

Dietary habits and *Helicobacter pylori* infection: is there an association?

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Abstract

Background: *Helicobacter pylori* (*H. pylori*) is a group 1 carcinogen and the etiological agent of gastric diseases such as gastritis, ulcers, and gastric cancer. It infects approximately half of the world's population. Risk factors associated with *H. pylori* infection include socioeconomic status, lifestyle, and diet.

Objectives: This study aimed to evaluate the association between eating habits and *H. pylori* infection in patients from a reference hospital in Central Brazil.

Design: This cross-sectional study included 156 patients from 2019 to 2022.

Methods: Data were collected using a structured questionnaire on sociodemographic and lifestyle characteristics and a validated food frequency questionnaire. The *H. pylori* infection status (positive versus negative) was determined using the histopathological method. After grams/day, foods were stratified into tertiles of consumption (low, medium, and high). Simple and multiple binary logistic regression models were used in the analysis of odds ratios (ORs) and their respective 95% confidence intervals (CIs), with a 5% significance level.

Results: The prevalence of *H. pylori* infection was 44.2% (69/156 patients). Infected individuals had a mean age of 49.6 ± 14.6 years; 40.6% were men, 34.8% were aged 60 years or older, 42.0% were unmarried, 7.2% had higher education, 72.5% were non-white, and 30.4% were obese. In the *H. pylori*-positive group, 55.1% were alcohol drinkers and 42.0% were smokers. The results of multiple analyses showed that the chance of *H. pylori* infection was higher among male participants (OR=2.25; CI=1.09–4.68) and individuals with obesity (OR=2.68; CI=1.10–6.51). Participants with moderate consumption of refined grains (bread, cookies, cakes, breakfast cereal) (OR=2.41; CI=1.04–5.62) and fruits (OR=2.53; CI=1.08–5.94) were more likely to be infected.

Conclusion: In this study, male sex, obesity, and the consumption of refined grains and fruits were positively associated with *H. pylori* infection. Further research is needed to investigate this association and elucidate the underlying mechanisms.

Keywords: dietary habits, *Helicobacter pylori*, Midwest Brazil, risk factors

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Background

Helicobacter pylori (*H. pylori*) is one of the most prevalent human pathogens, affecting half of the world's population. Prevalence can vary significantly by geographic region and is the highest in developing countries.¹ The prevalence of *H. pylori* infection in Latin America and the Caribbean is estimated to be 69.26%

in adults.² Brazil is the largest country in Latin America and has regional differences in the quality of life and health. This reflects a great variation in the prevalence of *H. pylori* infection, which is higher in the north than in the south of the country.^{3,4} In the Midwest region, a prevalence of 69.2% and 61.1% has been reported.^{5,6}

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Chronic infection caused by *H. pylori* can lead to gastrointestinal diseases such as gastritis, peptic ulcer, mucosa-associated lymphoid tissue lymphoma, and gastric adenocarcinoma (GAd).⁷ Since 1994, *H. pylori* has been recognized as a group 1 carcinogen by the International Agency for Research on Cancer.⁸ In addition, *H. pylori*-related extragastric manifestations have been reported in the scientific literature. Although for several of these supposed associations, the potential pathogenic mechanism remains obscure, the eradication of *H. pylori* is already a well-established therapeutic alternative for iron deficiency anemia, primary immune thrombocytopenia, and vitamin B12 deficiency.^{9–11}

The main known risk factors for *H. pylori* infection include conditions related to low socioeconomic status such as poor sanitation, inadequate hygiene practices, overcrowding, and intrafamily grouping. In addition, diet is an important risk factor for infection.¹²

Frequent consumption of fresh fruits and vegetables has been associated with protection against *H. pylori* infection.¹³ In addition, a diet consisting of a high intake of whole grains, roots and tubers, vegetables, mushrooms, various beans, vegetable oils, nuts, and seeds is associated with a decreased risk of infection.¹⁴ These associations may be attributed to the properties of some compounds present in these foods such as vitamin C, polyphenols, and flavonoids. These compounds may protect the gastric mucosa and inhibit the colonization of bacteria.¹⁵

In contrast, a diet high in carbohydrates, sweets, sausages, hamburgers, mayonnaise, and soft drinks was positively associated with *H. pylori* infection.^{16,17} Furthermore, a dietary pattern characterized by a high consumption of refined grains, pickled vegetables, bacon, salted fish, salted pickled eggs, processed and cooked meat, wine, and tea is associated with an increased risk of the infection.¹⁴

Brazil, especially in the Midwest region, has a high prevalence of *H. pylori* infection. Since the presence of this infection can be influenced by diet, it is important to know the foods that act as protective factors as well as those that may favor infection. However, data on the association between dietary habits and *H. pylori* infection in

this region are limited. Therefore, this study aimed to evaluate the association between eating habits and *H. pylori* infection in patients admitted to a reference hospital in the Brazilian Midwest.

Methods

Study design and participants

This was a cross-sectional study conducted from March 2019 to February 2022 at a referral hospital located in Central Brazil. Participants were recruited from the Department of Digestive Tracts.

Dyspeptic patients of both sexes aged between 18 and 75 years who underwent upper digestive endoscopy were included. Individuals who did not have an available histopathology report, those with cancer, or those without physical and/or psychological conditions to participate in the study were excluded.

Ethical aspects

The reporting of this study conforms to the STROBE statement.¹⁸

Data collection and evaluation

Data were collected using structured questionnaires completed during an interview and transferred online to the Research Electronic Data Capture (REDCap™) EPGC version 7.4.4.

