

A Survey Of Prevalence Of *Helicobacter pylori* Within Vietnamese Community Through The Vietnam Health Clinic

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Abstract

Helicobacter pylori (*H. pylori*) are gastric bacteria known to cause chronic gastritis, peptic ulcers, and can eventually lead to gastric adenocarcinoma in the long term. Prevalence of *H. pylori* among Caucasian, Hispanic and African-American populations has been surveyed in previous studies. However, prevalence and distribution of *H. pylori* among Asian communities has yet to be exhaustively profiled. While *H. pylori* exhibits worldwide circulation, studies have shown that infection is more prevalent in developing countries. The Vietnam Health Clinic (VHC) is a mobile clinic that provides free healthcare to underserved populations in Vietnam every year. The data collected from the roughly 3000 patients served from VHC trips between 2012 and 2014 presents an invaluable opportunity to learn more about *H. pylori* prevalence and distribution within the native Vietnamese community. By delving into the patient's background, medical history and previous treatment history of *H. pylori*, we have produced a level of granularity in our dataset that can facilitate novel prevention and treatment processes in future VHC trips. Tabulation of data and statistical analysis was done with Tableau and Excel. As this is a retrospective and exploratory study, a causal relationship cannot be confidently deduced. However, by providing insight into the relationship between patient demographic factors and *H. pylori*, our research can help to provide a foundation for causally directed studies in the future.

Keywords: *Helicobacter pylori*, Vietnam Health Clinic (VHC), Public Health

1. Introduction

Helicobacter pylori (*H. Pylori*) is among the most ubiquitous chronic bacterial infections in the world. Some questions have been posed about whether susceptibility to *H. Pylori* has to do with ethnicity or community factors. Many studies in the past have investigated *H. Pylori* cases within Mexican and African-American communities, but little research has been conducted with Asian communities. The survey data collected from the Vietnam Health Clinic in 2014 (Trip 6.0) hopes to shed light unto the characteristics of *H. Pylori* infection within Asian communities, specifically Vietnamese in this case, and provide valuable insight for the upcoming VHC trip in 2017 (Trip 8.0). This study focuses on whether intrinsic factors—age, gender, hereditary—or extrinsic factors—environment, living conditions—are associated with infection by *H. Pylori*, a classic tension between nature and nurture. The hypothesis is that *H. Pylori* susceptibility in Asian communities is associated with intrinsic factors.

2. Review Of Literature And Background

2.1 *Helicobacter Pylori*:

Transmission pathway, bacteriology, microbiology, and gastric adaptation

Helicobacter pylori (*H. Pylori*) is a gram negative bacterium that binds to the human gastric mucosa. Infection often occurs at a young age and through person-to-person contact. Identifying an infected person is difficult because acute symptoms are not immediately shown. In fact, the host may go through a majority of their life without any symptoms or signs of infection. People infected with *H. pylori* will begin developing symptoms as they age, if the bacteria accrue enough time to manifest and grow.^{1,3,5} Why this bacterium infects its host at a young age without causing any acute symptoms is not yet understood. Previous studies present mechanisms by which the bacterium infects the host and adapts to the host's defensive system.

H. pylori is sensitive to low pH levels within the human gastrointestinal system. *H. pylori* prefers environments with a pH of approximately 5-7. Within the stomach, the pH is 1-2 during the fasting period, and 4-5 when the host is consuming food. The bacteria can only survive several minutes within the gastric system when the pH is too acidic.^{2,5} There are 3 major factors that play into successful colonization by the bacterium: its physical attributes, the ability to produce urease, and the outer membrane proteins.

H. pylori has a helical morphology and moves through the environment through the use of flagella.⁵ Coupling the two attributes, the bacteria have the ability to swim, like a corkscrew, to the gastric epithelium. Studies show that *H. pylori* has chemotactic abilities to sense and orient itself to higher pH areas. This orients the bacterium toward areas with the desired pH levels and allows it to then move in the direction of the higher pH environment.¹ A meta-analysis by Harry Mobley concluded that all 3 structures must be present for successful colonization.^{2,3}

A crucial factor that determining *H. pylori*'s chance of successful infection is the massive production of urease. The bacteria are unable to adhere to the gastric epithelium without urease.^{2,4} Urease's primary purpose is aiding adherence of the bacterium to the gastric epithelium tissue by providing resources. The compound also aids in placing the bacteria in an environment that will provide the greatest chance of survival.

Once bound to the tip of the mucosal microvilli of the epithelial cell, *H. pylori* bacteria must continue to move deeper into the epithelium and evade the host's immune system. There is no central mechanism that gives *H. pylori* the ability to adhere and adapt to the epithelial cells; this ability is achieved through a combination of many factors—physical attributes, outer membrane proteins, and virulence factors—enabling the bacteria to infect and survive in the mucosa. The human gut epithelium has a natural process of preventing the adherence of *H. pylori* via production of Mucin-1 (MUC1) and antibodies. With the help of urease, and the bacteria's motility, again, *H. pylori* can bypass the MUC1 mucus layer by altering fluid properties. The other factors are responsible for exploiting the host's immune system: the outer membrane proteins—Lipopolysaccharide (LPS)—and virulence factors—Vacuolating cytotoxin A (VacA) and Cytotoxin-associated gene A (CagA).⁴

