

Risk Factors Associated with *Helicobacter pylori* Infection in Gaza, Palestine

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Abstract: *Helicobacter pylori* (*H. pylori*) infection is usually acquired in early childhood. *H. pylori* infection is associated with several upper gastrointestinal disorders. Local data on the epidemiology of the infection are scarce in Palestine. The purpose of this study is to measure the occurrence of infection and to explore the associated factors among the population living in Gaza strip. This study included 89 randomly selected participants from non-hospitalized patients. Age, sex, socioeconomic status and other potential risk factors were assessed using a structured interview. Ultra Rapid Urease Test was performed on biopsy specimens followed by histology examined with Methylene blue stain, HpSAG test to detect antigen in stool specimen and *Hp* IgM antibody was measured in blood using ELISA technique. Age ranged between 13-77 years, with mean age 37.03, (37.1%) were females and (62.9%) were males. The rate of *H. pylori* infection was (48.3%). There were variations between the different tests. There was a significant correlation between the type of drinking water consumed during childhood and *H. pylori* infection. *H. pylori* infection showed no significant correlation with age, sex, weight, marital status, smoking, education level, coffee drinking, oral hygiene, socioeconomic status including number of persons living in the accommodation, number of persons in each room, income, type of accommodation, consumption of drugs and antibiotics. Tea drinking proved to be a protective factor against *H. pylori* infection.

Keywords: *H. pylori*, URUT, HpSAG, ELISA, Biopsy specimen, Gaza, risk factors

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عوامل الخطر المصاحبة للعدوى بجراثيم الملوية البوابية في غزة، فلسطين

ملخص: العدوى ببكتيريا الملوية البوابية عادة ما تكتسب في الطفولة المبكرة، و يصاحب العدوى مجموعة من الأعراض في الجزء العلوي من الجهاز الهضمي. لا يوجد معلومات وبائية عن هذه البكتيريا والأمراض التي قد تصاحبها في فلسطين. الغرض من هذه الدراسة تحديد نسبة الإصابة و عوامل الخطر التي قد تساعد الإصابة بهذه البكتيريا. في هذه الدراسة تم اختيار 89 مريض بشكل عشوائي من عدة مستشفيات في قطاع غزة. جميع المرضى أجابوا عن مجموعة من الأسئلة: العمر، نمط الحياة، التاريخ المرضي بالإضافة لعدة أسئلة تشمل الوضع الاجتماعي الاقتصادي. تم أخذ 3 عينات من كل مريض عينة دم لفحص الأجسام المضادة IgM، عينة براز لفحص HpSAg و خزعة من المعدة للقيام بفحص اليوريا الفائق السرعة (URUT) وفحص نسيجي باستخدام صبغة أزرق الميثيلين. تراوحت أعمار المرضى بين 13-77 سنة. نسبة الإناث كانت (37.1%) و الذكور (62.9%)، نسبة انتشار الملوية البوابية كانت (48.3%)، هناك اختلاف بين نتائج الفحوصات الأربع. من أهم النتائج في هذه الدراسة وجود علاقة مهمة بين الإصابة بهذه البكتيريا ومصادر شرب الماء في مرحلة الطفولة. لم يكن هناك أي علاقة ذات دلالة إحصائية بين الإصابة بالبكتيريا والعمر، الجنس، الوضع الاقتصادي للمريض، عدد أفراد الأسرة، أنواع الأدوية التي يستخدمها، التدخين و شرب القهوة. أيضا من النتائج في هذه الدراسة وجد أن شرب الشاي يشكل عامل يحمي من الإصابة بالملوية البوابية.

Introduction

H. pylori, a spiral gram negative bacterium, colonizes the human stomach, can cause type B gastritis, is strongly associated with gastric and duodenal ulceration and has been implicated in the causation of gastric carcinoma and mucosa-associated lymphoid tissue (MALT) lymphomas. It has been reported that there is relationship between *H. pylori* infection and children's gastroenterological diseases [1].

H. pylori infection is one of the commonest infections worldwide, occurring in all regions and infecting at least half of the world's population [2]. The prevalence of *H. pylori* infection worldwide is approximately 50%, as high as 80%–90% in developing countries, and about 35%–40% in the United States [3]. While within countries, the prevalence is higher among groups with lower socioeconomic status [4]. *H. pylori* prevalence is generally found to increase with age, reaching 20-50% in adult populations in Europe and North America [5]. The prevalence of *H. pylori* infection was 10% among children in Egypt [6].