A food frequency questionnaire (FFQ), validated in Brazilian populations with patients treated for colorectal cancer and patients from a case-control study of GAd, was administered during the interview.^{19,20} The FFQ contained more than 120 food items, and each question was divided into three categories: (1) consumption frequency ranging from 1 to 10; (2) the unit of consumption (daily, weekly, monthly, or yearly); and (3) the size of the portion eaten (small, medium, or large) which were represented in grams or milliliters and their equivalents in home measures, for example teacups, cups, tablespoons, slices, among others. The responses were converted to food intake (grams per day or milliliters per day if applicable). The foods were selected and categorized according to Table 1, based on similar characteristics and nutritional content.

Table 1. Food groups analyzed between 2019 and 2022 in Goiania, Goiás, Brazil.

| | |
|----------------------------|---|
| Refined grains | French bread and flatbread; cookies without filling (sweet and salty); filled cookies, wafer, and buttery; simple cake; filled cake; breakfast cereal |
| Milk and dairy products | Milk; yogurt (natural, with fruit or diet); mozzarella cheese, queijo prato, parmesan and provolone; cheese mines, ricotta; Fruit smoothie |
| Fruit | Orange and tangerine, banana, apple and pear, papaya, melon and watermelon, pineapple, mango, avocado, guava, kaki, grape, berries (strawberry, blackberry, raspberry, blueberry), cupuaçu, bacuri, pupunha, graviola |
| Cereals and legumes | Beans (carioca, purple, black, green), Feijoada and Tropeiro's beans, lentil, white rice cooked with oil and seasonings, brown rice cooked with oil and seasonings, oat |
| Vegetables | Lettuce, spinach and endive, watercress and rocket, tomato, kale, cabbage, cauliflower and broccoli, cariru, jambu, carrot, eggplant, beetroot, chayote, pumpkin, cucumber, zucchini, onion |
| Salted and processed meats | Beef jerky and sun-dried meat; bacon; sausages; processed meats (ham, mortadella, salami); nuggets; meatballs, hamburgers; canned meat; salted/dried shrimp; hot dog, sandwich, and hamburger |
| Red meat and offal | Roasted beef/cooked/stewed meat, fried meat, meat with vegetables, pork (loin, beef), offal (beef or chicken), barbecue |
| White meats | Roasted/boiled/stewed chicken, fried chicken, roasted/boiled/stewed fish, fried fish, pirarucu and other salted fish |
| Sweets | Candies and lollipops; whipped cream, coconut milk and condensed milk; chocolate and bonbons; fruit candy; gelatin; confectionary sweets (pies, puddings, mousses); ice cream; chocolate powder |
| Sugar-sweetened beverages | Industrialized juice, natural juice, soda, soy-based beverage, cupuaçu natural juice, bacuri natural juice, graviola natural juice |
| Hot beverages | Coffee, tea |
| Alcoholic beverages | Beer, wine, caipirinha and distilled drinks |

During the interviews, a questionnaire regarding sociodemographic data and lifestyle habits was administered. In this study, data were collected regarding body mass index (BMI), age, marital status, ethnicity, level of education, and alcohol and tobacco consumption.

BMI was categorized according to the World Health Organization guidelines, which is calculated by dividing the weight (kg) by the square of the height (m). The adult population was categorized into underweight ($>18.5 \text{ kg/m}^2$), normal weight ($18.5\text{--}24.9 \text{ kg/m}^2$), overweight ($25.0\text{--}29.9 \text{ kg/m}^2$), and obese ($\geq 30.0 \text{ kg/m}^2$); and the elderly population (60 years and older) into underweight ($<23 \text{ kg/m}^2$), normal weight ($23\text{--}27.9 \text{ kg/m}^2$), overweight ($28\text{--}29.9 \text{ kg/m}^2$), and obese ($\geq 30 \text{ kg/m}^2$).^{21,22}

Marital status was categorized into married and unmarried (single, widowed, divorced/separated).²³ For ethnicity, two categories were created: white and non-white (black, brown).²⁴ Educational attainment was categorized into <12 years (illiterate, less than 5 years, 6–8 years, and 9–11 years), 12 years (high school), and ≥ 13 years (undergraduate and graduate).²³

Diagnosis of H. pylori infection

H. pylori status (positive versus negative) was determined by histopathological examination, which is considered the gold standard, with a sensitivity and specificity of approximately 94%.²⁵ Reports of the histopathological examinations performed at the Department of Digestive Tract were accessed. The examinations were carried

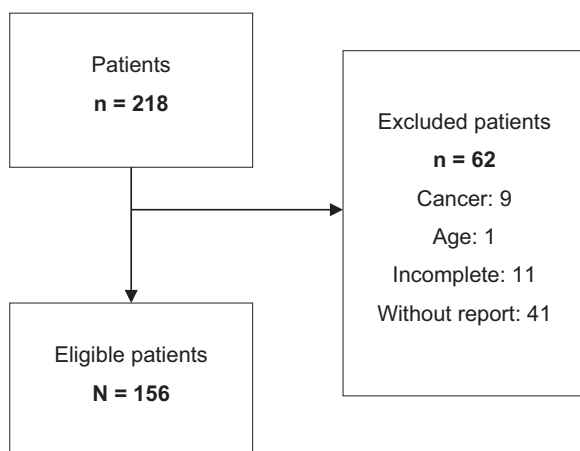


Figure 1. Flowchart of participant eligibility for the study conducted during the period 2019–2022, in Goiania, Goiás, Brazil.

out using biopsies of the gastric body and antrum stained with hematoxylin–eosin and Giemsa staining methods.

Statistical analyses

The variables are described using absolute and relative frequencies, and measures of central tendency and dispersion. After classification in grams per day, foods were stratified into tertiles of consumption (low, medium, and high). The chi-square test or Fisher’s exact test was used for association analysis of categorical variables when appropriate and Student’s *t*-test for continuous variables.

