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Inverse Relationship Between Alcohol Consumption and Active *Helicobacter pylori* Infection: The Bristol *Helicobacter* Project

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OBJECTIVE: The aim of this study was to examine whether smoking or consumption of alcohol or coffee is associated with active *Helicobacter pylori (H. pylori)* infection.

METHODS: This was a cross-sectional population study conducted as part of a randomized controlled trial of *H. pylori* infection eradication in southwest England. A total of 10,537 subjects, recruited from seven general practices, underwent ^{13}C-urea breath testing for active infection with *H. pylori* and provided data on smoking, usual weekly consumption of alcohol, and daily intake of coffee.

RESULTS: Smoking or coffee consumption were not related to active *H. pylori* infection. Total alcohol consumption was associated with a small, but not statistically significant, decrease in the odds of infection. After adjustment for age, sex, ethnic status, childhood and adult social class, smoking, coffee consumption, and intake of alcoholic beverages other than wine, subjects drinking 3–6 units of wine/wk had an 11% lower risk of *H. pylori* infection compared with those who took no wine: OR = 0.89, 95% CI = 0.80–0.99. Higher wine consumption was associated with a further 6% reduction in the risk of infection: OR = 0.83, 95% CI = 0.64–1.07. Intake of 3–6 units of beer (but no greater intake) was associated with a similar reduction in the risk of infection when compared to no beer intake (OR = 0.83, 95% CI = 0.75–0.91).

CONCLUSIONS: This study indicates that modest consumption of wine and beer (approximately 7 units/wk) protects against *H. pylori* infection, presumably by facilitating eradication of the organism. (Am J Gastroenterol 2002;97: 2750–2755. © 2002 by Am. Coll. of Gastroenterology)

INTRODUCTION

Helicobacter pylori (*H. pylori*) gastritis is one of the most prevalent bacterial infections in humans. The infection is usually acquired in childhood but may be acquired and eliminated spontaneously throughout adulthood (1). Little is known about the factors, apart from poor living conditions during childhood, that affect either acquisition or elimination of the organism (2). Lifestyle factors operating during adulthood such as smoking and alcohol consumption may influence spontaneous eradication of the organism. Studies investigating the relationship between antibody evidence of *H. pylori* infection and these factors have provided inconsistent findings (3–6), but serological tests may misclassify as still infected those individuals in which the organism has been eradicated. Recent work investigating subjects with evidence of active *H. pylori* infection, as measured by the ^{13}C-urea breath test, found no association with smoking, whereas coffee consumption was positively related to infection (7, 8). A strong graded inverse association between *H. pylori* infection (7) and alcohol consumption (especially wine) was seen, but a subsequent report indicated that the relationship was U-shaped and unrelated to the type of beverage consumed (9). Our aim was to examine the relationships between these lifestyle variables and active *H. pylori* infection in a large cross-sectional study in the United Kingdom.

MATERIALS AND METHODS

The Bristol *Helicobacter* Project is a large community-based, randomized, controlled trial of the effect of *H. pylori* eradication on dyspepsia, quality of life, and use of health services. Between 1996 and 1998, all eligible patients aged 20–59 yr who were registered with seven primary health care centers in southwest England (six located in a town north of Bristol and one within the city of Bristol) were invited to participate in the study. Participants fasted for at least 4 h before a ^{13}C-urea breath test. They were given 2.8 g of citric acid in 200 ml of commercially available orange juice (Tesco, Chesthunt, UK) before two breath samples were collected in stoppered tubes. Participants then ingested 100 mg of ^{13}C-labeled urea (BSIA, Southall, UK) dissolved in 50 ml of orange juice, and two more breath samples were taken 30 min later. Samples were analyzed using mass spectrometry. An excess of δ^{13} CO_2 of 3.5/ml was defined as indicating *H. pylori* positivity (10).

