

# The association of *H. pylori* and colorectal adenoma: does it exist in the US Hispanic population?

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**Background:** Although data on the association between colorectal adenomas and *Helicobacter pylori* (*H. pylori*) exists in White and Black patients, there is no data on this association in a US Hispanic population. Our aim was to study the association of adenoma detection and biopsy proven *H. pylori* infection in a cohort of US Hispanics.

**Methods:** Data were collected from Nassau University Medical Center, a 530-bed tertiary care teaching hospital in East Meadow, New York. Patients who underwent both an esophagogastroduodenoscopy (EGD) and colonoscopy from July 2009 to March 2011 were pulled from an electronic database. A total of 1,737 patients completed colonoscopies during this time with 95 excluded: 17 inflammatory bowel disease, 12 malignancy, 22 prior history of colorectal adenoma, and 44 incomplete. Among the colonoscopies, 799 patients had EGD's performed prior to colonoscopies that were eligible for our study.

**Results:** *H. pylori* prevalence was highest in Hispanics 40.9%, followed by Blacks 29.1% (OR 0.59, 95% CI: 0.42-0.84), then Whites 7.9% (OR 0.12, 95% CI: 0.06-0.24). The adenoma detection rate was significantly higher in Whites 23.2% and Blacks 21.8% compared to Hispanics 14.5%,  $P=0.0002$  respectively. Smoking and alcohol were lower in the *H. pylori* group, 18.6% ( $n=44$ ) vs. 26.1% ( $n=147$ ) for smoking ( $P=0.02$ ) and 14.4% ( $n=34$ ) vs. 19% ( $n=107$ ) for alcohol ( $P=0.12$ ), respectively. There was no evidence in the Hispanics for an association between adenoma detection and *H. pylori* infection. Furthermore size, location, and multiple polyps did not differ between the two groups.

**Conclusions:** While data has shown an association between *H. pylori* and colorectal adenomas, we did not find this in our Hispanic population. With the growing population of Hispanics in the U.S, large scale studies are needed to conclusively characterize the role of *H. pylori* infection in colorectal adenoma and adenocarcinoma in this group of patients.

**Keywords:** Hispanic; colorectal adenoma; neoplasia; colorectal cancer

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## Introduction

*Helicobacter pylori* (*H. pylori*), a spiral-shaped pathogenic bacterium found on the human gastric mucosa, was first isolated by Warren and Marshall (1). According to the CDC data, the estimated prevalence of *H. pylori* infection

is 70% in developing countries and 30-40% in the United States and other industrialized countries (2). Seroprevalence of *H. pylori* increases with age, lower socioeconomic class and race with a higher incidence and prevalence found in Hispanic and African Americans versus Whites (3).

H. pylori infection has been associated with inflammatory processes like chronic gastritis, peptic ulcer disease and gastric adenocarcinoma (4). An association between H. pylori and colorectal adenoma has been demonstrated in non-Hispanic populations (5,6). From *in vivo* and *in vitro* experiments, it appears that H. pylori infection promotes tumor formation in extra-gastric organs through at least two hypothesized mechanisms (7-9). One explanation may occur through a direct carcinogenic effect from the H. pylori's contact with the colonic mucosa. Another etiology may occur indirectly through increased gastrin levels caused by H. pylori infection, leading to a trophic effect on colonic epithelial cell growth and differentiation (10,11).

Data regarding colorectal adenomas and their association with H. pylori infection is scarce in the Hispanic population. Our aim was to study the association of adenoma detection and biopsy proven H. pylori infection in a cohort of Hispanic population.

## Methods

### Study population

Data were collected from Nassau University Medical Center, a 530-bed tertiary care teaching hospital in East Meadow, New York. Patients who underwent both an esophagogastroduodenoscopy (EGD) and colonoscopy from July 2009 to March 2011 were pulled from an electronic database. A total of 1,737 patients completed colonoscopies during this time with 95 excluded for the following reasons: 17 inflammatory bowel disease, 12 malignancy, 22 prior history of colorectal adenoma, and 44 were incomplete colonoscopies defined as no cecal intubation. Among the colonoscopies included for this study, 799 patients had EGD's performed prior to colonoscopies for evaluation of gastrointestinal bleeding, dysphagia, unexplained iron deficiency anemia, unintentional weight loss, persistent abdominal pain and esophageal reflux symptoms despite appropriate treatment (12). Of these individuals, 335 were Hispanic patients. The EGD biopsies were reviewed for H. pylori organisms with the results recorded as either H. pylori positive or negative. These 335 Hispanic patients also underwent either screening or diagnostic colonoscopy results were reviewed and recorded.

