Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/4qg17708

THE FREQUENCY OF *H. PYLORI* INFECTION AMONG DYSPEPTIC PATIENTS PRESENTING AT TERTIARY CARE HOSPITALS OF QUETTA

Safia Sana[1], Gulmina Kasi[1], Shama Noreen[1], Muhammad Essa Khan[1], Iqra Badar[2], Muhammad Waseem[3], Samreen Sana[4], Bahauddin[1], Abdul Qadir[1], Muhammad Kamran Taj[5*]

[1] Bolan Medical Complex Hospital, Quetta, Balochistan, Pakistan
[2]Peoples Medical Collage Hospital, Nawabshah Sindh, Pakistan
[3]M. Islam Medical and Dental Collage, Gujranwala Punjab, Pakistan
[4]Bolan Medical Collage, Quetta, Pakistan
[5*]CASVAB University of Balochistan, Quetta, Pakistan

***Corresponding Author**: Muhammad Kamran Taj,
*Email: kamrancasvab@yahoo.com

ABSTRACT

Background: *Helicobacter pylori* (*H. pylori*) infection is the most common cause of bacterial infection worldwide infecting more than 50% of the world's population predominantly in the developing countries. *H. pylori* is an important risk factor for gastritis, peptic ulcers, and gastric cancer likely due to the extensive inflammation in the stomach.
Objectives: To determine the frequency of *H. Pylori* infection among dyspeptic patients presenting at tertiary care hospital.
Study Design: Descriptive, cross-sectional study.
Study duration: 14th March 2023 to 13th February 2024.
Settings: Department of Gastroenterology, Bolan Medical Complex Hospital Quetta.
Methods: A study was conducted involving patients presenting with gastrointestinal symptoms. Participants were stratified into age groups (18–40 years and 41–60 years), gender (male and female), and by duration of symptoms (≤6 months and >6 months). The presence of *H. pylori* infection was assessed using diagnostic tests, and statistical analysis was performed to evaluate associations, with a p-value < 0.05 considered significant.
Results: In the 18–40 age group, 54 individuals tested positive and 15 negative, while in the 41–60 group, 47 were positive and 7 negative (p = 0.208), indicating no significant association with age. Among males, 52 tested positive and 17 negative while among females, 49 were positive and 5 negative. A statistically significant difference was found between genders (p = 0.027). Regarding symptom duration, 70 tested positive with symptoms ≤6 months, and 31 with symptoms >6 months (p = 0.918), showing no significant relationship.
Conclusion: The study found a statistically significant association between *H. pylori* infection and gender, but not with age or duration of symptoms. Gender-specific factors may influence infection prevalence and warrant further investigation.

Keywords: helicobacter, Pylori, Infection, Dyspepsia, Frequency.

