Seroprevalence and some correlates of *Helicobacter pylori* at adult ages in Gülveren Health District, Ankara, Turkey

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SUMMARY

The purpose of the study was to detect the frequency and distribution of *Helicobacter pylori* in the Gülveren Health Centre service area among residents aged between 25 and 64 years and to evaluate the relation of *H. pylori* infections with general health status, socioeconomic status, and some lifestyle habits. The study included a representative sample of Gülveren Health Centre residents, aged between 25 and 64 years. A stratified random sample of 1672 individuals was selected for study purposes out of 10 569 residents, stratified by age and gender. A standardized questionnaire was completed for all study participants using a face-to-face interview and all participants were invited to the local health centre for a thorough physical examination and blood tests. The overall prevalence of *H. pylori* was found to be 77.5% among individuals aged between 25 and 64 years. The frequency of *H. pylori* was higher among individuals with low socioeconomic status; those who migrated to Ankara after the age of 20 years; members of large families (household size of 4 or above); non-alcohol drinkers; and those who regularly drink tea.

INTRODUCTION

Helicobacter pylori is one of the most common bacterial infections in the world. Previous research indicates that *H. pylori* is the most common cause of type-B gastritis, and peptic and duodenal ulcers in adults [1]. In 1994 the WHO declared *H. pylori* to be a primary carcinogen for gastric ulcer [2]. Several studies have been conducted on the diagnosis and treatment of *H. pylori*-related diseases aiming for a decrease in the *H. pylori*-related disease burden. Although *H. pylori* prevalence has been found to be considerably high in many countries and several risk factors have been identified, evidence on transmission characteristics are still not conclusive. Evidence on transmission characteristics is mostly based on evaluation of the

association between *H. pylori* infection and potential risk factors. Thus, population-based studies are valuable to determine the epidemiological characteristics of *H. pylori* infections.

In recent years prevalence of *H. pylori* has not shown a significant change in developing countries, despite a decrease in its incidence in developed countries. Research has indicated that *H. pylori* seropositivity was significantly associated with age, gender, socioeconomic status (SES), and personal and environmental hygiene [3].

In developed countries, low H. pylori prevalence rates of approximately 8% among children show a considerable increase as age increases and 40-60% of the population become infected by the age of 60 years [4].

In developing countries, in contrast, exposure to *H. pylori* occurs mainly between the ages 1–7 years [5] and the prevalence rates in adults reaches up to 80%.

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A *H. pylori* study conducted in Germany found a prevalence rate of 82·1% among patients of Turkish ancestry aged 25–34 years whereas it was 40·0% in German patients with similar characteristics [6].

Several studies have been conducted in Turkey on patients admitted to hospitals and have found that frequency of H. pylori infections changed with age, educational status, SES, number of individuals in the household, and environmental factors. A hospitalbased study evaluated the association between H. pylori infection and age, SES, history of migration, family size, sharing the same bed, drinking of unboiled or well water, and disturbed environmental conditions. Out of these factors, low SES and migration from rural to urban settings were found as significant predictors of *H. pylori* infections [7]. The urban population of Turkey increased at an average of 4.5% in the 1980s, while the rural population was decreasing in absolute terms. Migration in general was directed to three big cities (Istanbul, Ankara, Izmir) and also to Adana and Bursa. The reason for the migration from rural areas to urban areas lies in the diminishing possibilities of existence in the rural regions, not in the attraction of the big cities [8]. This flow, by the creation of squatter areas around those cities results in a distorted growth of urban areas [9].

H. pylori infection is a priority public-health concern given its association with gastritis, peptic ulcer and gastric cancer. Population-based studies on the frequency of H. pylori infection and associated risk factors in local settings of low SES and disturbed environmental conditions are of value in Turkey, a developing country.

This study aimed (1) to determine the prevalence of *H. pylori* infection in the Gülveren Health Centre service area, a low-to-moderate socioeconomic setting, among residents aged 25–64 years; and, (2) to evaluate the relationship between *H. pylori* infection and some demographic characteristics (age, gender, migration history, size of household, etc.), and selected lifestyle habits (smoking, coffee- and tea-drinking habits, etc.).

METHODS

Gülveren Health Centre provides services to one of the oldest slum settings in Ankara. According to the health centre's records, the population was 22958 in 1998, the study year. In the same year the illiteracy rate in the area was 10·3%, the average socioeconomic level was low-to-moderate and the population

aged between 25 and 64 years was 10 567. For study purposes, a sample size of 1600 was required, using seroprevalence rates of *H. pylori* in some selected regions of Turkey [10]. A representative, stratified random sampling was established using eight gender and age (25–34, 35–44, 45–54 and 55–64 years) strata. Sampling was disproportionate to size and included 200 individuals from each stratum.

Out of all study participants, blood samples could be obtained from 1182 (73.9%) and 1089 (68.1%) samples could be evaluated serologically for *H. pylori* infection. All analyses were corrected for sampling strategy, using sampling weights, calculated as inverses of sampling fractions. Weighted and unweighted analyses were compared and contrasted to check for the robustness of sample weights. Individuals lacking *H. pylori* serological test results were treated as missing observations in the calculation of sampling weights.

