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Helicobacter Pylori Infection in Texas Hispanic and Non-Hispanic White Men: Implications for Gastric Cancer Risk Disparities

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Abstract

Chronic *Helicobacter pylori* (*H. pylori*) infection is a major gastric adenocarcinoma (GA) risk factor. GA disproportionately affects U.S. Hispanics compared with non-Hispanic Whites (NHWs). Since *H. pylori* infection studies in Hispanics are few, infection rates in Hispanic and NHW men in Bexar County were compared, and relationships with ethnicity and obesity examined. Age- and zip code-matched participants from a community-dwelling cohort were randomly selected. Sera from 284 men were analyzed by enzyme immunoassay for *H. pylori* antibodies. Adjusted risk ratio estimation for matched data was conducted to identify differences. Hispanics had a markedly higher prevalence of infection (30.3%) than NHWs (9.2%). Matched risk ratio (mRR) analyses revealed a strong association between *H. pylori* seropositivity and Hispanic ethnicity (mRR = 3.31; 95% CI [1.91, 5.73], adjusted by BMI, smoking status, and family history of cancer (mRR range = 3.28-3.89). BMI mRRs (range = 1.19-1.22) were significant in all models. In this cohort, Hispanic men had higher *H. pylori* infection rates than NHWs, and parallel the disproportionately higher rates of GA; obesity contributes to this higher prevalence. Future studies should address country of origin, acculturation, and other factors influencing obesity to further elucidate risk of GA in Hispanic populations.

Keywords

health inequality/disparity, cancer prevention, quantitative research, men of color, risk factors

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Introduction

Gastric adenocarcinoma (GA), which accounts for over 90% of all gastric cancers (Blaser et al., 1995), affects U.S. Hispanics disproportionately compared with non-Hispanic Whites (NHWs; American Cancer Society, 2015). Studies in Texas have reported that GA incidence among Hispanics is more than twice as high as among NHWs (11.4 vs. 4.7 per 100,000 from 2005 to 2009; Ramirez, Thompson, & Vela, 2013). Data from 1995 to 2010 indicate that Hispanics have a fourfold higher risk of GA compared with NHWs (Munoz, Westin, Long Parma, Suarez, & Ramirez, 2015; Texas Department of State Health Services, 2013), and these results mirror national data (National Cancer Institute, 2013). Hispanics are one of the largest minority groups in the United States, comprising 17.1% of the general population (U.S. Census Bureau, 2014), and will grow to an estimated 31% by 2060 (U.S. Census Bureau, 2013a). In 2009, cancer surpassed cardiovascular disease as the primary cause of mortality in Hispanics (Siegel, Naishadham, & Jemal, 2012). Thus, the causes and control of GA in Hispanics should be a major public health focus (Ramirez, 2013; Ramirez et al., 2005).

Chronic infection with the bacterium *Helicobacter pylori* (*H. pylori*) is thought to be a major cause of GA

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(Blaser et al., 1995; Graham, 2015). GA can be subdivided into two distinct morphologic types, intestinal and diffuse, both of which have been previously associated with *H. pylori* infection (Gonzalez et al., 2012). *H. pylori* is also the most important pathogenic factor for the development of gastric mucosa-associated lymphoid tissue (MALT) lymphoma. In one report, 92% to 98% of patients diagnosed with MALT lymphoma were also positive for *H. pylori* (Testerman & Morris, 2014). As with GA, rates of gastric MALT lymphoma are significantly elevated in Hispanics who reside in South Texas compared with NHWs (RR = 1.82; Munoz et al., 2015). Thus, the study of *H. pylori* infection in Hispanic populations is of compelling interest.

H. pylori infection has also been linked to other cancers, including colorectal, lung, and prostate (Adriani, Repici, Hickman, & Pellicano, 2014; Franceschi, Tortora, Gasbarrini, & Gasbarrini, 2014; Garcia-Gonzalez et al., 2015; Wu, Yang, Xu, Gao, & Fan, 2013). However, the data are conflicting, particularly for colorectal cancer (Kapetanakis et al., 2012; Patel et al., 2014; Tatishchev, Vanbeek, & Wang, 2012).

