Odds ratio | 95% CI* | *P* value |
|---|---|---|---|
| Never smoked** | | | |
| Ex-smokers | 1.8 | 0.91–3.3 | 0.09 |
| Current smokers | 1.9 | 1.2–2.8 | 0.003 |

*95% confidence interval; **reference group.

quency of *H. pylori* infection in males was significantly higher than in females after controlling for age and other factors. Similar findings also occurred in a study of young adults from California.[10] The reasons for this difference and the clinical relevance is unclear. It is of interest to note that duodenal ulcer disease is more common in males than in females. Similarly, gastric adenocarcinoma is generally more common in males. Possible explanations for gender difference may relate to genetic factors, methods of hygiene and other social factors. A recent study from USA has shown that *H. pylori* infection was a significant risk factor for adenocarcinoma in women to a greater extent than in men.[11] These gender differences in *H. pylori* status and in clinical diseases require further study.

### *Socio-economic status*

In accordance with other studies, we have found that the prevalence of *H. pylori* is more common in lower household income level subjects. Women with lower education levels had a higher infection rate of *H. pylori*; however, when adjusted for other factors including age, occupation, alcohol consumption and smoking, this difference was not significant. In an Arkansas study of 247 healthy children, *H. pylori* infection was more common in low-income families than in high-income families.[12] Another study from the USA has shown that *H. pylori* seroprevalence was inversely associated with levels of income and education.[3] In Wales, the prevalence of *H. pylori* infection was highest in the lower social classes after adjustment for age.[13] In Saudi Arabia, *H. pylori* infection was less common in highly educated subjects.[14] This inverse relationship to socio-economic status is seen with gastritis[15] and other faecal–oral transmitted diseases including hepatitis A and poliomyelitis.[16,17]

Studies of *H. pylori* prevalence in the Japanese suggest the presence of at least two cohorts that differ in risk factors for *H. pylori* infection.[18] The high prevalence of *H. pylori* infection in persons over the age of 40 years is consistent with an early age of acquisition of *H. pylori* infection in Japan. It is postulated that this infection occurred in many individuals born prior to World War II when Japan was an under-developed, agrarian country. Since World War II, the country has become a major economic power with a higher standard of living and good sanitation. The frequency of *H. pylori* infection in those under the age of 30 years follows a pattern similar to that currently seen in the USA[3] and Australia. The prevalence of *H. pylori* infection in those 30–39 years of age appears to represent a transition between the pre-Westernized pattern and one of a developed Western country.

### *Smoking*

The association between *H. pylori* infection and smoking habits is controversial. A study from the USA has shown that there was no relationship between *H. pylori* infection and smoking.[3] A Danish study, however, found a trend relating *H. pylori* infection with smokers of tobacco ($P < 0.07$). This difference was not related to amounts of tobacco smoked.[19]

A study from China observed a significant correlation between *H. pylori* seropositivity and subjects who were ex-smokers and current smokers, as well as with the total current daily consumption of tobacco.[20] Our study has shown that the frequency of *H. pylori* was associated with both ex-smokers and current smokers; however, this association was only observed in current smokers after adjustment for age and sex. There was no association with the age at which subjects started smoking or daily consumption of cigarettes. The precise explanations for these associations are unclear. Smokers have a greater prevalence of peptic ulcer disease which is considered to be due to an increase in gastric acid secretion. Other effects of nicotine on the gastric mucosa, including altered mucosal blood flow and change in bicarbonate secretion, may also theoretically influence *H. pylori* colonization.

### *Other factors*

We found that alcohol consumption and NSAID use were not related to *H. pylori* status. These findings are similar to those of other studies[3,20] and confirm previous studies on chronic gastritis.[21] Our study also found that *H. pylori* infection was not associated with subjects' upper gastrointestinal tract symptoms, a finding similar to that reported from Denmark.[19] *Helicobacter pylori* may thus be present with histological evidence of gastritis without changes in symptoms, endoscopic findings

or degree of histological inflammation.[22] There are no data available for a relationship between *H. pylori* and the type of food intake. There is no evidence to suggest seasonal variation in rates of infection.

### *Transmission of* H. pylori

Human-to-human transmission of *H. pylori* infection has been postulated and studies showing a higher prevalence of *H. pylori* infection in family members of *H. pylori*-infected subjects support this concept. A recent study from China has shown a higher prevalence of *H. pylori* in urban populations as compared with subjects from rural areas. It was suggested that this is due to the high population density of urban areas.[23] However, in our study, the prevalence of *H. pylori* was not associated with the number of people living in the same household. The reason may be due to the lower population density and higher living standards in Australia, as well as to the higher percentage of older subjects living alone.

In summary, *H. pylori* is a common infection of the gastric mucosa in the Australian Anglo-Celtic population. This infection is present in both sexes with increasing prevalence with age. Because of the strong association with peptic ulcer disease and gastric malignancy, further studies are required to evaluate modes of transmission, effective treatment strategies and possible immunization against the infection.

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