

RESEARCH ARTICLE

Editorial Process: Submission:12/15/2017 Acceptance:06/27/2018

Clinical and Socio- Demographic Risk Factors for Acquisition of *Helicobacter pylori* Infection in Nigeria

Stella Smith^{1*}, Tolu Jolaiya², Muinah Fowora¹, Pia Palamides³, Favour Ngoka¹, Moses Bamidele¹, Olufunmilayo Lesi⁴, Charles Onyekwere⁵, Rose Ugiagbe⁶, Ifeanyi Agbo⁶, Dennis Ndububa⁷, Olusegun Adekanle⁷, Abimbola Adedeji⁸, Isaac Adeleye², Ute Harrison³

Abstract

Background: The aim of the study was to assess clinical and socio-demographic characteristics as well as prior drug usage as risk factors for *Helicobacter pylori* (*H. pylori*) infection in Nigeria. **Methods:** A total of 347 respondents were surveyed by assessing their clinical and socio-demographic characteristics in comparison with the non-invasive gold standard for *H. pylori* diagnosis, the urea breath test (UBT). Chi-square test and odds ratio analyses were conducted in order to assess if variables such as socio-demographic factors, drug intake, and history of ulcer/gastritis/gastric cancer within the family significantly predicted test results. **Results:** A total of 130 (37.5%) respondents were positive for *H. pylori* by the UBT. Living with more than three people in an apartment and a history of ulcer/gastritis within the family were significantly associated with *H. pylori* ($p \leq 0.05$), as well as current antibiotic intake ($p \leq 0.05$). Nationality, stay outside Nigeria, level of education, main occupation, smoking and drinking habits, sources of drinking water, number of children and history of gastric cancer had no significant association with *H. pylori* infection ($p \geq 0.05$). **Conclusion:** The results of the questionnaire revealed that most socio-demographic characteristics of the respondents had no significant association with *H. pylori*. Overcrowding, having siblings/parents with history of ulcer/gastritis as well as prior antibiotic usage had a significant association.

Keywords: *Helicobacter pylori*- risk- factors- acquisition- Nigeria

Asian Pac J Cancer Prev, **19** (7), 1851-1857

Introduction

Helicobacter pylori (*H. pylori*) is a Gram-negative, microaerophilic, highly motile fastidious bacterium which resides in the human gastric mucus layer. *H. pylori* is the causative agent of gastritis, peptic ulcer disease, mucosa associated lymphoid tissue (MALT) lymphoma and is a risk factor in the development of gastric cancer (Warren and Marshall, 1983).

In 1994, the International Agency for Research in Cancer (IARC) and arm of WHO identified *H. pylori* as a class I carcinogen (IARC, 1994). World-wide gastric cancer has been reported to be the fifth most common cancer and third leading causes of cancer related deaths (Torre et al., 2015).

H. pylori colonizes 50% of the world's population (Mitchell et al., 1992) and its prevalence in the human population ranges from 20% in developed

countries to as high as 80% in the developing countries (Frenck and Clemens, 2003). The high prevalence of *H. pylori* in developing countries as compared to developed countries is a result of increased risk of the infection in the former. Factors that predispose individuals to increased risk of *H. pylori* infection have been reported to include precarious hygiene standards, cultural background, dietary, deficient sanitation as well as crowded households (Ahmed et al., 2007).

Other risk factors include interaction with potentially contaminated environmental sources such as local drinking water, ingestion of faecal- contaminated vegetables and swimming in rivers (Ahmed et al., 2007). In Nigeria, all of these conditions abound with prevalence rates as high as 80% (Smith et al., 2001). *H. pylori* produces several virulence factors including cytotoxin associated gene (cagA) and vacuolating cytotoxin A (vacA), both of these are associated with marked increase in the risk of

¹Department of Molecular Biology and Biotechnology, ⁸Monitoring and Evaluation Unit, Nigerian Institute of Medical Research, Yaba, Lagos, ²Department of Microbiology, University of Lagos, Akoka, ⁴Lagos University Teaching Hospital, Idi-Araba, ⁵Lagos State University Teaching Hospital, Ikeja, ⁶University of Benin Teaching Hospital, Benin, ⁷Obafemi Awolowo University Teaching Hospitals Complex, Ile-Ife, Nigeria, ³Max von Pettenkofer-Institute, Munich, Germany. *For Correspondence: stellaismith@yahoo.com

disease development (Kusters et al., 2006).