Data collection

Data were collected by trained interviewers using a face-to-face interview, and a standardized question-naire. Study participants were interviewed at their homes and were subsequently invited to the local health centre for blood sampling and ECG evaluation. *H. pylori* seroprevalence was evaluated using frozen serum samples, stored at $-20\,^{\circ}\text{C}$ for 3 years. *H. pylori* anti-IgG was detected using the ELISA (enzymelinked immunosorbent assay) technique, and Trinity Biotech kit (Co. Wicklow, Ireland). The sensitivity and specificity of this method are 96% and 93% respectively. In serological analyses, the cut-off point was chosen as $1\cdot1$ U IgG/ml, and individuals with values above this were considered as *H. pylori*-positive.

Statistical analyses

Analyses included univariate, bivariate, stratified analyses and multiple logistic regression modeling. For study purposes, two composite indices were created. The first one, the 'SES index', is based on individuals' educational status, occupation, vehicle ownership, housing tenure and the number of household members working outside home. The SES index was categorized into 'high', 'moderate' and 'low' levels, corresponding to scores of 0–2, 3–5, and \geqslant 6. Another composite index, the 'risk score for H. pylori' included data on age, size of household, age at

| Age (yr)* | Total no. | | Seropositivity (%)† | | OR (95% CI) | |
|-----------|-----------|--------|---------------------|--------|------------------|------------------|
| | Male | Female | Male | Female | Male | Female |
| 25–34 | 93 | 157 | 73.1 | 72.0 | 1 | 1 |
| 35-44 | 130 | 183 | 79.2 | 74.9 | 1.40 (1.20-1.63) | 1.16 (0.99–1.35) |
| 45-54 | 109 | 165 | 75.3 | 83.6 | 1.12 (0.92–1.35) | 1.99 (1.59–2.48) |
| 55-64 | 109 | 143 | 79.8 | 81.2 | 1.45 (1.14–1.86) | 1.69 (1.34–2.13) |
| Total | 441 | 648 | 77.1 | 77.7 | | |

Table 1. Distribution of Helicobacter pylori seroprevalence by age and gender (Gülveren Health Centre area, 1998)

migration to Ankara, SES (three groups), cigarette smoking, tea-drinking and alcohol consumption. The maximum score possible was 8.

In multivariate analyses, the dependent variable was seropositivity for *H. pylori* (positive vs. negative) and covariate variables included sociodemographic characteristics, lifestyle habits, and variables representing health/disease status. Stratified analyses suggested potential effect modification of gender on the association between H. pylori seropositivity and SES, and alcohol consumption respectively. Therefore, interaction terms for such factors were tested in multivariate logistic regression models. Inter-variable correlations were evaluated prior to modelling: none of the correlations were above 0.25. Dummy variables were created to study various risk factors. Odds ratios (OR) and relevant 95% confidence intervals (CI) were presented for potential risk factors of interest. All analyses were conducted using SPSS statistical software package, version 10.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

The study included 441 male and 648 female residents of the Gülveren Health Centre service area, Ankara. In the study population the average value for H. pylori IgG (\pm standard deviation) was $1\cdot54\pm0\cdot91$ U IgG/ml, ranging between $1\cdot34$ and $3\cdot4$ U IgG/ml. The H. pylori seroprevalence in the study group was $77\cdot5\%$. Prevalence rates were $77\cdot1$ and $77\cdot7\%$ in males and females respectively and there was no statistically significant difference among gender groups ($P=0\cdot79$). In males the average age was $38\cdot6\pm10\cdot0$ years (median age 44 years) and $38\cdot0\pm9\cdot2$ years (median age 44 years) in individuals with H. pylori infection and those free of infection respectively. In females, in contrast, age of H. pylori positives

was significantly higher $(39.4\pm10.4 \text{ years})$ than that of females free of *H. pylori* $(36.9\pm9.9 \text{ years})$ (t=50.5, P<0.001). The median ages were 45 and 41 years among *H. pylori*-seropositive females and males respectively. In males seropositivity showed a peak in age groups 35–44 and 55–64 years (Table 1), whereas seropositivity steadily increased with age in females, and reached 80% for >45 years.

Using the composite index for SES, no study participant had 'high' SES (Table 2). *H. pylori* seropositivity was higher among individuals with 'low' SES than those with 'moderate' SES, regardless of gender. However, results of stratified analyses suggested a modifying effect of gender on the association between SES and *H. pylori* seropositivity. The seroprevalence of *H. pylori* was 84·0% in males with low SES, and significantly higher [OR 2·42 (95% CI 2·11–2·77)] than that in males with moderate SES level. In females, the odds of *H. pylori* infection were 1·77 (95% CI 1·55–2·03) times more in those with low SES compared to those with moderate SES.