H. pylori prevalence is associated with increasing age, non-white skin color, lower family income, lower education level, higher size of the family, low socio-economic conditions in childhood, higher number of siblings and

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attendance to day-care centers in childhood, and presence of dyspeptic symptoms. Socioeconomic conditions in childhood besides ethnicity and presence of dyspeptic symptoms were the factors significantly associated with the infection [7].

There are several methods for diagnosing *H. pylori* infection including invasive procedures using mucosa biopsies taken during endoscopy (mainly culture, histology and the rapid urease test) and noninvasive procedures [8]. The success of treatment is usually dependent on early detection. Moreover, prevention of *H. pylori* infection seems to be a wise strategy. Prevention strategies require deep understanding of the transmission risks. Thus the aim of this study is to determine possible risk factors for *H. pylori* infection.

Patients and Methods

Patients

An eligible patient with *H. pylori* infection was defined as the patient independently assessed by attending physician based on clinical symptoms [9]. No age limitation was imposed in this study.

Sample size

One hundred twenty two randomly selected eligible patients were subjected to endoscopy for exploration and gastric or duodenal biopsy collection by their attending physicians. In addition, blood and stool sample were collected. All patients were interviewed. Verbal consent was obtained from all patients.

Sample collection

Gastric biopsies collection was performed by the attending specialist. Ultra rapid urease test and methylene blue staining tests were performed on biopsies. About 5 ml of venous blood was collected for *H. pylori* IgM serum determination and about 20 grams of stool were collected into a sterile container.

***H. pylori* IgM,**

Purified *H. pylori* antigen (DRG, Germany) coated on the surface of microwells. 1:40 dilution of patient's serum was added to the wells, and the *H. pylori* IgM- specific antibody, if present, binds to the antigen. Enzyme conjugate was added, which binds to the antibody-antigen complex. The enzyme conjugate catalytic reaction is stopped at a specific time. Reading of color absorbance and result interpretation was done according to the manufacturer recommendations.

Ultra Rapid Urease Test (URUT)

Each biopsy tissue was placed immediately into capped Eppendorf tube containing 0.5 ml of freshly prepared solution of 10% urea in deionized water, to which had been added two drops of 1% phenol red as pH indicator. A positive result was indicated by change in the color of the solution from yellow to pink within one minute [10].

Methylene Blue staining of biopsies

After reading the result of the ultra rapid urease test (URUT) the biopsy was removed from the urea solution and imprint smears were made by lightly rolling it on a clean glass slide, using a hypodermic needle. The imprint smear was air dried and fixed in absolute methanol. Imprint smears were stained by loeffler's methylene blue stain. Slides were read using the oil immersion objective to search for the curved bacilli [11].

HpSAg test

All reagents of the kit (DRG, Germany) were brought to room temperature and stool sample was extracted as recommended by the manufacturer. Calibrators were included in each run. Following the addition of the extracted stool and enzymatic conjugate, the microtiter plates were incubated for 120 minutes at 37 °C. After washing, chromogen/substrate were added into all wells and incubated for 20 minutes. Stop reagent was added and the color intensity was measured using ELISA reader at 450 nm and 620-630nm filter.

Questionnaire

The questionnaire was designed to obtain demographic data such as age, sex, occupation, educational level, and place of birth. Socioeconomic status was also assessed.

Analysis of data

Data generated from the study was tabulated as Microsoft Excel sheets and uploaded to Statistical Package for Social Sciences (SPSS version 11). Cross tabulations of variables were generated. Chi square was used to detect statistically significant differences among variables.

Results

Study Sample Description

This study was conducted during the period from September 2006 to March 2007. During the study period, 122 patients undergoing upper gastrointestinal endoscopy were interviewed and a serum, biopsy and stool specimens were collected. Among them only 89 patients provided the three specimen types. Those patients are non-hospitalized patients from different hospitals across Gaza strip. Approximately (10%) from patients were from

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Al-shifa hospital, (45%) from Balsam-hospital, (30%) from the European-hospital and (15%) from the Military-hospital. Table (1) is a concise description the study sample.