Table 1. Sociodemographic and Lifestyle Characteristics of the Study Population

	H. pylori Negative, N (%)	*H. pylori* Positive, N (%)	All Participants, N
Age, yr*			
<40	1005 (30.8)	250 (15.3)	1255 (25.6)
40–49	1121 (34.3)	522 (32.0)	1643 (33.5)
50–59	1142 (34.9)	862 (52.8)	2004 (40.9)
All ages	3268	1634	4902
Sex*			
Male	1473 (45.1)	798 (48.8)	2271 (46.3)
Female	1795 (54.9)	836 (51.2)	2631 (53.7)
Ethnic origin*			
White	3199 (99.4)	1579 (97.4)	4778 (98.7)
Other	18 (0.6)	42 (2.6)	60 (1.2)
Social class*			
I	214 (6.6)	97 (6.0)	311 (6.3)
II	919 (28.4)	395 (24.5)	1314 (26.8)
IIINM	1015 (31.4)	423 (26.2)	1438 (29.7)
IIIM	562 (17.4)	366 (22.7)	928 (19.1)
IV	403 (12.5)	254 (15.8)	657 (13.4)
V	121 (3.7)	76 (4.7)	197 (4.0)
Total intake of alcohol (units/wk)			
None	495 (15.8)	282 (18.3)	777 (13.1)
1 or 2	398 (12.7)	212 (13.8)	610 (13.1)
3–6	727 (23.2)	321 (20.9)	1048 (22.5)
7–13	720 (23.0)	339 (22.1)	1059 (22.7)
14–20	387 (12.4)	189 (12.3)	576 (12.3)
≥21	401 (12.8)	194 (12.6)	595 (12.8)
Intake of beer, lager, or cider (units/wk)*			
None	1442 (45.1)	767 (48.2)	2209 (46.1)
1 or 2	461 (14.4)	192 (12.1)	653 (13.6)
3–6	555 (17.3)	230 (14.5)	785 (16.4)
7–13	346 (10.8)	181 (11.4)	527 (11.0)
≥14	396 (12.4)	221 (13.9)	617 (12.9)
Intake of wine (units/wk)*			
None	1096 (34.2)	621 (38.9)	1717 (35.8)
1 or 2	881 (27.5)	445 (27.9)	1326 (27.6)
3–6	806 (25.2)	355 (22.2)	1161 (24.2)
≥7	421 (13.2)	175 (10.9)	596 (12.4)
Intake of spirits (units/wk)			
None	1960 (61.5)	979 (61.7)	2939 (61.5)
1 or 2	676 (21.2)	314 (19.8)	990 (20.7)
≥3	549 (17.2)	294 (18.5)	843 (17.7)
Cigarette smoking			
Never smoked	1758 (54.1)	834 (51.5)	2592 (53.3)
Ex-smoker	743 (22.9)	384 (23.7)	1127 (23.2)
Current smoker, <20/day	467 (14.4)	232 (14.3)	699 (14.4)
Current smoker, ≥20/day	281 (8.6)	168 (10.4)	449 (9.2)
Coffee consumption (cups/day)			
None	641 (19.8)	339 (20.9)	980 (20.1)
1–4	1733 (53.4)	844 (52.0)	2577 (53.0)
≥5	869 (26.8)	440 (27.1)	1309 (26.9)

* $p < 0.05$ based on χ^2 test.

Participants also completed a self-administered questionnaire that gathered lifestyle information including data on smoking history, current weekly alcohol intake, and daily coffee consumption. Intake of alcohol was provided in three groups: 1) beer, lager, and cider, 2) wine, and 3) spirits. Alcohol intake was calculated in units/week, with 1 unit equal to one glass of wine, a standard measure of spirits, or half a pint of beer or cider. These variables were categorized as in Table 1. Other data collected by questionnaire included information on living conditions experienced during childhood (tenure of accommodation and sharing of a bedroom) and measures of adult socioeconomic status (occupation, tenure of accommodation, number of cars in the household, and highest educational qualification). Occupation in adulthood was coded into nonmanual social classes I, II, and IIINM, and manual social classes IIIM, IV, and IV accord-

ing to the Standard Occupational Classification of The Office of Population Censuses and Surveys, Government Statistical Service, UK. This study includes data relating to all participants who tested positive for *H. pylori* infection and a random sample of *H. pylori* negative subjects to give a *H. pylori* negative to positive ratio of 2:1.

