Helicobacter pylori prevalence in non-ulcer dyspepsia — ethnic and socio-economic differences

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Abstract ***Helicobacter pylori*** **is an important cause of gastritis and a number of therapeutic trials suggest that it may be important in the genesis of duodenal ulcer recurrence. The reported prevalence of gastric colonisation by the organism varies considerably. The aim of this cross-sectional survey was to determine its prevalence in non-ulcer dyspeptics and to determine whether this is influenced by age, race, sex, socio-economic status, educational level and the number of persons sharing accommodation. One hundred and sixty-nine patients underwent endoscopy; biopsy specimens were taken from the antrum and *H. pylori* status was determined histologically. Gastric colonisation was found in 106 patients (63%). The prevalence showed a marked ethnic difference: 40% in whites and 71% in coloureds ($P < 0{,}001$). The ethnic groups were characterised by significant differences in socio-economic status ($P < 10^{-6}$), educational level ($P < 10^{-6}$), number of persons sharing accommodation ($P < 10^{-6}$) and age ($P < 0{,}001$). These same differences were found when comparing the *H. pylori*-positive and negative groups, but were less marked and could be attributed to the marked differences between ethnic groups. We conclude that *H. pylori* prevalence differs between the ethnic groups studied. This may be because of varying degrees of exposure risk.**

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Helicobacter pylori is accepted as the major cause of non-auto-immune (type B or antral) gastritis[1] and evidence from a number of therapeutic trials suggests that it may be an important factor in the pathogenesis of duodenal ulcer disease.[2-5]

The epidemiology of *H. pylori* infection is currently not well defined and crucial information, especially with regard to the mode of transmission of the organism, is still lacking. However, certain facts have been established. It is known that *H. pylori* is found in 90 - 100% of duodenal ulcer patients, 50 - 60% of patients with non-ulcer dyspepsia, 20 - 50% of asymptomatic subjects, and up to 75% of gastric ulcer sufferers in whom drug-induced disease has been excluded.[6] Furthermore, it has been well documented that the prevalence of the infection increases with age in asymptomatic subjects and non-ulcer dyspeptics.[7-8]

These observations apply, for the most part, to 'Western' populations; there is evidence that they may not be applicable in an African context. A number of studies have shown both an extremely high prevalence rate and an apparent early exposure to the organism in the African setting; up to 90% of teenagers studied were *H. pylori* positive.[9-11] Furthermore, it has been suggested that a number of factors may influence *H. pylori* prevalence. These include race,[12,13] socio-economic status[12,14] and close personal contact, e.g. as experienced by institutionalised subjects.[15]

Three South African studies have reported on the prevalence of gastric *H. pylori* colonisation in the local population.[16-18] These studies demonstrated a high overall prevalence of the organism that ranged from approximately 70%[16] to 82%.[17] However, they examined a number of gastroduodenal conditions and were not designed specifically to investigate factors which may influence the prevalence of *H. pylori* colonisation.

The aim of this cross-sectional study was, therefore, to determine the prevalence of *H. pylori* colonisation in a group of non-ulcer dyspeptic patients, and to investigate the influence of a number of factors related to socio-economic development on the prevalence of *H. pylori* infection.

Patients and methods

All dyspeptic patients presenting to the Gastro-enterology Clinic, Groote Schuur Hospital, and a Cape Town-based gastro-enterological practice were eligible for the study. The sample reflects the referral pattern of the clinic and practice in question and was not intended to be a population survey. Patients with peptic ulcer disease, gastric carcinoma and endoscopically detectable oesophagitis were excluded. All patients gave informed consent, and the study was approved by the Ethics and Research Committee of the University of Cape Town.

H. pylori status and the presence of gastritis were determined by histological assessment of adequate antral biopsy specimens, taken within 5 cm of the pylorus and stained by the Giemsa method. All histological assessments were carried out by one pathologist, experienced in the technique (K.J.), and classified as previously reported.[19]

All patients were interviewed by the participating endoscopist, and the following data collected:

Demographic information. This included details of age, racial group and sex.