2.2 Pathophysiology: Indications And Diagnostic Tests

According to the American College of Gastroenterology, diagnostic testing should be performed in patients with gastric mucosa-associated lymphoid tissue (MALT) lymphoma, active peptic ulcer disease, or a past history of documented peptic ulcer. The strategy of testing and treating if positive is indicated in populations under 55 who don't have any red flags, which include: bleeding, anemia, early satiety, unexplained weight loss, progressive dysphagia, odynophagia, recurrent vomiting, family history of gastrointestinal cancer and previous esophagogastric malignancy.⁶ In low-risk patients below 55, using a non-invasive method, such as the outlined Urea Breath Test (UBT) or Stool Antigen Test, is recommended. Conversely, endoscopic biopsies are more expensive and invasive and should thus be reserved for the populations whose diagnostic endoscopies revealed gastric or duodenal ulcers.⁶

Antibacterial treatment for *H. Pylori* for ulcer patients without initial diagnostic confirmation is not recommended despite the fact that the infection is highly prevalent in those populations. *H. Pylori* negative ulcer cases have been increasingly recognized, and the empirical treatment for such cases causes adverse patient outcomes. Endoscopic methods, particularly antral biopsy urease tests, are the primary diagnostic routes for determining if a patient is infected.⁶ While the actual antral biopsy kits are inexpensive, obtaining the tissue necessary for the procedure is generally expensive.⁸ Antral biopsies have a sensitivity between 90 and 95 percent, and a specificity between 95 and 100 percent, making false positives uncommon.⁶ A non-invasive alternative is the rapid UBT, which can detect *H.*

Pylori liberate CO₂ using a carbon isotope. In both methods, false negatives can occur in populations with recent gastrointestinal bleeding, or with the use of Proton Pump Inhibitors (PPIs), antibiotics, or bismuth-containing compounds.⁸ It is even more expensive to obtain the tissue necessary for an antral biopsy in those populations. Negative results should be confirmed with histology (expensive), serology or fecal antigen testing in order to more accurately ascertain *H. Pylori* infection status.⁶

Serology is an inexpensive and noninvasive diagnostic method that is well suited for a primary care setting but is inherently limited by its inferior specificity and sensitivity.⁷ This method should especially be avoided in low prevalence populations. Serology is found to be most useful in confirming eradication of *H. Pylori* in populations that have been treated for the infection for at least a year. A decision analysis evaluating cost-effectiveness of eradication confirmation options found stool testing to be more cost effective than UBT and rapid urease testing.⁹ Like other tests that use urea as a marker, fecal antigen testing suffers from the production of false positives in patients using PPI's or bismuth-containing compounds, albeit at a smaller rate.⁸

American College of Gastroenterology advises that all patients receiving treatment for *H. Pylori* confirm the eradication of the infection considering the widespread availability of accurate, noninvasive tests.⁶ The panel especially recommends confirmation of eradication in patients who have persistent symptoms after *H. pylori* treatment for dyspepsia or had an *H. pylori*-associated ulcers, gastric MALT lymphomas, or resections for early gastric cancer.⁶

2.3 Epidemiology

Helicobacter pylori affects 50% of the world's population.¹⁰ The organism is common among adults and children. However, rates of infection vary significantly across the globe. Infection is more common in developing countries. Prevalence in these parts of the world is higher than developed nations by two-fold.¹¹

There is no association between *H. pylori* infection and gender.¹² Epidemiological studies across Southeast Asia suggest acquisition of *H. pylori* often occurs during childhood. High rates of infection are evident in children within the first few years of life. A more drastic increase of infection is apparent between ages three and six. This trend continues to increase gradually throughout adulthood.^{13,14} By age sixty, prevalence of *H. pylori* in adults reaches about 80% in most developing countries.¹¹ These results are consistent with similar studies conducted across the globe.^{12,15,16}

H. pylori is associated with socioeconomic status and poor living conditions during childhood. Incidence of *H. pylori* is higher among children living in communities of lower socioeconomic status (SES).¹⁷ Children in these communities also experience higher rates of infection at a younger age.¹⁷ High density living conditions during childhood is another risk factor for *H. pylori*. In Japan, children who attend nursery programs or live in households with seven or more members have a higher chance of infection.^{18,19} In England, researchers concluded that children who share beds for most of their childhood are also more prone to infection.²⁰

Poor hygiene is also linked to acquisition of *H. pylori*. Prevalence is higher for those who report often drinking well water and bathing in ponds.^{21,22} This is also true for those reporting that they infrequently boil feeding bottles and do not wash hands often.^{17,21,22} In contrast, there are studies showing that drinking tap water and using flush toilets reduces rates of infection.²² Evidence of reduced infection through improved water sanitation has been demonstrated in Japan. The implementation of the public water service in 1956 greatly contributed to decreasing the rate of *H. pylori* infection throughout the country.¹⁸ The seroprevalence of *H. pylori* in Japanese children alone dropped from 25% in 1997 to 1.8% in 2010 across several villages.²¹

2.4 Hereditary Susceptibility

Hereditary susceptibility to *H. pylori* infection is possible, but has not been proven. Recent twin studies suggest the concordance rate for *H. pylori* infection is higher in monozygotic twins than dizygotic twin pairs. Sharing the same rearing environment during childhood also influences *H. pylori* infection.²³ Some studies indicate ethnicity as an emerging risk factor. For instance, Hispanics and Southeast Asians have a higher rate of infection than Caucasians.^{11,16} However, confounding environmental factors make it difficult to definitively determine causality.