Relationships between lifestyle variables (smoking, alcohol, and coffee intake) and active *H. pylori* infection were assessed using logistic regression (Stata version 7; Stata, College Station, TX). We accounted for the clustered nature of the data by defining the general practice at which participants were registered as the primary sampling unit in the logistic regression models. Models were constructed for total alcohol intake with adjustment for age, sex, ethnic origin (white/nonwhite), childhood living conditions, and measures of adulthood socioeconomic status. The final total alcohol model also included adjustment for smoking and coffee consumption. Separate models were then constructed for alcohol intake in its various forms with adjustment as for total alcohol intake, but the final model also included adjustment for intake of the other forms of alcohol. Tests for linear trends across categories were applied.

RESULTS

A total of 27,536 individuals were eligible to participate in the study. The main reason for ineligibility to participate was known sensitivity to the constituents of the *H. pylori* eradication regimen (ranitidine bismuth citrate, and clarithromycin). In all, 10,537 subjects (38.3%) gave informed consent to take part, underwent a ^{13}C-urea breath test, and completed the questionnaire. Of the participants, 1,634 (15.5%) tested positive for *H. pylori* infection. Data relating to 3,268 of the *H. pylori* negative subjects were randomly selected, providing a total study population of 4,902 subjects. Table 1 shows the lifestyle and sociodemographic characteristics of the study population. Nonmanual social classes were slightly over-represented, with 62.8% of participants from nonmanual social classes compared to 52.3% for the general population in this region of England; the proportions of the working age population in the southwest region in 1998 in social classes I, II, IIINM, IIIM, IV, and V were 5.0%, 26.0%, 21.3%, 17.9%, 15.3%, and 4.9%, respectively (11). Very few of the participants (1.2%) were of nonwhite ethnic origin. The majority of participants (83.4%) consumed some alcohol, with more taking wine (64.2%) than beer (53.9%) or spirits (38.5%). More than half of the participants had never smoked, and one fifth did not drink coffee. The mean number of cups of coffee consumed per day was 3.8 and the median 3.0. Smoking and coffee consumption were both related to alcohol intake: 27.1% of participants consuming 10 or more units of alcohol/wk were current smokers, compared to 21.6% of subjects taking less alcohol (χ^2 18.1, df 1, $p < 0.01$). Similarly, 31.1% of consumers of 10 or more units of alcohol/wk took five or more cups of coffee/day, compared to 24.6% of consumers of lower amounts of alcohol (χ^2 22.6, df 1, $p < 0.01$).

Active *H. pylori* was positively associated with increasing age, male sex, lower social class, nonwhite ethnic origin, and sharing a bedroom with other children during childhood (Table 1). Total alcohol consumption was inversely associated with active *H. pylori* infection. The OR and 95% CI for infection among consumers of 1–2, 3–6, 7–13, 14–20, and ≥21 units of alcohol/wk were as follows: OR = 0.93, 95% CI = 0.71–1.23; OR = 0.77, 95% CI = 0.62–0.97; OR = 0.83, 95% CI = 0.70–0.98; OR = 0.86, 95% CI = 0.73–1.01; OR = 0.85, 95% CI = 0.69–1.04 ($p < 0.01$ for trend). This apparent protective effect of alcohol consumption was attenuated on adjustment for age, sex, measures of childhood and adult socioeconomic status, smoking, and coffee consumption; OR and 95% CI for infection in the alcohol consumption groups were as follows: OR = 1.12, 95% CI = 0.81–1.56; OR = 0.92, 95% CI = 0.72–1.18; OR = 0.95, 95% CI = 0.75–1.20; OR = 0.97, 95% CI = 0.81–1.16; and OR = 0.94, 95% CI = 0.73–1.20 ($p = 0.08$ for trend).