In Nigeria, several reports have shown that irrespective of the disease outcome most strains produce CagA and VacA and reports have also shown that these virulence factors have also had their proteins expressed in a majority of cases irrespective of the disease outcome (Smith et al., 2002; Harrison et al., 2017; Seriki et al., 2017).

The triple therapy (combination of PPI with clarithromycin and amoxicillin or metronidazole) has been recommended for the first line treatment of *H. pylori* or a bismuth quadruple-based therapy for the treatment of *H. pylori* in areas of high (>15%) clarithromycin resistance (Malfertheiner et al., 2016).

In Nigeria, antibiotic resistance rates can get as high as 100%, which is a major reason for treatment failure (Smith et al., 2001; Aboderin et al., 2007; Harrison et al., 2017). In addition there are no standard treatments for *H. pylori* in Nigeria which adds to the burden of disease in the country.

To better understand the risk factors of *H. pylori* infection in Nigeria, the study was aimed to assess the clinical, socio-demographic characteristics, history of ulcer/gastritis/gastric cancer as well as prior drug usage as a risk factor for *H. pylori* infection amongst dyspeptic patients in Nigeria.

Materials and Methods

Study design

The study participants consisted of 347 adults that visited six tertiary hospitals in Nigeria with various dyspeptic cases with symptoms like upper abdominal pain, bloating, nausea or early satiety. All the study participants underwent upper endoscopy to enable appropriate disease diagnosis. Ethical approval for this study under a bigger study; was obtained by the Institutional Review Board of the Nigerian Institute of Medical Research (reg no IORG0002656) as well as Ludwig-Maximilians-University Munich (reg no 335-08). Informed consent was obtained from all study participants.

A total of 347 study participants were randomly selected for this study. Individuals were questioned concerning their socio-demographic characteristics such as stay outside the country for more than one month during the last five years, the highest education obtained, their habits, main occupation and sources of drinking water.

Other areas include drugs currently being taken, while a history of gastric cancer, ulcer or gastritis within the family was asked. This information was contained in a questionnaire.

A disease prevalence was determined based on test results from Urea breath test (UBT) method which is the gold standard for the non-invasive diagnosis of *H. pylori*.

Detection of *H. pylori* using the UBT test

H. pylori status was determined by the UBT, the gold standard for the non-invasive diagnosis of *H. pylori*. The UBT test conducted in this study was performed according to the method provided by Harrison et al., (2017).

Data analysis

Data analysis by Chi-square test and odds ratio were carried out to assess the relationship and measure the strength of association between related factors and outcome of diagnosis of *H. pylori*. Univariate analysis was carried out using frequencies to determine the number of subjects tested at the different groups of variable of interest. Also, bivariate analysis was conducted using Chi-Square test and Odds ratio with 95% Confidence Interval to assess relationship between two variables (associated risk factor and outcome of diagnosis of *H. pylori*) as well as measure the strength of association between the two variables tested. Statistical significance was set at p-value ≤ 0.05 .