The overall prevalence of *H. pylori* infection in the United States ranges from 14% to 31% in gastric biopsy specimens (Sonnenberg, Lash, & Genta, 2010), and according to National Health and Nutrition Examination Survey data (Grad, Lipsitch, & Aiello, 2012), Hispanics were three times as likely to be infected as NHWs (64% vs. 21%; American Cancer Society, 2015). In a prospective cohort study of over 1,200 Mexican Americans in San Antonio, Texas, the *H. pylori* seroprevalence rate was 57%. Being older and having a lower education level were significant predictors of *H. pylori* seropositivity (Rubicz et al., 2011). Despite the growing awareness of the high prevalence of *H. pylori* infection in Hispanics, few studies have examined risk factors related to these infection rates among Hispanics. This paucity of data is likely due to the fact that *H. pylori* infection is not reportable (Adams et al., 2013), and testing is not recommended in asymptomatic individuals (Malfertheiner et al., 2012).

Changes in gut microbiota have been implicated in development of metabolic disorders like obesity, but how *H. pylori* and gut microbiota act together to regulate human metabolism is unknown (Yang & Sheu, 2016). In one study of patients undergoing gastric banding, *H. pylori* infection was twice as common in Hispanics as in their NHW counterparts (36% vs. 15%; Portocarrero, Olafsson, Jackson, Doss, & Malamud, 2012). However, the groups did not differ with respect to other risk factors, such as alcohol or tobacco consumption, or proton pump inhibitor medication use.

The purpose of the present study was to determine and compare *H. pylori* infection rates in a cohort of healthy Hispanic and NHW men from San Antonio, Texas, and to correlate these rates with known and potential GA risk factors, including age and obesity (Lin et al., 2014), low socioeconomic status (Malfertheiner et al., 2012; Sokic-Milutinovic, Alempijevic, & Milosavljevic, 2015), tobacco use (Ladeiras-Lopes et al., 2008; Ramirez et al., 2013), and family history of specific cancers (Sokic-Milutinovic et al., 2015). The study focused on men because of their higher rates of gastric cancer (American Cancer Society, 2015), in an existing community-living cohort with a large proportion of Hispanic participants (Beuten et al., 2010).

Method

Frozen serum samples were obtained from men enrolled in the San Antonio center for Biomarkers of Risk of prostate cancer (SABOR), an Early Detection Research Network–sponsored Clinical Validation Center supported by the National Cancer Institute (Beuten et al., 2010). Using simple random sampling without replacement, a cohort of 332 men were identified (166 Mexican American/Hispanic, 166 NHW) who were still alive at the time samples were obtained and had no personal history of prostate cancer, and matched for age and zip code. SABOR policy restricts access to blood samples of prostate cancer patients, so those were excluded from this analysis. Otherwise, participants with at least four blood draws were included to ensure adequate serum availability and maximum availability of data. The second-most recent annual study visit was selected to obtain survey data and sera. Data for this study were collected from September 2013 through May 2014. The institutional review board of the University of Texas Health Science Center at San Antonio approved this study.

Of the 332 cohort members selected for this study, serum samples were available for 284 (85.5%) participants from the selected study visit. These samples were analyzed by enzyme immunoassay (ELISA) to determine anti-*H. pylori* IgG antibody titers (HpG Screen ELISA Kit; ALPCO, Salem, NH). Briefly, the samples were diluted 1:200 and run in duplicate 100 µL volumes. As per the manufacturer's instructions, optical densities (measured at 450 nm) greater than the standard (6.25 U/mL + 10% = 6.875) were considered positive.

Deidentified clinical data were abstracted from study charts and the SABOR electronic database. Data elements assessed included age, ethnicity, and body mass index (BMI). Additional risk factor information, like income, tobacco use history, and family history of cancer, was gathered from survey data of a subset of 99 participants (matched pairs), 55 of whom were *H. pylori* antibody-positive.