The majority of the residents in the area migrated in from rural areas, but the migration times were considerably different. H. pylori seropositivity was studied by age at the time of migration to Ankara. The average age at the time of migration to Ankara was statistically significantly different for H. pylori seropositives $(26.7 \pm 11.8 \text{ years})$ and H. pylori seronegatives $(24.8 \pm 13.0 \text{ years})$, the median age was 28 years for seropositive and 24.5 years for seronegatives. Table 3 presents the results of logistic regression model for *H. pylori* seropositivity to study the association with age at the time of migration to Ankara and size of household, controlling for age and gender. Seropositivity rates were similar for individuals born in Ankara, and those who migrated to Ankara before reaching 9 years of age. Thus, these

^{*} Average age \pm standard deviation (median) were 38.6 ± 10.0 (36) and 38.2 ± 9.2 (36) years in males and females respectively.

[†] Weighted percentages are presented.

Table 2. Distribution of Helicobacter pylori seroprevalence by gender and socioeconomic status (SES) (Gülveren Health Centre area, 1998)

| | Total no. | | Seropositivity (%)† | | OR (95% CI) | |
|-----------------|------------|------------|---------------------|--------------|-----------------------|-----------------------|
| SES* | Male | Female | Male | Female | Male | Female |
| Moderate Low | 313 128 | 303 345 | 68·9 84·0 | 70·4 80·4 | 1 2·42 (2·11–2·77) | 1 1·77 (1·55–2·03) |
| Total | 441 | 648 | 75.9 | 75.6 | 2 72 (2 11 2 77) | 1 // (1 33 2 03) |

^{*} None of the study participants had high socioeconomic level.

Table 3. Distribution of Helicobacter pylori seroprevalence by age at the time of migration and the household size (Gülveren Health Centre area, 1998)

| | No. of | Seropositivity | Total | | |
|--------------------|---------------|----------------|-------|--------------------|---------|
| | seropositives | (%)* | no. | OR (95% CI) | P value |
| Age at the time of | | | | | |
| migration (years) | | | | | |
| 0–9 | 210 | 66·1 | 289 | 1 | |
| 10-19 | 125 | 69.2 | 171 | 0.89 (0.79 - 1.06) | 0.06 |
| 20-29 | 197 | 82.8 | 242 | 1.89 (1.64–2.18) | 0.00 |
| ≥30 | 312 | 81.7 | 387 | 1.73 (1.48–2.01) | 0.00 |
| Size of household | | | | | |
| 3–4 | 428 | 70.6 | 570 | 1 | |
| 5–6 | 318 | 74.9 | 394 | 1.74 (1.56–1.93) | 0.061 |
| ≥7 | 98 | 83.6 | 125 | 1.36 (1.15–1.60) | 0.000 |
| Total | 844 | | 1089 | | |

^{*} Weighted percentages are presented.

two groups were combined in analyses. The sero-positivity rates were $66\cdot1$, $69\cdot2$, $82\cdot8$ and $81\cdot7\%$ for those who migrated to Ankara before the age of 9 years, at age 10-19 years, 20-29 years and $\geqslant 30$ years respectively.

Distribution of *H. pylori* seropositivity was studied by total number of individuals sharing the same house with the study participant. The lowest level of seropositivity rate was 70.6%, and for households sized ≤ 4 . A statistically significant association was detected between *H. pylori* seropositivity and household size. Compared to individuals from households of size ≤ 4 , those from households of 5–6 individuals, and households of at least 7 members were significantly more likely to have *H. pylori* infection: the ORs (95% CI) were 1.74 (1.56-1.93) and 1.36 (1.15-1.60) respectively.

The association between *H. pylori* seropositivity and some lifestyle habits and health status was studied by a logistic regression model that included

coffee- and tea-drinking, cigarette smoking, alcohol intake, presence of a disease, ECG finding, age, and gender (Table 4).

Coffee-drinking was not common in the study population. The average daily coffee consumption was found as 0.13 ± 65 cups/day, and 0.23 ± 0.62 cups/day in *H. pylori* seropositives, and seronegatives respectively, suggesting a non-significant difference between the groups. Tea-drinking, in contrast, was found to be statistically significantly associated with *H. pylori* seropositivity, and *H. pylori* positivity was 68.2% among non-regular tea-drinkers. A dose-response effect was also observed for tea-drinking and seropositivity increased as the amount of tea drunk per day increased. Compared to *H. pylori* seropositivity among individuals who did not drink tea, the OR in drinkers of 1-5 glasses of tea per day was 4.53 (95% CI 2.58-4.17).

H. pylori seropositivity was 76·1, 75·9, 72·9 and 78·7% among never smokers, current smokers,

[†] Weighted percentages are presented.