Results

A total of 347 samples were analysed for the diagnosis of *H. pylori*. Out of these, 37.5% were positive for *H. pylori* according to the UBT. 151 (43.5%) males and 196 (56.5%) females took part in this study, with 38.4% and 36.7% positivity for *H. pylori* respectively. However, there was no statistical difference in *H. pylori* prevalence with respect to gender (Table 1). The median age of the study participants was 46 years. The age group with the highest prevalence (43%) of *H. pylori* was the 31 – 40 years. The majority of the subjects (208; 59.9%) had post-secondary education, while the remaining 139 (40.1%) had at most a secondary education or no formal education. The prevalence of *H. pylori* according to occupation amongst study participants was analysed. Those who worked with animals accounted for 50% prevalence followed by other occupation (38.7%), although there was no statistical significance in terms of occupation and *H. pylori* prevalence. The socio-demographic characteristics of the study participants are presented in the Table 1.

Prevalence of *H. pylori* was significantly lower among study participants who stayed outside the country for more than one month during the last 5 years (16.7%) compared to those who did not stay outside the country (39.4%) during the same period. Odds ratio (0.31) with 95% CI (0.115, 0.824) and p-value (0.014).

People who drank unprotected surface water were more likely to get infected with *H. pylori* compared to those who drank pipe borne/borehole water. However, this was not statistically significant.

Study participants who grew up with three people in one apartment/house showed a significantly higher prevalence (71.4%) compared to less than 34% prevalence among those who grew up with one or two persons (Fisher's exact test, p-value = 0.036; Table 2).

Study participants who had a parent (either mother or father) and siblings with a history of ulcer/gastritis had a 3.58 times higher risk of developing *H. pylori* infection compared to those whose children did not have a history of ulcer/gastritis, 95% CI (1.31, 9.78) (p-value = 0.009; Table 3).

Those who were currently on Omeprazole medication were 6.11 times less likely to develop *H. pylori* infection than those who were currently not taking Omeprazole, 95% CI (0.39, 0.95), (p-value = 0.029).

Table 1. Prevalence of *Helicobacter pylori* among Study Participants with Respect to Socio-demographic Variables, N = 347

Variable	Total subjects tested	<i>H. pylori</i>		p-value
		Positive No (%)	Negative No (%)	
Age				
< 20	12	2 (16.7)	10 (83.3)	0.413
21 - 30	33	11 (33.3)	22 (66.7)	
31 - 40	86	37 (43.0)	49 (57.0)	
41 - 50	73	29 (39.7)	44 (60.3)	
> 50	143	51 (35.7)	92 (64.3)	
Sex				
Male	151	58 (38.4)	93 (61.6)	0.74
Female	196	72 (36.7)	124 (63.3)	
Educational attainment				
No formal education	12	7 (58.3)	5 (41.7)	0.55
koranic education	1	0 (0.0)	1 (100)	
primary completed	45	15 (33.3)	30 (66.7)	
Secondary completed	81	31 (38.3)	50 (61.7)	
Post secondary completed	208	77 (37.0)	131 (63.0)	
Occupational				
Working with the sick	20	7 (35.0)	13 (65.0)	0.252
Working with children	31	7 (22.6)	24 (77.4)	
Working with animal	14	7 (50.0)	7 (50.0)	
Others (such as environmentalist/ occupation not specified)	282	109 (38.7)	173 (61.3)	
Occupational exposure to people with gastric disorders				
Yes	33	11 (33.3)	22 (66.7)	0.77
No	255	95 (37.3)	160 (62.7)	
Don't know	59	24 (40.7)	35 (59.3)	

Table 2. Prevalence of *H. pylori* According to Household. N = 347

Variable	Total subjects tested	<i>H. pylori</i>		P - Value
		Positive No (%)	Negative No (%)	
Number of children subjects have				
None	72	26 (36.1)	46 (63.9)	0.998
One	25	9 (36.0)	16 (64.0)	
Two	52	20 (38.5)	32 (61.5)	
Three	46	17 (37.0)	29 (63.0)	
More than three	153	58 (38.2)	94 (61.8)	
Number of people subjects grew up with (within one apartment/house)				
None	1	0 (0.0)	1 (100.0)	0.036
One	3	1 (33.3)	2 (66.7)	
Two	7	1 (14.3)	6 (85.7)	
Three	14	10 (71.4)	4 (28.6)	
More than three	322	118 (36.6)	204 (63.4)	
Number of people subjects currently live together with (within one apartment/house)				
None	16	8 (50.0)	8 (50.0)	0.353
One	35	17 (48.6)	18 (51.4)	
Two	30	11 (36.7)	19 (63.3)	
Three	35	15 (42.9)	20 (57.1)	
More than three	231	79 (34.2)	152 (65.8)	

