ORIGINAL ARTICLE

Helicobacter pylori infection in Ontario: Prevalence and risk factors

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BACKGROUND: *Helicobacter pylori* has been classified by the World Health Organization as a type I carcinogen. Nearly 50% of the world's population is estimated to be infected with *H pylori*. Prevalence patterns of the infection are different between developing and developed countries. The present study had two objectives – to estimate the prevalence of *H pylori* infection in Ontario, and to evaluate the relationship between the infection and various demographic characteristics and selected lifestyle factors.

METHODS: Ten microlitres of plasma were aliquoted from stored blood of 1306 men and women, 50 to 80 years of age, from Ontario. The blood samples belonged to control patients of a colorectal cancer population-based study group. Serological testing was used to detect *H pylori* infection; information was obtained on dietary intake and lifestyle habits, as well as past and present medical history, education, income, number of siblings, ethnicity and place of birth.

RESULTS: The overall weighted seroprevalence of *H pylori* was 23.1% (95% CI 17.7% to 29.5%), with men having higher infection rates (29.4%, 95% CI 21.1% to 39.3%) than women (14.9%, 95% CI 10.1% to 21.4%). Seroprevalence of the infection increased significantly with age and number of siblings. Increased risk was also associated with being nonwhite, being born outside of Canada and immigrating at 20 years of age or older. An inverse association with seroprevalence was found for education and alcohol consumption.

CONCLUSION: The prevalence of *H pylori* infection in Ontario is comparable with that of other developed countries. Age, sex, number of siblings, ethnicity, place of birth and age at immigration are among the factors associated with *H pylori* infection.

Une infection à *Helicobacter pylori* en Ontario: La prévalence et les facteurs de risque

HISTORIQUE : L'Organisation mondiale de la santé a classé le *Helicobacter pylori* dans les carcinogènes de type 1. On estime que près de 50 % de la population mondiale est infectée par le *H pylori*. Les modèles de prévalence de l'infection diffèrent entre les pays en voie de développement et les pays industrialisés. La présente étude comporte deux objectifs : estimer la prévalence d'infection à *H pylori* en Ontario et évaluer le lien entre l'infection, diverses caractéristiques démographiques et des facteurs précis reliés au mode de vie.

MÉTHODOLOGIE: Dix microlitres de plasma ont été aliquotés du sang entreposé de 1 306 hommes et femmes ontariens de 50 à 80 ans. Les échantillons de sang appartenaient à des patients témoins d'un groupe d'étude du cancer colorectal en population. L'essai sérologique était utilisé pour déceler une infection à *H pylori*. On a obtenu l'information relative à l'apport alimentaire et aux habitudes reliées au mode de vie, de même que l'anamnèse courante et passée, l'éducation, le revenu, le nombre de frères et sœurs, l'ethnie et le lieu de naissance.

RÉSULTATS : La séroprévalence pondérée globale du *H pylori* était de 23,1 % (95 % IC 17,7 % à 29,5 %), les hommes présentant un taux d'infection plus élevé (29,4 %, 95 % IC 21,1 % à 39,3 %) que les femmes (14,9 %, 95 % IC 10,1 % à 21,4 %). La séroprévalence de l'infection augmentait considérablement avec l'âge et le nombre de frères et sœurs. Un risque accru s'associait également au fait de ne pas être blanc, d'être né à l'extérieur du Canada et d'avoir immigré à 20 ans ou plus tard. De plus, la séroprévalence était inversement proportionnelle à l'éducation et à la consommation d'alcool.

CONCLUSION : La prévalence d'infections à *H pylori* en Ontario est comparable à celle des autres pays industrialisés. L'âge, le sexe, le nombre de frères et sœurs, l'ethnie, le lieu de naissance et l'âge au moment de l'immigration font partie des facteurs associés à l'infection à *H pylori*.

Key Words: Age; Helicobacter pylori; Seroprevalence; Sex

Helicobacter pylori is a spiral-shaped, Gram-negative, microaerophilic rod with four to seven flagella. *H pylori* infection is an important risk factor for gastric cancer, which is the second leading cause of cancer-related deaths worldwide (1). In 1994, *H pylori* was classified as a class I carcinogen by the International Agency for Research on Cancer – World Health Organization (2). Host genetics, host immune response and bacterial virulence appear to play critical roles in the development of clinical disease in *H pylori*-infected patients (3).

H pylori infection is the most common infectious disease in the world (4). Nearly 50% of the world's population is estimated to be infected (5). While the prevalence of the infection has dropped significantly in many parts of North America and Western Europe, no such decline has been noted in the developing world (6).