### Statistical analysis

The following variables were recorded for each subject: age, sex, race, BMI, diabetes, hypertension, dyslipidemia, alcohol use, tobacco use, family history of colorectal cancer

and H. pylori status. Additionally, presence, number, size and location of colorectal adenomas were recorded.

Analyses were conducted using SAS version 9.1 (SAS Institute, Cary, NC).

Associations between groups were tested using a Chi-Square statistic for categorical variables and a *t*-test for continuous variables. Age, sex, race, alcohol use and tobacco use -adjusted odds ratios and their 95% confidence intervals (CIs) were estimated using unconditional logistic regression models. Statistical significance was assessed using a two sided test at the alpha =0.05 level for all studies.

## Results

Table 1 shows the characteristics of the H. pylori negative and positive patient populations meeting the criteria for our study. The mean age of the H. pylori positive and negative groups was 53.7 and 55.3 respectively but this did not reach statistical significance (P=0.06). The mean body mass index (BMI) between the two groups was in the overweight range of 29.1 and 28.8 respectively and was also not different (P=0.43). The racial discrepancy in the H. pylori positive group was significantly different between the groups (P<0.0001). In the H. pylori positive population, 58.1% (n=137) were Hispanic *vs.* 35.3% (n=198) in the H. pylori negative patients. In contrast, 4.7% were White in the H. pylori positive group *vs.* 23.0% (n=129) in the H. pylori negative population. The majority of patients in our population were Hispanics followed by Blacks and Whites respectively (P<0.001). Smoking and alcohol were lower in the H. pylori group, 18.6% (n=44) *vs.* 26.1% (n=147) for smoking (P=0.02) and 14.4% (n=34) *vs.* 19% (n=107) for alcohol (P=0.12), respectively. Diabetes, dyslipidemia and hypertension distribution did not differ between the two groups.

Table 2 shows the association between H. pylori and combined colonoscopic findings. There were no statistically significant associations seen between H. pylori infection and colonoscopic findings. Table 3 shows the demographic characteristics of the Hispanic population meeting inclusion criteria. There were no statistically significant differences between H. pylori positive and negative individuals in terms of mean age, BMI, sex, smoking, alcohol, diabetes, hypertension, dyslipidemia, or history of colorectal cancer. Table 4 shows that there were no significant associations between H. pylori infection and colonoscopic findings in the Hispanic study population. Table 5 shows the total adenoma detection rate (ADR) was highest in the Whites

**Table 1** Characteristics of H. pylori (+) and H. pylori (-) patients undergoing colonoscopy

Characteristics	H. pylori (-) (n=562)	H. pylori (+) (n=236)	P value*
Age	55.3±11.7	53.7±11.0	0.06
BMI	28.8±6.0	29.1±6.3	0.43
Sex-female	283 (50.5%)	135 (57.2%)	0.08
Race			<0.0001
White	129 (23.0%)	11 (4.7%)	
Black	185 (32.9%)	76 (32.2%)	
Hispanic	198 (35.2%)	137 (58.1%)	
Other	50 (8.9%)	12 (5.1%)	
Smoking-yes	147 (26.1%)	44 (18.6%)	0.02
Alcohol-yes	107 (19.0%)	34 (14.4%)	0.12
Diabetes-yes	137 (24.3%)	55 (23.3%)	0.76
Hypertension-yes	277 (49.2%)	109 (46.2%)	0.44
Dyslipidemia-yes	171 (30.4%)	72 (30.5%)	0.97
Family history of colorectal cancer	38 (6.8%)	13 (5.5%)	0.65

\*, Use  $\chi^2$  test for categorical variables, and *t*-test for continuous variables. BMI, body mass index; H. pylori, Helicobacter pylori.