Table 3. Prevalence of *H. pylori* in Relation to History of Ulcer/gastritis within the Family. N = 347

Variable	Total subjects tested	<i>H. pylori</i>		P-value	Odds ratio	95% CI
		Positive No (%)	Negative No (%)			
Father						
Yes	26	9(34.6)	17(65.4)	0.755	0.88	(0.378, 2.025)
No	321	121(37.7)	200(62.3)			
Mother						
Yes	42	16(38.1)	26(61.9)	0.923	1.03	(0.53, 2.004)
No	305	114(37.4)	191(62.6)			
Siblings						
Yes	48	12(25.0)	36(75.0)	0.055	0.51	(0.256, 1.023)
No	299	118(39.5)	181(60.5)			
Grandparents						
Yes	6	2(33.3)	4(66.7)	0.833	0.832	(0.15, 4.607)
No	341	128(37.5)	213(62.5)			
Children						
Yes	18	12(66.7)	6(33.3)	0.009	3.58	(1.308, 9.775)
No	329	118(35.9)	211(64.1)			

Also, those who were currently taking antibiotics were five times likely to develop *H. pylori* infection than those who were currently not taking antibiotics drugs 95% CI (0.29, 0.86) (p-value = 0.011, Table 4).

Similarly, those who were currently taking hypertension/diabetics drugs were 5.6 times less likely to develop *H. pylori* infection than those who were currently not taking hypertension/diabetics drugs, 95% CI (0.32, 0.99), (p-value = 0.046).

Table 4. Prevalence of *H. pylori* According to Those Regularly Taking Antibiotics for any Reason. N = 347

Variable	Total subjects tested	<i>H. pylori</i>		p-Value
		Positive No (%)	Negative No (%)	
Metronidazole				
Yes	20	7 (35.0)	13 (65.0)	0.815
No	327			
Amoxicillin				
Yes	28	10 (35.7)	18 (64.3)	0.842
No	319	18 (64.3)	199 (62.4)	
Clarithromycin				
Yes	6	0 (0)	6 (100)	0.088
No	341	130 (38.1)	211 (61.9)	
Tetracycline				
Yes	3	2 (66.7)	1 (33.3)	0.559
No	344	128 (37.2)	216 (62.8)	
Ciprofloxacin				
Yes	7	4 (57.1)	3 (42.9)	0.432
No	340	126 (37.1)	214 (62.9)	
Ampicillin				
Yes	2	1 (50.0)	1 (50.0)	1
No	345	129 (37.1)	216 (62.6)	
Augmentin				
Yes	2	1 (50)	1 (50)	1
No	345	129 (37.4)	216 (62.6)	

However, occupation, smoking/drinking habits, drugs taken regularly and so on, had no statistical significance on the prevalence of *H. pylori* infection using the UBT method.

Discussion

The prevalence of *H. pylori* in this study was 37.5% which is similar to a previous study in the same region by Harrison et al., (2017). Higher prevalence levels of 69% have also been reported in other African countries (Asrat et al., 2004; Tanih et al., 2011; Archampong et al., 2015). Environmental factors as well as socio-economic/cultural practices of the study participants could have accounted for this. However, different detection methods were used in the reported studies.