In Canada, few studies have estimated the prevalence of H pylori infection. In Nova Scotia, the seroprevalence of this infection increased from 21% for subjects in their thirties, to 50% for those in their eighties (7). However, there are certain populations in Canada with much higher infection rates. One study found that 95% of a First Nations community in Manitoba was infected (8), and 67% of children from this community tested positive for H pylori by two years of age (9). The Canadian Adult Dyspepsia Empiric Treatment – Prompt Endoscopy (CADET-PE) study found that approximately 30% of dyspeptic patients were infected (10). The results of the aforementioned studies could not be applied to the general Canadian population due to their limitations in sample size and the types of populations studied.

Among the risk factors associated with *H pylori* infection, poor socioeconomic status, crowded living conditions, smoking,

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TABLE 1

Frequency distribution, unweighted and weighted percentage seropositivity for men and women by age groups

	Total, n	% positve (95% CI)	% positive* (95% CI)
Men			
Age, years†			
<60	218	29.4 (23.7–35.8)	27.7 (17.6–41.0)
60 to 70	321	32.1 (27.2–37.4)	31.96 (27.0–37.4)
>70	194	38.7 (32.0-45.7)	38.7 (32.0–45.8)
Total	733	33.0 (29.7–36.5)	29.4 (21.1–39.3)
Women			
Age, years†			
<60	191	17.3 (12.5–23.3)	10.5 (5.9–18.0)
60 to 70	243	24.3 (19.3–30.1)	24.3 (19.3–30.1)
>70	139	36.0 (28.4-44.3)	36.8 (29.3–45.3)
Total	573	24.8 (21.4–28.5)	14.9 (10.1–21.4)

*Weighted values presented; [†]Average age of the population studied was 64.04 years, with men being slightly older than women: 64.7±8.0 years versus 63.2±9.4 years, respectively

higher number of siblings and a lower consumption of fruits are the most cited in the literature (11-13).

The present study had two objectives – to estimate the prevalence of *H pylori* infection in Ontario, and to evaluate the relationship between *H pylori* infection and a number of demographic characteristics and selected lifestyle habits.

METHODS

The present study was approved by the Research Ethics Boards of the University of Toronto (Toronto, Ontario) and Mount Sinai Hospital (Toronto, Ontario). Blood samples from the Ontario Familial Colon Cancer Registry (OFCCR) were used to estimate the prevalence of H pylori infection. The OFCCR collects family history information, epidemiologic data and blood samples from a population-based sample of colorectal cancer patients and controls. Population controls are identified using random selection through Info-direct (Bell Canada, Canada), a service of Bell Canada that provides a listing of residential telephone numbers in Ontario. If there is more than one eligible household member (matched by sex and five-year age group with OFCCR case distribution), then one person is randomly selected and asked to participate. The methodology for the OFCCR has been described in detail elsewhere (14). For the purpose of the present study, the blood samples taken from the control population, which were stored in the biospecimen repository of Mount Sinai Hospital, were used. Ten microlitres of plasma were aliquoted from each of the 1306 samples (adults aged 50 to 80 years). H pylori-specific immunoglobulin G antibody titres were measured by a validated ELISA using the DRG kit (DRG International Inc, USA) in the robotics laboratory at Mount Sinai Hospital. Performance data for this kit showed a sensitivity and specificity of 99% and 97%, respectively (A Azad, personal communication).

Using family history and epidemiological questionnaires, information was obtained on past and present medical history, smoking and drinking habits, socioeconomic status, number of siblings, education level, ethnicity and place of birth, as well as the consumption of fruits, vegetables and meat. Nonwhites included blacks (from Africa, the Caribbean and North America) and those from the Middle East and Asia. Low education levels corresponded to completion of high school or lower, moderate education levels corresponded to completion of technical school or college, and higher education levels corresponded to completion of bachelor's degree or higher. Data on fruit, vegetable and meat intake referred to patient diets two years before completion of the questionnaire. The alcohol consumption for patients between 30 and 40 years of age, 41 and 59 years of age and 60 years of age or older referred to the consumption during their 20s, 30s and 40s, and since they had turned 50 years of age, respectively.

The prevalence of H pylori infection was estimated separately for each sex. Weighted prevalence estimates were obtained using sampling weights calculated as the inverse of the sampling fractions to correct for the sampling strategy. In weighting, the distribution of the 2003 Ontario population by sex and five-year age group was used as a reference (Statistics Canada, 2003).