Simple and multiple binary logistic regression models were used to analyze odds ratios (ORs) and their respective 95% confidence intervals (CI). In the construction of the multiple model, simple regression analysis were conducted employing a *p* value of ≤ 0.20 as an entry criterion in the modelling process. A stepwise forward methodology was used to select the most appropriate model. The Hosmer–Lemeshow test was used to verify the adequacy of the model.

The significance level adopted was 5%. All statistical analysis were performed using IBM Statistical Package for the Social Science (IBM SPSS) version 25.0.

Results

A total of 218 individuals were recruited; however, 62 individuals were excluded because they

did not meet the eligibility criteria. After exclusion, 156 participants were eligible (Figure 1).

In this study, the prevalence of *H. pylori* infection was 44.2% (69/156). Infected individuals had a mean age of 49.6 ± 14.6 years; 40.6% were men, 34.8% were 60 years or older, 42.0% were unmarried, 7.2% had higher education, 72.5% were non-white, and 30.4% were obese (Table 2).

Non-infected individuals had a mean age of 48.2 ± 15.7 years; 27.6% were men, 28.7% were 60 years or older, 40.2% were unmarried, 8.0% had higher education, 75.9% were non-white, and 17.2% were obese. There was no significant difference between the positive and negative groups for bacterial infection regarding the sociodemographic variables evaluated (Table 2).

In the *H. pylori*-positive group, 55.1% were alcohol drinkers and 42.0% were smokers. In the negative group, the frequencies of individuals who consumed alcohol and tobacco were 44.8% and 37.9%, respectively. There was no difference in lifestyle habits between the groups (Table 2).

Regarding food consumption, there was no difference between infected and non-infected individuals for the intake of the food groups refined grains, milk and dairy products, fruits, cereals and legumes, vegetables, sweets, salted and processed meats, red meats and offal, white meats, sugar-sweetened beverages, hot beverages, and alcoholic beverages (Table 3).

In the univariate analysis, sociodemographic and lifestyle variables were not associated with the presence of *H. pylori* infection (Table 4).

Regarding eating habits, in the univariate analysis, individuals with a moderate consumption of refined grains had a higher chance of being infected with *H. pylori* (OR=2.40; 95% CI=1.08–5.32). Patients with moderate fruit consumption also had a higher chance of being infected with *H. pylori* (OR=2.36; 95% CI=1.07–5.20). The consumption of milk and dairy products, cereals and legumes, vegetables and legumes, sweets, salted and processed meats, red meats and offal, white meats, sugar-sweetened beverages, hot beverages, and alcoholic beverages was not associated with *H. pylori* infection (Table 5).

Table 2. Sociodemographic characteristics and lifestyle habits of the study participants from 2019 to 2022 in Goiania, Goiás, Brazil.

| Characteristics | <i>H. pylori</i> positive <i>n</i> = 69 (44.2%) | <i>H. pylori</i> negative <i>n</i> = 87 (55.8%) | <i>p</i> Value ^a |
|--|---|---|-----------------------------|
| Sex | | | |
| Female | 41 (59.4) | 63 (72.4) | 0.124 |
| Male | 28 (40.6) | 24 (27.6) | |
| Age (years) | | | |
| ≤45 | 27 (39.1) | 37 (42.5) | 0.721 |
| 46–59 | 18 (26.1) | 25 (28.7) | |
| ≥60 | 24 (34.8) | 25 (28.7) | |
| Marital status | | | |
| Married | 40 (58.0) | 52 (59.8) | 0.950 |
| Not married | 29 (42.0) | 35 (40.2) | |
| Level of education | | | |
| <12 years | 40 (58.0) | 52 (59.8) | 0.937 |
| 12 years | 24 (34.8) | 28 (32.2) | |
| ≥13 years | 5 (7.2) | 7 (8.0) | |
| Ethnicity | | | |
| White | 19 (27.5) | 21 (24.1) | 0.766 |
| Non-white | 50 (72.5) | 66 (75.9) | |
| BMI (kg/m ²) | | | |
| Underweight and eutrophic | 28 (40.6) | 42 (48.3) | 0.152 |
| Overweight | 20 (29.0) | 30 (34.5) | |
| Obesity | 21 (30.4) | 15 (17.2) | |
| Alcohol | | | |
| Yes | 38 (55.1) | 39 (44.8) | 0.267 |
| No | 31 (44.9) | 48 (55.2) | |
| Tobacco | | | |
| Yes | 29 (42.0) | 33 (37.9) | 0.723 |
| No | 40 (58.0) | 54 (62.1) | |
| ^a Pearson's chi-square test. BMI, body mass index. | | | |

Table 3. Dietary habits of the study participants from 2019 to 2022 in Goiania, Goiás, Brazil.