Several reports abound concerning the prevalence of *H. pylori* with respect to the age of participants. Majority of studies reported statistical significance with increasing prevalence of *H. pylori* with age (Ahmed et al., 2007; Alizadeh et al., 2009; Tanih et al., 2010; Ameri and Alkadasi 2013; Abebaw et al., 2014) while others reported increased prevalence with age but without any statistical difference (Dorji et al., 2013; Zhang et al., 2013). In present study the age group with the highest *H. pylori* prevalence was the 31 – 40 but there was no statistical significance. Increasing age did not affect *H. pylori* prevalence, in fact it was observed that those who were 51 and above were the third highest. Studies have also shown that people who are >60 years in the developing countries were more prone to severe disease conditions such as gastric cancer, peptic ulcer disease with significantly higher mortality (Pilotto et al., 2000; Pilotto and Salles, 2002; Pilotto and Franceschi, 2014), most likely as a result of lower socio-economic status, poor hygiene, crowding and lack of safe water while their counterparts in the developed countries are likely due to birth cohort effect of an earlier generation exposed to poor sanitation (Abebaw et al., 2014; Pilotto

and Franceschi, 2014). In present study most of the study participants showed a normal stomach flora with very few cases of ulcer and no case of gastric cancer which could account for the reduced prevalence of *H. pylori* in the higher age groups.

In present study, the prevalence of *H. pylori* was slightly higher in males than females (38.4% vs 36.7%) which was not statistically significant. Several studies have supported our findings of similar prevalence of *H. pylori* in males and females (Tanih et al., 2011; Dorji et al., 2013), while Abebaw et al., (2014) reported a slightly higher prevalence in females than males with Valliani et al., (2013) reporting a higher prevalence in males than females. Risk factors such as smoking and alcohol consumption had been attributed to the higher prevalence of *H. pylori* in males than females (Chen et al., 2000).

Socio-economic status has been reported to be a reliable marker to reflect the level of household hygiene as possible risk factors for *H. pylori* transmission (Ahmed et al., 2007). In our study, those with no formal education were found to have the highest prevalence of *H. pylori* (58.3%) although this was not statistically significant. The result of our study was corroborated with earlier studies by Torres et al., (1998) and Ahmed et al., (2007) but in contrast to that of Abebaw et al., (2014).

Large family size has been accepted as a risk factor in *H. pylori* transmission (McCallion et al., 1996; Herbath et al., 2010). In our study, participants who grew up with more than three people in one apartment had a significantly higher prevalence (71.4%) of *H. pylori* compared to those less than three (34%). Our result was corroborated by Ahmed et al., (2007); Dorji et al., (2013), but contrasted with Abebaw et al., (2014).

Residing in a developing country has been associated as one of the risk factors of *H. pylori* infection (Salih, 2009), our study supported the findings of Salih (2009) as there was a significant difference amongst the participants who stayed outside the country (16.7%) for more than one month in the last five years compared to those that stayed in the country (37.5%).

Smoking and alcohol consumption have been reported as risk factors for *H. pylori* infection (Ogihara et al., 2000). However our study showed that both parameters were not statistically significant although there was a slightly higher prevalence with alcohol consumption. Our result is in consonance with Ogihara et al., (2000); Mukhopadhyay (2007) and in contrast to those of Abebaw et al., (2014) and Shamseya et al., (2015).

Likewise from our study, sources of drinking water had no significant association with *H. pylori* infection although drinking water from unprotected surface waters had a higher *H. pylori* prevalence than piped water (45.5% vs 35.56%). Other studies (Ahmed et al., 2007; Abebaw et al., 2014) corroborated our study but strongly posited a link between surface waters and *H. pylori* transmission. Lindkivist et al., (1998) was however of a contrary opinion.

Our study shows a 3.58 times higher risk of *H. pylori* acquisition amongst study participants who had parent/siblings with history of ulcer/gastritis. In Nigeria, most mothers pre-masticate food, blow food to cool, taste food with infant spoon before giving to their babies

(Anigo et al., 2009; Ogunshe et al., 2013; Samuel, 2013) and this could be one of the strongest route of *H. pylori* acquisition. Our study was corroborated by Goodman et al., (1996); Malaty et al., (2000); Salih (2009), where their report buttressed the key role mothers had in the strong transmission of *H. pylori* infection.