The relationships between the prevalence of infection and the various sociodemographic factors were assessed by calculating age-adjusted ORs and 95% CIs, using logistic regression with incidence of infection as the outcome measure. In all the regression analyses, age was used as a categorical variable because the reference population that was used for weighting the estimates was grouped by age and sex.

In multivariate analyses, the dependent variable was seropositivity for H pylori, and covariate variables included available sociodemographic and lifestyle factors. The stepwise solution was used, which combined forward and backward solutions and therefore overcame the limitations associated with each. Stratified analyses suggested potential effect modification by sex on the association between H pylori seropositivity and various factors; thus, interaction terms for such factors were tested in multivariate logistic models. Intervariable correlations were evaluated before modelling. Place of birth and age at immigration were significantly correlated ($r^2=0.98$) and were combined. The new variable had three categories: born in Canada, immigrated to Canada at younger than 20 years of age and immigrated to Canada at 20 years of age or older. The associations between H pylori infection and the various factors considered in the present study were not weighted.

Most of the data analyses were performed using SPSS version 12.1 (SPSS Inc, USA). STATA version 8.0 (StataCorp LP, USA) was used to calculate the weighted prevalence estimates and their 95% CIs.

RESULTS

In the study sample, the overall *H pylori* seroprevalence was 29.4% (95% CI 27.5% to 31.9%). Seroprevalence rates were different between sexes; male subjects had significantly higher seroprevalence rates (33.0%, 95% CI 29.7% to 36.5%) than female subjects 24.8%, 95% CI 21.4% to 28.5%). Weighted analysis yielded an overall lower estimate than the unweighted analysis (23.1%, 95% CI 17.7% to 29.5%), with male subjects still having higher prevalence (29.4%, 95% CI 21.1% to 39.9%) than female subjects (14.9%, 95% CI 10.1 to 21.4). For both sexes, prevalence rates increased with age and peaked after 70 years of age (Table 1).

Analysis of the factors associated with infection is shown in Table 2. With regard to place of birth, prevalence rates were higher among male subjects born outside Canada than those born in Canada (OR 2.2, 95% CI 1.6 to 3.0). Furthermore, the pattern of H pylori prevalence in relation to age was different in

TABLE 2				
Frequency distribution,	percentage seropositivities,	age-adjusted OR estimates	and 95% Cls i	in men and women