| Food groups | <i>H. pylori</i> positive <i>n</i> = 69 (44.2%) | <i>H. pylori</i> negative <i>n</i> = 87 (55.8%) | <i>p</i> Value |
|------------------------------------|---|---|--------------------|
| Refined grains (g/day) | | | |
| ≤43.04 | 17 (24.6) | 35 (40.2) | 0.089 ^a |
| 43.05–83.87 | 28 (40.6) | 24 (27.6) | |
| ≥83.88 | 24 (34.8) | 28 (32.2) | |
| Milk and dairy products (g/day) | | | |
| ≤37.25 | 19 (27.5) | 33 (37.9) | 0.363 ^a |
| 37.26–132.52 | 26 (37.7) | 26 (29.9) | |
| ≥132.53 | 24 (34.8) | 28 (32.2) | |
| Fruit (g/day) | | | |
| ≤105.97 | 19 (27.5) | 33 (37.9) | 0.056 ^a |
| 105.98–235.18 | 30 (43.5) | 22 (25.3) | |
| ≥235.19 | 20 (29.0) | 32 (36.8) | |
| Cereals and legumes (g/day) | | | |
| ≤240.30 | 25 (36.2) | 27 (31.0) | 0.679 ^a |
| 240.31–426.58 | 20 (29.0) | 24 (27.6) | |
| ≥426.59 | 24 (34.8) | 36 (41.4) | |
| Vegetables (g/day) | | | |
| ≤78.24 | 23 (33.3) | 28 (32.2) | 0.985 ^a |
| 78.25–132.47 | 23 (33.3) | 29 (33.3) | |
| ≥132.48 | 23 (33.3) | 30 (34.5) | |
| Sweets (g/day) | | | |
| ≤13.62 | 22 (31.9) | 29 (33.3) | 0.658 ^a |
| 13.63–47.87 | 26 (37.7) | 27 (31.0) | |
| ≥47.88 | 21 (30.4) | 31 (35.6) | |
| Salted and processed meats (g/day) | | | |
| ≤8.57 | 21 (30.4) | 33 (37.9) | 0.607 ^a |
| 8.58–39.28 | 23 (33.3) | 27 (31.0) | |
| ≥39.29 | 25 (36.2) | 27 (31.0) | |

(Continued)

Table 3. (Continued)

| Food groups | <i>H. pylori</i> positive <i>n</i> = 69 (44.2%) | <i>H. pylori</i> negative <i>n</i> = 87 (55.8%) | <i>p</i> Value |
|--|---|---|--------------------|
| Red meats and offal (g/day) | | | |
| ≤78.01 | 25 (36.2) | 27 (31.0) | 0.732 ^a |
| 78.02–132.29 | 23 (33.3) | 29 (33.3) | |
| ≥132.30 | 21 (30.4) | 31 (35.6) | |
| White meats (g/day) | | | |
| ≤24.17 | 20 (29.0) | 32 (36.8) | 0.227 ^a |
| 24.18–57.86 | 28 (40.6) | 24 (27.6) | |
| ≥57.87 | 21 (30.4) | 31 (35.6) | |
| Sugar-sweetened beverages (ml/day) | | | |
| ≤102.86 | 22 (31.9) | 33 (37.9) | 0.646 ^a |
| 102.87–274.29 | 22 (31.9) | 28 (32.2) | |
| ≥274.30 | 25 (36.2) | 26 (29.9) | |
| Hot beverages (ml/day) | | | |
| ≤98.57 | 22 (31.9) | 31 (35.6) | 0.780 ^a |
| 98.58–229.74 | 22 (31.9) | 29 (33.3) | |
| ≥229.75 | 25 (36.2) | 27 (31.0) | |
| Alcoholic beverages (ml/day) | | | |
| 0 | 57 (65.5) | 40 (58.0) | 0.339 ^b |
| 0.01–3.83 | 5 (5.7) | 2 (2.9) | |
| ≥3.84 | 25 (28.7) | 27 (39.1) | |
| ^a Pearson's chi-square test. ^b Fisher's exact test. | | | |

In the multiple logistic regression analysis, male individuals had a higher chance of being infected with *H. pylori* (OR=2.25; 95% CI=1.08–4.67). Participants with obesity also had a higher chance of being infected (OR=2.67; 95% CI=1.10–6.51), compared to underweight and eutrophic individuals. Regarding dietary habits, participants with moderate consumption of refined grains had a higher chance of being infected with *H. pylori* (OR=2.41; 95% CI=1.03–5.617). Individuals with moderate fruit consumption also had a

higher chance of being infected with *H. pylori* (OR=2.53; 95% CI=1.08–5.93) (Table 6).

Discussion

In this study, the prevalence of *H. pylori* infection was 44.2%. This value is considered high when compared with data from developed countries such as Switzerland and Australia, which have prevalence rates of 18.9% and 24.6%, respectively.²⁶ A recent meta-analysis reported a

Table 4. Univariate logistic regression analysis of *H. pylori* infection, sociodemographic characteristics, and lifestyle habits from 2019 to 2022 in Goiania, Goiás, Brazil.

| Characteristics | Univariate OR (95% CI) | p Value |
|-------------------------------|------------------------|---------|
| Sex | | |
| Female | 1 | |
| Male | 1.79 (0.91–3.51) | 0.089 |
| Age (years) | | |
| ≤45 | 1 | |
| 46–59 | 0.98 (0.45–2.15) | 0.973 |
| ≥60 | 1.31 (0.62–2.78) | 0.472 |
| Marital status | | |
| Not married | 1 | |
| Married | 0.92 (0.48–1.76) | 0.821 |
| Level of education | | |
| <12 years | 1 | |
| 12 years | 1.11 (0.56–2.20) | 0.756 |
| ≥13 years | 0.92 (0.27–3.14) | 0.905 |
| Ethnicity | | |
| White | 1 | |
| Non-white | 0.83 (0.40–1.72) | 0.629 |
| BMI (kg/m²) | | |
| Underweight and eutrophic | 1 | |
| Overweight | 1.00 (0.47–2.09) | 1.000 |
| Obesity | 2.10 (0.92–4.75) | 0.075 |
| Alcohol | | |
| No | 1 | |
| Yes | 1.50 (0.79–2.84) | 0.205 |
| Tobacco | | |
| No | 1 | |
| Yes | 1.18 (0.62–2.26) | 0.604 |

BMI, body mass index; CI, confidence interval; OR, odds ratio.

prevalence of 69.26% in adults in Latin America and the Caribbean.² In Brazil, the prevalence is heterogeneous in different regions, with proportions of 31.7%, 61.1%, and 91% in the Southeast, Midwest, and North regions, respectively.^{3,4,6} The prevalence of infection depends on different factors, and it is necessary to consider that these values may have been influenced by the living conditions of the population, different diagnostic methods, genetic characteristics of patients and bacteria, and lifestyle habits, among others.