Table 4. Distribution of Helicobacter pylori by some lifestyle habits and some reported diseases and ECG findings*

| | Helicobacter pylori seropositivity | | | | |
|----------------------------|------------------------------------|------|-------|-------------------|---------|
| | \overline{n} | %† | Total | OR (95% CI) | P value |
| Coffee-drinking | | | | | |
| No | 674 | 77.9 | 865 | 1 | |
| Yes | 66 | 74.2 | 89 | 0.96 (0.74–1.24) | 0.060 |
| Total | 740 | 77.5 | 954 | | |
| Tea-drinking (glasses/day) | | | | | |
| No | 134 | 55.7 | 182 | 1 | |
| 1–5 | 283 | 72.8 | 358 | 4.53 (2.58–7.98) | < 0.001 |
| 6–10 | 289 | 76.5 | 371 | 4.62 (2.62–8.14) | < 0.001 |
| 11+ | 138 | 79.4 | 178 | 6.98 (3.85–12.67) | < 0.001 |
| Total | 844 | 77.5 | 1089 | | |
| Cigarette smoking | | | | | |
| Never | 427 | 76.1 | 543 | 1 | |
| Current | 245 | 75.9 | 323 | 0.96 (0.77-1.20) | 0.741 |
| Seldom | 43 | 72.9 | 55 | 1.19 (0.82–1.72) | 0.370 |
| Quitter | 128 | 78.7 | 167 | 2.15 (1.55–2.99) | < 0.001 |
| Total | 843 | 77.5 | 1088 | | |
| Alcohol intake | | | | | |
| Yes | 59 | 70.1 | 83 | 1 | |
| No | 658 | 77:1 | 839 | 2.15 (1.55–2.66) | 0.002 |
| Total | 717 | 77.5 | 922 | | |
| Disease | | | | | |
| No | 373 | 74.1 | 495 | 1 | |
| Cardiovascular | 106 | 88.4 | 120 | 2.39 (1.85–3.07) | < 0.001 |
| Gastrointestinal | 55 | 77.3 | 72 | 1.18 (0.98–1.42) | 0.087 |
| Other | 310 | 75.4 | 402 | 1.03 (0.92–1.14) | 0.637 |
| ECG finding | | | | | |
| Normal | 488 | 71.8 | 651 | 1 | |
| Pathologic | 356 | 81.6 | 438 | 1.25 (1.14 –1.38) | 0.000 |
| Total | 844 | | 1089 | , | |

^{*} All analyses were controlled for age and gender.

seldom smokers, and quitters respectively and statistically significant difference was observed in *H. pylori* seropositivity between the groups. The prevalence was higher among quitters than among never smokers, current smokers and seldom smokers.

Drinking alcoholic beverages was found to be negatively but significantly associated with *H. pylori* infection. *H. pylori* seropositivity rates were 77·1 and 70·1% in never drinkers and current drinkers respectively, indicating an OR of 2·15 (95% CI 1·55–2·66).

H. pylori seropositivity was also evaluated by presence of self-reported cardiovascular disease, gastro-intestinal disease and presence of any cardiac rhythm or coronary heart disease sign in ECG, controlling for lifestyle habits, age, and gender. H. pylori seropositivity was significantly associated with the

presence of any cardiovascular system disease (P < 0.001) but not with 'other' diseases (i.e. arthralgia) (Table 4). H. pylori seropositivity was 88.4% in individuals with cardiovascular disease and compared to their counterparts without any cardiovascular disease, the odds of H. pylori seropositivity was 2.39 times higher (95% CI 1.85-3.07) in these individuals. H. pylori seropositivity was 81.6% in individuals who were detected to have abnormal ECG findings, regardless of presence of any reported disease: taking individuals with normal ECG as the comparison group the OR was 1.25 (95% CI 1.14-1.38).

A composite index was calculated to score the total risk for *H. pylori* seropositivity. This index included age, SES, age at migration to Ankara, household size, tea-drinking, coffee-drinking, alcohol intake, and

[†] Weighted percentages are presented.

| Risk score | Male | Male | | ; | Total | Total | |
|---------------|------|------------------|-----|------------------|----------------|------------------|--|
| | n | Seropositive (%) | n | Seropositive (%) | \overline{n} | Seropositive (%) | |
| ≤ 4 | 29 | 55.4 | 35 | 71.8 | 64 | 62.9 | |
| 5 | 118 | 74.8 | 171 | 72·1 | 289 | 73.6 | |
| 6 | 225 | 80.8 | 346 | 78.0 | 571 | 79.4 | |
| ≥7 | 69 | 77.0 | 96 | 78.4 | 165 | 77.5 | |
| Total | 441 | 77·1 | 648 | 77.8 | 1089 | 77.5 | |

Table 5. Distribution of Helicobacter pylori seropositivity by gender and risk score

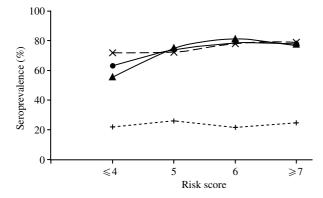


Fig. Distribution of *H. pylori* seroposivity by risk score. $- \bullet -$, Total *H. pylori* seropositivity; $- \cdot + \cdot -$, total *H. pylori* seronegativity; $- \bullet -$, *H. pylori* seropositivity in male, $- \cdot \times - -$, *H. pylori* seropositivity in female.

cigarette smoking. The maximum score that could be obtained from this index was 8. The risk scores obtained ranged between 3 and 8 and the average risk score was 5.6 ± 0.9 (median=6) for both males and females. In Table 5, *H. pylori* prevalence in males with a risk score below 4 was 55.4%: seropositivity increased steadily as the score increased until the risk score reached 7, where seropositivity decreased slightly (Fig.). In females, risk scores ranged between 3 and 8 with a mean value of 5.6 ± 0.8 (median=6). The *H. pylori* seropositivity rate was 71.8% for females with a risk score <4 and the seroposivity rate increased as the risk score increased.