		Men			Women		
	Total, n	% positive (95% CI)	OR (95% CI)	Total, n	% positive (95% CI)	OR (95% CI)	
Age, years							
<60	218	29.4 (23.7-35.8)	1	191	17.3 (12.5–23.3)	1	
60 to 70	321	32.1 (27.2–37.4)	1.1 (0.8–1.6)	243	24.3 (19.3–30.1)	1.5 (1.0–2.5)	
>70	194	38.7 (32.0-45.7)	1.5 (1.1–2.3)	139	36.0 (28.4-44.3)	2.7 (1.6-4.5)	
Marital status							
Not married	90	40.0 (30.4–50.4)	1	194	27.8 (22.0–34.6)	1	
Married	633	32.1 (28.5–35.8)	0.7 (0.4–1.1)	374	23.3 (19.2–27.8)	0.9 (0.6–1.3)	
Place of birth							
Canada	462	26.6 (22.8-30.8)	1	396	25.3 (21.2–29.8)	1	
Other	271	43.9 (38.1–49.9)	2.2 (1.6–3.0)	177	23.7 (18.0–30.6)	0.8 (0.5–1.3)	
Ethnicity							
White	676	32.0 (28.5–35.6)	1	534	24.7 (21.2–28.6)	1	
Nonwhite	56	46.4 (33.9–59.5)	1.8 (1.1–3.1)	38	23.7 (12.8–39.6)	0.9 (0.4–2.1)	
Age of immigration, yea	Irs	, , , , , , , , , , , , , , , , , , ,	, , , , , , , , , , , , , , , , , , ,		· · · ·	, , , , , , , , , , , , , , , , , , ,	
<20	111	34.2 (26.0-43.5)	1	62	21.1 (12.6–32.9)	1	
≥20	152	51.3 (43.4–59.2)	1.9 (1.1–3.2)	111	26.1 (18.8–35.1)	1.2 (0.6–2.6)	
Education			, , , , , , , , , , , , , , , , , , ,		, , , , , , , , , , , , , , , , , , ,	· · · ·	
Low	281	35.2 (29.8–41.0)	1	225	29.3 (23.7–35.6)	1	
Middle	232	37.9 (31.9-44.4)	1.1 (0.8–1.7)	215	25.6 (20.1–31.8)	0.9 (0.6–1.4)	
High	212	25.0 (19.6–31.3)	0.6 (0.4–0.9)	123	15.4 (10.0–23.0)	0.5 (0.3–0.9)	
Income							
<20.000	185	42 2 (35 2-49 4)	1	207	26 6 (21 0-33 0)	1	
20.000 to 40.000	230	30.9 (25.2–37.1)	0.7 (0.4–1.0)	159	27.7 (21.2–35.1)	1.2 (0.7–1.9)	
>40 000	19	26.3 (0.2–1.4)	0.5	24	25.0 (11.7-45.7)	0.9(0.3-2.3)	
Missing	299	29.4 (24.5–34.9)	0.6 (0.4–0.9)	183	20.2 (15.3–68.0)	0.8 (0.5–1.3)	
Number of siblings	200	2011 (2110 0110)	0.0 (0.1 0.0)				
<2	316	28.8 (24.1–34.0)	1	255	21.5 (16.9–27.1)	1	
_ 2 to 4	180	36 1 (29 4–43 4)	1 4 (1 0–2 1)	139	27.3 (20.1–35.4)	1 4 (0 9–2 3)	
>4	158	38.6 (31.3–46.4)	1.5 (1.0-2.3)	116	37 1 (28 8–46 2)	2 0 (1 2–3 4)	
Missina	79	31.6 (22.3–42.7)	1 1 (0 6–1 9)	63	9.5 (4.3–19.7)	0.3 (0.1–0.8)	
Regular use of antacids		0.10 (22.0 12.1.)	(0.0)				
No	620	31 3 (27 7–35 1)	1	480	24 8 (21 1–28 9)	1	
Yes	100	44.0 (34.6–53.9)	1 7 (1 1–2 6)	88	26.1 (18.0–36.3)	1 1 (0 6–1 8)	
Regular use of multivita	mins	(()			(
No	445	35 7 (31 4-40 3)	1	317	26 2 (21 6-31 3)	1	
Yes	278	28 4 (23 4–34 0)	0.7(0.5-1.0)	247	23.9 (19.0–29.6)	0 8 (0 6–1 3)	
Regular use of aspirin	2.0	2011 (2011 0110)	0.1 (0.0 1.0)		2010 (1010 2010)		
No	393	33 6 (29 1–38 4)	1	385	23 4 (19 4–27 9)	1	
Yes	327	31.5 (26.7–36.7)	0.9 (0.6–1.2)	180	27 8 (21 7-34 8)	1 1 (0 7–1 7)	
Regular use of acetamir	nophen	0.10 (2011 0011)	0.0 (0.0 1.2)		2.1.0 (2.1.1 0.1.0)	(0)	
No	638	32 0 (28 4–35 7)	1	455	237 (200–279)	1	
Yes	81	40.7 (30.6–51.7)	1.5 (0.9–2.3)	111	30.6 (22.7–39.8)	14(09-23)	
Fruit intake/day	01		1.0 (0.0 2.0)		00.0 (22.7 00.0)	1.1 (0.0 2.0)	
<1	190	31.3 (24.9–38.0)	1	76	28 9 (19 9–40 1)	1	
1 to 2	424	33 3 (28 9–37 9)	1 1 (0 7–1 5)	327	25.7 (21.2–30.7)	0.8 (0.5–1.5)	
>2	90	33 3 (24 3-43 7)	1.1 (0.6–1.8)	151	21.2 (18.4 - 28.5)	0.6 (0.3-1.2)	
Vegetable intake/day	50	00.0 (24.0 40.7)	1.1 (0.0 1.0)	101	21.2 (10.4 20.0)	0.0 (0.0 1.2)	
<1	116	34 5 (26 4-43 6)	1	34	32 4 (18 9-49 6)	1	
1 to 2	178	33.1(20.0, 37.4)	00(06.14)	208	32.4 (10.3 - 43.0)	07(0315)	
>2	122	30.3 (22.0-37.4)	0.3 (0.0 - 1.4) 0.8 (0.4 - 1.3)	230	23.0 (21.2-31.1)	0.7 (0.3 - 1.3) 0.5 (0.2 - 1.2)	
Red meat intake serving		00.0 (22.0-03.1)	0.0 (0.4-1.3)	202	22.0 (11.1-21.0)	0.0 (0.2-1.2)	
<2	95, WEEK 959	365 (30 8 12 6)	1	2/1	286 (22 2 24 7)	1	
~5 3 to 5	202	30.3 (30.0-42.0)	1 08/06 1 2)	24 I 224	20.0 (23.3-34.1)		
5 to 5	150	31.7 (20.0-37.1)	0.0(0.0-1.2)	22 4 00	22.3(11.3-20.3)	0.7(0.0-1.1)	
-0	100	29.1 (23.1-31.3)	0.7(0.3-1.1)	90	21.4 (14.4-30.7)	0.7 (0.4 - 1.2)	