INTRODUCTION

Helicobacter pylori (commonly referred to as *H. pylori*) is a spiral-shaped, flagellated bacterium that lives in the human stomach. It thrives in low-oxygen, highly acidic environments, making it well-suited to survive in the stomach lining. Globally, it is one of the most widespread bacterial infections, affecting nearly half of the world's population.[1, 2] The discovery of *H. pylori* in 1984 by Barry Marshall and Robin Warren marked a major breakthrough in medical science, fundamentally changing the understanding and treatment of various gastric diseases. Prior to their work, conditions like stomach ulcers were largely attributed to stress or diet, but it is now well-established that *H. pylori* plays a central role in the development of gastritis, peptic ulcers, and even stomach cancer.[3,4] Helicobacter pylori (H. pylori) infection is the most common cause of bacterial infection worldwide[4,5] infecting more than 50% of the world's population predominantly in the developing countries.[6] H. pylori is an important risk factor for gastritis, peptic ulcers, and gastric cancer likely due to the extensive inflammation in the stomach.[6,7] H. pylori has been associated with the development of two forms of cancer and has led to the World Health Organization (WHO) classifying it as the only bacterial Class-I carcinogen. Unless treated, colonization usually persists for life, indicating that H. pylori are well adapted to the gastric environment. Prevalence of H. pylori infection varies among countries and within different racial groups within the same country. The highest rates of infection are generally associated with low socio economic status, crowding, poor sanitation and unclean water supplies.[8] In the developed countries the prevalence of H. pylori is lower than 40% but in some developing countries more than 50% of children are infected by the age of 10 years with prevalence of infection rising to more than 80% in young adults.[9] In Ivory Coast, 55 % of children aged <10 years have been reported to be infected, while in northern Nigeria and Gambia, 50% of children under 5 years are infected. A recent study in Soweto found 46% of children at 1 year and 100% of children at 12 years to be infected with H. pylori.[10] The prevalence of H. pylori in dyspeptic patients has been reported to be 75%, 91.3% in Ivory Coast and 72-91% in Nigeria.[11] The diagnosis of H. pylori infection relies on the various testing methods which may be invasive or non-invasive. There are invasive techniques such as histological examination, bacterial cultures, rapid urease test, use of deoxyribonucleic acid probes, and polymerase chain reaction (PCR) analysis all of which require an endoscopy and a biopsy. Non-invasive techniques such as urea breath tests, immunoglobulin G and M serology, Stool antigen test, gastric juice PCR, and urinary excretion of Nl5 ammonia do not require endoscopy.[12,13] These various methods have been found to be sensitive and specific' but require skilled persons to perform them correctly. H. pylori urease test is fast, simple and does not need highly experienced laboratory staff to accurately identify H. pylori infection in the endoscopy unit within few hours of the procedure.[14] The aim of this study is to evaluate the current incidence of *H. pylori* infection in our local population and to assess how effective and feasible the HUT is as a routine diagnostic tool during endoscopy. In regions like Ghana and Balochistan, where socioeconomic conditions are gradually improving and the use of antibiotics and proton pump inhibitors (PPIs) is on the rise, it's possible that infection rates are not as high as they once were. By integrating the HUT test into standard endoscopic procedures, especially in under-resourced areas, healthcare providers can make faster and more cost-effective diagnoses. This could significantly reduce complications from long-term *H. pylori* infections, including ulcers and gastric cancer, ultimately improving patient outcomes and public health.

MATERIAL AND METHODS

Study Design: Cross-sectional study.
Setting: Department of Gastroenterology, Bolan Medical Complex Hospital Quetta.
Duration of Study: 14th March 2023 to 13th February 2024.
Sample Size: Total of 123 patients sample size calculated, using WHO formula for sample size calculation. 91.3% prevalence of H. pylori in dyspeptic,[11] 95% confidence level and 5% absolute precision.
Sample Technique: Non-probability, consecutive sampling.
Sample Selection

a. Inclusion Criteria:

1. All patients presenting with dyspepsia bloating, discomfort, nausea, and burping as per-operational definition.
2. The age at Patients more than 18 less than 60 years
3. Both gender (Male & Female) were included.

b. Exclusion Criteria:

1. Patients taking antibiotics in past 8 weeks.
2. Proton pump inhibitors in past 2 weeks or H2 blocker agents in past 1 week.
3. Taking immunosuppressive agents.
4. Active GI bleeding.
5. Pregnancy.
6. Breast feeding and history of gastrectomy.

Data Collection Procedure:

The study conducted at the Bolan Medical Complex Hospital Quetta. The study was approved by Ethical Committee of the BMCH, Quetta and all participating patients or their relatives provided written informed consent. Baseline patient's history obtained from all patients and diagnosis of gastric disorders made base on the presence of atrophic mucosa, oedematous mucosa, red spots or streaks and erosions suggesting inflammation and active or healing ulcers. Endoscopic diagnosis was made at the discretion of the endoscopist. Gastric antral mucosal biopsies of adequate size was then taken for H. pylori urease testing. In regions of atrophic gastritis, the biopsies were taken more proximally. Biopsy samples approximately 2-3 mm each was taken from the antralgastric mucosa and placed on the yellow color well containing urea and a pH indicator. The production of the urease enzyme by H. pylori results in the decomposition of urea into bicarbonate and ammonia which causes the pH to rise and the colour of the dot to change from yellow to red or pink. Positive results were read within 5 to 30 min. Samples that were weakly positive took up to 1 h to develop and no colour change at I h were regarded negative. All the above mentioned information was recorded in a predesigned proforma.