A final logistic regression model was created to study the significant predictors of *H. pylori* sero-positivity in the study population. Using the backward elimination method, all sociodemographic characteristics, lifestyle habits, variables on health/disease status, and interaction terms for gender and SES, and gender and alcohol intake were simultaneously studied to check for their association with *H. pylori* seropositivity. In the final model, controlling for other variables, no significant multiplicative interaction was detected. The final model included

variables with significant association with H. pylori seropositivity at $\alpha = 0.05$ (Table 6). Accordingly, age, gender, SES, age at migration to Ankara, household size, coffee- and tea-drinking, alcohol consumption, presence of a disease, and a pathological ECG finding were all significant predictors of H. pylori seropositivity.

DISCUSSION

Clinical diagnosis of *H. pylori* infection involves the use of invasive techniques. Alternative techniques of detecting *H. pylori*-specific IgG antibody levels serologically have been in use for diagnosis of current or past *H. pylori* infections in population-based studies. Such serological methods are cheap, easy to implement, repeatable, and have a sensitivity and specificity of 80–95 % [11]. Serological tests can be used for diagnosis of *H. pylori* at primary health-care settings [12].

The seropositivity rate for *H. pylori* was found to be 77.5% in our study population of individuals aged between 25 and 64 years. *H. pylori* prevalence varies by geographic regions worldwide with a seropositivity of higher than 70% in developing countries but less than 40% in developed countries.

Studies from developed countries indicate a higher seropositivity rate for *H. pylori* in males compared to females [13–15]. In developing countries, in contrast, seropositivity rates in males and females seem to be similar [16–19]. Gender-specific *H. pylori* seropositivity rates often vary by the developmental index of the country, and its geographic location. Socioeconomic welfare and improved hygiene conditions are acclaimed as the cause of a decrease in *H. pylori* seropositivity [20].

In our study, *H. pylori* seropositivity did not vary significantly by gender but was observed to peak at ages 35–44 and 55–64 years in males whereas in females seropositivity increased in parallel with increasing age. This suggests that exposure to *H. pylori*

Table 6. Logistic regression model for H. pylori seropositivity in the study population

| 0.134 | 0.072 | 0.060 | 1.146 (0.994–1.320) |
|--------|--|---|--|
| -0.305 | 0.106 | 0.004 | 0.737 (0.599–0.907) |
| -0.186 | 0.132 | 0.160 | 0.831 (0.641–1.076) |
| | | | |
| -0.291 | 0.070 | < 0.001 | 0.747 (0.652-0.857) |
| | | | |
| | | | |
| 0.641 | 0.063 | < 0.001 | 1.898 (1.676-2.149) |
| 0.284 | 0.092 | 0.002 | 1.325 (1.107–1.586) |
| | | | |
| | | | |
| -0.086 | 0.073 | 0.239 | 0.918 (0.796–1.059) |
| 0.688 | 0.084 | < 0.001 | 1.990 (1.686–2.348) |
| 0.501 | 0.098 | < 0.001 | 1.650 (1.362–2.000) |
| | | | , |
| -0.724 | 0.058 | < 0.001 | 0.485 (0.433-0.543) |
| 0 /21 | 0 050 | (0 001 | 0 105 (0 155 0 5 15) |
| | | | |
| -0.302 | 0.064 | < 0.001 | 0.739 (0.653–0.838) |
| | | | (|
| | | | |
| 0.778 | 0.207 | < 0.001 | 2.178 (1.453-3.266) |
| 1.061 | 0.207 | < 0.001 | 2.889 (1.926–4.331) |
| 1.096 | 0.213 | < 0.001 | 2.993 (1.971–4.544) |
| | | | |
| | | | |
| -0.412 | 0.089 | < 0.001 | 1.510 (1.218–1.798) |
| | | | (|
| | | | |
| 0.243 | 0.118 | 0.040 | 1.275 (1.011–1.608) |
| 0.138 | 0.075 | 0.067 | 1.148 (0.990–1.330) |
| 0.201 | 0.097 | 0.039 | 1.223 (1.011–1.481) |
| | | | |
| 0.761 | 0.138 | < 0.001 | 2.140 (1.633–2.803) |
| 0.020 | 0.111 | 0.860 | 1.020 (0.820–1.269) |
| 0.147 | 0.065 | 0.024 | 1.158 (1.020–1.315) |
| | | | , / |
| | | | |
| 0.298 | 0.061 | < 0.001 | 1.347 (1.196–1.517) |
| | -0·305 -0·186 -0·291 0·641 0·284 -0·086 0·688 0·501 -0·724 -0·302 0·778 1·061 1·096 -0·412 0·243 0·138 0·201 0·761 0·020 | -0·305 0·106 -0·186 0·132 -0·291 0·070 0·641 0·063 0·284 0·092 -0·086 0·073 0·688 0·084 0·501 0·058 -0·302 0·064 0·778 0·207 1·061 0·207 1·096 0·213 -0·412 0·089 0·243 0·118 0·138 0·075 0·201 0·097 0·761 0·138 0·020 0·111 0·147 0·065 | $\begin{array}{cccccccccccccccccccccccccccccccccccc$ |

^{*} P value for Wald test.