Male individuals were more likely to be infected with *H. pylori*. Some studies have found no relationship between sex and infection.^{1,2} However, the results of this study corroborate a robust meta-analysis that showed a male predominance in the prevalence of *H. pylori* in adult populations from all continents.²⁷ Differences in sex in the prevalence of *H. pylori* infection among adults may be explained by distinct exposure to environmental factors such as smoking, which has been associated in the literature with an increased risk of *H. pylori* infection.²⁸ It has also been hypothesized that physiological differences, especially sex hormones, may affect immunity and inflammatory responses to *H. pylori* differently in males and females. These hormones may interfere directly or indirectly with cell receptors, thereby altering the immune response.²⁹

In this study, obesity was found to increase the risk of *H. pylori* infection. However, data on the association between this infection and obesity are conflicting. Some studies have suggested an inverse association between *H. pylori* prevalence and obesity.³⁰ Consistent with the findings of the present study, meta-analyses have indicated a positive correlation between the risk of *H. pylori* infection and prevalence of obesity.^{31,32} Furthermore, a cohort study demonstrated that *H. pylori* infection was positively associated with increased BMI regardless of socioeconomic status and other confounding factors.³³

The mechanisms involved in the relationship between *H. pylori* infection and obesity are not well established. However, it has been suggested that regulation of the gastrointestinal hormones ghrelin and leptin may be involved. Individuals positive for *H. pylori* have lower plasma leptin

Table 5. Univariate logistic regression analysis of *H. pylori* infection and eating habits from 2019 to 2022 in Goiania, Goiás, Brazil.

| Food groups | Univariate OR (95% CI) | p Value |
|------------------------------------|------------------------|--------------|
| Refined grains (g/day) | | |
| ≤43.04 | 1 | |
| 43.05–83.87 | 2.40 (1.08–5.32) | 0.031 |
| ≥83.88 | 1.76 (0.79–3.91) | 0.162 |
| Milk and dairy products (g/day) | | |
| ≤37.25 | 1 | |
| 37.26–132.52 | 1.73 (0.79–3.80) | 0.167 |
| ≥132.53 | 1.48 (0.67–3.26) | 0.320 |
| Fruit (g/day) | | |
| ≤105.97 | 1 | |
| 105.98–235.18 | 2.36 (1.07–5.20) | 0.032 |
| ≥235.19 | 1.08 (0.49–2.40) | 0.840 |
| Cereals and legumes (g/day) | | |
| ≤240.30 | 1 | |
| 240.31–426.58 | 0.90 (0.40–2.01) | 0.798 |
| ≥426.59 | 0.72 (0.34–1.52) | 0.391 |
| Vegetables (g/day) | | |
| ≤78.24 | 1 | |
| 78.25–132.47 | 0.96 (0.44–2.10) | 0.929 |
| ≥132.48 | 0.93 (0.43–2.02) | 0.861 |
| Sweets (g/day) | | |
| ≤13.6 | 1 | |
| 13.63–47.87 | 1.26 (0.58–2.74) | 0.545 |
| ≥47.88 | 0.89 (0.40–1.95) | 0.777 |
| Salted and processed meats (g/day) | | |
| ≤8.57 | 1 | |
| 8.58–39.28 | 1.33 (0.61–2.92) | 0.464 |
| ≥39.29 | 1.45 (0.67–3.14) | 0.341 |

(Continued)

Table 5. (Continued)

| Food groups | Univariate OR (95% CI) | p Value |
|--|------------------------|---------|
| Red meats and offal (g/day) | | |
| ≤78.01 | 1 | |
| 78.02–132.29 | 0.85 (0.39–1.85) | 0.694 |
| ≥132.30 | 0.73 (0.33–1.59) | 0.430 |
| White meats (g/day) | | |
| ≤24.17 | 1 | |
| 24.18–57.86 | 1.86 (0.85–4.07) | 0.117 |
| ≥57.87 | 1.08 (0.49–2.38) | 0.841 |
| Sugar-sweetened beverages (ml/day) | | |
| ≤102.86 | 1 | |
| 102.87–274.29 | 1.17 (0.54–2.56) | 0.678 |
| ≥274.30 | 1.44 (0.66–3.11) | 0.351 |
| Hot beverages (ml/day) | | |
| ≤98.57 | 1 | |
| 98.58–229.74 | 1.06 (0.49–2.32) | 0.867 |
| ≥229.75 | 1.30 (0.60–2.82) | 0.499 |
| Alcoholic beverages (ml/day) | | |
| 0 | 1 | |
| 0.01–3.83 | 0.57 (0.10–3.08) | 0.514 |
| ≥3.84 | 1.53 (0.78–3.03) | 0.212 |
| CI, confidence interval; OR, odds ratio. Bold values are statistically significant. | | |

levels. The reduction in this hormone delays the feeling of satiety, favoring obesity.³⁴ Low ghrelin levels have also been identified in *H. pylori*-infected obese patients. The reduced production of this hormone may be the result of atrophic gastritis.³⁵ Another mechanism that may contribute to the development of obesity in *H. pylori*-infected individuals is increased insulin resistance identified in these patients.³⁶ In addition, impaired immune function of the gastrointestinal tract in the obese population may favor *H. pylori* survival.³⁷ Few studies have investigated the frequency of *H. pylori* virulence factors in the obese