Data Analysis Procedure:

Data was entered and analyzed in computer software IBM-SPSS version 17. Qualitative variables like gender, H. pylori and endoscopic diagnosis were measured as frequency and percentages in both groups. Quantitative variables like age and duration of symptoms were measured as mean ± standard deviation. Effect modifiers like age, gender and duration of symptoms were controlled by the stratification. Post stratification Chi-square test was applied and P-value ≤0.05 was taken as significant.

RESULTS

The *H. pylori* infection screening was checked on 124 patients from tertiary care hospitals of Quetta city. According to result data 101 patients were positive for *H. pylori* infection while 23 patients were negative for *H. pylori* infection as shown in Figure-1.

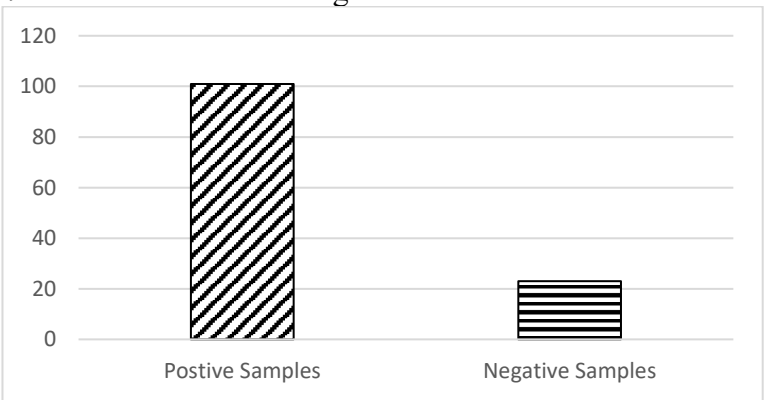

Figure-1: Overall prevalence of H. Pylori infection in Quetta City.

The H. pylori infection in two age groups (18–40 years and 41–60 years), were checked and data releveled that in the 18–40 **years** group, 54 individuals were infected, and 15 were not while In the 41–60 **years** group, 47 were infected, and 7 were not. Although the number of infections is slightly higher in the younger age group, the difference is not statistically meaningful**,** and age is not significantly associated with infection status as shown in Table-1.

Table-1: Stratification of *H. Pylori* infection with respect to age groups.

	H. Pylori infection		**p-value**
Age (years)	Yes	No	
18-40	54	15	**0.208**
41-60	47	08	
Total	101	23	

The distribution of *Helicobacter pylori* (*H. pylori*) infection among male and female participant's shows that 52 male were tested positive for *H. pylori*, and 17 male tested negative while 49 female**,** were tested positive, and only 5 tested negative as shown in Table-2.

Table-2: Stratification of H. Pylori infection with respect to gender.

	H. Pylori infection		**p-value**
Gender	Yes	No	
Male	52	17	**0.027**
Female	49	06	
Total	101	23	

This table shows how *H. pylori* infection is distributed among patients based on how long they have been experiencing symptoms (in months). Among patients with symptoms lasting 6 months or less, 70 were positive for *H. pylori*, and 15 were negative. Among those with symptoms for more than 6 months, 31 tested positive and 7 tested negative. Although a higher number of infections is seen in patients with symptoms lasting ≤6 months, the difference is likely due to the larger number of people in that group, not because duration affects infection risk as shown in Table-3.

Table-3: Stratification of *H. Pylori* infection with respect to duration of symptoms.