Furthermore, the study participants that were on Omeprazole were 6.11 times less likely to develop *H. pylori* infection. An explanation for this could be that first Omeprazole is one of the drugs used for the treatment of *H. pylori* and second other factors such as nutrition might be involved. A report by Langtry and Wilde, (1998) showed the effective use of Omeprazole as a monotherapy for GORD or NSAID induced gastrointestinal damage.

Similarly, the use of hypertension and diabetes drugs reduced the chances of *H. pylori* acquisition amongst our study participants. The report by Nagata et al., (2015) corroborated our study, although the study by Migneco et al., (2003) was to the contrary. Another study suggested treating patients with Type 2 Diabetes Mellitus (T2DM) with *H. pylori* drugs as targets of intervention in high-risk communities (He et al., 2014).

Surprisingly, our study showed that participants who were currently on antibiotics were five times more likely to develop *H. pylori* infection. One reason that could account for this is the indiscriminate use of antibiotics in Nigeria (Harrison et al., 2017). In Nigeria across -the- counter purchase of drugs are a common phenomenon and this is in addition to the fact that the dosages are usually not completed resulting in treatment failure due to drug resistances.

We can therefore conclude from our study that risk factors associated with *H. pylori* acquisition in Nigeria are overcrowding, parents/siblings with history of gastritis/ulcer and antibiotic usage. Sources of drinking water, education attainment, occupation, smoking/drinking habits were not significantly associated with *H. pylori* acquisition. It is therefore pertinent to monitor the antibiotic usage in the populace as well as conduct educational and enlightenment programs to educate people about the risk of overcrowding as well as behavioural practices such as blowing of food to cool and pre-mastication of foods from mothers to babies in order to further reduce the risk of *H. pylori* infection in Nigeria.

Acknowledgments

The Authors wish to acknowledge funding from the DFG (HA 2697/18-1) awarded to Prof. Rainer Haas and SIS to Dr. S. Smith.

References

- Abebaw W, Kibret M, Abera B (2014). Prevalence and risk factors of *H. pylori* from dyspeptic patients in NorthWest Ethiopia: A hospital based cross-sectional study. *Asian Pac J Cancer Prev*, **15**, 4459-63.
- Aboderin OA, Abdu AR, Odetoyin B, et al (2007). Antibiotic resistance of *Helicobacter pylori* from patients in Ile-Ife, South-west Nigeria. *Afr Health Sci*, **7**, 143 – 7.