**Table 2** Association between H. pylori infection and colonoscopy findings

Findings	H. pylori (-) (n=562)	H. pylori (+) (n=236)	P value*	OR (95% CI)*
Adenoma	102 (18.1%)	38 (16.1%)	0.87	1.04 (0.67-1.61)
Adenoma ≥10 mm	13 (2.3%)	6 (2.5%)	0.52	1.43 (0.49-4.16)
Adenoma ≥2	32 (5.7%)	10 (4.2%)	0.94	0.97 (0.44-2.12)
Adenoma present in proximal colon	61 (10.8%)	18 (7.6%)	0.64	0.87 (0.49-1.56)
Adenoma present in distal colon	53 (9.4%)	23 (9.8%)	0.46	1.23 (0.71-2.12)
Largest adenoma in proximal colon	54 (9.6%)	18 (7.6%)	0.81	0.93 (0.51-1.70)
Largest adenoma in distal colon	50 (8.9%)	23 (9.8%)	0.33	1.32 (0.76-2.30)

\*, Use logistic model controlling age, sex, BMI, race, alcohol, and tobacco. BMI, body mass index; H. pylori, Helicobacter pylori.

**Table 3** Demographics of H. pylori (+) and H. pylori (-) Patients Undergoing Colonoscopy (HISPANIC)

Demographics	H. pylori (-) (n=198)	H. pylori (+) (n=137)	P value*
Age	52.9±11.4	52.2±11.4	0.60
BMI	29.6 ± 6.0	29.4 ± 5.8	0.66
Sex-female	130 (65.7%)	79 (57.7%)	0.14
Smoking-yes	23 (11.6%)	19 (13.9%)	0.54
Alcohol-yes	18 (9.1%)	20 (14.6%)	0.12
Diabetes-yes	40 (20.2%)	27 (19.7%)	0.91
Hypertension-yes	78 (39.4%)	51 (37.2%)	0.69
Dyslipidemia-yes	66 (33.3%)	43 (31.4%)	0.71
Family history of colorectal cancer	15 (7.6%)	5 (3.7%)	0.14

\*, Used  $\chi^2$  for categorical variables and *t*-test for continuous variables. BMI, body mass index; H. pylori, Helicobacter pylori.

**Table 4** Association between H. pylori infection and colonoscopy findings in Hispanic population

Findings	H. pylori (-) (n=198) (%)	H. pylori (+) (n=137) (%)	P value*	OR (95% CI)*
Adenoma	27 (13.6)	15 (11.0)	0.53	0.81 (0.41-1.59)
Adenoma ≥10 mm	1 (0.5)	5 (3.7)	0.09	6.73 (0.75-60.69)
Adenoma ≥2	6 (3.0)	5 (3.7)	0.79	1.18 (0.34-4.07)
Adenoma present in proximal colon	14 (7.1)	8 (5.8)	0.75	0.86 (0.35-2.14)
Adenoma present in distal colon	15 (7.6)	9 (6.6)	0.80	0.89 (0.38-2.12)
Largest adenoma in proximal colon	12 (6.1)	7 (5.1)	0.74	0.85 (0.32-2.24)
Largest adenoma in distal colon	15 (7.6)	9 (6.6)	0.80	0.89 (0.38-2.12)

\*, Use logistic model controlling age, sex, BMI, alcohol, and tobacco. BMI, body mass index; H. pylori, Helicobacter pylori.

**Table 5** Adenoma detection rate in different ethnicities

Race	Total	P value	Screening	P value	Diagnostic	P value
White	23.2%	0.0002	30.6%	0.0003	18.4%	0.06
Black	21.8%	0.0002	24.8%	0.006	19.1%	0.02
Hispanic*	14.5%		16.4%		12.9%	

\*, Reference group.

**Table 6** Prevalence of H. pylori in different ethnicities

Race	H. pylori (-) (n=562)	H. pylori (+) (n=236)	Prevalence (%)	P value	OR (95% CI)
White	129	11	7.9	0.0001	0.12 (0.06-0.24)
Black	185	76	29.1	0.003	0.59 (0.42-0.84)
Hispanic*	198	137	40.9		1
Other	50	12	19.4	0.001	0.35 (0.18-0.68)

\*, Reference group.

(23.2%, P=0.0002) followed by Blacks (21.8%, P=0.0002) in comparison to the Hispanics (14.5%). *Table 6* compares prevalence of H. pylori in Hispanics with other races. It shows that highest prevalence was seen in Hispanics 40.9% *vs.* Blacks (29.1%, P=0.003) followed by other races (19.4%, P=0.001) then Whites (7.9%, P=0.0001).