	H. Pylori infection		**p-value**
Duration (months)	Yes	No	
≤6 months	70	15	**0.918**
>6 months	31	08	
Total	101	23	

People from the Pathan and Baloch groups have the highest rates of H. pylori infection. The Hazara group has the lowest infection rate. The statistically significant p-value suggests that ethnic differences matter and may influence infection risk — possibly due to differences in lifestyle, hygiene, living conditions, or genetic factors as shown in Table-4.

Table-4: Ethnicity wise Distribution of H. Pylori infection.

	H. Pylori infection		**p-value**
Ethnic Groups	Yes	No	
Pathan	29	02	**0.029**
Baloch	27	04	
Punjabi	25	06	
Hazara	20	11	
Total	101	23	

Ethnicity and socioeconomic status are both strong predictors of H. pylori infection in Quetta city. The data also revealed that socioeconomic status plays a major role. Lower-class individuals in Quetta city were significantly more infected with *H. pylori* than those from the middle and higher classes as shown in Figure-2.

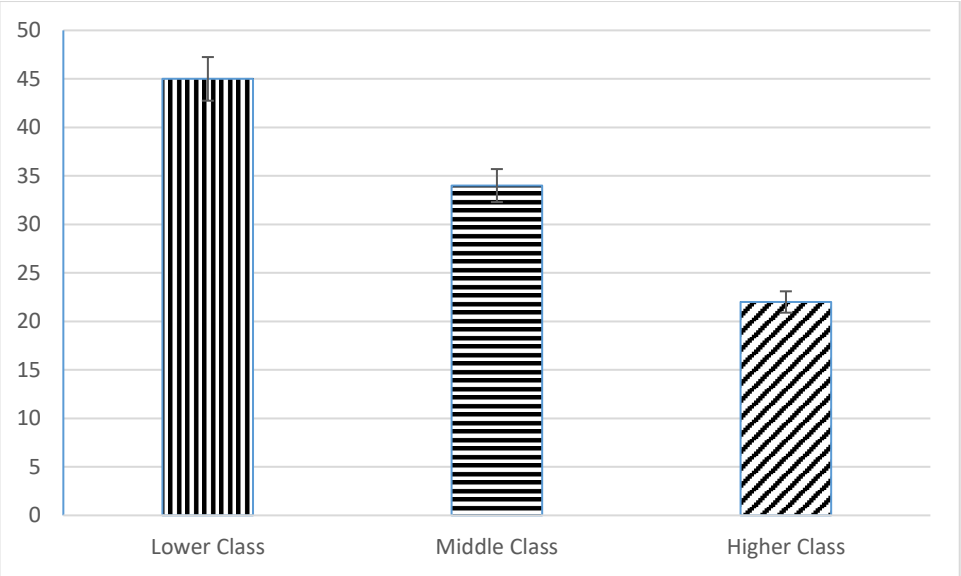

Figure-2: Socioeconomic Class wise Distribution of H. Pylori infection.

DISCUSSION

Since it was first isolated from gastric mucosa in 1990, *Helicobacter pylori* has been reported to be the cause of a variety of gastrointestinal (GI) diseases, including dyspeptic ulcers and gastric cancer. The vast majority of cases are the result of the ingestion of *H. pylori*-infected foods. Nonetheless, person-to-person transmission is currently considered to be the main route of contagiousness *H. pylori* infection and the proposed routes are mainly oral-oral and fecal-oral.[15] The prevalence of *H. pylori* infection is higher in developing countries than in developed countries, mostly due to a lack of safe drinking water and a lack of basic hygiene; this high prevalence may also be due to poor diet and overcrowded living conditions. Turkey reportedly has a high prevalence of *H. pylori* infection as well.[16] Although there may be geographical differences in prevalence, the majority of patients with *H. pylori* infection have been found to live under poor hygienic conditions, including poor urban infrastructure. It is generally accepted that this infection is mainly prevalent among lower social classes as well as in poor countries, likely due to the favorable circumstances for contagion between persons living under overcrowded and unhygienic conditions. Relatively socially deprived populations may also harbor *H. pylori* infection.[17]