Continued on next page

	Men		Women			
	Total, n	% positive (95% CI)	OR (95% CI)	Total, n	% positive (95% CI)	OR (95% CI)
Smoking, years						
Never smoked	259	32.4 (27.4–39.0)	1	293	24.6 (20.0–29.8)	1
<10	62	29.0 (19.1–41.5)	0.8 (0.5–1.6)	53	17.0 (9.1–29.6)	0.7 (0.3–1.4)
10 to 25	164	34.1 (27.3–41.7)	1.1 (0.7–1.6)	70	25.7 (16.8–37.2)	1.2 (0.6–2.2)
26 to 40	134	35.1 (27.5–43.5)	1.1 (0.7–1.7)	83	26.5 (18.1–37.0)	1.3 (0.7–2.2)
>40	68	30.9 (21.1–42.8)	0.8 (0.5–1.5)	50	36.0 (23.0–50.1)	1.4 (0.7–2.7)
Number of cigarettes/day						
Never smoked	259	32.4 (27.4–38.4)	1	293	24.6 (20-29.8)	1
<10	130	32.3 (24.8–40.8)	0.9 (0.6–1.5)	113	18.6 (12.4–26.9)	0.7 (0.4–1.2)
10 to 20	189	36.0 (29.4–43.1)	1.1 (0.8–1.7)	88	30.7 (21.9–41.1)	1.4 (0.8–2.5)
>20	124	29.8 (22.4–38.5)	0.9 (0.5–1.4)	57	28.1 (17.9–41.1)	1.4 (0.7–2.7)
Alcohol intake, drinks/week						
Never	140	45 (32.0-48.9)	1	237	31.2 (27.6–39.4)	1
≤10	362	28.7 (24.4–34.9)	0.5 (0.3–0.7)	172	19.1 (13.7–26.6)	0.5 (0.4–0.8)
>10	187	30.5 (22.9–36.7)	0.5 (0.3–0.8)	43	20.9 (2.3–30.0)	0.6 (0.3–1.4)
Polyps						
No	629	32.8 (29.2–36.5)	1	497	23.9 (20.4–27.9)	1
Yes	89	36.0 (26.7-46.4)	1.1 (0.7–1.8)	56	35.7 (24.3-49.0)	1.6 (0.9–3)
Inflammatory bowel disease						
No	693	33.0 (29.6–36.6)	1	480	26.3 (22.5–30.4)	1

TABLE 2 – CONTINUED Frequency distribution, percentage seropositivities, age adjusted OR estimates and 95% CIs in men and women

male subjects born in Canada than those born outside Canada. While the prevalence seemed to steadily increase in men born outside Canada, an increase was only seen in the older age group among those born in Canada. Prevalence rates for ages – younger than 60 years of age, between 60 and 70 years of age, and older than 70 years of age - were 25.4%, 25% and 31% for Canadian-born men and 36.3%, 45.1% and 50% for non-Canadian-born men, respectively. Among the non-Canadianborn men, those who immigrated at 20 years of age or older were more likely to be infected (OR 1.9, 95% CI 1.1 to 3.2) than those who immigrated at a younger age. Nonwhite men had higher prevalence rates than white men (OR 1.8, 95% CI 1.1 to 3.1). Patients coming from large families (more than four siblings) had higher infection rates (men, OR 1.5, 95% CI 1.0 to 2.3; women, OR 2.0, 95% CI 1.2 to 3.4). Education and alcohol intake were negatively associated with infection. The results showed that higher education and increased consumption of alcohol correlated with lower infection prevalence. Dietary factors, smoking, incidence of polyps, diabetes and presence of any cancer were not significantly associated with infection.

Multivariate modelling included sex, age, place of birth and age at immigration to Canada, number of siblings, alcohol intake and two interaction terms (sex*age and sex*age at immigration). The interaction between sex and age was found to be significant; thus, separate models were generated for each sex. For men, higher prevalence rates were observed among those who immigrated to Canada at 20 years of age or older, followed by those who immigrated younger than 20 years of age with the lowest values for Canadian-born (reference category) (OR 2.9, 95% CI 1.9 to 4.2; OR 1.6, 95% CI 1.0 to 2.5, respectively) (Table 3). Intake of antacids was associated with higher rates of infection. For women, the odds of infection decreased with increased consumption of alcohol (Table 4). The number of siblings was positively associated with the infection. Smoking, fruit and vegetable intake, and incidence of diabetes were included because they were associated with *H pylori* seropositivity in other studies. None of these variables had an effect, and the models discussed above were not changed.