Table (1): Personal factors effect on *H. pylori* infection

Variable		<i>H. pylori</i> infection				P value
		Negative		Positive		
		No.	%	No.	%	
Age	13-20y	4	66.7	2	33.3	0.832
	21-35y	23	48.9	24	51.1	
	36-50y	11	50.0	11	50.0	
	>51y	8	57.1	6	42.9	
Sex	Male	31	54.4	26	45.6	0.323
	Female	15	46.9	17	53.1	
Weight	35-55	1	12.5	7	87.5	0.125
	56-76	23	57.5	17	42.5	
	77-97	20	52.6	18	47.4	
	98-118	2	66.7	1	33.3	
Marital status	Married	38	50.7	37	49.3	0.440
	Single	8	57.1	6	42.9	

H. pylori infection

There were variations in the percentage of positive results in the four employed tests. A true positive was assumed if URUT and/or Methylene blue tests were positive. According to this criterion only 48.3% were considered positive. All the subsequent correlations between possible risk factors and *H. pylori* infection were done with the true positive.

Risk factors For *H. pylori* Infection.

Personal factors

The study population age ranged between 13-77 years, with mean age 37.03. 37.1% are females and 62.9% are males. Among the 89 subjects who completed data, the highest positive result was found in the age group 21-35yr (51.1%) while the highest negative result was in age group 13-20yr (66.7%). The highest positive result was in female and it constituted 53.1%, while in male it was 45.6%.

The highest positivity result was found in weight group 35-55kg it was 87.5% but the lowest results were in weight group 98-118kg. With regard to marital status, there were no significant differences. Both married and single subjects were approximately equally effected (Table1).

Life style variables

As shown in Table (2), there is no significant difference between smokers and non smokers with regard to *H. pylori* infection. Even the number of cigarettes seems not to affect or increase the possibility of infection. There is a statistically significant difference among subjects who drinks tea and those who do not with a *P* value = 0.045.

Subjects who drink coffee are less likely to develop *H. pylori* infection (41.0%) as compared to those who don't drink coffee (54.0%). However, the difference is not statistically significant. With regard to the number of cups per day, lower percentage of *H. pylori* infection was observed among those who consume more than 5 cups per day.

From statistical analysis of data, the type of water consumed during childhood could be considered as a risk factor with *P* value=0.018. As shown in Table (2), the positive results were high in subjects who consumed municipality or well water during childhood (53.2%) while subjects who consumed filtered (purified) water during childhood have 16.7% positive results. However, the drinking water sources during adulthood did not influence the outcome of *H. pylori* infection.

Oral hygiene status of the subjects was assessed by asking the patient if he/she had a dental complains. *H. pylori* infection was high in subjects with dental complains 64.3%, but this result was not statistically significant.

Contact with animals and travelling abroad were also evaluated as possible risk factors. However, these factors did not show any significant effect on *H. pylori* infection.

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Table (2): Life style variables and *H. pylori* infection

Variable		<i>H. pylori</i> infection				P value
		Negative		Positive		
		No.	%	No.	%	
Smoking	No	29	50.9	28	49.1	0.507
	Yes	17	53.1	15	46.9	
No. of cigarettes/day	1-20	13	48.1	14	51.9	0.208
	>20	4	80.0	1	20.0	
Drink tea	No	1	14.3	6	85.7	0.045
	Yes	45	54.9	37	45.1	
How many cups/day	1-5	36	54.5	30	45.5	0.564
	>5	9	56.3	7	43.8	
Drinking coffee	No	23	46.0	27	54.0	0.158
	Yes	23	59.0	16	41.0	
How many cups per day	1-5	20	55.6	16	44.4	0.194
	>5	3	100	-	-	
Type of drinking water (childhood)	Municipality or well	36	46.8	41	53.2	0.018
	Filtered water	10	83.3	2	16.7	
Type of drinking water (adulthood)	Municipality or well	8	50.0	8	50.0	0.548
	Filtered water	37	50.7	34	46.5	
Dental complains	Yes	5	35.7	9	64.3	0.156
	No	41	54.7	34	45.3	
Consumed drugs	No.	28	49.1	29	50.9	0.336
	Yes	18	56.3	14	43.8	
Consumed antibiotics during previous month	No	23	46.9	26	53.1	0.218
	Yes	23	57.5	17	42.5	
Contact with animals	No	36	52.2	33	47.8	0.532
	Yes	10	50.0	10	50.0	
Traveling abroad	No	36	50.0	36	50.0	0.351
	Yes	10	58.8	7	41.2	

Drugs history

As shown in Table (3), the history of medication with aspirin, other anti-inflammatory drugs and gastric medication (PPI, H₂ antagonist) was evaluated and the results showed no significant differences.