I have conducted this study to determine the frequency of *H. pylori* infection among dyspeptic patients. Age range in this study was from 18 to 60 years with mean age of 41.53 ± 9.23 years. Majority of the patients 69 (56.10%) were between 18 to 40 years of age. Out of 123 patients, 69 (56.10%) were male and 54 (43.90%) were female with male to female ratio 1.3:1. In our study, frequency of *H. Pylori* infection among dyspeptic patients was found in 101 (82.11%) patients. In Ivory Coast, 55 % of children aged <10 years have been reported to be infected, while in northern Nigeria and Gambia, 50% of children under 5 years are infected. A recent study in Soweto found 46% of children at 1 year and 100% of children at 12 years to be infected with H. pylori.[10] The prevalence of *H. pylori* in dyspeptic patients has been reported to be 75%, 91.3% in Ivory Coast and 72-91% in Nigeria.[11]

Similarly, the prevalence we found in this study was lower than the previously reported 89% rate from Addis Ababa, Ethiopia,[18] 87% in Ugana,[19] 85% in Tanzania,[20] 83.5% in Nigeria,[21] 84.5% in Kenya,[22] and 85.4% in Ghana.[23] The difference in the prevalence might be due to diverse contributing factors including socioeconomic status, geographical or living conditions, as well as ethnicity or location of each population. However, the prevalence of H. pylori obtained in this study was higher

than the prevalence reported in Japan (34.9%),[24] Canada (23.1%),[25] and the US (9.4%).[26] The difference in prevalence could be due to variations in the socioeconomic status, the level of environmental sanitation, and difference in hygiene conditions. The other possible explanation for the higher prevalence of *H. pylori* infection found in our study may be due to the test method we used. In Tanzania, seroprevalence of *H. pylori* was 39.1% in dyspeptic patients.[27] Fluctuating trends in seroprevalence in dyspeptic patients were observed in Ethiopia.[28,] In Cameroon, the seroprevalence in symptomatic patients in hospital settings in the North West region was found to be 27.5%.[29] Higher prevalence rates have been reported in hospital-based studies where endoscopy and biopsy methods were used for diagnosis. A study conducted at the teaching hospital in Yaoundé, showed a *H. pylori* infection prevalence of 72.5% among symptomatic patients referred for upper gastrointestinal endoscopy.[30] Additionally, Ankouane and colleagues found prevalence rates of 71.2% among patients with atrophic gastritis, 75% among those with follicular gastritis, and 80% among those with intestinal metaplasia.[31] Among HIV patients presenting with gastro-intestinal symptoms, the prevalence of *H. pylori* infection was 50%.[32] A population-based study carried out in two health districts in Cameroon among asymptomatic children (0–10 years) reported a prevalence of 52.3% for stool H. pylori antigen, suggesting that infection in our population is acquired at a very early ages.[33] A seroprevalence of 62.4% in the Democratic Republic of Congo[118], 50.6% in the North of South Africa (Venda)[119], about 58% in Guatemala[34], 68% in Turkey[35], 61% in Saudi Arabia[36], 62% in Kuwait[37], 66.7 to 85% in some regions of Iran[38], and 61.7% in Brazil[49] was observed among the examined individuals. Also, an infection rate of 97 and 94.5% were reported in Ghana[40] and in Mozambique[41] respectively.

Several studies showed conflicting findings about the association of *H. pylori* infection and age of the patients. Studies conducted in the People's Republic of China[42] and Bhutan[43] reported that there is no statistically significant association between age of the patients and H. pylori infection. However, several other studies conducted elsewhere found a significant association between age of the patients and the prevalence of *H. pylori* infection.[44] Consistent with the earlier studies, this study found an increase in the prevalence of *H. pylori* infection as age of the patient's increases.