Table 1. Sample Characteristics by Ethnicity (*N* = 284).

	Hispanic (*n* = 142)		NHW (*n* = 142)		Total (*N* = 284)		
	n	%	*n*	%	*N*	%	*p*
Age groups[a] (years)							.927
49 to 64	71	50.0	69	48.6	140	49.3	
65 to 69	28	19.7	31	21.8	59	20.8	
70 to 79	43	30.3	42	29.6	85	29.9	
Age (years, *M, SD*)[a]	65.0	7.5	65.1	7.5	65.0	7.5	.887
BMI classification							.002
Normal (20-24)	14	10.1	36	25.9	50	18.0	
Overweight (25-29)	67	48.2	59	42.5	126	45.3	
Obese (≥30)	58	41.7	44	31.7	102	36.7	
BMI (kg/m^2, *M, SD*)	29.6	4.8	28.0	4.8	28.7	4.8	.007
Income[b,c]							1.000
<$36,000	10	20.4	10	20.0	20	20.2	
$36,001-$40,000	7	14.3	7	14.0	14	14.1	
$40,001-$50,000	12	24.5	13	26.0	25	25.3	
$50,001-$60,000	9	18.4	9	18.0	18	18.2	
>$60,000	11	22.5	11	22.0	22	22.2	
Smoking status[b]							.544
Never	22	44.9	19	38.0	41	41.4	
Past/current	27	55.1	31	62.0	58	58.6	
Family history of cancer[b]							
Colon, lung, prostate, or stomach[d]	20	40.8	21	42.0	41	41.4	1.000

Note. NHW = non-Hispanic White; BMI = body mass index. *p* Values obtained from independent two-sample *t* test for continuous variables and chi-square test for categorical variables.
[a]Matching criteria variables. [b]Data available only for 49 Hispanics and 50 non-Hispanic Whites. [c]Income was imputed as median income of the zip code of residence (*Source*. U.S. Census Bureau, 2013b). [d]Five participants reported more than one relative with history of cancer.

Statistics

Descriptive statistics were used to summarize sample characteristics, and covariate balance across groups was confirmed by performing independent two-sample *t* and chi-square tests for continuous and categorical variables, respectively. Matched and covariate-adjusted risk ratios (mRR) and their corresponding 95% confidence intervals (95% CI) were estimated using conditional Poisson regression models with a robust variance estimator (Cummings, 2011) to evaluate the relationship between *H. pylori* serostatus and contributing variables. All analyses were conducted using Stata version 13 (StataCorp. 2014, College Station, TX).

Results

Demographics for men whose samples were used in this study are reported in Table 1. The mean age and BMI were 65.0 (*SD* = 7.5) and 28.7 (*SD* = 4.8), respectively. Most participants were from Bexar County (92.3%). There were no significant differences between Hispanics and NHWs with respect to matching variables (age and county of residence). However, mean BMI was significantly higher in Hispanic participants compared with NHW participants, while significantly more NHWs than Hispanics had a BMI in the normal range. The analysis of additional risk factor data obtained from a subset of 49 Hispanics and 50 NHWs showed no significant differences between ethnic groups in relation to income, smoking status, or family history of cancer. Mean age (65.5; *SD* = 6.4) and BMI (28.2; *SD* = 3.8) of the participants in this subset did not differ from the larger cohort (data not shown).

Almost 20% (56) of samples tested were positive for the *H. pylori* IgG antibody. Infection rates among Hispanics were three times higher than among NHWs, and a strong association between *H. pylori* seropositivity and Hispanic ethnicity was observed, in both the full sample (mRR [95% CI] 3.31 [1.91, 5.73]) and the subsample (3.23 [1.86, 5.6]; Table 2). Both younger (age 49-64; mRR [95% CI] 4.25 [1.69, 10.67]) and older (70-79; 5.67 [1.89, 17]) Hispanics were at significantly higher risk for infection than NHWs. In addition, the subsample analysis demonstrated that Hispanics with lower annual incomes had 4 to 5 times higher risk of infection than NHWs (4.5 [1.13, 18] for income <$36,000; 5.5 [1.38, 22] for $40,000-$50,000). Hispanics with family history of cancer were at higher risk of infection compared with NHWs (4.5 [1.13, 18]).