Table (3): Medication intake in relation to *H. pylori* infection

Under Medication	<i>H. pylori</i> infection				Total		P value
	Negative		Positive				
	No.	%	No.	%	No.	%	
No	28	49.1	29	50.9	57	100.0	0.336
Yes	18	56.3	14	43.8	32	100.0	
Total	46	51.7	43	48.3	89	100.0	

Antibiotics intake during the last month

As shown in Table (4) the history of antibiotics intake in the last month was evaluated and the results showed no significant results.

Table (4): Antibiotics intake in relation to *H. pylori* infection

Do you take antibiotics	<i>H. pylori</i> infection				Total		P value
	Negative		Positive				
	No.	%	No.	%	No.	%	
No	23	46.9	26	53.7	49	100.0	0.218
Yes	23	57.5	17	42.5	40	100.0	
Total	46	51.7	43	48.3	89	100.0	

Socioeconomic status

As shown in Table (5), none of the socioeconomic variables showed significant effect on *H. pylori* infection.

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Table (5): Effects of socioeconomic status on *H. pylori* infection

Variable		<i>H. pylori</i> infection				P value
		Negative		Positive		
		No.	%	No.	%	
Education	University	12	63.2	7	36.8	0.311
	High school	19	52.8	17	47.2	
	Intermediate	6	33.3	12	66.7	
	Uneducated	9	56.3	7	43.8	
Monthly income (NIS)	Below 1000	5	45.5	6	54.5	0.370
	1000-2000	22	46.8	25	53.2	
	2000-3000	12	54.5	10	45.5	
	>3000	7	77.8	2	22.2	
Garbage collection system	No	8	44.4	10	55.6	0.336
	Yes	38	53.5	33	46.5	
Type of accommodation	Flat	23	51.1	22	48.9	0.134
	House	19	47.5	21	52.5	
	Villa	4	100	-	-	
Number of rooms in the accommodation	>5	1	50.0	1	50.0	0.768
	3-5	39	53.4	34	46.6	
	2	6	42.9	8	57.1	
Number of persons in the accommodation	>10	13	76.5	4	23.5	0.073
	5-10	27	46.6	31	53.4	
	1-4	6	42.9	8	57.1	
Number of persons in each room	>5	3	100.0	0	-	0.227
	2-4	43	50.7	33	49.3	
	1	9	47.4	10	52.6	
Type of water supply	Municipality	41	51.9	38	48.1	0.586
	Well	5	50.0	5	50.0	
Sewage system	Poor	12	48.0	13	52.0	0.421
	Good	34	53.1	30	46.9	

P value <0.05 significant

Discussion

There is no significant result pointing to the increase in infection with age. This may be due to the limited number of old age participants. Our result was similar to a study in Brazil (2005) which showed that the prevalence of infection did not increase significantly ($P=0.147$) with age. There were no significant differences in the prevalence of *H. pylori* infection, when

patients were classified by age. This has been explained as being due to a reduction in the specific serological response among older individuals and/or to a decreased number of microorganisms as a consequence of gastric atrophy [12].

The current study showed that there is no significant difference in the overall prevalence of *H. pylori* infection between males and female. And both of them appear to be equally exposed. This result is in agreement with other studies. In Brazil, two hundred and four individuals participated in the study, 49 males and 155 females, with ages ranging from 18 to 80 years. Overall, 165 of 204 participants (80%) were *H. pylori* positive, with no significant gender differences ($P= 0.49$) [12].

The highest positive result was in weight group 35-55 Kg (87.5%) and this percentage did not increase with increasing weight. It is possible that *H. pylori* infection may have some detrimental effect on growth, especially during the pubertal growth spurt [13].

A slightly higher percentage of *H. pylori* infection was observed among married than single subjects. But the increase was not statistically significant. This increase may be attributed to age and personal contact. A Libyan study showed that higher prevalence of *H. pylori* in married subjected (84%) compared to single subjects (68%) [14].

Smoking is considered as a risk factor for many diseases and is implicated by several studies in the literature as a risk factor for *H. pylori* infection. However, the results of this study showed no statistical differences between smokers and non-smokers (15 (46.9%), 28 (49.1%) respectively). Brenner and colleagues documented the effects of lifestyle on *Helicobacter pylori* infection in 447 patients in a German rural area. They found that the *H. pylori* infection rate appeared to non-significantly increased by smoking. In the past, when idiopathic gastric hyperacidity was considered to be the chief cause of dyspeptic symptoms, smoking, was often implicated as exacerbating the condition and advice given to eliminate this habit [15].