The role of sex as a risk factor for H. pylori infection is much argued. Some studies found no association between sex and H. pylori infection, [45,46] and some others reported female predominance. However, in this study, we found a higher prevalence of *H. pylori* infection in females compared to males. Similar to our current findings, many other studies found higher prevalence in females compared to males.[47, 48] Thus, this may explain why peptic ulcer diseases and gastric cancers, which are the diseases that have high association with H. pylori infection, occur -predominantly in female.

REFERENCES

1. Darko RYA, Osei V, Owusu-Ansah J, Aluze-Ele S. Changing partterns of the prevalence of Heleicobacter pylori among patients at a corporate hospital in Ghana. Ghana Med J. 2015;49(3):147–53.
2. Ayana SM, Swai B, Maro VP. Upper gastrointestinal endoscopic findings and prevalence of Helicobacter pylori infection among adult patients with dyspepsia in northern Tanzania. Tanzan J Health Res. 2014;16(1):16–22
3. Korkmaz H, Kesli R, Karabagli P, Terzi Y. Comparison of the diagnostic accuracy of five different stool antigen tests for the diagnosis of Helicobacter pylori infection. Helicobacter. 2013;18(5):384-91.
4. Thung I, Aramin H, Vavinskaya V, Gupta S, Park JY, Crowe SE. et al. The global emergence of Helicobacter pylori antibiotic resistance. AP&T. 2016 Feb;43(4):514-33.
5. Graham DY. Helicobacter pylori update: gastric cancer, reliable therapy, and possible benefits. Gastroenterology. 2015 Apr 1;148(4):719-31.
6. Torre LA, Bray F, Siegel RL, Ferlay J, Lortet' Tieulent J, Jemal A. Global cancer statistics, 201--2-.CA: a cancer jo;urnal for cliniciari:s:a.- 2015 Mar;65(2):87-108.
7. Sugano K, Tack J, Kuipers EJ, Graham DY, El-Omar EM, Miura S, et al. Kyoto global consensus report on Helicobacter pylori gastritis. Gut. 2015 Sep 1;64(9):1353-67.