Table 2. Proportion and Matched Risk Ratios of *H. Pylori* Diagnosis by Ethnicity.

A. Full sample					
	Hispanics (*n* = 142)		NHW (*n* = 142)		
	H. pylori diagnosis (*n*)	Proportion diagnosed (%)	*H. pylori* diagnosis (*n*)	Proportion diagnosed (%)	Hispanics vs. NHW, mRR [95% CI]
All participants	43	30.3	13	9.2	3.31 [1.91, 5.73]
Age group (years)					
49-64	18	25.4	4	5.8	4.25 [1.69, 10.67]
65-69	8	28.6	6	19.4	1.60 [0.57, 4.47]
70-79	17	39.5	3	7.1	5.67 [1.89, 17]
BMI classification					
Normal	4	28.6	0	0.0	—
Overweight	23	34.3	6	10.2	1.8 [0.71, 4.53]
Obese	16	27.6	6	13.6	3.0 [0.31, 28.84]
B. Subsample					
	Hispanics (*n* = 49)		NHW (*n* = 50)		
	H. pylori diagnosis (*n*)	Proportion diagnosed (%)	*H. pylori* diagnosis (*n*)	Proportion diagnosed (%)	Hispanics vs. NHW, mRR [95% CI]
Subsample	42	85.7	13	26.0	3.23 [1.86, 5.6]
Income ($)					
<36,000	9	90.0	2	20.0	4.50 [1.13, 18]
36,001-40,000	6	85.7	2	28.6	3.00 [0.75, 12]
40,001-50,000	11	91.7	2	15.4	5.50 [1.38, 22]
50,001-60,000	8	88.9	2	22.2	4.00 [1.00, 16]
>60,000	8	72.7	5	45.5	1.60 [0.63, 4.05]
Smoking status					
Never	20	90.9	2	10.5	—
Past/current	22	81.5	11	35.5	2.33 [0.99, 5.49]
Family history of cancer					
Colon, lung, prostate, or stomach	17	85.0	4	19.0	4.50 [1.13, 18]

Note. NHW = non-Hispanic White; BMI = body mass index; *H. pylori* = *Helicobacter pylori*; mRR = pair-matched risk ratio; CI = confidence interval. Dashes indicate the ratio was not estimable for that category due to absence of pairs with a non-Hispanic White *H. pylori*-seropositive member.

[a]Reference group.

Likewise, there was a trend toward significant infection risk for past and current Hispanic smokers relative to their NHW counterparts (2.33 [0.99, 5.49]).

Hispanic ethnicity remained a significant predictor of *H. pylori* seropositivity after adjusting for BMI, smoking status, and family history of cancer. Hispanics were almost four times more likely to be *H. pylori* seropositive than NHWs, after adjusting for all three risk factors (Table 3). BMI remained a significant factor independent of ethnicity (mRR 1.19 [1.07, 1.32]), and in all the models where it was included.

Discussion

In an existing cohort of Hispanic and NHW men, an overall *H. pylori* infection rate of 20% was identified, which was consistent with recent regional findings (Patterson, Straten, & Jimenez, 2012). Sonnenberg et al. (2010) reported that gastric biopsies from Texas residents had an overall infection rate of 12.7%. A study done in central Texas reported a prevalence of 24% in persons aged 41 to 60 years compared with other age groups (Patterson et al., 2012), but results were not stratified by ethnicity. As expected, the infection rate among Hispanics was much higher than NHWs (30% vs. 9%), consistent with previous reports of national data (Grad et al., 2012). The elevated infection rate among Hispanics was independent of BMI, smoking status, or family history of cancer. After adjusting for all other contributing variables, Hispanics had almost 4:1 risk of *H. pylori* infection relative to NHWs. The overall infection rate in the current sample was 20%, lower than national (31%) and regional (24%)

Table 3. Matched Risk Ratio of Hispanic Versus NHW (Reference) With *H. Pylori* Diagnosis Adjusted by BMI,[a] Smoking Status, and Family History of Cancer.