DISCUSSION

To our knowledge, the present study is the first of its kind to offer an estimate of *H pylori* prevalence in an adult, asymptomatic population in Ontario. We found an overall weighted seroprevalence of 23.1%, with men having higher rates of infection than women (29.4% versus 14.9%). The unweighted seroprevalence was 29.4%, which was comparable with prevalence estimates from other developed countries, such as 32.5% in the United States (15) and 32% in Australia (16). In Canada, the CADET-PE study found that approximately 30% of dyspeptic patients were infected (13).

Our results indicated a significant effect of sex on prevalence. Men were found to have significantly higher infection rates than women. The literature regarding the relationship between sex and *H pylori* infection is conflicting (17-22). It is possible that women are more likely to have infection eradicated with antimicrobials used for other illnesses (23,24). In British Columbia, women consumed 17% more antibiotics than men (25).

Worldwide, two characteristic, age-specific patterns of *H pylori* seroprevalence have been described. In developing countries, infection appears to occur early in life with chronic infection continuing into adulthood, while in developed countries, the prevalence among children is low but rises in proportion throughout adult life at a rate of approximately 1% per year (26). In our study, seroprevalence rates followed the pattern of other developed countries and increased with age to peak after 70 years. This increase may be explained by a constant infection rate over time or by a birth cohort effect, with decreasing rates in subsequent generations. When considering place of birth, we

TABLE 3

Logistic regression model with OR estimates and 95% CIs for *Helicobacter pylori* seropositivity in the study population (men)

Characteristic	OR (95% CI)
Age, years	
<60	1
60–70	1.1 (0.8–1.7)
>70	1.3 (0.9–2.1)
Age at immigration to Canada	
Born in Canada	1
<20 years	1.6 (1.0–2.5)
≥20 years	2.9 (1.9-4.2)
Number of siblings	
<2	1
2 to 4	1.4 (1.0–2.2)
>4	1.6 (1.1–2.4)
Intake of antacids	
Have not consumed	1
Have consumed (two times per week for a	
minimum of one month)	0.6 (0.4–1.0)

found that prevalence rates were higher among immigrants. These findings resonate with findings from the United States, where it was shown that being born outside the country increased infection odds 2.53-fold (15). When we looked at the effect of age at immigration, we found that higher prevalence estimates were observed in those who immigrated at 20 years of age or older. This finding points to the importance of H pylori acquisition early in life.

Increased number of siblings is among the well-established risk factors for *H pylori* infection (29). In our data, the number of siblings was positively associated with infection rates. Some reports suggest that the number of siblings and socioeconomic status are highly correlated, and that the former effect is confounded by socioeconomic variables such as education and income. However, in our data, the number of siblings remained a significant risk factor even after adjustment for income and education. It is not known whether this effect was due to sharing a common exposure source or transmission among individuals.

Among lifestyle factors, we found that alcohol intake is associated with a decrease in H pylori prevalence. Several crosssectional studies (7,28-31) have specifically assessed the relationship between alcohol consumption and H pylori infection, and have found an inverse association between moderate alcohol consumption and prevalence of infection. This suggests that moderate alcohol consumption may facilitate elimination of H pylori, given the antibacterial effects of wine, which have been clearly demonstrated in vitro (32).

A potential limitation to the generalizability of the results of the present study is the age of the patients. Because the population studied was age-matched to colon cancer patients, the distribution was skewed to older-aged patients. However, this limitation is not too severe because *H pylori* infection occurs early in life and seems to be persistent in adulthood in most cases (33,34). In addition, spontaneous elimination rates appear to be low (35).

Serological testing, being a noninvasive, inexpensive method that has a high sensitivity and specificity, has been widely used in population-based studies to diagnose *H pylori* infection (36). In addition, the accessibility of blood samples from the population

TABLE 4

Characteristic	OR (95% CI)
Age, years	
<60	1
60 to 70	1.4 (0.9–2.3)
>70	2.8 (1.6–4.7)
Number of siblings	
<2	1
2 to 4	1.3 (0.8–2.2)
>4	1.9 (1.1–3.1)
Alcohol intake, drinks/week	
Never	1
≤10 drinks/week	0.5 (0.3–0.9)
>10 drinks/week	0.2 (0.0–1.1)

Logistic regression model with OR estimates and 95% Cls

controls of the OFCCR in the present study made serological testing the method of choice for diagnosis of infection.