2.5 Diseases Relevant To *Helicobacter Pylori*: Gerd, Ulcers, Cancers

Helicobacter pylori is associated with gastritis, or inflammation of gastric mucosa. There are two types of gastritis: acute and chronic. Acute gastritis occurs after ingestion of *H. pylori*, however it is very uncommon.²⁴ Chronic gastritis, in contrast, occurs when *H. pylori* resides in the lining of the gastric mucosa and affects two-thirds of the global population.²⁵

H. pylori is strongly linked to gastric adenocarcinoma and gastric lymphoma. Prevalence of *H. pylori* in intestinal-type gastric cancer is significantly higher than diffuse cancer. This supports the theory that most infections are attributed to infection by *H. pylori*.²⁶ Furthermore, global-scale serological studies show that there is a six-fold increased risk of gastric cancer in populations with 100% of *H. pylori* infection compared to populations not infected at all.²⁷ In gastric lymphoma, or MALToma, the presence of *H. pylori* causes mucosa-associated lymphoid tissue (MALT) to form in the stomach, which otherwise would not exist.²⁸ Serum immunoassays from several studies indicate that the majority of patients with MALToma are also infected by *H. pylori*.²⁸

H. pylori is also associated with peptic ulcer disease. This mainly takes the form of duodenal ulcers and gastric ulcers. Immunoassays in Chinese and Japanese-American populations show high association between *H. pylori* infection and the presence of both types of peptic ulcer disease.^{29,30} Although some believe *H. pylori* is associated with Gastroesophageal reflux disease (GERD), conflicting results among different studies suggest there is currently no direct link between *H. pylori* and GERD.²⁹

2.6 Treatment Regimens

Over time, numerous treatment techniques have been developed to treat *H. pylori*. First-line *H. pylori* eradication therapy varies in regimen and duration of treatment from country to country. While there is no consistent bona fide first-line treatment for *H. pylori*, there are general guidelines for treatment. The challenges to treatment success are cost, side effects, and patient compliance. Treatments being halted due to side effects occurs only rarely, and pregnant patients normally hold off treatment until after pregnancy. The most common first-line therapy is triple therapy (TT). This consists of two antibiotics and a Proton Pump Inhibitor (PPI) for 7-14 days.^{32,35} Common antibiotics used are amoxicillin, clarithromycin, or metronidazole. Developing countries tend to place patients on triple therapy for 7 days, while patients in industrialized countries are placed on therapy for 14 days. If initial triple therapy is ineffective, the patient will have the option of sequential therapy or quadruple therapy for retreatment.^{32,35}

Sequential therapy (ST) is similar to triple therapy, but differs in durations and sequences of the medication taken. In sequential therapy, the patient will take an antibiotic and a PPI for 5 days. Following the initial 5 days, the patient switches to taking 2 other antibiotics for another 5 days. In Bojan Tepes' meta-analysis, Israel, Korea, and Taiwan reported a higher eradication rate in initial treatments using TT. However, a 95% success in re-eradication was reported after initial treatment using ST compared to a 70% using TT.³⁵ Subsequent studies by Luigi Gatta et al. concluded that ST was more effective than TT on antibiotic resistant strains.³² ST has been shown to be equal in terms of efficacy for treating *H. pylori* as quadruple therapy (QT). Deng-Chyang Wu and colleagues verified these claims in their clinical study in 2010. They noted both techniques are effective, but QT is recommended for rare cases of double antibiotic resistance.³⁶ While still viable, higher rates of error in patient compliance prevents ST from being a reliable first line treatment.

Quadruple therapy (QT) consists of 2 antibiotics, a PPI, and bismuth subsalicylate. QT showed a cure rate of 88-91% in patients with and without antibiotic resistance.³⁵ Only a 3% difference was shown in favor of those without antibiotic resistance than those with.³¹ Tepes' experiment in China compared the success rate of QT versus TT in antibiotic resistant strains. They found QT had a rate of 82.1%, while TT had only 66.7%. No differences in success rate between duration of treatment was noted in Tepes' study. Quadruple therapy is known to be difficult in respect to patient compliance. To simplify compliance, the FDA approved a new treatment called combination capsule. These capsules contain bismuth subcitrate 140mg, Metronidazole 125 mg, & tetracycline 125 mg, and is taken with a PPI.³⁴ It is similar to receiving QT but simpler for patient compliance as opposed to taking 4 separate medications. Though scarce in data, several experiments have shown the combination pill to have the same efficacy compared to QT.³³

At this time, QT is recommended as the first line treatment for *H. pylori* with antibiotic resistance. When it comes to *H. pylori* with no resistance, QT and ST were demonstrated to have equivalent efficacy, but proper compliance complicates those options. In 2007, L. Fischback concluded TT continues to be a viable treatment if sensitivity to the antibiotics exist. If the strains have resistance, success in turn will plummet.³¹ Triple therapy remains the most often used first line treatment because identification of resistance is difficult, especially in developing countries. The treatment's effectiveness hinges on patient compliance and resistance susceptibility. Many believe the future of treating *H. pylori* is heading towards the combination pill. However, more comparisons between QT, ST, TT, and the combination pill are needed to further elucidate a single generally recommended first line therapy option.