Models	Hispanic vs. NHW, mRR[b] [95% CI]	BMI, mRR[a,b] [95% CI]
Ethnicity (Hispanic vs. NHW)[c]	3.31 [1.91, 5.73]	
Ethnicity and		
BMI	3.89 [2.1, 7.19]	1.19 [1.07, 1.32]
Smoking status	3.43 [1.89, 6.2]	
Family history of cancer	3.28 [1.87, 5.77]	
BMI, smoking status	3.79 [2.06, 6.97]	1.19 [1.05, 1.35]
BMI, family history of cancer	3.62 [1.97, 6.64]	1.22 [1.08, 1.37]
BMI, Smoking status, family history of cancer	3.67 [2.0, 6.73]	1.21 [1.06, 1.37]

Note. NHW = non-Hispanic White; BMI = body mass index (kg/m^2); *H. pylori* = *Helicobacter pylori*; mRR = pair-matched risk ratio; CI = confidence interval.
[a]Exponentiated coefficient for BMI is reported for the models where it applies. [b]Matched and covariate-adjusted risk ratios (mRR). [c]Unadjusted matched risk ratio.

seroprevalence rates despite similar timing of exposure to *H. pylori* across studies. Thus, the current results warrant further investigation into environmental and biological factors that could influence infection in heterogeneous populations.

The seropositivity rate of the Hispanics in this study (30%), although similar to that in another study including obese Hispanics (Portocarrero et al., 2012), is lower than a previous report of 57% seropositivity using the same antibody test among Mexican Americans in the San Antonio Family Heart Study (Rubicz et al., 2011). The discrepancy between these results may be explained by a previous comparative study between SABOR and San Antonio Family Heart Study cohorts by Beuten et al. (2011). Analyses of genetic admixtures of Hispanics from both cohorts revealed significant differences in the proportions of European and Native American ancestry. This variation is a potential contributing factor to susceptibility to *H. pylori* infection. Analysis of ethnic ancestry and seropositivity in Hispanics and NHWs in the SABOR cohort could help elucidate the contribution of ethnicity to *H. pylori* infection rate and risk of gastric cancer.

Elevated BMI was also observed to increase the risk for *H. pylori* seropositivity. This may seem counterintuitive given *H. pylori*'s negative effects on ghrelin, and therefore appetite, levels (Weigt & Malfertheiner, 2009). *H. pylori* eradication studies have reported increases in both ghrelin levels and growth in infected children (Yang, Sheu, Yang, Lu, & Chuang, 2012); however, the overall interaction of infection with the brain–gut axis is likely more complex (Budzynski & Klopocka, 2014). Nevertheless, future studies should be geared toward investigating the link between obesity and *H. pylori* as a potential mediator for the development of GA in this region, given the high obesity rates in STX (37.9% of Hispanics vs. 24.6% of NHWs) and Texas (35.2% of Hispanics vs. 25.2% of NHWs; Ramirez et al., 2013; Texas Department of State Health Services, 2013).