Table 2 shows the unadjusted and adjusted relationships between active *H. pylori* infection and cigarette smoking, coffee consumption, and intake of alcohol in its various forms. Infection was not related to cigarette smoking, either before or after adjustment for potential confounders. Coffee consumption was associated with slightly reduced odds of active *H. pylori* infection, but conventional statistical significance was not reached. Intake of wine was inversely related to active *H. pylori* infection. The unadjusted OR and 95% CI for infection among subjects who consumed 1–2, 3–6, and ≥7 units of wine/wk compared with those taking no wine were as follows: OR = 0.89, 95% CI = 0.79–1.01; OR = 0.78, 95% CI = 0.63–0.97; and OR = 0.73, 95% CI = 0.51–1.05. This inverse trend, although slightly attenuated, survived adjustment for age, sex, ethnic origin, childhood living conditions, adult socioeconomic status, smoking, coffee consumption, and intake of alcohol in other forms. The adjusted ORs between active *H. pylori* infection and categories of beer, lager, and cider intake indicated a U-shaped relationship. The OR and 95% CI for infection among consumers of 1–2, 3–6, 7–13, and ≥14 units of beer per week were as follows: OR = 0.89, 95% CI = 0.76–1.05; OR = 0.83, 95% CI = 0.75–0.91; OR = 1.02, 95% CI = 0.91–1.15; and OR = 1.05, 95% CI = 0.80–1.41. There was no relationship between intake of spirits and *H. pylori* infection.

Sex specific models did not show the relationships to differ between the sexes, and exclusion of participants with a history of peptic ulceration (n = 236, 132 *H. pylori* positive) made little difference in the observed ORs.

DISCUSSION

To date, there have been very few population-based studies that have examined the relationships between active *H. pylori* infection, as determined by the ^{13}C breath test, and

Table 2. Unadjusted and Adjusted Relationships Between Alcohol, Cigarette Smoking, and Coffee Intake and Active *H. pylori* Infection

Alcohol/Nicotine Use	Model 1 (Unadjusted): OR α (95% CI)	Model 2: OR α (95% CI)	Model 3: OR α (95% CI)
Intake of alcohol (units/wk)			
None	1.00*	1.00*	1.00*
1 or 2	0.93 (0.71–1.23)	1.12 (0.80–1.56)	1.12 (0.81–1.56)
3–6	0.77 (0.62–0.97)	0.93 (0.72–1.20)	0.92 (0.72–1.18)
7–13	0.83 (0.70–0.98)	0.94 (0.75–1.18)	0.95 (0.75–1.20)
14–20	0.86 (0.73–1.01)	0.95 (0.79–1.14)	0.97 (0.81–1.16)
≥20	0.85 (0.69–1.04)	0.95 (0.73–1.24)	0.94 (0.73–1.20)
			p for trend = 0.08
Intake of wine (units/wk)			
None	1.00*	1.00*	1.00*
1 or 2	0.89 (0.79–1.01)	1.02 (0.90–1.16)	1.03 (0.87–1.23)
3–6	0.78 (0.63–0.97)	0.89 (0.81–0.98)	0.89 (0.80–0.99)
≥7	0.73 (0.51–1.05)	0.85 (0.63–1.15)	0.83 (0.64–1.07)
			p for trend = 0.03
Intake of beer, lager and cider (units/wk)			
None	1.00*	1.00*	1.00*
1 or 2	0.78 (0.66–0.93)	0.90 (0.76–1.06)	0.89 (0.76–1.05)
3–6	0.78 (0.69–0.88)	0.81 (0.71–0.91)	0.83 (0.75–0.91)
7–13	0.98 (0.88–1.10)	0.99 (0.85–1.14)	1.02 (0.91–1.15)
≥14	1.05 (0.84–1.31)	1.03 (0.79–1.35)	1.05 (0.80–1.41)
			p for trend = 0.64
Intake of spirits (units/wk)			
None	1.00*	1.00*	1.00*
1 or 2	0.93 (0.72–1.21)	1.02 (0.76–1.37)	1.07 (0.79–1.43)
≥3	1.07 (0.88–1.31)	1.00 (0.84–1.19)	1.05 (0.89–1.23)
			p for trend = 0.49
Cigarette smoking			
Never smoked	1.00*	1.00*	1.00*
Ex-smoker	1.09 (0.87–1.36)	0.96 (0.80–1.15)	0.96 (0.82–1.13)
Current smoker, <20/day	1.05 (0.95–1.15)	0.97 (0.85–1.11)	0.94 (0.82–1.08)
Current smoker, ≥20 day	1.26 (1.01–1.57)	1.06 (0.71–1.57)	1.04 (0.73–1.50)
			p for trend = 0.95
Coffee consumption (cups/day)			
None	1.00*	1.00*	1.00*
1–4	0.92 (0.68–1.25)	0.93 (0.70–1.24)	0.92 (0.66–1.30)
≥5	0.96 (0.83–1.10)	0.99 (0.87–1.12)	0.97 (0.82–1.15)
			p for trend = 0.73