3. Methods

3.1 Data Collection And Measures

Patients were selected by socioeconomic status through a third-party Vietnamese non-profit organization prior to visiting the Vietnam Health Clinic (VHC). A pre-survey was collected in person and/or through public record. For data collection, nurses or student volunteers asked patients to take public health survey questionnaires during clinic transition time. Public health data was recorded on paper during clinic. The public health survey questionnaire and VHC Intake Form Final will be submitted separately in the supplementary data.

The public health data was later computerized for database storage using Microsoft Access. The fragmented data from Access was compiled and data of interest was extracted to Microsoft Excel. Data analysis was performed using Microsoft Excel and Tableau Desktop.

3.2 Public Health Questionnaire³⁸

Besides patient's health information (age, gender, health history), the public health survey's specific questionnaires are:

a. HH1 – HHTotal: How many people live in your household?

Answer options: Numerical value

b. HH2 – HHincome: In the past 12 months, what is the estimated income per month for the entire household? (Thousand or Million Vietnamese Dong per Month)

Answer options: 1 = <100K (< \$4.48); 2 = 100K - 500K (= \$4.48 - \$22.41); 3 = 500K - 1M (= \$22.41 - \$44.81); 4 = 1M - 3M (= \$44.81 - \$134.43); 5 = >3M (> \$134.43); -99 = Don't know/Refused to answer [Currency conversion rate is \$1 = 22,306.49 Vietnamese Dong]

c. HH3 – HHwater: What is the main source of cooking/drinking water for your household?

Answer options: 1 = Tap Water; 2 = Rain Water; 3 = River Water; 4 = Self-dug Well Water; 5 = Public Well Water; -99 = Don't know/Refused to answer

d. HH4 – HHfreq_doc: In the past 12 months, how many times has your family visited the doctor, dentist, or pharmacist?

Answer options: 1 = 0 visits; 2 = 1 – 2 visits; 3 = 3 – 4 visits; 4 = 5 – 7 visits; 5 = 8+ visits; -99 = Don't know/Refused to answer

e. HH5 – Literate and Education: Can you read and write?

Answer options: 1 = yes; 0 = no

3.3 Inclusion And Exclusion Criteria

All data were obtained voluntarily and patients had the option of refusing to answer the public health survey. For the *H. pylori* infected patient group, percentages were calculated with the overall population of 52 patients who had *H. pylori*. The total is considered as the total of *H. pylori* patients who answered the public health survey, excluding the patients who chose the "Don't know/Refused" option and the missing data points (both are highlighted yellow in Table 1).

For example, in Table 1, under Household Water category, with 52 *H. pylori* patients, only 29 data points are available, with 22 data points that were missing, and 1 patient refusing to answer. Therefore, the total *H. pylori* patients for the calculation is 29 patients; for percentages of patients who have access to Tap Water, the calculation will be 2 divided by 29 and multiplied by 100.

3.4 Tools

3.4.1 primary literature resource

Access and download permissions were through the licenses of University of Washington (UW) Library. Primary literature search engines are from UpToDate, PubMed, Google Scholar, and UW Library.

3.4.2 software

Tableau Desktop Professional Edition 9.3 (Version 9300.16.0520.1152)

Microsoft Office 365 (Version 16.0.6001.1078): Microsoft Access and Microsoft Excel for Database

4. Results

The Vietnam Health Clinic (VHC) 6.0 in 2014 was between August 24, 2014 to September 03, 2014. VHC provided free health care at 8 clinic sites: Site Number 1 – Lộc Sơn, Phú Lộc; Site Number 2 – Thủy Thanh, Hương Thủy; Site Number 3 – Quảng An, Quảng Điền; Site Number 4 – Phong Sơn, Phong Điền; Site Number 5 – Phong Xuân, Phong Điền; Site Number 6 – Quảng Phú, Quảng Điền; Site Number 7 – Thượng Nhật, Nam Đông; Site Number 8 – Thượng Long, Nam Đông.³⁷ Each clinic site has a unique demographic of patients.

The total patients visited the VHC 6.0 was 1967 patients, according to database records. The compiled data of 52 *H. pylori* patients' profiles are summarized in Table 1. The term patients from this point on and below are considered as patients from the 52 *H. pylori* patients group.

4.1 overview

Table 1a. Patient demographics and clinic sites

HELICOBACTER PYLORI SUMMARY			
AGE	ALL (n = 52)	MALE (n = 16)	FEMALE (n = 36)
Min		2	19
Max		94	79
Mean		55.7	62.8
Median		56.5	63.5
Standard Deviation		18.297	16.771
		18.061	
HHTotal (n = 51)			
Min		1	
Max		10	
Mean		4.8	
Median		5	
Standard Deviation		1.9	
SITE SUMMARY			
SiteNumber	Total Patients	Site Village	Site District
1	9	Lộc Sơn	Phú Lộc
2	9	Thủy Thanh	Hương Thủy
3	2	Quảng An	Quảng Điền
4	4	Phong Sơn	Phong Điền
5	0	Phong Xuân	Phong Điền
6	8	Quảng Phú	Quảng Điền
7	2	Thượng Nhật	Nam Đông
8	18	Thượng Long	Nam Đông
Total	52		
GERD (n = 52)		TOTAL	
	12		23.08%
SUMMARY		52 out of 1967 patients have H. Pylori	2.64%
		57 Student Volunteers	
		19 Health Professionals	

Clinic site 1 (9 patients), 2 (9 patients), 6 (8 patients), and 8 (18 patients) have the highest number of patients with *H. pylori*. The clinic sites are accessible through main roads, but are at great distances to each other so no overlapped households nor population.