This study has some limitations. Data on tobacco use, income, and family history of cancer was obtained from only 99 participants. The sample size was too small to determine risk differences for specific cancers, with the exception of prostate for which no difference between ethnic groups was observed. Due to imputation of income from geographic location, which was matched for each pair of participants in the study, the analysis in Table 3 could not be adjusted by income. Such an analysis in a future study would be of great interest. Data on acculturation, generational status, and country of origin were not available. These variables can influence diet, gut bacteria, and metabolic processes, such as obesity, that are associated with cancer, and should be included in future studies. In addition, the study cohort excluded women, and gastric cancer is more than twice as prevalent in Hispanic women compared with NHW women (American Cancer Society, 2015; Ramirez et al., 2013). The minimum eligible age of SABOR participants (50 years) means the finding of gastric cancer among younger Hispanics (Al-Refaie et al., 2008) cannot be linked to *H. pylori* serostatus nor can other findings (Patterson et al., 2012) of higher infection rates among 41- to 60-year-olds compared with other age groups be corroborated. Additional studies with larger samples and multicohort comparisons are needed to further characterize *H. pylori* infection among Hispanics. Case-control studies are also needed to determine the proportion of gastric cancer cases related to *H. pylori* in Hispanics. The results of these future investigations could help inform effective primary surveillance and prevention approaches, not only for reducing risk of gastric cancer but more proximal health outcomes like peptic ulcer disease and atrophic gastritis (a precursor of gastric cancer; Malfertheiner et al., 2012) in this underserved population.

Declaration of Conflicting Interests

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References

Adams, D. A., Gallagher, K. M., Jajosky, R. A., Kriseman, J., Sharp, P., Anderson, W. J., . . . Abellara, J. P. (2013). Summary of notifiable diseases—United States, 2011. *Morbidity and Mortality Weekly Reports*, 60(53), 1-117. Retrieved from http://www.cdc.gov/mmwr/preview/mmwrhtml/mm6053a1.htm

Adriani, A., Repici, A., Hickman, I., & Pellicano, R. (2014). *Helicobacter pylori* infection and respiratory diseases: Actual data and directions for future studies. *Minerva Medica*, 105(1), 1-8.

Al-Refaie, W. B., Tseng, J. F., Gay, G., Patel-Parekh, L., Mansfield, P. F., Pisters, P. W., . . . Feig, B. W. (2008). The impact of ethnicity on the presentation and prognosis of patients with gastric adenocarcinoma: Results from the National Cancer Data Base. *Cancer*, *113*, 461-469. doi:10.1002/cncr.23572

American Cancer Society. (2015). *Cancer facts and figures for Hispanics/Latinos 2015-2017*. Retrieved from http://www.cancer.org/acs/groups/content/@research/documents/document/acspc-046405.pdf

Beuten, J., Gelfond, J. A., Franke, J. L., Shook, S., Johnson-Pais, T. L., Thompson, I. M., & Leach, R. J. (2010). Single and multivariate associations of *MSR1, ELAC2*, and *RNASEL* with prostate cancer in an ethnic diverse cohort of men. *Cancer Epidemiology, Biomarkers & Prevention*, *19*, 588-599. doi:10.1158/1055-9965.EPI-09-0864

Beuten, J., Halder, I., Fowler, S. P., Groing, H. H., Duggirala, R., Arya, R., . . . Lehman, D. M. (2011). Wide disparity in genetic admixture among Mexican Americans from San Antonio, TX. *Annals of Human Genetics*, *75*, 529-538. doi:10.1111/j.1469-1809.2011.00655.x

Blaser, M. J., Perez-Perez, G. I., Kleanthous, H., Cover, T. L., Peek, R. M., Chyou, P. H., . . . Nomura, A. (1995). Infection with *Helicobacter pylori* strains possessing *cagA* is associated with an increased risk of developing adenocarcinoma of the stomach. *Cancer Research*, *55*, 2111-2115.

Budzynski, J., & Klopocka, M. (2014). Brain-gut axis in the pathogenesis of *Helicobacter pylori* infection. *World Journal of Gastroenterology*, *20*, 5212-5225. doi:10.3748/wjg.v20.i18.5212

Cummings, P. (2011). Estimating adjusted risk ratios for matched and unmatched data: An update. *Stata Journal*, *11*, 290-298.

Franceschi, F., Tortora, A., Gasbarrini, G., & Gasbarrini, A. (2014). *Helicobacter pylori* and extragastric diseases. *Helicobacter*, *1*, 52-58.

Garcia-Gonzalez, M. A., Bujanda, L., Quintero, E., Santolaria, S., Benito, R., Strunk, M., . . . Lanas, A. (2015). Association of PSCA rs2294008 gene variants with poor prognosis and increased susceptibility to gastric cancer and decreased risk of duodenal ulcer disease. *International Journal of Cancer*, *137*, 1362-1373.