There was no statistically significant difference between those who consume or don't consume coffee. In fact a little bit lower percentage of positive *H. pylori* was found in those who consume coffee (41.0%) than those who did not (54.0%). A survey in Germany in 1997 on 447 patients with an overall prevalence of 21%., coffee consumption showed a positive dose-response relation with active infection. The positive relation between coffee

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consumption and *H. pylori* infection identified in that study is consistent with results from a cohort study among epidemiologists in which the risk of seroconversion (change from negative to positive results for antibodies to *H. pylori* in serum) was 4.6 times higher among those who drank more than 2 cups of caffeinated drinks a day than among the others. The mechanism underlying this association, however, is not clear [16].

A significant finding of this study is that (as shown in table 4.13) tea consumption is a protective factor. Only 45.1% of those who consume tea were infected, a very much higher percentage was found among those who do not drink tea (85.7%). This finding is supported by a Japanese study (1999) on the benefits of tea. Recent studies have presented data that show a variety of biological activities of tea catechins, compounds which constitute about 15% (dry weight) of tea against *H. pylori* [17].

Type of drinking water during childhood proved to be a detrimental factor with a statistically significant result (P value=0.018). *H. pylori* infection rate was higher in patients who drank well water during childhood (53.2%) as compared to patients who used bottled water during childhood (16.7%). This result is in agreement with other studies in developed and developing countries. They implicated the type of drinking water during childhood as the main risk factor for *H. pylori* infection. The microorganism is transmitted by the fecal-oral route in the infected water to the child and persists through life and as the results showed that the type of drinking water during adulthood does not affect the infection rate.

In a study in Leipzig, Germany (2004), which consisted of a self-administered or parent-completed questionnaire (age-dependent), eliciting information on lifestyle habits and their use/drinking the well water as well as the *H. pylori* infection. A total of 91 subjects (44 users of *H. pylori* positively and 47 negatively tested wells) were screened for their *H. pylori* status. The group was comprised of 42 males and 49 females, i.e., 73 adults and 19 children under the age of 18 (mean age 39.5 years with a range between 3-80 years). Logistic regression analyses identified the drinking of well water as the significant risk factor for a positive colonization status [Odds Ratio (OR)=8.3; 95% confidence interval (95% CI) 2.4-29.0; $P<0.001$]. Water supplies have been identified as possible reservoirs to acquire the bacterium [18].

Higher percentage of *H. pylori* infection was observed among those with poor oral hygiene (64.3%) than those with good hygiene (45.3%). The

difference is not statistically significant. In a study in Australia by Hedley (1997) on 217 adults randomly selected from the electoral roll, they found that the positive *H. pylori* status was significantly associated with increasing number of tooth surfaces with a high plaque score (odds ratio, 1.7; 95% confidence interval, 1.1-2.7) [19].

Among the study subjects, 40 took antibiotics one day to one month prior sample collection, 57.5% of them were shown to be negative while among the 49 who did not consume antibiotics, only 46.9% of them were negative for *H. pylori*. This reduction could be explained by the fact that both URUT and Methylene blue depend on the activity and the presence of *H. pylori* in the biopsy and antibiotic consumption may reduce the number and activity of the organism, therefore interfering with the result outcome producing false negative results. Among the study subjects, 32 took drugs, 56.3% of them were shown to be negative while among the 57 who did not consume drugs, only 49.1% of them were negative for *H. pylori* (not statistically significant).

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Proton pump inhibitors are known to decrease the activity of *H. pylori* organisms within the stomach and to shift their distribution proximally. This effect may reduce the sensitivity of histological examination and rapid urease testing for *H. pylori* on biopsies taken from recommended sites. It is of particular relevance if a proton pump inhibitor has been prescribed before the patient has undergone diagnostic endoscopy [20].

In our study the prevalence of *H. pylori* infection was not associated with Socio economic status including education, income, number of persons in the house and type of accommodations.

It can be concluded that drinking and/or using *H. pylori* contaminated water is a risk for the acquisition of *H. pylori*. Given the public health impact of *H. pylori* infection, this should be taken into account when measures of prevention are considered. We recommend that further investigations should be conducted to detect the possible sources in Gaza strip of *H. pylori* infection, especially drinking water.