Regarding patient demographics, 36 patients (69.23%) are female and 16 patients (30.77%) are male. Average age of male is 62.8, which is older than the average age of female of 52.6. The youngest *H. pylori* patient is a 2-year-old female and the oldest *H. pylori* patient is a 94 years old male.

In this group of 52 patients, the bell curve, graph not shown, is slightly skewed to the left with the peak on the right. The median is higher than the mean (56.5 > 55.7)—the *H. pylori* patients group (n = 52 patients) belongs to the older group within the Vietnamese population. Outliers are the 2 years old female and 94 years old male.

Table 1b. Education, water access, income, and other factors

HELICOBACTER PYLORI SUMMARY		
NUMBER OF PATIENTS (n = 52)		
Male	16	30.77%
Female	36	69.23%
LITERATE (n = 52)		
Yes	32	61.54%
No	20	38.46%
EDUCATION (n = 51)		
0 = No formal schooling	20	39%
1 = Primary school (1-5)	20	39%
2 = Middle School (6-9)	7	14%
3 = High School (10-12)	4	8%
4 = College or Postgrad	0	0%
-99 = Don't know/Refused	1	
HOUSEHOLD WATER (n = 29)		
1 = Tap Water	2	7%
2 = Rain Water	11	38%
3 = River Water	15	52%
4 = Self-dug well Water	1	3%
5 = Public Well Water	0	0%
-99 = Don't know/Refused	1	
0 = Missing Data	22	
HOUSEHOLD INCOME (n = 16)		
1 = < 100K = < \$4.48	8	50%
2 = 100K - 500K = \$4.48 - \$22.41	2	13%
3 = 500K - 1M = \$22.41 - \$44.81	3	19%
4 = 1M - 3M = \$44.81 - \$134.43	3	19%
5 = > 3M = > \$134.43	0	0%
-99 = Don't know / refuse	23	
0 = Missing Data	13	
HOUSEHOLD FREQ DOCTOR VISIT		
1 = 0 visit	11	23%
2 = 1-2 visits	6	13%
3 = 3-4 visits	6	13%
4 = 5-7 visits	17	35%
5 = 8+ visits	0	0%
-99 = Don't know/Refuse	8	17%
0 = Missing Data	4	

On literacy and education, most patients can write, read, and understand Vietnamese (61.54%). However, the majority of patients have no formal schooling (39%) or stopped at primary school (39%). Most patient household income is less than \$4.48 per year, 8 patients out of 16 patients reported (50%).

On the number of people per household, out of 51 *H. pylori* patients (1 data point missing), the maximum people per household is 10, the average people per household is 4.8, and the median is 5 people per household. Out of 52 *H. pylori* patients, 12 patients (23.08%) have GERD. For access to water resource, the majority of patient use river water, 15 patients (52%), and 11 patients (38%) use rain water; only 2 patients (7%) have access to tap water. Finally, for the frequency of doctor visit, the distribution peaks at either 0 visits, 11 patients out of 48 patients (23%), or 5 – 7 visits, 17 patients out of 48 (35%).

5. Conclusion And Discussion

5.1 Data Analysis And Implication

The Vietnam Health Clinic 5.0 (VHC 5.0), which visited the same areas in 2012, had 24 *H. pylori* patients out of a total of 1962 patients (1.25%).^{37, 39} Comparing to the VHC 5.0 in 2012, the VHC 6.0 in 2014 has a higher prevalence of *H. pylori* cases at 52 cases out of 1967 patients (2.64%). With the same patient population—Hue, Vietnam—the increase in local population and crowding may have contributed to the increase in *H. pylori* cases.

Overwhelming evidence indicates higher rates of *H. pylori* infection among Hispanic and African American populations. This led us to believe that susceptibility to *H. pylori* infection is hereditary. However, scientific literature reveals that this has not yet been confirmed. High prevalence of infection within certain ethnic groups is instead attributed to environmental risk factors.

These factors include poor hygiene and consumption of contaminated water. This is supported by data obtained from the Vietnam Health Clinic 6.0 (VHC 6.0). Of the patients diagnosed with *H. pylori*, 52% reported the river and 38% reported rain as their main water source. These environmental conditions are associated with low socioeconomic status and are more common among developing countries—fifty percent of patients within this group belong to the lowest income tier within Vietnamese society, with less than \$4.48 to live off of for 12 months. Along with low economic status, which is consistent with past studies, the average number of people per household in the study is 4.8 people per household, suggesting that the crowding effect may also contribute to the spreading of *H. pylori*.

There is a high frequency of doctor visits, up to 5-7 visits, among this group of *H. pylori* patients (35%). This may be due to the discomfort caused by *H. pylori* infection—the chronicity of *H. pylori* results in long term discomfort, reduction in productivity, and costly management and treatment plans. *H. pylori* infection thus puts a heavy burden on patients who have already have low socioeconomic status and low quality of life.