## Discussion

In our Hispanic cohort we did not find an association between H. pylori infection and the presence of colorectal adenoma. Meta-analysis have previously shown a small to modest association between colorectal adenoma and H. pylori prevalence in non-Hispanic populations OR 1.66, 95% CI: 1.39-1.97 and OR 1.36, 95% CI: 1.10-1.68 (13,14). In addition to different genetic susceptibility or environmental factors, inconsistent and discrepancies maybe explained by small sample sizes, uncontrolled biases, differences in selection of controls and inadequate consideration of confounding in

previously published data (15-20).

Per the Centers for disease control and prevention (CDC) data in 2010 (21), Blacks had the highest rate of colorectal cancer, followed by Whites, Hispanics, Asian/Pacific Islanders, and American Indian/Alaska Natives. Siegel *et al.* (22) reported that Hispanics had a lower risk of colorectal cancer when compared to Whites. Conflicting data exists on the association between race and colorectal adenomas. One retrospective analysis of 1,656 patients indicated a lower prevalence of colorectal adenomas in Hispanic versus non-Hispanic subjects (14.8% *vs.* 22.5%, P<0.01) (23). Conversely, an analysis of over 5,000 patients reported a higher prevalence of larger adenomas (>10 mm in size) in Hispanics; and the overall prevalence was not lower in Hispanics compared to White patients (24). The difference in the findings may be explained by the predominant South American ancestry versus the Dominican and Caribbean descent found in the later study. The Veterans Administration Colonoscopy Screening study found that

African Americans had a higher risk of polyps larger than 9 mm (8%) in comparison to the White population (6%) (25). This difference was especially pronounced in African American women compared with White women.

Despite a high prevalence of *H. pylori* infection in our Hispanic population, there was no association between *H. pylori* and colonoscopic findings. One explanation could be confounding factors such as genetic differences that result in lower adenoma prevalence in Hispanics or perhaps no association at all. Additionally, discrepancies between studies may be the result of assay differences in the detection of *H. pylori* infection. Most studies reporting a positive association between *H. pylori* and colorectal adenomas and adenocarcinomas have utilized seroprevalence of *H. pylori* while our study utilized histologically proven *H. pylori* infection. Although there are genetic and environmental factors that may play a role in differing colorectal cancer etiologies in different races, further studies assessing the relationship between *H. pylori* infection and colonoscopic findings are needed. Biological details such as the specific strain of *H. pylori* infection may address the differences between studies.

In an analysis of 7,465 participants (National Health and Nutritional Examination Survey database), age-adjusted prevalence of *H. pylori* was found to be substantially higher among Hispanic Americans (61.6%) than non-Hispanic Blacks (52.7%) and non-Hispanic Whites (26.2%) (26). Despite the reported association of *H. pylori* and colorectal adenoma (13,14), and a higher prevalence of *H. pylori* in Hispanics the global prevalence of colorectal cancer in Hispanics compared to Whites and Blacks remains lower (22). This is comparable to our data where Hispanics had the highest prevalence around 40.9% compared to Blacks 29.1% then non-hispanic Whites 7.9%, and our adenoma detection rate was also lower in this group.

We found that the *H. pylori* positive population had a statistically lower prevalence of smoking compared to *H. pylori* negative population hinting towards a protective effect of smoking. While reports in the literature associate smoking with *H. pylori* infection, other studies have yielded an inverse relationship similar to our findings (26-31). In a Japanese study of 8,837 subjects (32), smoking was negatively associated with *H. pylori* infection. The risk of *H. pylori* seropositivity decreased linearly with cigarette consumption per day. A possible basis of smoking's protective effect may be due to an increased smoking related gastric acidity which then protects against *H. pylori*.

Limitations of our study include the small sample size,

and retrospective design. Furthermore, the ethnic diversity of the Hispanic population also limits generalizations. Most of our patients were South American; however this data was not available in medical records. Major strengths of our study included the selection of a diverse patient population sample. Additionally, our statistical analysis took into account multiple factors that could influence adenoma growth and detection, thus limiting confounders. Finally, we diagnosed *H. pylori* by biopsy, which is more accurate for diagnosing infection.

## Conclusions

While previous literature has shown an association between *H. pylori* positive patients and colorectal adenomas, we did not find this in our Hispanic population. With the growing population of Hispanics in the U.S, large scale studies are needed to conclusively characterize the role of *H. pylori* infection in colorectal adenoma and adenocarcinoma. Our findings may be due to different environmental factors such as dietary/colonic flora, or genetic susceptibility. Our results support further investigation of these findings in Hispanics to further characterize the risk associated with *H. pylori* and colorectal adenoma in this population.

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