Table 6. Multiple logistic regression analysis of *H. pylori* infection, sociodemographic characteristics and eating habits from 2019 to 2022 in Goiania, Goiás, Brazil.

| Variables | Adjusted OR (95% CI) | p Value |
|--|----------------------|--------------|
| Sex | | |
| Female | 1 | |
| Male | 2.25 (1.08–4.67) | 0.029 |
| BMI (kg/m ²) | | |
| Underweight and eutrophic | 1 | |
| Overweight | 1.10 (0.50–2.42) | 0.813 |
| Obesity | 2.67 (1.10–6.51) | 0.030 |
| Refined grains (g/day) | | |
| ≤43.04 | 1 | |
| 43.05–83.87 | 2.41 (1.03–5.61) | 0.041 |
| ≥83.88 | 1.49 (0.64–3.46) | 0.351 |
| Fruit (g/day) | | |
| ≤105.97 | 1 | |
| 105.98–235.18 | 2.53 (1.08–5.93) | 0.032 |
| ≥235.19 | 1.17 (0.48–2.81) | 0.721 |
| BMI, body mass index; CI, confidence interval; OR, odds ratio. Bold values are statistically significant. | | |

patients. A recent observational study revealed a high prevalence of *vacA* and *cagE* followed by *cagA*, *dupA*, *iceA1*, *oipA*, and *babA2* genotypes in *H. pylori* strains isolated from obese patients diagnosed with gastric ulcer, duodenal ulcer, and gastric cancer.³⁸

Moderate consumption of the ‘refined grains’ food group, characterized by breads, cakes, cookies, and breakfast cereal, was positively associated with *H. pylori* infection. These foods are rich in carbohydrates with a high glycemic index and are present in the diet of Brazilians.³⁹ Different studies have also shown a positive relationship between the intake of breads, refined grain products, and carbohydrates in the diet and the risk of *H. pylori* infection.^{16,17,40} In addition, Sohoul *et al.* showed that a high glycemic load and a high glycemic index in the diet significantly increased the risk of *H. pylori* infection.⁴⁰

The intake of carbohydrates and acquisition of *H. pylori* infection may be related to the effect of these compounds on blood glucose levels. High blood glucose levels can lead to the development of enteric neuropathy, increasing the production of pro-inflammatory cytokines, and eventually causing neurodegeneration. Consequently, delayed gastric emptying, decreased acid secretion, and increased bacterial colonization favor *H. pylori* infection.⁴¹ In addition, several recent studies have shown an association between *H. pylori* infection and metabolic syndrome.³⁶ Other studies have reported a higher prevalence of this infection in individuals with diabetes.⁴² Considering that carbohydrate intake is associated with metabolic syndrome and diabetes, these findings reinforce a possible link between carbohydrates and *H. pylori* infection.⁴³

Moderate fruit consumption was positively associated with *H. pylori* infection. This result contrasts with studies demonstrating that fruit consumption has a protective effect against this bacterium.¹³ However, it is worth considering that the consumption of raw fruits and vegetables, often irrigated by water contaminated by feces, has been associated with *H. pylori* transmission.^{44,45} Bacteria can form a biofilm, which favors the persistence of microorganisms in the environment for long periods. Moreover, the cells in biofilms are highly resistant to elimination by sanitizing agents. Thus, raw vegetables can potentially serve as vehicles for the transmission of this pathogen.⁴⁶

Food can also be contaminated with *H. pylori* owing to poor hygiene practices during processing. Food handling can contribute to contamination *via* hands, nails, and oral and nasal secretions. Previous studies have shown that some types of raw ready-to-eat foods, such as fruit salad, can be sources of resistant and virulent *H. pylori* strains.⁴⁷

Some limitations of this study should be considered when interpreting these results. The sample comprised individuals who sought health care; these patients are probably more likely to have been diagnosed with *H. pylori* and thus changed their eating habits. Owing to the nature of the self-reported questionnaire, there is information and memory bias, and the amount of food intake may not be accurate. Another limiting factor was the lack of information regarding

the previous treatment of the infection. Finally, the cross-sectional design precludes inference of causality. However, this study has several strengths. This is the first study to examine the association between *H. pylori* infection and dietary habits in this region. A previously validated FFQ (food frequency questionnaire) was used to assess dietary intake.^{19,20} In addition, the method used to assess the presence of infection is considered the gold standard.²⁵

Conclusion

The prevalence of *H. pylori* infections was 44.2%. Males and obese individuals are more likely to be infected with *H. pylori*. The findings of this study indicated that dietary habits influence the presence of *H. pylori* infection. Consumption of refined grains increases the chance of infection, which may reinforce the role of *H. pylori* in metabolic diseases. Interestingly, fruit consumption also had a positive association with the presence of bacteria, probably because of its role as a vehicle for transmission of this pathogen. Further research is required to elucidate the mechanisms underlying the associations identified in this study.

Declarations

Ethics approval and consent to participate

This study was approved by the Research Ethics Committees of Fundação Antônio Prudente – A.C. Camargo Cancer Center, consolidated opinion 3.174.666 (CAAE:53166915.9.1001.5432), and Hospital das Clínicas da Universidade Federal de Goiás, consolidated opinion 3.005.621 (CAAE:53166915.9.3003.5078). Written informed consent was obtained from all individuals who agreed to participate in the study.

Consent for publication

Written informed consent was obtained from all individuals who agreed to participate in the study.

Author contribution(s)

Giovana Alice Sampaio Soares: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Resources; Visualization; Writing – original draft; Writing – review & editing.

Felipe Augusto de Sousa Moraes: Data curation; Investigation; Methodology; Resources; Writing – review & editing.