Model 2: Adjusted for age sex, ethnicity, and measures of childhood and adult socio-economic status. Model 3: Further adjustment for variables in table.
* Reference category.

lifestyle factors (7–9, 12), and no studies investigating residents in the UK have been published. Important strengths of this study were its large size (the largest study to date) and inclusion of a wide range of measures of socioeconomic status, which is potentially a major confounder of the relationship between alcohol intake and *H. pylori* infection. As in all community-based studies, participants in this study were self-selected and cannot be assumed to be representative of the population. Some care must therefore be taken in generalizing the findings beyond the study population. The response rate seems low, but it must be borne in mind that this study was designed as an interventional study and the overall response rate was 12.5% higher than that of a comparable study recently undertaken in Leeds, UK (13). We found what seems to be a low overall prevalence of *H. pylori* infection (15.5%). This was due in part to the inclusion of young subjects, but selection bias may also have contributed to this low figure. However, with one exception (14), previous studies in the UK (3, 15) showing a high prevalence of *H. pylori* infection were performed a decade before this study and employed serology, not the ^{13}C breath test, to diagnose the infection.

The cross-sectional nature of this study presents some limitations, particularly with respect to providing evidence that the consumption of alcohol precedes eradication of the organism. It is possible that eradication of the organism (either spontaneous or after specific therapy) could lead to a reduction in dyspeptic symptoms and allow subjects to increase the amount of alcohol that they usually consume. We did not collect data on whether subjects had received specific anti–*H. pylori* therapy, and the relationship between alcohol consumption and infection may have been more

clearly observed if such individuals had been excluded. However, it should be noted that the subjects were recruited from the general population and were not necessarily symptomatic, and the proportion of subjects who previous received successful eradication therapy is likely to have been low. The cross-sectional nature of the study also confined the collection of data on alcohol consumption to one time point, which may have resulted in an incomplete assessment of usual alcohol intake.