SES, Socioeconomic status; CVS, cardiovascular system disease; GIS, gastro-intestinal disease.

infections increases with age in females. In a study of North American missionaries, of 65% females residing in sub-Saharan Africa the annual seroconversion incidence rate was 1.9% in *H. pylori* seronegatives. There was an increase in age-specific *H. pylori* prevalence rates in parallel to age, regardless of gender [15].

Over a 30-month follow-up, H. pylori prevalence increased from 20.5% to 40.3% in a Japanese population and suggests that females are more prone to H. pylori exposure than males [21].

Several studies worldwide have suggested a negative association between SES and *H. pylori* seroprevalence

[22, 23]. In developing countries, inequalities in social status of males and females adversely affects the SES of females which, in turn, leads to an increase in *H. pylori* prevalence rates in females. In the Gülveren study, SES was found to be negatively associated with *H. pylori* seropositivity but the odds of *H. pylori* were higher in males than in females: OR (95 % CI) comparing low SES to moderate SES were 2·42 (2·11–2·77) and 1·77 (1·55–2·03) for males and females respectively. Earlier studies on *H. pylori* infection also indicated a higher risk in males than in females [18].

Number of children is the main reason of an increase in household size. Household size, in turn, would increase the likelihood of *H. pylori* infection. In this study, the seropositivity of *H. pylori* was 74·9% in households of 5–6 members, and was 83·6% in households of at least 7 members. In large families, children often share their bedroom, or even beds, with their siblings. Sharing beds increases the risk of *H. pylori* infection exposure significantly [13]. *H. pylori* seropositivity rates are also high in adults living in large households [24].

In our study, average age at migration to Ankara was 26.7 ± 11.8 and 24.8 ± 13.0 years in *H. pylori* seropositives and seronegatives respectively. Considering those who migrated to Ankara before 20 years of age as the reference group, the odds of *H. pylori* infection were calculated as 1.89 (95% CI 1.64-2.18) and 1.73 (95% CI 1.48-2.01), in study participants who reported their age at time of migration to Ankara as 20-29 years and $\geqslant 30$ years respectively.

Gülveren district, Ankara, houses many residents who migrated to the area from rural areas or from other cities, in different years. In our study, total duration of stay in Ankara was not found to be significantly associated with H. pylori infection rates. However, a significant association was observed between H. pylori seropositivity and age at the time of migration to Ankara. H. pylori seropositivity was 82.8% among those who migrated to Ankara in the 20-29 years age group, and 81.7% in those who migrated to Ankara after reaching 30 years of age. This finding suggested that most of the residents are likely to be exposed to H. pylori before they migrated to Ankara. Earlier research in this field suggests that seroconversion mostly occurs during the ages of 5–6 years [10]. This also suggests that most of the in-migrated residents are likely to have been exposed to H. pylori even before they migrated to Ankara. However, seroconversion may occur at different ages depending on area of residence and further studies are required to find the ages of *H. pylori* seroconversion in the Turkish population.

A multivariate analysis was conducted to evaluate the independent contributions of various predictor variables on H. pylori seropositivity in the study population. Several sociodemographic characteristics, factors associated with lifestyle habits (cigarette smoking, alcohol intake, tea- and coffee-drinking), and variables related to disease status and ECG were simultaneously studied for their individual effects on H. pylori seropositivity. Daily coffee consumption was not common and a significant association was not detected between coffee consumption and H. pylori infection. However, a significant association was observed between H. pylori seropositivity and tea consumption: H. pylori seroprevalence was 77.9 and 55.7% in regular tea-drinkers and non-drinkers respectively. The average amount of tea drunk per day was 7.3 ± 3.9 glasses/day in *H. pylori* seropositives whereas the corresponding amount in seronegatives was 6.7 ± 3.7 glasses/day. Daily tea intake was both plentiful and common in the study population. Although tea is prepared using boiled water, boiling of the water does not always decrease the risk of H. pylori transmission [24]. Further, preparation and service of tea is not always performed using proper hygienic techniques. Tea glasses, teaspoons and sugar may not always be clean, either.