- Ahmed KS, Khan AA, Ahmed I, et al (2007). Impact of Household hygiene and water source on the prevalence and transmission of *Helicobacter pylori*: a South Indian perspective. *Singapore Med J*, **48**, 543 – 9.
- Ameri GA, Alkadasi MN (2013). The prevalence of *Helicobacter pylori* and risk factors of infection associated in Taiz city, Yemen. *Int J Curr Microbiol Appl Sci*, **2**, 226 - 13
- Anigo KM, Ameh DA, Ibrahim S, Solomon SD (2009). Infant feeding practices and nutritional status of Children in North Western Nigeria. *Asian J Clin Nutr*, **1**, 12 – 22.
- Alizadeh AHM, Ansari S, Ranjba M, et al (2009). Seroprevalence of *Helicobacter pylori* in Nahavand: a population based study. *East Mediterr Health J*, **15**, 129 – 5.
- Archampong TNA, Asmah RH, Wiredu EK, et al (2015). Epidemiology of *Helicobacter pylori* infection in dyspeptic Ghanaian patients. *Pan Afr Med J*, **20**, 178.
- Asrat D, Nilsson I, Mengistu Y, et al (2004). Prevalence of *Helicobacter pylori* infection among adult dyspeptic patients in Ethiopia. *Trop Med Parasitol*, **98**, 219 – 1.
- Chen T, Chang F, Lee S (2000). Smoking and male gender rather than CagA protein are associated with increased risk of duodenal ulcer in *Helicobacter pylori*-infected patients in Taiwan. *J Dig Dis Sci*, **44**, 2076 – 80.
- Dorji D, Dendup T, Malaty HM et al (2013). Epidemiology of *Helicobacter pylori* in Bhutan: the role of environment and geographic location. *Helicobacter*, **19**, 69 – 73.
- Frenck RW, Clemens J (2003). Helicobacter in the developing world. *Microb Infect*, **5**, 705-13.
- Goodman KJ, Correa P, Tengana Aux HJ, et al (1996). *Helicobacter pylori* infection in the Columbian Andes: a population-based study of transmission pathways. *Am J Epidemiol*, **144**, 290 – 9.
- Harrison U, Muinah AF, Abiodun TS, et al (2017). *Helicobacter pylori* infection in Nigeria is associated with low prevalence and divergent antibiotic resistance patterns. *PLoS One*, **12**, e0176454.
- He C, Yang Z, Lu N (2014). *Helicobacter pylori* infection and diabetes: Is it a myth or fact?. *World J Gastroenterol*, **20**, 4607 – 17.
- Herbath O, Krumbiegel P, Fritz GJ, et al (2010). *Helicobacter pylori* prevalence and risk factors among school beginners in a German urban centre and its rural country. *Environ Health Perspect*, **109**, 573 – 7.
- International Agency for Research on Cancer (1994). IARC Monographs on the evaluation of carcinogenic risks to humans volume 61. Schistosomes, Liver flukes and *Helicobacter pylori*. Lyon, France: IARC.
- Kusters KG, van Vliet AH, Kuipers EJ (2006). Pathogenesis of *Helicobacter pylori* infection. *Clin Microbiol Rev*, **19**, 449 – 90.
- Langtry HD, Wilde MI (1998). Omeprazole. A review of its use in *Helicobacter pylori* infection, gastro-oesophageal reflux disease and peptic ulcers induced by nonsteroidal anti-inflammatory drugs. *Drugs*, **56**, 447 – 86.
- Lindkvist P, Enquesselassie F, Asrat D, et al (1998). Risk factors for infection with *Helicobacter pylori*: a study of children in rural Ethiopia. *Scand J Infect Dis*, **30**, 371 – 6.
- Malaty HM, Graham DY, Isaksson I, Engstrabd L, Pedersen NL (2000). Are genetic and environmental components of peptic ulcer disease: a study of twins reared apart and twins reared together?. *Arch Intern Med*, **160**, 105 – 9.
- Malfertheiner P, Megraud F, O'Morain CA, et al (2016). On behalf of the European *Helicobacter* and microbiota study group and consensus panel management of *Helicobacter pylori* infection-the Maastricht V/Florence consensus report. *Gut*, **0**, 1–25.
- McCallion WA, Murray LJ, Bailie AG, et al (1996). *Helicobacter pylori* infection in children: relation with current household living conditions. *Gut*, **39**, 18 -21.
- Migneco A, Ojetti V, Franceschi F, et al (2003). Eradication of *Helicobacter pylori* infection improves blood pressure values in patients affected by hypertension. *Helicobacter*, **8**, 585 – 9.