Recent sero-epidemiological studies in diverse populations (5, 6, 16) indicate a negative association between *H. pylori* infection and alcohol intake. An Italian population–based study (17) has shown a positive association between active *H. pylori* infection and frequency of alcohol intake, but no adjustment was made for age or socioeconomic status. Two German studies have demonstrated a strong negative association between alcohol consumption and active *H. pylori* infection (7, 8), particularly among wine drinkers. However, when the two German studies were pooled with one additional study (also in Germany), the relationship between alcohol intake and *H. pylori* infection was U-shaped and seemed to be unrelated to beverage type (9). We did not find the relationship between alcohol consumption and active *H. pylori* infection to be U-shaped; instead there seemed to be a weak protective effect with increasing consumption of alcohol that did not reach conventional statistical significance. Examination of alcohol intake by type of beverage showed an inverse relationship with wine consumption, with a 17% reduction in the risk of infection among subjects consuming 7 or more units of wine/wk. Drinking 3–6 units of beer, lager, or cider was associated with the same reduction in risk of infection, which was not seen in subjects consuming higher amounts of beer. Consumption of spirits was not related to active *H. pylori* infection. These results indicate that consumption of moderate amounts of alcohol in the form of wine, beer, lager, or cider may protect against *H. pylori* infection. As this infection is predominately acquired in childhood (17), it is likely that alcoholic beverages exert this effect by facilitating eradication of the organism rather than by preventing its acquisition. Consumption of alcoholic beverages increases gastric acid secretion and speeds gastric emptying, which could aid eradication of *H. pylori* (18). These effects are most apparent for wine and beer consumed in moderate amounts (19, 20), and distilled products do not seem to stimulate acid secretion (21), which is in keeping with our findings. Wine has long been known to protect against acute GI infection; and, more recently, the antibacterial effect of wine against enteropathogenic organisms (*e.g.*, *Salmonellae*) has been proved through *in vitro* studies (22, 23) and in an outbreak situation (24). It also seems that the antibacterial effect of wine exceeds that of ethanol of the same dilution and pH, and this effect has been shown to be extended to *H. pylori* (25, 26). It has recently been suggested that the active antibacterial compound in red wine is resveratrol (3,4,5-trihydroxy-trans-stilbene), a natural diphenolic antioxidant (26). It is not known whether beer displays a similarly increase in antibacterial activity beyond that of ethanol alone; but, like wine, it is a rich source of polyphenolic compounds (27), which may have antibacterial activity. It is therefore possible that the antibacterial effects of wine and beer may be unrelated to their alcohol content.

A socioeconomic gradient in *H. pylori* infection is well recognized, with lower infection rates in subjects with higher social status (3, 15); and, in the UK, wine consumption is more common in higher (nonmanual) social classes (28, 29). The same may be true for moderate beer consumption. It is possible that the apparent protective effect of wine or beer consumption against *H. pylori* infection observed in this study merely reflects the socioeconomic distribution of the infection. Although we adjusted the relationship for an extensive range of measures of childhood and adult socioeconomic status, residual confounding by this factor cannot be excluded with certainty. Alternatively, it may be inappropriate to adjust this relationship for socioeconomic status if part of the observed socioeconomic gradient in *H. pylori* infection results from a protective effect of moderate wine or beer consumption.

In this study, cigarette smoking was not associated with active *H. pylori* infection. In fact, smoking was not associated with even the modest (but not statistically significant) increases in odds of active infection seen by Brenner *et al.* in a recent study of patients presenting in primary care (7). A subsequent study by Brenner *et al.* (8) involving employees at a health insurance company and their families found no association between smoking and active *H. pylori* infection. In contrast to these two studies, in which consumption of coffee was associated with approximately a 2-fold increase in odds of active *H. pylori* infection, the relationship between coffee consumption and *H. pylori* infection observed in this study was negative (although not statistically significant). Mean and median coffee consumption in our study was very similar to that seen in the primary care study by Brenner *et al.*, and recategorization of daily consumption of coffee using these investigators' thresholds made little difference to the OR that we observed (data not shown). It is unlikely that smoking or coffee consumption are either risk factors for, or protective factors against, active *H. pylori* infection.

Our data indicate that modest consumption of wine or beer (approximately 7 units/wk) protects against active *H. pylori* infection, presumably by facilitating eradication of the organism. However, the data do not enable us to comment on the relevance of patterns of wine and beer consumption.

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