Furthermore, data obtained from the Vietnam Health Clinic suggests a link between *H. pylori* and gastroesophageal reflux disease (GERD) with 12 patients out of the 52 *H. pylori* patients having comorbid GERD. However, conflicting studies and several confounding factors lead researchers to believe that there is no association between the two.

Finally, 69% of VHC patients diagnosed with *H. pylori* were female, while only 31% were male. Our data conflicts with other studies suggesting there is no association between *H. pylori* and gender, no causal link can be drawn due to the limitation of study design. For this dataset, sampling bias must be taken into account due to the initial socioeconomic filter of patients prior to clinic visit. Gender inequality and lower socioeconomic status of females⁴⁰ may contribute to the higher presence of *H. pylori* female patients within our dataset.

The majority of the survey data from this research matches with the *H. pylori* prevalence studies in other demographics and ethnicities. Therefore, we suggest that *H. pylori* prevalence within Vietnamese community is more related to socioeconomic status, living conditions, and sanitation rather than ethnicity or race. Future research should focus more on elucidating the infectious and spreading mechanisms of *H. pylori*. Future studies can additionally take a look at the actual pathways in which extrinsic factors affect infection rates. In conclusion, the data analysis above does not support the initial hypothesis that susceptibility to *H. pylori* within Asian communities is due to intrinsic factors.

5.2 Limitations And Future Perspective

One limitation of this study is that the Vietnam Health Clinic was not able to follow-up with the patients treated in order to ensure sustainability of patient care. Vietnam Health Clinic 4.0 visited certain populations within a span of two weeks in 2014, but the group will not return until summer 2017. Due to these limitations, there were no reports on the percentage of treatment regimens successfully eradicating the bacteria. A lack of treatment efficacy data prevents this study from ascertaining whether the symptoms are of *H. pylori* pathology—through the clearance of symptoms after triple therapy treatment—or if *H. pylori* antibiotic resistance exists. Other sources of bias come from the survey questioning method, partial database corruption, and small dataset.

This research study is based on survey data and is thus retrospective and exploratory, not a controlled experimental-design research. Therefore, no causation conclusion can be draw from the data. However, the study can provide a mean to suggest future directions for other studies by providing insights on the *H. pylori* prevalence within the Asian community. The research will also help to inform better data collection practices for future VHC trips.

In future studies of *Helicobacter pylori* in the Vietnamese population, studies should focus on the strain of the bacteria involving resistance to certain antibiotics. During the VHC trip, only triple therapy was prescribed to the patients who were diagnosed with *H. pylori*. Because the clinic is not a specialized gastrointestinal clinic, but rather a mobile clinic, treatment options available to the patient were limited. Furthermore, the clinic did not have the resources to assess if the bacteria had antibiotic resistance. Thus, there was no way to assess if the treatment was the optimal one. VHC adhered treatment to the guidelines of the most commonly prescribed treatment. As discussed, if the bacteria strain showed any signs of resistance to the antibiotics used, the efficacy of triple therapy will drop substantially. Furthermore, if treatment is continued whilst resistance is disregarded, patients are put at higher risk to develop additional resistance. If VHC is able to assess the antibiotic resistance of the population they serve, they can use different varieties of regimens to more effectively treat patients.

5.3 Implications For Healthcare Providers And For Future Data Collection Process

During the patient health questioning and public health survey, nurses and volunteer students were trained to ask patients about their chief complaint and what was causing the highest level of discomfort for the patient—this is understandable due to the limited time and resources of the mobile clinic. However, the questioning method meant that many patients might not have had the chance to discuss their less pressing chronic complaints, such as symptoms of *H. pylori* infection. Besides data collection, many cases of *H. pylori* could have been under-reported as patients have more pressing chief complaints. There could also be missing diagnoses for *H. pylori* infection from physicians.

Instead of asking patients to voice their chief concern, nurses and student volunteers should allow patients to share all complaints during clinic. There is a delicate balance between the limited time and resources of crowded mobile clinic, the patient's well-being, and proper data collection method.

6. Conflict Of Interest

The authors have no conflict of interests to declare.

7. Acknowledgements

The author(s) wish to express their appreciation to Andrew Wilmington, Benjamin Blumberg, and Johnson Tran for technical and editing help. We would like to thank Scott Fung for his support. We acknowledge and grateful for the database contribution from the Vietnam Health Clinic non-profit organization.