Gonzalez, C. A., Megraud, F., Buissonniere, A., Lujan Barroso, L., Agudo, A., Duell, E. J., . . . Riboli, E. (2012). *Helicobacter pylori* infection assessed by ELISA and by immunoblot and noncardia gastric cancer risk in a prospective study: The Eurogast-EPIC project. *Annals of Oncology*, *23*, 1320-1324. doi:10.1093/annonc/mdr384

Grad, Y. H., Lipsitch, M., & Aiello, A. E. (2012). Secular trends in *Helicobacter pylori* seroprevalence in adults in the United States: Evidence for sustained race/ethnic disparities. *American Journal of Epidemiology*, *175*, 54-59. doi:10.1093/aje/kwr288

Graham, D. Y. (2015). *Helicobacter pylori* update: Gastric cancer, reliable therapy, and possible benefits. *Gastroenterology*, *148*, 719-731.

Kapetanakis, N., Kountouras, J., Zavos, C., Michael, S., Tsarouchas, G., Gavalas, E., . . . Moschos, I. (2012). Re: *Helicobacter pylori* infection and colorectal cancer risk: Evidence from a large population-based case-control study in Germany. *American Journal of Epidemiology*, *176*, 566-567. doi:10.1093/aje/kws302

Ladeiras-Lopes, R., Pereira, A. K., Nogueira, A., Pinheiro-Torres, T., Pinto, I., Santos-Pereira, R., & Lunet, N. (2008). Smoking and gastric cancer: Systematic review and meta-analysis of cohort studies. *Cancer Causes & Control*, *19*, 689-701. doi:10.1007/s10552-008-9132-y

Lin, X. J., Wang, C. P., Liu, X. D., Yan, K. K., Li, S., Bao, H. H., . . . Liu, X. (2014). Body mass index and risk of gastric cancer: A meta-analysis. *Japanese Journal of Clinical Oncology*, *44*, 783-791. doi:10.1093/jjco/hyu082

Malfertheiner, P., Megraud, F., O'Morain, C. A., Atherton, J., Axon, A. T., Bazzoli, F., . . . Kuipers, E. J. (2012). Management of *Helicobacter pylori* infection—the Maastricht IV/Florence Consensus Report. *Gut*, *61*, 646-664. doi:10.1136/gutjnl-2012-302084

Munoz, E., Westin, G. F., Long Parma, D., Suarez, L., & Ramirez, A. G. (2015, October). *Abstract B13: Gastric cancer disparities in Latino populations in South Texas, Texas and the United States*. Paper presented at the American Association for Cancer Research 7th Annual Conference on the Science of Cancer Health Disparities, San Antonio, TX.

National Cancer Institute. (2013). *Surveillance, Epidemiology, and End Results (SEER) Program SEER*Stat Database: Incidence-SEER 13 Regs Research Data, Nov 2013 Sub (1992-2011)*. Bethesda, MD: Author.

Patel, S., Lipka, S., Shen, H., Barnowsky, A., Silpe, J., Mosdale, J., . . . Krishnamachari, B. (2014). The association of *H. pylori* and colorectal adenoma: Does it exist in the US Hispanic population? *Journal of Gastrointestinal Oncology*, *5*, 463-468.

Patterson, T., Straten, E., & Jimenez, S. (2012). The prevalence of *Helicobacter pylori* antibody in different age groups in Central Texas. *Clinical Laboratory Science*, *25*, 102-106.