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References

1. **Marshall B.,** - *Helicobacter pylori* physiology and genetics. One hundred years of discovery and rediscovery of *Helicobacter pylori* and its association with peptic ulcer disease. Mobley H., Mendz G., and Hazell S.,(eds):American Society for Microbiology.: 19-24 (2001).
2. **Parsonnet J.,** *Helicobacter pylori: the size of the problem.* Gut. 43 (1):6–9 (1998).
3. **Lacy B., semore J.,** *Helicobacter pylori: ulcers and more: the beginning of an era.* Journal of Nutrition. 131(27) :89-93 (2001).
4. **Murray L., McCrum E., Evans A. ,Bamford K.,** *Epidemiology of Helicobacter pylori infection among 4742 randomly selected subjects from Northern Ireland.* International Journal of Epidemiology. 26(1) :880–887 (1997).
5. **Bergenzaun P., Kristinsson KG., Thjodleifsson B., Sigvaldadottir E., Mölstað S., et al.,** *Seroprevalence of Helicobacter pylori in south Sweden and Iceland.* Scandinavian Journal of Gastroenterology. 31(12):1157–1161 (1996).
6. **Chun D., Shun N., Shi H., Jia Y.,** *Seroepidemiology of Helicobacter pylori infection among asymptomatic Chinese children.* World Journal of Gastroenterology. 6(5) :759-761 (2000).
7. **Blaser M., Atherton J.,** *Helicobacter pylori persistence: Biology and disease.* The Journal of Clinical Investigation. 113(3) : 321-333 (2004).
8. **Suerbaum S., Michetti P.,** *Helicobacter pylori infection.* The New England Journal of Medicine. 347(15): 1175-1186 (2002).
9. **Bergey B., Marchildon P., Peacock J., Mégraud F.,** *What is the role of serology in assessing Helicobacter pylori eradication?.* Alimentary Pharmacology & Therapeutics. 18 (6) :635–639 (2003).
10. **Ogata S., Kawakami E., Reis F.,** *Evaluation of invasive method to diagnosis Helicobacter pylori infection in children and adolescents with dyspepsia invasive method to diagnose Hp infectio .* Faculdade de Medicina de Ribeirão Preto, 35(1) : 24-29 (2002).
11. **Misra S., Misra V., Dwivedi M., Singh P., Bhargava V., et al.,** *Evaluation of the one minute ultra rapid urease test diagnosing helicobacter pylori .* Postgraduate Medical Journal 75 (881):154-156 (1999).
12. **Rodrigues M., Queiroz D., Rodrigues R., Rocha A., Braga Neto M., et al.,** *Helicobacter pylori infection in adults from a poor urban community in northeastern Brazil: demographic, lifestyle and*

- environmental factors*. The Brazilian Journal of Infectious Diseases . 9(5):405-410 (2005).
13. **Sood M., Joshi S., Akobeng A., Mitchell J., Thomas G.,** *Growth in children with Helicobacter pylori infection and dyspepsia*. Archives of Disease. 90(10):1025-1028 (2005).
 14. **Bakka A., Salih B.,** *Prevalence of Helicobacter pylori infection in asymptomatic subjects in Libya*. Diagnostic Microbiology and Infectious Disease. 43(4):265-268 (2002).
 15. **Jenkins D.,** *Helicobacter pylori and its interaction with risk factors for chronic disease*. British Medical Journal. 315(7121):1481-1482 (1997).
 16. **Brenner H., Rothenbacher D., Bode G., Adler G.,** - *Relation of smoking and alcohol and coffee consumption to active Helicobacter pylori infection: cross sectional study*. British Medical Journal. 315(7121):1489-1492 (1997).
 17. **Mabe K., Yamada M., Oguni I., Takahashi T.,** - *In Vitro and In Vivo Activities of Tea Catechins against Helicobacter pylori*. Antimicrobial Agents and Chemotherapy. 43(7):1788-1791 (1999).
 18. **Rolle-Kampczyk U., Fritz G., Diez U., Lehmann I., Richter M., et al,** *Contaminated well water: a risk factor for Helicobacter pylori infection* International Journal of Hygiene and Environmental Health. 207(4):363-368 (2004).
 19. **Peach H., Pearce D., Farish S.,** *Helicobacter pylori infection in an Australian regional city: prevalence and risk factors*. Medical Journal of Australia. 167(6):310-313 (1997).
 20. **Dickey W, Kenny B., McConnell J.,** *Effect of proton pump inhibitors on the detection of Helicobacter pylori in gastric biopsies*. Alimentary Pharmacology & Therapeutics, 10(3):289-293 (1996).