8. References

- [1] Manuel R Ameiva, Emad M El-Omar. Host-bacteria Interaction in *Helicobacter pylori* Infection. *Gastroenterology*. 2008, Nov 09; 134:306-323
- [2] H. L. T. Mobley. The role of *Helicobacter pylori* urease in the pathogenesis of gastritis and peptic ulceration. *Alimentary Pharmacology and Therapeutics*. 1996; 10 (Suppl. 1); 57-64.
- [3] H. L. T. Mobley. Defining *Helicobacter pylori* as a Pathogen: Strain Heterogeneity and Virulence. *The American Journal of Medicine*. 1996 May 20; (Suppl 5A) Volume 100.
- [4] Jones KR, Whitmire JM, Merrell DS. A tale of two toxins: *Helicobacter pylori* CagA and VacA modulate host pathways that impact disease. *Front Microbiol*. 2010;1(NOV):1-17. doi:10.3389/fmicb.2010.00115.
- [5] Rhee KH, Park JS, Cho MJ. *Helicobacter pylori*: Bacterial strategy for incipient stage and persistent colonization in human gastric niches. *Yonsei Med J*. 2014;55(6):1453-1466. doi:10.3349/ymj.2014.55.6.1453.
- [6] Chey, W. D., Wong, B. C. Y., & Practice Parameters Committee of the American College of Gastroenterology. (2007). American College of Gastroenterology Guideline on the Management of *Helicobacter pylori* Infection. *The American Journal of Gastroenterology*, 102(8), 1808–1825. doi:10.1111/j.1572-0241.2007.01393.x
- [7] Gisbert, J. P., & Pajares, J. M. (2004). Stool antigen test for the diagnosis of *Helicobacter pylori* infection: a systematic review. *Helicobacter*, 9(4), 347–368.

doi:10.1111/j.1083-4389.2004.00235.x

- [8] Gatta, L., Vakil, N., Ricci, C., Osborn, J. F., Tampieri, A., Perna, F., ... Vaira, D. (2004). Effect of proton pump inhibitors and antacid therapy on ¹³C urea breath tests and stool test for *Helicobacter pylori* infection. *American Journal of Gastroenterology*, 99(5), 823–829. doi:10.1111/j.1572-0241.2004.30162.x
- [9] Vakil, N., Rhew, D., Soll, a, & Ofman, J. J. (2000). The cost-effectiveness of diagnostic testing strategies for *Helicobacter pylori*. *The American Journal of Gastroenterology*, 95(7), 1691–1698. doi:10.1111/j.1572-0241.2000.02193.x
- [10] Goodwin, C S, M M Mendall, and T C Northfield. "*Helicobacter Pylori* Infection." *Lancet* (London, England) 349, no. 9047 (1997): 265-9.
- [11] Zhu Y, Zhou X, Wu J, Su J, Zhang G. Risk factors and prevalence of *helicobacter pylori* infection in persistent high incidence area of gastric carcinoma in yangzhong city. *Gastroenterol Res Pract*. 2014;2014. doi:10.1155/2014/481365.
- [12] Mitchell AHM, Li YY, Hu PJ, et al. Epidemiology of *Helicobacter pylori* in Southern China : Identification of Early Childhood as the Critical Period for Acquisition Published by : Oxford University Press Stable URL : <http://www.jstor.org/stable/30112245> Accessed : 11-06-2016 04 : 00 UTC Epi. 2016;166(1):149-153.
- [13] Nguyen B V, Nguyen KG, Phung CD, et al. Prevalence of and factors associated with *Helicobacter pylori* infection in children in the north of Vietnam. *Am J Trop Med Hyg*. 2006;74(4):536-539. <http://www.ncbi.nlm.nih.gov/pubmed/16--606980>.
- [14] Perez-perez AGI, Taylor DN, Bodhidatta L, et al. Seroprevalence of *Helicobacter pylori* Infections in Thailand Published by : Oxford University Press Stable URL : <http://www.jstor.org/stable/30129587> Accessed : 11-06-2016 04 : 02 UTC Your use of the JSTOR archive indicates your acceptance of the Terms & . 2016;161(6):1237-1241.
- [15] Braga ABC, Fialho AMN, Rodrigues MN, Queiroz DMM, Rocha AMC, Braga L. *Helicobacter pylori* colonization among children up to 6 years: Results of a community-based study from Northeastern Brazil. *J Trop Pediatr*. 2007;53(6):393-397. doi:10.1093/tropej/fmm051.
- [16] Torres J, Leal-herrera Y, Perez-perez G, et al. A Community-Based Seroepidemiologic Study of *Helicobacter pylori* Infection in Mexico Muñoz Source : *The Journal of Infectious Diseases* , Vol . 178 , No . 4 (Oct ., 1998), pp . 1089-1094 Published by : Oxford University Press Stable URL : <http://www.jsto>. 2016;178(4):1089-1094.
- [17] Muhsen K, Jurban M, Goren S, Cohen D. Incidence, age of acquisition and risk factors of *Helicobacter pylori* infection among Israeli Arab infants. *J Trop Pediatr*. 2012;58(3):208-213. doi:10.1093/tropej/fmr068.
- [18] Miyaji H, Azuma T, Ito S, et al. *Helicobacter pylori* infection occurs via close contact infected individuals in early childhood. *J Gastroenterol Hepatol*. 2000;15(3):257-262. doi:10.1046/j.1440-1746.2000.02070.x.
- [19] Okuda M, Osaki T, Lin Y, et al. Low Prevalence and Incidence of *Helicobacter pylori* Infection in Children: A Population-Based Study in Japan. *Helicobacter*. 2015;20(2):133-138. doi:10.1111/hel.12184.
- [20] Webb, P. M., Knight T., Greaves S., Wilson A., Newell D. G., Elder J., and Forman D. "Relation Between Infection With *Helicobacter Pylori* And Living Conditions In Childhood: Evidence For Person To Person Transmission In Early Life." *BMJ: British Medical Journal* 308.6931 (1994): 750-53. Web.
- [21] Brown LM, Thomas TL, Ma J-L, et al. *Helicobacter pylori* infection in rural China: demographic, lifestyle and environmental factors. *Int J Epidemiol*. 2002;31(3):638-645. <http://www.ncbi.nlm.nih.gov/pubmed/12055167>.
- [22] Ueda, Mitsue, Shogo Kikuchi, Tatsuzo Kasugai, Tuuki Shunichi, and Chieko Miyake. "*Helicobacter Pylori* Risk Associated with Childhood Home Environment." *Cancer Science* 94, no. 10 (2003): 914-18.
- [23] Malaty HM, Engstrand L, Pedersen NL, Graham DY. *Helicobacter pylori* infection: Genetic and environmental influences. A study of twins. *Ann Intern Med*. 1994;120(12):982-986.
- [24] Morris, A., and G. Nicholson. "Ingestion of *Campylobacter Pyloridis* Causes Gastritis and Raised Fasting Gastric PH." *American Journal of Gastroenterology* 82, no. 3 (1987): 192-99.
- [25] Odze RD, Goldblum JR. Inflammatory disorders of the stomach. In: *Surgical Pathology of the GI Tract, Liver, Biliary Tract, and Pancreas*, Lash RH, Lauwers GY, et al. (Eds), Saunders, Philadelphia 2009. P.285.
- [26] Parsonnet, Vandersteen, Goates, Sibley, Pritikin, & Chang. (1991). HELICOBACTER-PYLORI INFECTION IN INTESTINAL-TYPE AND DIFFUSE-TYPE GASTRIC ADENOCARCINOMAS. *Journal Of The National Cancer Institute*, 83(9), 640-643.
- [27] The EUROGAST Study Group. An International Association Between *Helicobacter pylori* Infection and Gastric Cancer. *Lancet*. 1993;341(8857):1359-1363. doi:10.1016/0140-6736(93)90938-D.
- [28] Wotherspoon, A., & Falzon, M. (1991). *Helicobacter pylori*-Associated Gastritis and Primary B-Cell Gastric Lymphoma. *The Lancet*, 338(8776), 1175-6.