CONCLUSION AND RECOMMENDATIONS

The weighted prevalence of *H pylori* infection in a sample of Ontario adults aged 50 to 80 years was 29.4% for men and 14.9% for women.

Given its complications (eg, atrophic gastritis and gastric cancer), *H pylori* infection endangers public health. The results of the present study helped to define a high-risk population of older immigrants from large families. Educational programs could be planned and implemented on topics such as personal hygiene, nutritional hygiene, transmission routes of *H pylori* and relevant preventive measures. Further research is needed to study the effectiveness of screening and treating immigrants upon landing in Canada.

Whether the observed increase of infection rates with age is a result of a higher rate of acquisition or a birth cohort effect is still to be determined. Future cohort studies looking at infection rates over time may answer the question.

Women tend to have lower infection rates and lower gastric cancer incidence; however, to date, we have no explanation for this observation. Therefore, research on both physiological and behavioural levels is warranted.

The present study is the first step in the demonstration project proposed by Sullivan et al (37). Given the importance of the problem, work is needed to evaluate the merits of screening, and if warranted, develop a full-blown population screening and treatment program.

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REFERENCES

- Lambert R, Guilloux A, Oshima A, et al. Incidence and mortality from stomach cancer in Japan, Slovenia and the USA. Int J Cancer 2002;97:811-8.
- 2. The International Agency for Research on Cancer. Schitosomes, Liver, Flukes and *Helicobacter pylori*: IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Lyon, France: IARC Press, 1994.
- 3. Figueiredo C, Machado JC, Yamaoka Y. Pathogenesis of *Helicobacter pylori* infection. Helicobacter 2005;10:14-20.
- 4. Pounder RE, Ng D. The prevalence of *Helicobacter pylori* infection in different countries. Aliment Pharmacol Ther 1995;9:33-9.
- 5. Cave DR. How is *Helicobacter pylori* transmitted? Gastroenterology 1997;113:S9-14.
- 6. Frenck RW Jr, Clemens J. *Helicobacter* in the developing world. Microbes Infect 2003;5:705-13.
- Veldhuyzen Van Zanten S, Lauritsen K, Delchier JC, et al. One-week triple therapy with esomeprazole provides effective eradication of *Helicobacter pylori* in duodenal ulcer disease. Aliment Pharmacol Ther 2000;14:1605-11.
- Bernstein CN, McKeown I, Embil JM, et al. Seroprevalence of Helicobacter pylori, incidence of gastric cancer, and peptic ulcer-associated hospitalizations in a Canadian Indian population. Dig Dis Sci 1999;44:668-74.
- 9. Sinha SK, Martin B, Sargent M, McConnell JP, Bernstein CN. Age of acquisition of *Helicobacter pylori* in a pediatric Canadian First Nations population. Helicobacter 2002;7:76-85.
- Thomson AB, Barkun AN, Armstrong D, et al. The prevalence of clinically significant endoscopic findings in primary care patients with uninvestigated dyspepsia: The Canadian Adult Dyspepsia Empiric Treatment – Prompt Endoscopy (CADET-PE) study. Aliment Pharmacol Ther 2003;17:1481-91. (Erratum in 2004;20:702).
- Moayyedi P, Axon AT, Feltbower R, et al. Relation of adult lifestyle and socioeconomic factors to the prevalence of *Helicobacter pylori* infection. Int J Epidemiol 2002;31:624-31.
- Woodward M, Morrison C, McColl K. An investigation into factors associated with *Helicobacter pylori* infection. J Clin Epidemiol 2000;53:175-81.
- 13. Olafsson S, Berstad A. Changes in food tolerance and lifestyle after eradication of *Helicobacter pylori*. Scand J Gastroenterol 2003;38:268-76.
- Cotterchio M, McKeown-Eyssen G, Sutherland H, et al. Ontario familial colon cancer registry: Methods and first-year response rates. Chronic Dis Can 2000;21:81-6.
- Everhart JE, Kruszon-Moran D, Perez-Perez GI, Tralka TS, McQuillan G. Seroprevalence and ethnic differences in *Helicobacter pylori* infection among adults in the United States. J Infect Dis 2000;181:1359-63.
- Robertson MS, Cade JF, Savoia HF, Clancy RL. Helicobacter pylori infection in the Australian community: Current prevalence and lack of association with ABO blood groups. Intern Med J 2003;33:163-7.
- Replogle ML, Glaser SL, Hiatt RA, Parsonnet J. Biologic sex as a risk factor for *Helicobacter pylori* infection in healthy young adults. Am J Epidemiol 1995;142:856-63.
- Perez-Perez GI, Witkin SS, Decker MD, Blaser MJ. Seroprevalence of *Helicobacter pylori* infection in couples. J Clin Microbiol 1991;29:642-4.
- Fawcett JP, Shaw JP, Cockburn M, Brooke M, Barbezat GO. Seroprevalence of *Helicobacter pylori* in a birth cohort of 21-year-old New Zealanders. Eur J Gastroenterol Hepatol 1996;8:365-9.