- Mitchell HM, Li YY, Hu PJ, et al (1992). Epidemiology of *Helicobacter pylori* in southern China: identification of early childhood as the critical period for acquisition. *J Infect Dis*, **166**, 149 – 53.
- Mukhopadhyay S (2007). Smoking habits amongst Slum Dweller and the impact on health among the population of West Bengal. *J Gastroenterol Hepatol*, **10**, 633-8.
- Nagata N, Niikura R, Sekine K, et al (2015). Risk of peptic ulcer bleeding associated with *Helicobacter pylori* infection, nonsteroidal anti-inflammatory drugs, low-dose aspirin, and antihypertensive drugs: a case control study. *J Gastroenterol Hepatol*, **30**, 292 – 8.
- Ogihara A, Kikuchi S, Hasegawa A, et al (2000). Relationship between *Helicobacter pylori* infection and smoking and drinking habits. *J Gastroenterol Hepatol*, **15**, 271 – 6.
- Ogunshe AO, Doris AL, Oghenekaro JD, Olayinka PV (2013). Parental perceptions and microbial / public health implications of pre-chewed weaning foods. *Food Public Health*, **3**, 315-22.
- Pilotto A, Franceschi M, Costa MC, Di Mario F, Valerio G (2000). *Helicobacter pylori* test-and-eradication strategy. *Lancet*, **356**, 1683-4.
- Pilotto A, Salles N (2002). *Helicobacter pylori* infection in geriatrics. *Helicobacter*, **7**, 56-62.
- Pilotto A, Franceschi M (2014). *Helicobacter pylori* infection in older people. *World J Gastroenterol*, **20**, 6364 – 73.
- Salih BA (2009). *Helicobacter pylori* infection in developing countries: the burden for how long?. *Saudi J Gastroenterol*, **15**, 201 -7.
- Samuel OO (2013). Infant mortality in Nigeria: Assessing knowledge of predisposing risk factors among mothers and bacteriological profile of the weaning foods. *Am J Food Nutr*, **13**, 22-6.
- Seriki AT, Smith SI, Adeleye AI, et al (2017). *Helicobacter pylori* cytotoxin-associated gene A protein among adult dyspeptic patients in South-West Nigeria. *Afr J Microbiol Res*, **11**, 681-6.
- Shamseya AM, Shamseya MM, Salem MA, Ahmed AS, Abdelfatah DA (2015). Assessment of some health-related practices and knowledge among a group of Egyptian patients with peptic ulcer disease. *J Med Sci Clin Res*, **3**, 8186 - 92.
- Smith SI, Oyediji KS, Arigbabu AO, Atimomo C, Coker AO (2001). High amoxicillin resistance in *Helicobacter pylori* isolated from gastritis and peptic ulcer patients in western Nigeria. *J Gastroenterol*, **36**, 67-8.
- Stella IS, Christian K, Kola S, et al (2002). Prevalence of *Helicobacter pylori* vacA, cagA and iceA genotypes in Nigerian patients with duodenal ulcer disease. *J Med Microbiol*, **51**, 851 –4.
- Tanih NF, Ndip LM, Clarke AM, et al (2010). An overview of pathogenesis and epidemiology of *Helicobacter pylori* infection. *Afr J Microbiol Res*, **4**, 426 – 6.
- Tanih NF, Okeley BI, Ndip LM, et al (2011). *Helicobacter pylori* prevalence in dyspeptic patients in the Eastern Cape province and disease status. *South Afr Med J*, **100**, 734 – 7.
- Torres J, Leal-Herrera Y, Perez-Perez G, et al (1998). A community-based seroepidemiologic study of *Helicobacter pylori* infection in Mexico. *J Infect Dis*, **178**, 1089 – 94.
- Torre LA, Bray F, Siegel RL, et al (2015). Global cancer statistics, 2012. *CA Cancer J Clin*, **65**, 87–108.
- Valliani A, Khan F, Chagani B, et al (2013). Factors associated

- with *Helicobacter pylori* infection: results from a developing country-Pakistan. *Asian Pac J Cancer Prev*, **14**, 53 – 6.
- Warren JR, Marshall B (1983). Unidentified curved bacilli on gastric epithelium in active chronic gastritis. *Lancet*, **1**, 1273 – 5.
- Zhang B, Hao GY, Gao F, et al (2013). Lack of association of common polymorphisms in MUC 1 gene with *Helicobacter pylori* infection and non-cardia gastric cancer risk in a Chinese population. *Asian Pac J cancer Prev*, **14**, 7355 – 8.



This work is licensed under a Creative Commons Attribution-Non Commercial 4.0 International License.