Portocarrero, D. J., Olafsson, S., Jackson, C. S., Doss, L., & Malamud, A. (2012). Obese minorities have a higher prevalence of *H. pylori* than do whites, but nonsignificant differences in upper gastrointestinal tract findings, before laparoscopic adjustable gastric banding. *Journal of Clinical Gastroenterology*, *46*, 431-432. doi:10.1097/MCG.0b013e31824c0f45

Ramirez, A. G. (2013). The dire need for cancer health disparities research. *Health Education Research*, *28*, 745-747. doi:10.1093/her/cyt089

Ramirez, A. G., Gallion, K. J., Suarez, L., Giachello, A. L., Marti, J. R., Medrano, M. A., . . . Trapido, E. J. (2005). A national agenda for Latino cancer prevention and control. *Cancer*, *103*, 2209-2215. doi:10.1002/cncr.21053

Ramirez, A. G., Thompson, I. M., Jr., & Vela, L. (Eds.). (2013). *The South Texas health status review: A health disparities roadmap*. New York, NY: Springer.

Rubicz, R., Leach, C. T., Kraig, E., Dhurandhar, N. V., Grubbs, B., Blangero, J., . . . Goring, H. H. (2011). Seroprevalence of 13 common pathogens in a rapidly growing U.S. minority population: Mexican Americans from San Antonio, TX. *BioMed Central Research Notes*, *4*, 433. doi:10.1186/1756-0500-4-433

Siegel, R., Naishadham, D., & Jemal, A. (2012). Cancer statistics for Hispanics/Latinos, 2012. *CA: A Cancer Journal for Clinicians*, *62*, 283-298. doi:10.3322/caac.21153

Sokic-Milutinovic, A., Alempijevic, T., & Milosavljevic, T. (2015). Role of *Helicobacter pylori* infection in gastric carcinogenesis: Current knowledge and future directions. *World Journal of Gastroenterology*, *21*, 11654-11672.

Sonnenberg, A., Lash, R. H., & Genta, R. M. (2010). A national study of *Helicobactor pylori* infection in gastric biopsy specimens. *Gastroenterology*, *139*, 1894-1901.

Tatishchev, S. F., Vanbeek, C., & Wang, H. L. (2012). *Helicobacter pylori* infection and colorectal carcinoma: Is there a causal association? *Journal of Gastrointestinal Oncology*, *3*, 380-385. doi:10.3978/j.issn.2078-6891.2012.058

Testerman, T. L., & Morris, J. (2014). Beyond the stomach: An updated view of *Helicobacter pylori* pathogenesis, diagnosis, and treatment. *World Journal of Gastroenterology*, *20*, 12781-12808.

Texas Department of State Health Services. (2013). *Texas Cancer Registry SEER*Stat Database, 1995-2010 Limited-Use Incidence, Texas statewide based on NPCR-CSS Submission, cut-off 11/30/12*. Austin, TX: Author.

U.S. Census Bureau. (2013a). *2012 National Population Projections*. Retrieved from https://www.census.gov/population/projections/data/national/2012.html

U.S. Census Bureau. (2013b). *American FactFinder: Community facts*. Retrieved from http://factfinder.census.gov/faces/nav/jsf/pages/index.xhtml

U.S. Census Bureau. (2014). *State and County QuickFacts*. Retrieved from https://www.census.gov/quickfacts/table/PST045216/00

Weigt, J., & Malfertheiner, P. (2009). Influence of *Helicobacter pylori* on gastric regulation of food intake. *Current Opinion in Clinical Nutrition and Metabolic Care*, *12*, 522-525. doi:10.1097/MCO.0b013e32832eb56e

Wu, Q., Yang, Z. P., Xu, P., Gao, L. C., & Fan, D. M. (2013). Association between *Helicobacter pylori* infection and the risk of colorectal neoplasia: A systematic review and meta-analysis. *Colorectal Disease*, *15*, e352-e364.

Yang, Y. J., & Sheu, B. S. (2016). Metabolic interaction of *Helicobacter pylori* infection and gut microbiota. *Microorganisms*, *4*, 15. doi:10.3390/microorganisms4010015

Yang, Y. J., Sheu, B. S., Yang, H. B., Lu, C. C., & Chuang, C. C. (2012). Eradication of *Helicobacter pylori* increases childhood growth and serum acylated ghrelin levels. *World Journal of Gastroenterology*, *18*, 2674-2681.