Socio-economic class and occupation. Patients were either classified according to their own, or the family breadwinner's, status. Classification was according to the British Registrar-General's guidelines.[20] However, because a large section of the South African population falls mainly within the Registrar-General's classes IV and V, we used a modification of this system in which classes I, II and III are combined and called MRC class I. Class IV becomes MRC class II and class V becomes MRC class III. Pensioners were classified according to

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their previous occupation and unemployed patients were classified as MRC III.

Educational level. This was determined in terms of the number of successful years spent in school, tertiary education and in-service training.

The total number of people sharing accommodation with the patient.

Statistical analysis

Categorical data were analysed by means of the χ^2-test and continuous data by Student's *t*-test using the SAS, BMDP and EPISTAT statistical packages. Discrete multivariate analysis (DMA), which applies the principles of a logistic regression model, was used to control for race, sex, social class and number of persons sharing accommodation. A *P*-value of less than 0,05 was regarded as significant.

Results

The results are summarised in Tables I and II.

Adequate antral biopsy specimens were obtained from 169 of 179 patients studied — 47 white, 109 coloured and 13 black. Because of their small number and the compatibility of their demographic characteristics, the black patients were included with the coloured group for analysis.

TABLE I.
Summary of main differences between infected v. non-infected subjects

	H. pylori-positive	*H. pylori*-negative	*P*
Total sample	106 (63%)	63	
Male	55 (64%)	31	
Female	51 (61%)	32	NS
Coloured	87 (71%)	35	< 0,001*
White	19 (40%)	28	
Socio-economic classification			
MRC I	43 (53%)	38	
MRC II	27 (75%)	09	< 0,05‡
MRC III	36 (69%)	16	
Age (mean ± SD)	44 ± 13	53 ± 18	< 0,01†
Education			
≤ 10 yrs	77 (73%)	33 (52%)	
> 10 yrs	29 (27%)	30 (48%)	0,01*
Persons sharing accommodation			
≤ 3	47 (44%)	37 (59%)	
> 3	59 (56%)	26 (41%)	NS

* = χ^2-test.
† = Student's *t*-test.
‡ = Bartholomew's test for order.
NS = not significant.

TABLE II.
Summary of main differences between ethnic groups

	White	Coloured	*P*
Age (mean ± SD)	56 (±16)	44 (±14)	< 0,001*
Education			
≤ 10 yrs	4 (9%)	106 (87%)	
> 10 yrs	43 (91%)	16 (13%)	< 10^{-6}†
No. of persons sharing accommodation			
≤ 3	42 (89%)	42 (34%)	
> 3	5 (11%)	80 (66%)	< 10^{-6}†
Socio-economic classification			
MRC I	46	35	
MRC II	1	35	< 10^{-6}†
MRC III	0	52	

* Student's *t*-test.
† χ^2-test.

Overall prevalence

One hundred and six (63%) patients were infected with *H. pylori*.

H. pylori prevalence and sex

No significant difference in the prevalence of the infection was found between the sexes; 64% of male patients were infected compared with 61% of female patients.

H. pylori and age

In total, the infected individuals were younger than those not infected, the mean age (± SD) being 44 ± 13 years in the infected group, and 53 ± 18 years in the non-infected group ($P < 0,01$). This difference reflects the sample composition, where the mean age in the white group was 56 ± 16 years, and in the coloured group 43 ± 14 years ($P < 0,001$). Significantly more coloured patients than whites were infected before the age of 45 years (59% v. 33%; $P < 0,05$).

H. pylori and race

A marked racial difference in prevalence was found. The coloured group had a prevalence of 71% and the white group one of 40% ($P < 0,001$). When we applied the multivariate model, controlling for the factors noted above, race remained a predictive factor for *H. pylori* infection.