Amanda Ferreira Paes Landim Ramos: Investigation; Methodology; Resources; Writing – review & editing.

Silvana Barbosa Santiago: Investigation; Methodology; Resources; Writing – review & editing.

Janaina Naiara Germano: Data curation; Formal analysis; Methodology; Resources; Software; Validation; Writing – review & editing.

Gisele Aparecida Fernandes: Supervision; Validation; Writing – review & editing.

Maria Paula Curado: Conceptualization; Funding acquisition; Investigation; Methodology; Project administration; Writing – review & editing.

Mônica Santiago Barbosa: Conceptualization; Investigation; Methodology; Project administration; Supervision; Validation; Writing – review & editing.

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Competing interests

The authors declare that there is no conflict of interest.

Availability of data and materials

Not applicable.

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References

1. Zamani M, Abramabad F, Zamani V, *et al.* Systematic review with meta-analysis: the worldwide prevalence of *Helicobacter pylori*

- infection. *Aliment Pharmacol Ther* 2018; 47: 868–876.
2. Curado MP, de Oliveira MM and de Araújo Fagundes M. Prevalence of *Helicobacter pylori* infection in Latin America and the Caribbean populations: a systematic review and meta-analysis. *Cancer Epidemiol* 2019; 60: 141–148.
 3. Vinagre ID, Queiroz AL, Silva Júnior MR, *et al.* *Helicobacter pylori* infection in patients with different gastrointestinal diseases from northern Brazil. *Arq Gastroenterol* 2015; 52: 266–271.
 4. Rodrigues MF, Guerra MR, Alvarenga AVR, *et al.* *Helicobacter pylori* infection and gastric cancer precursor lesions: prevalence and associated factors in a reference laboratory in southeastern Brazil. *Arq Gastroenterol* 2019; 56: 419–424.
 5. Barbosa MS, Ramos AFPL, Silva LL de L, *et al.* *Helicobacter pylori* infection and risk factors in the development of gastroduodenal diseases in a population from the Central-west region of Brazil. *Sapiencia* 2019; 8: 181–196.
 6. Borges SS, Ramos AFPL, Moraes Filho AV, *et al.* [Article partial retraction] Prevalence of *Helicobacter pylori* infection in dyspeptic patients and its association with clinical risk factors for developing gastric adenocarcinoma. *Arq Gastroenterol* 2019; 56: 66–70. Erratum in: *Arq Gastroenterol* 2019; 56: 110.
 7. de Brito BB, da Silva FAF, Soares AS, *et al.* Pathogenesis and clinical management of *Helicobacter pylori* gastric infection. *World J Gastroenterol* 2019; 25: 5578–5589.
 8. Schistosomes, liver flukes and *Helicobacter pylori*. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Lyon, 7–14 June 1994. *IARC Monogr Eval Carcinog Risks Hum* 1994; 61: 1–241. <https://pubmed.ncbi.nlm.nih.gov/7715068/>
 9. Gravina AG, Zagari RM, De Musis C, *et al.* *Helicobacter pylori* and extragastric diseases: a review. *World J Gastroenterol* 2018; 24: 3204–3221.
 10. Franceschi F, Covino M and Roubaud Baudron C. Review: *Helicobacter pylori* and extragastric diseases. *Helicobacter* 2019; 24: e12636.
 11. Malfertheiner P, Megraud F, O’Morain CA, *et al.* Management of *Helicobacter pylori* infection—the Maastricht V/Florence Consensus Report. *Gut* 2017; 66: 6–30.
 12. Sjomina O, Pavlova J, Niv Y, *et al.* Epidemiology of *Helicobacter pylori* infection. *Helicobacter* 2018; 23: e12514.
 13. Zhang F, Pu K, Wu Z, *et al.* Prevalence and associated risk factors of *Helicobacter pylori* infection in the Wuwei cohort of north-western China. *Trop Med Int Health* 2021; 26: 290–300.
 14. Shu L, Zheng PF, Zhang XY, *et al.* Dietary patterns and *Helicobacter pylori* infection in a group of Chinese adults ages between 45 and 59 years old: an observational study. *Medicine* 2019; 98: e14113.
 15. Ullah H, Di Minno A, Santarcangelo C, *et al.* Vegetable extracts and nutrients useful in the recovery from *Helicobacter pylori* infection: a systematic review on clinical trials. *Molecules* 2021; 26: 2272.
 16. Mard SA, Khadem Haghghighian H, Sebghatulahi V, *et al.* Dietary factors in relation to *Helicobacter pylori* infection. *Gastroenterol Res Pract* 2014; 2014: 826910.
 17. Xia Y, Meng G, Zhang Q, *et al.* Dietary patterns are associated with *Helicobacter pylori* infection in Chinese adults: a cross-sectional study. *Sci Rep* 2016; 6: 32334.
 18. Von Elm E, Altman DG, Egger M, *et al.* The strengthening of reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. *PLOS Med* 2007; 4: e296.
 19. Lameza MMS. *Validation of a food frequency questionnaire for patients treated for colorectal cancer.* Master’s Thesis, Fundação Antônio Prudente, São Paulo, Brazil, 2010.
 20. Peres SV, Silva DRM, Coimbra FJF, *et al.* Consumption of processed and ultra-processed foods by patients with stomach adenocarcinoma: a multicentric case-control study in the Amazon and southeast regions of Brazil. *Cancer Causes Control* 2022; 33: 889–898.
 21. *Obesity: preventing and managing the global epidemic. Report of a WHO consultation.* World Health Organ Technical Report Series 2000; 894: 1–253. <https://pubmed.ncbi.nlm.nih.gov/11234459/>
 22. Multicenter survey aging, health and wellbeing in Latin America and the Caribbean (SABE): preliminary report. Meeting of the Advisory Committee on Health Research, 36. Pan American Health Organization, 9–11 July 2001.
 23. Brazilian Institute of Geography and Statistics. *National survey by household sample: summary of indicators 2015.* Rio de Janeiro: IBGE, 2016.
 24. Brazilian Institute of Geography and Statistics. *National health survey 2013: perception of health status, lifestyles, and chronic diseases - Brazil, major*