Many studies have suggested a positive association with cigarette smoking and H. pylori seropositivity, however, results are not yet conclusive [25]. In our study, bivariate analyses suggested a higher H. pylori seropositivity among quitters than among individuals who were current smokers or never smokers. Reasons for quitting was studied to investigate whether these individuals quit smoking due to a health condition. An earlier study in the same region found that 89.7% of quitters did so because of a health problem [26]. Grouping individuals in multivariate analysis as those 'never smoked' or 'ever smoked' showed that ever smokers were 1·185 (95 % CI 1·038–1·353) times more likely than never smokers to be positive for H. pylori (P=0.012). When smoking was evaluated as a dummy variable, with never smokers as the reference group, again a positive association between H. pylori and cigarette smoking was found, but no dose-response association was detected (Table 6). In our study H. pylori seropositivity was higher in individuals who had never used any alcoholic beverages (77·1 %) than in current consumers of alcohol (70·1%) and this finding was in accordance with other work [12, 13, 27]. Wine, in particular, has been suggested to have a protective effect against *H. pylori* infection [28]. However, a study from Russia found a positive association between vodka intake and *H. pylori* seropositivity [29]. Studies in favour of a protective effect of alcohol intake for *H. pylori* infection claim that it is through an increase in gastric acidity [28]. Further studies are warranted to study the association between alcohol intake and risk of *H. pylori* infection, and mechanism of protection (if any).

In the study, H. pylori seropositivity was 77.3% among those with gastrointestinal disease, and 88.4% in cardiac patients. Patients without any clinical symptoms but who were detected to have cardiac problems in ECGs were found to have a higher seropositivity of *H. pylori*, confirming the findings of earlier studies [30]. A meta-analysis of 20 studies has implied that H. pylori seropositivity is not associated with coronary heart disease [31]. Our finding of a positive association of *H. pylori* with cardiac and gastrointestinal disease could, at least, be partially explained due to the confounding effects of some lifestyle and nutritional habits accompanying such diseases. In multiple logistic regression analyses for adjustment for age, gender, lifestyle habits and disease status, the effect size of cigarette smoking on H. pylori seropositivity changed slightly, but did not disappear.

A composite index was calculated and used in this study and the cut point was determined as 4. *H. pylori* seropositivity was found to increase steadily as this composite risk score increased, with a *H. pylori* seropositivity of 62·9 % in those with a risk score of 4 or below. These findings suggest that *H. pylori* infection risk increases as several risk factors are present simultaneously. In developing countries, in particular, *H. pylori* seropositivity increases due to poor personal, environmental, and residential hygiene conditions, in-migrations, high fertility rates, overcrowding, and poor residential facilities.

Health personnel should recognize that several factors may affect the prevalence of *H. pylori* infection at particular settings. Algorithms could be prepared to identify patients at high risk for *H. pylori* infection and to choose diagnostic and therapeutic modalities appropriately. Early diagnosis and proper treatment of *H. pylori* infections would provide effective secondary prevention methods against *H. pylori*-associated malignancies. Finally, health education of the public on preventable risk factors of *H. pylori* such as personal hygiene and sanitation should be emphasized and promoted.

CONCLUSION

H. pylori infection is common in populations with poor SES, and low personal hygiene. Given its complications such as atrophic gastritis and gastric cancers, H. pylori infections endanger public health. Therefore, studies aimed at improving behaviours related to personal hygiene are worthwhile. Screening for H. pylori in high-risk groups, early diagnosis and treatment of the infected, and prevention of further transmission of the infection should be encouraged in high-risk countries, in particular. In developing countries, H. pylori infection occurs at early ages. Therefore, children with gastrointestinal symptoms should be examined for the presence of H. pylori using non-invasive diagnostic tests. This would not only enable the control of the infection at an early stage but might avoid complications due to the persistence of infection throughout later ages.

H. pylori infection is known to be associated with SES and personal hygiene. Thus, studies conducted in areas of low SES and adverse hygiene conditions would be of value to investigate the natural course of the disease and to identify potential prevention modalities.

Study results suggest an inverse association between *H. pylori* infection and alcohol intake. Further studies to evaluate this association are clearly warranted. Seroprevalence studies, including the childhood period, would be beneficial when investigating the age(s) that seroconversion occurs.

Educational programmes should be planned and implemented for mothers on topics such as personal hygiene, nutritional hygiene, healthy nutrition, transmission routes of *H. pylori* and relevant preventive measures.

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REFERENCES

- 1. Dubois A. Spiral bacteria in the human stomach: the gastric Helicobacter. Emerg Infect Dis 1995; 1: 79–85.
- Kadayıfçı A. Helicobacter pylori: current approach in pathogenesis. Diagnosis and treatment [in Turkish]. Gastroenterology Update 1997; 1: 7–12.
- 3. Veldhuyzen van Zanten SJO, Pollak PT, Best LM, Bezanson GS, Marric T. Increasing prevalence of