H. pylori and socio-economic class

A significant difference in prevalence was found between the social classes, with an apparent inverse relationship between socio-economic classification and *H. pylori* infection ($P < 0,05$). However, with these particular data, especially in the South African context, it is difficult to separate the influence of racial grouping and socio-economic status and we found that this apparent significant difference in prevalence between our three social groupings could be ascribed to the racial distribution in these groups. In terms of the DMA model, racial grouping and social class were so strongly associated that social class could not be implicated as an independent predictor of *H. pylori* infection.

H. pylori and education

There were significant differences in the educational levels of the infected and non-infected groups. In the infected group, 77 (73%) of the patients had had less than 10 years of education (the equivalent of a Standard 8 or Junior Certificate), compared with only 33 (52%) in the non-infected group ($P = 0,01$). Once again, this reflects the sample composition and the marked difference in educational level between the two racial groupings (Table II).

H. pylori and the sharing of accommodation

The non-infected subjects appeared to have fewer persons sharing their accommodation than the infected patients. Thirty-seven (59%) of the non-infected group had 3 or fewer persons sharing accommodation, compared with 47 (44%) of the infected group. However, this difference did not reach significance ($P = 0,09$), and again reflects the marked differences noted between racial groups. When investigated further according to

the DMA model, with control for race, sex and social class, no significant differences could be shown between infected and non-infected subjects.

Table II highlights some of the key differences between the two race groups and clearly demonstrates the marked differences with regard to age, level of education, the number of persons sharing accommodation and their distribution in the socio-economic groups.

Discussion

This study confirms the high prevalence of *H. pylori* infection documented by other South African workers.[16-18] It has, however, demonstrated a marked difference in the racial distribution of the infection, a finding that supports the results of the Houston[12] and Singapore[13] groups. Unfortunately, but not unexpectedly, socio-economic status is strongly linked to racial grouping in this sample, so that the effect of either cannot be distinguished in the present study design; this makes it impossible to attribute causality. However, while a genetically based difference in the susceptibility of individuals to *H. pylori* colonisation cannot be excluded, it seems more likely that the marked racial differences in prevalence shown reflect different exposure risks to the organism. The significant differences noted between our racial groups with regard to socio-economic class, level of education and number of persons sharing accommodation, suggest that these factors may be important in determining the prevalence of the infection, possibly by raising the risk of exposure to the organism. Living habits, personal hygiene and the availability of basic sanitation and clean water are known to influence the epidemiology of most communicable diseases. The effect of apparent socio-economic deprivation on the increased prevalence of *H. pylori* infection had of course already been noted by the Oxford and Houston-based groups.[12,14]

The finding in this sample that the infected patients were younger than their non-infected counterparts, although contrary to documented Western experience, is in line with other African studies, which have documented a high prevalence of infection in young subjects,[10,11] and suggest that infection occurs early in 'Third-World' environments.

H. pylori has, of course, not been isolated from the environment, and available data suggest that it is spread by human association. To what extent the spread is determined by close personal contact or contamination of the individual's immediate environment is not clear. Both factors may be important. *H. pylori* has been isolated from the dental plaque of a patient[21] and is prevalent among institutionalised patients; this prevalence increases with the length of the institutionalisation.[15] It is known to occur in familial clusters[22] and this suggests transmission by intimate contact. On the other hand, it has also been shown to survive for at least a week in river water under laboratory conditions[23] and in a recent Peru-based study, the water supply of a community was implicated as a possible source of the infection.[24] These factors may, of course, all be influenced by low socio-economic standards.

In conclusion, the results of this study suggest that the prevalence of *H. pylori* infection may be influenced by socio-economic factors. Spread of the organism may be brought about by direct interpersonal contact, or via contamination of the individual's immediate environment. Further studies, aimed at detecting the organism in the environment by means of technology not available before (such as DNA amplification techniques), are needed to identify possible sources of the infection.

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