8. Cover. TL. Helicobacter pylori diversity and gastric cancer risk. MBio. 2016 Mar 2,7(1).1869-15.
9. Li Y, Xia R, Zhang B, Li C. Chronic atrophic gastritis: view. JEP Toxi & Onco. 2018;37(3).
10. Archampong TN, Asmah RH, Wiredu EK, Gyasi RK, Nkrumah KN, Rajakumar K. Epidemiology of Helicobacter pylori infection in dyspeptic Ghanaian patients. Pan Afr Med J. 2015;20(1).
11. Malfertheiner P, Megraud F, O'morain CA, Gisbert JP, Kuipers EJ, Axon AT. Et al. Management of Helicobacter pylori infection-the Maastricht V/Florence consensus report. Gut. 2017 Jan 1;66(1):6-30.
12. Wang YK, Kuo FC, Liu CJ, Wu MC, Shih HY, Wang SS, et al. Diagnosis of Helicobacter pylori infection: Current options and developments.e.9015 Oct 28;21(40):11221.
13. Kalali B, Formichella L, Gerhard M. Diagnosis of Helicobacter pylori: Changes towards the future diseases. Disease. 2015;3(3):122-35.
14. Ferwana M, Abdulmajeed I, Alhajiahmed A, Madani W, Firwana B, Hasan R, et al. Accuracy of urea breath test in Helicobacter pylori infection: meta-analysis. World J Gastroenterol. 2015;21(4):1305.
15. Delport W. The transmission of Helicobacter pylori: The effects of analysis method and study population on inference. Best Pract Res Clin Gastroenterol. 2007;21(2):215–36.
16. World Gastroenterology Organisation. World Gastroenterology Organisation Global Guideline: Helicobacter pylori in developing countries. J Clin Gastroenterol. 2011;45(5):383–8.
17. Woodward M, Morrison C, McColl K. An investigation into factors associated with Helicobacter pylori infection. J Clin Epidemiol. 2000;53(2):175–81.
18. Desta K, Asrat D, Derbie F. Seroprevalence of Helicobacter pylori infection among health blood donors in Addis Ababa, Ethiopia. Can J Gastroenterol. 2007;21:501–506.
19. Newton R, Ziegler J, Casabonne D, et al; Uganda Kaposi's Sarcoma Study Group. Helicobacter pylori and cancer among adults in Uganda. Infect Agent Cancer. 2006;7(1):5.
20. Ayana SM, Swai B, Maro VP, Kibiki GS. Upper gastrointestinal endoscopic findings and prevalence of Helicobacter pylori infection among adult patients with dyspepsia in northern Tanzania. Tanzan J Health Res. 2014;16(1):16–22.
21. Abiodun C, Jesse A, Samuel O, et al. Prevalence of Helicobacter pylori among Nigerian patients with dyspepsia in Ibadan. Pan Afr Med J. 2011;6:18.
22. Ogutu EO, Kang'ethe SK, Nyabola L, Nyong'o A. Endoscopic findings and prevalence of Helicobacter pylori in Kenyan patients with dyspepsia. East Afr Med J. 1998;75(2):85–89.
23. Baako BN, Darko R. Incidence of Helicobacter pylori infection in Ghanaian patients with dyspeptic symptoms referred for upper gastrointestinal endoscopy. West Afr J Med. 1996;15(4):223–227.
24. Fujisawa T, Kumagai T, Akamatsu T, Kiyosawa K, Matsunaga Y. Changes in seroepidemiological pattern of Helicobacter pylori and hepatitis A virus over the last 20 years in Japan. Am J Gastroenterol. 1999;94(8):2094–2099.
25. Naja F, Kreiger N, Sullivan T. Helicobacter pylori infection in Ontario: prevalence and risk factors. Can J Gastroenterol. 2007;21(8):501–506.
26. Jackman RP, Schlinchting C, Carr W, Dubois A. Prevalence of Helicobacter pylori in United States navy submarine crews. Epidemiol Infect. 2006;134(3):460–464.
27. Jaka H, Mushi MF, Mirambo MM, Wilson L, Seni J, Mtebe M, et al. Sero-prevalence and associated factors of helicobacter pylori infection among adult patients with dyspepsia attending the gastroenterology unit in a tertiary hospital in Mwanza, Tanzania. Afr Health Sci. 2016;16:684–9.
28. Workineh M, Andargie D. A 5-year trend of helicobacter pylori seroprevalence among dyspeptic patients at Bahir Dar Felege Hiwot referral hospital, Northwest Ethiopia. Res Rep Trop Med. 2016;7:17–22.
29. Mathewos B, Moges B, Dagnew M. Seroprevalence and trend of helicobacter pylori infection in Gondar University hospital among dyspeptic patients, Gondar, north West Ethiopia. BMC Res