- Staat MA, Kruszon-Moran D, McQuillan GM, Kaslow RA. A population-based serologic survey of *Helicobacter pylori* infection in children and adolescents in the United States. J Infect Dis 1996;174:1120-3.
- 21. Graham DY, Malaty HM, Evans DG, Evans DJ Jr, Klein PD, Adam E. Epidemiology of *Helicobacter pylori* in an asymptomatic population in the United States. Effect of age, race, and socioeconomic status. Gastroenterology 1991;100:1495-501.
- Lin SK, Lambert JR, Nicholson L, Lukito W, Wahlqvist M. Prevalence of *Helicobacter pylori* in a representative Anglo-Celtic population of urban Melbourne. J Gastroenterol Hepatol 1998;13:505-10.
- Luknarova N, Slezakova M, Blahutova A. [Antibiotic consumption in relation to sex and age.] Cesk Farm 1992;41:162-5.
- 24. Vanden Eng J, Marcus R, Hadler JL, et al. Consumer attitudes and use of antibiotics. Emerg Infect Dis 2003;9:1128-35.
- 25. Patrick DM, Marra F, Hutchinson J, Monnet DL, Ng H, Bowie WR. Per capita antibiotic consumption: How does a North American jurisdiction compare with Europe? Clin Infect Dis 2004;39:11-7.
- Parsonnet J, Harris RA, Hack HM, Owens DK. Modelling cost-effectiveness of *Helicobacter pylori* screening to prevent gastric cancer: A mandate for clinical trials. Lancet 1996;348:150-4.
- Goodman KJ, Correa P. Transmission of *Heliobacter pylori* among siblings. Lancet 2000;355:358-62.
- Ogihara A, Kikuchi S, Hasegawa A, et al. Relationship between Helicobacter pylori infection and smoking and drinking habits. J Gastroenterol Hepatol 2000;15:271-6.
- Brenner H, Bode G, Adler G, Hoffmeister A, Koenig W, Rothenbacher D. Alcohol as a gastric disinfectant? The complex relationship between alcohol consumption and current *Helicobacter pylori* infection. Epidemiology 2001:12:209-14.
- Murray LJ, Lane AJ, Harvey IM, Donovan JL, Nair P, Harvey RF. Inverse relationship between alcohol consumption and active *Helicobacter pylori* infection: The Bristol Helicobacter project. Am J Gastroenterol 2002;97:2750-5.
- 31. Kuepper-Nybelen J, Rothenbacher D, Brenner H. Relationship between lifetime alcohol consumption and *Helicobacter pylori* infection. Ann Epidemiol 2005;15:607-13.
- Weisse ME, Eberly B, Person DA. Wine as a digestive aid: Comparative antimicrobial effects of bismuth salicylate and red and white wine. BMJ 1995;311:1657-60.
- Sipponen P, Kosunen TU, Samloff IM, Heinonen OP, Siurala M. Rate of *Helicobacter pylori* acquisition among Finnish adults: A fifteen year follow-up. Scand J Gastroenterol 1996;31:229-32.
- 34. Rothenbacher D, Inceoglu J, Bode G, Brenner H. Acquisition of *Helicobacter pylori* infection in a high-risk population occurs within the first 2 years of life. J Pediatr 2000;136:744-8.
- 35. Xia HH, Talley NJ. Natural acquisition and spontaneous elimination of *Helicobacter pylori* infection: Clinical implications. Am J Gastroenterol 1997;92:1780-7.
- Logan RP, Walker MM. ABC of the upper gastrointestinal tract: Epidemiology and diagnosis of *Helicobacter pylori* infection. BMJ 2001;323:920-2.
- Sullivan T, Ashbury FD, Fallone CA, et al. *Helicobacter pylori* and the prevention of gastric cancer. Can J Gastroenterol 2004;18:295-302.