- regions and federation units. Rio de Janeiro: IBGE, 2014.
25. Abadi ATB. Diagnosis of *Helicobacter pylori* using invasive and noninvasive approaches. *J Pathog* 2018; 2018: 9064952.
 26. Hooi JKY, Lai WY, Ng WK, *et al.* Global prevalence of *Helicobacter pylori* infection: systematic review and meta-analysis. *Gastroenterology* 2017; 153: 420–429.
 27. Ibrahim A, Morais S, Ferro A, *et al.* Sex-differences in the prevalence of *Helicobacter pylori* infection in pediatric and adult populations: systematic review and meta-analysis of 244 studies. *Dig Liver Dis* 2017; 49: 742–749.
 28. Monno R, De Laurentiis V, Trerotoli P, *et al.* *Helicobacter pylori* infection: association with dietary habits and socioeconomic conditions. *Clin Res Hepatol Gastroenterol* 2019; 43: 603–607.
 29. Taneja V. Sexual dimorphism, aging and immunity. *Vitam Horm* 2021; 115: 367–399.
 30. Lender N, Talley NJ, Enck P, *et al.* Review article: associations between *Helicobacter pylori* and obesity—an ecological study. *Aliment Pharmacol Ther* 2014; 40: 24–31.
 31. Xu X, Li W, Qin L, *et al.* Relationship between *Helicobacter pylori* infection and obesity in Chinese adults: a systematic review with meta-analysis. *PLoS One* 2019; 14: e0221076.
 32. Baradaran A, Dehghanbanadaki H, Naderpour S, *et al.* The association between *Helicobacter pylori* and obesity: a systematic review and meta-analysis of case-control studies. *Clin Diabetes Endocrinol* 2021 10;7: 15–.
 33. Suki M, Leibovici Weissman Y, Boltin D, *et al.* *Helicobacter pylori* infection is positively associated with an increased BMI, irrespective of socioeconomic status and other confounders: a cohort study. *Eur J Gastroenterol Hepatol* 2018; 30: 143–148.
 34. Romo-González C, Mendoza E, Mera RM, *et al.* *Helicobacter pylori* infection and serum leptin, obestatin, and ghrelin levels in Mexican schoolchildren. *Pediatr Res* 2017; 82: 607–613.
 35. Nweneka CV and Prentice AM. *Helicobacter pylori* infection and circulating ghrelin levels - a systematic review. *BMC Gastroenterol* 2011; 11: 7.
 36. Azami M, Baradaran HR, Dehghanbanadaki H, *et al.* Association of *Helicobacter pylori* infection with the risk of metabolic syndrome and insulin resistance: an updated systematic review and meta-analysis. *Diabetol Metab Syndr* 2021; 13: 145.
 37. Arslan E, Atilgan H and Yavaşoğlu I. The prevalence of *Helicobacter pylori* in obese subjects. *Eur J Intern Med* 2009; 20: 695–697.
 38. Farsimadan M, Heravi FS, Emamvirdizadeh A, *et al.* Evaluation of *Helicobacter pylori* genotypes in obese patients with gastric ulcer, duodenal ulcer, and gastric cancer: an observational study. *Dig Dis* 2022; 40: 355–361.
 39. Brazilian Institute of Geography and Statistics. *Family budget survey 2017–2018: analysis of food security in Brazil*. Rio de Janeiro: IBGE, 2020.
 40. Sohoulí MH, Haghshenas N, Pouladi F, *et al.* Association between glycemic index and *Helicobacter pylori* infection risk among adults: a case-control study. *Nutrition* 2021; 83: 111069.
 41. Brown CT, Davis-Richardson AG, Giongo A, *et al.* Gut microbiome metagenomics analysis suggests a functional model for the development of autoimmunity for type 1 diabetes. *PLoS One* 2011; 6: e25792.
 42. Zhou X, Zhang C, Wu J, *et al.* Association between *Helicobacter pylori* infection and diabetes mellitus: a meta-analysis of observational studies. *Diabetes Res Clin Pract* 2013; 99: 200–208.
 43. Schwingshackl L, Hoffmann G, Lampousi AM, *et al.* Food groups and risk of type 2 diabetes mellitus: a systematic review and meta-analysis of prospective studies. *Eur J Epidemiol* 2017; 32: 363–375.
 44. Atapoor S, Safarpour Dehkordi F and Rahimi E. Detection of *Helicobacter pylori* in various types of vegetables and salads. *Jundishapur J Microbiol* 2014; 7: e10013.
 45. Yahaghi E, Khamesipour F, Mashayekhi F, *et al.* *Helicobacter pylori* in vegetables and salads: genotyping and antimicrobial resistance properties. *Biomed Res Int* 2014; 2014: 757941.
 46. Ng CG, Loke MF, Goh KL, *et al.* Biofilm formation enhances *Helicobacter pylori* survivability in vegetables. *Food Microbiol* 2017; 62: 68–76.
 47. Hemmatinezhad B, Momtaz H and Rahimi E. *VacA*, *cagA*, *iceA* and *oipA* genotypes status and antimicrobial resistance properties of *Helicobacter pylori* isolated from various types of ready to eat foods. *Ann Clin Microbiol Antimicrob* 2016; 15: 2.