Notes. 2013;6:346.
30. Abongwa LE, Samje M, Sanda AK, Signang A, Elvis M, Bernadette L, et al. Knowledge, practice and prevalence of helicobacter pylori infection in the north west region of Cameroon. Clin Biotechnol Microbiol. 2017;1:135–43.
31. Andoulo FA, Noah DN, Tagni-Sartre M, Ndam EC Blackett KN. Epidémiologie de l'infection à Helicobacter pylori à Yaoundé: de la particularité à l'énigme Africaine. Pan Afr Med J. 2013;16:115.
32. Ankouane F, Noah DN, Enyime FN, Ndjollé CM, Djapa RN, Nonga BN, et al. Helicobacter pylori and precancerous conditions of the stomach: the frequency of infection in a cross-sectional study of 79 consecutive patients with chronic antral gastritis in Yaoundé, Cameroon. Pan Afr Med J. 2015;20:52.
33. Andoulo FA, Kowo M, Ngatcha G, Ndam AN, Awouoyiegnigni B, Sida MB, et al. Prevalence of helicobacter pylori prevalence and upper gastrointestinal endoscopy in HIV/AIDS patients with gastrointestinal symptoms in the university teaching hospitals in Cameroon. Med Sante Trop. 2016;26:278–82.
34. Ndip RN, Malange AE, Akoachere JFT, MacKay WG, Titanji VPK, Weaver LT. Helicobacter pylori antigens in the faeces of asymptomatic children in the Buea and Limbe health districts of Cameroon: a pilot study. Tropical Med Int Health. 2004;9:1036–40.
35. Longo-Mbenza B, Nsenga JN, Ngoma VD. Prevention of metabolic syndrome insulin resistance and atherosclerotic in diseases in Africans infected by Helicobacter pylori infection and treated with antibiotics. Inter J Cardiol. 2007;121:229–38.
36. Samie A, Obi CL, Barrett LJ, Powell SM, Guerrant RL. Prevalence of Campylobacter species, Helicobacter pylori and Arcobacter species in stool samples from the Venda region, Limpopo, South Africa: studies using molecular diagnostic methods. J Inf Secur. 2007;54:558–66.
37. Dowsett AS, Archila L, Segreto AV, Gonzalez RC, Silva A, Vastola AK, Bartizek DR, Kowolik JM. Helicobacter pylori infection in indigenous families of central America: Serostatus and oral and fingernail carriage. J Clin Microbiol. 1999;37(8):2456–60.
38. Seyda T, Derya C, Füsun A, Meliha K. The relationship of Helicobacter pylori positivity with age, sex, and ABO/rhesus blood groups in patients with gastrointestinal complaints in Turkey. Helicobacter. 2007;12:244–50.
39. Khan MA, Ghazi HO. Helicobacter pylori infection in asymptomatic subjects in Makkah, Saudi Arabia. J Pak Med Assoc. 2007;57:114–7.
40. Alazmi WM, Siddique I, Alateeqi N, Al-Nakib B. Prevalence of Helicobacter pylori infection among new outpatients with dyspepsia in Kuwait. BMC Gastroenterol. 2010;10:14.
41. Farshad SH, Japoni A, Alborzi A-V, Zarenezhad M, Ranjbar R. Changing prevalence of Helicobacter pylori in south of Iran Iranian. J. Clin Infect Dis. 2010;5:65–9.
42. Massarrat S, Saberi-Firoozi M, Soleimani A, Himmelmann GW, Hitzges M, Keshavarz H. Peptic ulcer disease, irritable bowel syndrome and constipation in two populations in Iran. Eur J Gastroenterol Hepatol. 1995;7:427–33.
43. de Mattos LC, Cintra JR, Sanches FE, et al. ABO, Lewis, secretor and non-secretor phenotypes in patients infected or uninfected by the Helicobacter pylori bacillus. Sao Paulo Med J. 2002;120:55–8.
44. Kidd M, Louw JA, Mark NI. Helicobacter pylori in Africa: observation on an 'enigma within an enigma. J Gastroenterol Hepatol. 1999;14:851–8.
45. Carrilho C, Modcoicar P, Cunha L, Ismail M, Guisseve A, Lorenzoni C, Fernandes F, Peleteiro B, Almeida R, Figueiredo C, David L, Lunet N. Prevalence of Helicobacter pylori infection, chronic gastritis, and intestinal metaplasia in Mozambican dyspeptic patients. Virchows Arch. 2009;54:153–60.
46. Zhang B, Hao GY, Gao F, et al. Lack of association of common polymorphisms in MUC1 gene with Helicobacter pylori infection and non-cardia gastric cancer risk in a Chinese population. Asian Pac J Cancer Prev. 2013;14(12):7355–7358.
47. Dorji D, Dendup T, Malaty HM. Epidemiology of Helicobacter pylori in Bhutan: the role of

environment and geographic location. Helicobacter. 2013;19(1):69–73.
48. Wizla-Derambure N, Michaud L, Ategbo S, et al. Familial and community environmental risk factors for Helicobacter pylori infection in children and adolescents. J Pediatr Gastroenterol Nutr. 2001;33(1):8–63.