- [29] Li Z, Zou D, Ma X, et al. Epidemiology of peptic ulcer disease: endoscopic results of the systematic investigation of gastrointestinal disease in China. *Am J Gastroenterol*. 2010;105(12):2570-2577. doi:10.1038/ajg.2010.324.
- [30] Nomura A, Stemmerman GN, Chyou PH, Perez-Perez GI, Blaser MJ. *Helicobacter pylori* infection and the risk for duodenal and gastric ulceration. *Ann Intern Med*. 1994;12(12):977-981.
- [31] L. Fischback, E. L. Evans. Meta-analysis: the effect of antibiotic resistance status on the efficacy of triple & quadruple first-line therapies for *Helicobacter pylori*. *Alimentary Pharmacology and Therapeutics*. 2007, May 18. 10.1111/j.1365-2036.2007.03386.x
- [32] Gatta L, Vakil N, Leandro G, Di Mario F, Vaira D. Sequential therapy or triple therapy for *Helicobacter pylori* infection: systematic review and meta-analysis of randomized controlled trials in adults and children. *Am J Gastroenterol*. 2009;104(12):3069-3079; quiz 1080. doi:10.1038/ajg.2009.555.
- [33] Loren Laine, Richard Hunt, et al. Bismuth-Based Quadruple Therapy Using a Single Capsule of Bismuth Biscalcitrate, Metronidazole, and Tetracycline Given with Omeprazole Versus Omeprazole, Amoxicillin, and Clarithromycin for Eradication of *Helicobacter pylori* in Duodenal Ulcer Patients: A Prospective, Randomized, Multicenter, North American Trial. *The American Journal of Gastroenterology*. 2003; Vol. 98, No. 3.
- [34] Malfertheiner P, Bazzoli F, Delchier JC, et al. *Helicobacter pylori* eradication with a capsule containing bismuth subcitrate potassium, metronidazole, and tetracycline given with omeprazole versus clarithromycin-based triple therapy: A randomised, open-label, non-inferiority, phase 3 trial. *Lancet*. 2011;377(9769):905-913. doi:10.1016/S0140-6736(11)60020-2.
- [35] Tepes B, O'Connor A, Gisbert JP, O'Morain C. Treatment of *Helicobacter pylori* infection 2012. *Helicobacter*. 2012;17(SUPPL.1):36-42. doi:10.1111/j.1523-5378.2012.00981.x.
- [36] Deng-Chyang Wu, Ping-I Hsu, et al. Sequential and Concomitant Therapy with 4 drugs are Equally Effective for Eradication of *H. pylori* Infection. *Clinical Gastroenterol Hepatology*. 2010 Jan; 8(1): 36-41.
- [37] Vietnam Health Clinic. VHC 7.0 Newsletter. 2016
- [38] Vietnam Health Clinic. VHC Intake Form Final. 2013 – 2014
- [39] Scott Fung. Results from VHC 4.0 (2012 Trip) Public Health Survey & Clinic Data. Vietnam Health Clinic. June 28, 2013.
- [40] Helle Rydstrom. Gendered inequalities in Asia: configuring, contesting and recognizing women and men. Copenhagen: NIAS. 2010. In Print