- Helicobacter pylori infection with age: continuous risk of infection in adults rather than cohort effect. J Infect Dis 1994; **169**: 434–437.
- 4. Greenberg RE, Bank S. The prevalence of *Helicobacter pylori* in nonulcer dyspepsia importance of stratification according to age. Arch Intern Med 1990; **150**: 2053–2055.
- Perez-Perez G, Taylor DN, Bodhidatta L, et al. Seroprevalence of *Helicobacter pylori* infections in Thailand. J Inf Dis 1990; 161: 1237–1241.
- Sen C. Prevalence of *Helicobacter pylori* in Turkish population living in Germany [in Turkish]. Turkish Journal of Gastroenterology 1996; 7: 61–64.
- Karabiber N. Epidemiologic characteristics of *Helico-bacter pylori* infections [in Turkish]. Bulletin of Microbiology 1992; 26: 297–300.
- Koray S. Dynamic of demography and development in Turkey: implication to the potential for migration to Europe. Turkish J Popul Studies 1997; 19: 37–55.
- 9. The situation analysis of mothers and children in Turkey, Government of Turkey and UNICEF Programme Cooperation, Country Programme 1991–1995. Ankara: Yenicag Printing House, 1991: 25–45.
- Us D, Hascelik G. Seroprevalence of *Helicobacter pylori* infection in an asymptomatic Turkish Population. J Infect 1998; 37: 148–150.
- Logan R, Walker M. ABC of the upper gastrointestinal tract epidemiology and diagnosis of *Helicobacter pylori* infection. Br Med J 2001; 323: 920–922.
- 12. Mitchell H, Megraud F. Epidemiology and diagnosis of *Helicobacter pylori* infection. Helicobacter 2002; 7: 8-16.
- Moayyedi P, Axon ATR, Feltbower R, et al. Relation of adult lifestyle and socioeconomic factors to the prevalence of *Helicobacter pylori* infection. Int J Epidemiol 2002; 31: 624–631.
- Everhart JE, Kruszon-Moran D, Perez-Perez G, et al. Seroprevalence and ethnic differences in *Helicobacter* pylori infection among adults in the United States. J Infect Dis 2000; 181: 1359–1363.
- 15. Woodward M, Morrison C, McColl K. An investigation into factors associated with *Helicobacter pylori* infection. J Clin Epidemiol 2000; **53**: 175–181.
- Bakka AS, Salih BA. Prevalence of Helicobacter pylori infection in asymptomatic subjects in Libya. Diagn Microbiol Infect Dis 2002; 43: 265–268.
- 17. Mbengue M, Diouf ML, Dangou JM, et al. Frequency of *Helicobacter pylori* infection in symptomatic patients in Senegal. Med Trop 1997; **57**: 256–258.

- Dogan YE, Gurgoze MK, Unal S. Seroprevalence of Helicobacter pylori infection among children and their parents in Eastern Turkey. J Paediatr Child Health 2002; 38: 183–186.
- Kalkan A, Bulut A, Bahcecioglu TH, Demirdag K, Aral M. Seroprevalence of *Helicobacter pylori* in Elazig region. Turkish J Microbiol Soc 1997; 27: 28–32.
- The EUROGAST Study Group. Epidemiology of and risk factors for *Helicobacter pylori* infection among 3194 asymptomatic subjects in 17 populations. Gut 1993; 34: 1672–1676.
- 21. Urite Y, Hike K, Torii N, et al. High rate seroconversion for *Helicobacter pylori* older patient. Gastroenterology 2001; **120**: A229 [Abstract].
- Brown LM. Helicobacter pylori: epidemiology and route of transmission. Epidemiol Rev 2000; 22: 283–297.
- 23. Dominici P, Bellentani S, Di Biase AR, et al. Familial clustering of *Helicobacter pylori* infection: population based study. Br Med J 1999; **319**: 537–541.
- 24. Mitchell HM, Li YY, Hu PJ, et al. Epidemiology of *Helicobacter pylori* in southern of China: identification of early childhood as the critical period of acquisition. J Infect Dis 1992: **166**: 149–153.
- Brenner H, Rothenbacher D, Bode G, et al. Relation of smoking and alcohol and coffee consumption to active *Helicobacter pylori* infection: cross sectional study. Br Med J 1997; 315: 1489–1492.
- Tezcan S, Guciz DB, Sonmez R, Altintas H. Prevalence of cardiovascular risk factors in Gülveren, Project Report Supported by European Union. Ankara, 2000
- 27. Klontz KC. Does imbibing alcohol protect against enteric pathogens? Epidemiology 1999; 10: 207–209.
- Brenner H, Berg G, Lappus N, Kliebsch V, Bode G, Boeing H. Alcohol consumption and *Helicobacter* pylori infections: results from the German National Health and Nutrition Survey. Epidemiology 1999; 10: 214–218.
- 29. Webb PM, Knight T, Greaves A, et al. Relation between infection with *Helicobacter pylori* and living condition in childhood: evidence for person to person transmission in early life. Br Med J 1994; **308**: 750–753.
- Strachan DP, Mendall MA, Carrington D, et al. Relation of *Helicobacter pylori* infection to 13-year mortality and incident ischaemic heart disease in the caerphilly prospective heart disease study. Circulation 1998; 98: 1286–1290.
- 31. Danesh J. Is there a link between chronic *Helicobacter pylori* infection and coronary heart disease? Eur J Surg (Suppl) 1998; **582**: 27–31.