



Prevalence of *Helicobacter pylori* in Pregnancy related to birth weight and micronutrient deficiency

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ABSTRACT

Infection with the bacterium *Helicobacter pylori* is common, especially in underdeveloped regions. hyperemesis gravidarum, foetal neural tube abnormalities, and other pregnancy-related complications. Miscarriage and stunted foetal development that occurs within the uterus. In this study, we aimed to collect evidence of how *H. pylori* infection affects Low-income mothers' risk of having babies with low birth weight, nutritional deficiencies, and anaemia on unfavourable results during pregnancy. Of critical importance, there are Problems with our current knowledge of how *H. pylori* infection and stomach problems are connected caused by an insufficiency of essential nutrients during pregnancy.

Methods: In Iraq, researchers used a prospective cohort design to examine participants' health histories between July 2020 and July 2021. The Individuals included both first-time and repeat mothers who tested negative for hepatitis due to either a positive or negative *H. pylori* result on a hepatitis screen. Participants were recruited at 18 weeks' gestation, and they were evaluated again at 13 and 18 weeks. period of time between the weeks before labour and the days after it. Stool *H. pylori* antigen testing was used to confirm the presence of *H. pylori* infection. Maternal Body mass index (BMI) and total weight increase during pregnancy were determined by taking measurements of height and weight. The study included only full-term, healthy newborns. Those with a birth weight of less than 2,500 grammes were considered to have a low birth weight (LBW).

Results: The average age of the 110 registered individuals was 20.9 2.7 years (SD = 4.9 years). Births occurred at a mean SD gestational age of 39.4 1.0 weeks. There were 61.5% (n = 94) primates and 52.9% One hundred and eighty-eight people tested positive for *H. pylori* infection. Weight-for-height ratio (WHR) was (14.6%) were short (n = 14), whereas 38.2% were tall (n = 36). As a percentage of newborns, Low birth weight (2500 g) was seen in 13.6% (n = 13) of the study population. Having a mother who tested positive for *H. pylori* was a significant predictor of LBW (odds ratio [OR], 3.60, 95% CI, 2.70–5.10). 3.4, 95% CI 1.4-8.2; P = 0.008) and maternal height at recruitment 156 cm (OR 1.1, 95% CI 1.1-11.5; P = 0.031) and maternal height at recruitment 156 cm (OR 3.4, 95% pregnant women who gain less than 0.3 kg per week during the second and third trimesters (OR 3.8, 95% CI 1.0-14.1; P = 0.044).

Conclusion *H. pylori* infection is linked to LBW in both first-time and repeat mothers, we conclude. Infection with *H. pylori* has been linked to risk of IDA rises, although not always in tandem with other nutritional. During pregnancy, vitamins are essential.

Keywords:

H. pylori infection, pregnancy, birth weight, and gestational weight increase are some of the key concepts.

Introduction

Helicobacter pylori (*H. pylori*) infection affects approximately one half of the world population and it is more prevalent in developing countries[1,2]. This microorganism colonizes the stomach. Typically, it is acquired during childhood and causes asymptomatic chronic infection[2]. A small portion of *H. pylori* infected subjects develop peptic ulcers and gastric carcinoma, usually during late adulthood[2]. *H. pylori* pathogenicity depends on several strain-specific factors. Some *H. pylori* strains express specific genes conferring pro-inflammatory, cytotoxic and vacuolating properties which could enhance the *in vivo* pathogenicity[3]. Virulence factors such as urease and flagella are present in all strains and they are pivotal for pathogenesis and colonization[4]. Adhesins, such as Outer inflammatory protein and Sialic acid-binding adhesin, facilitate bacterial attachment to the host epithelium and often induce its inflammatory response[5,6]. *H. pylori*-strains can also express Cytotoxin-associated antigen A (CagA) and Vacuolating cytotoxin A (VacA), the most investigated cytotoxins among *H. pylori* virulence factors. CagA is directly injected into the cytoplasm of epithelial cells, affecting cell morphology, proliferation and apoptosis[7]. *H. pylori* strains carrying CagA have been associated with both duodenal ulcer and gastric cancer[8], and infection with CagA-positive strain is generally associated to higher levels of inflammatory mediators compared to CagA negative strains[3]. VacA is a proteic pore-forming toxin crucial to promote and maintain bacterial colonization[9]. It disrupts cell polarity, promotes epithelial cells apoptosis and inhibits T cell proliferation and effector function[10]. Interestingly, combined seropositivity for both CagA and VacA directly correlates with elevated morbidity[11-13]. During the past decades, several reports indicated a correlation between *H. pylori* infection and various extragastric disorders[14]. Such manifestations include ischemic heart disease, diabetes mellitus, idiopathic thrombocytopenia, urticaria, and

sideropenic anemia[14]. Lanciers *et al*[15] (1999) found a significantly increased incidence of pregnant subjects with high *H. pylori* IgM (marker for recently acquired infection) compared to non pregnant women. These Authors suggested that pregnancy itself may increase the susceptibility to *H. pylori* infection[15].

This is probably due to the fact that there are immunologic adaptations in pregnancy to ensure maternal tolerance towards the semi-allogeneic fetus. In general, pregnancy is characterized by a decreased cell-mediated cytotoxic immune response with preservation of humoral and innate immunity[16]. Nowadays, no follow-up study was conducted to describe

the complete immune response against *H. pylori* infection during pregnancy. Most studies on the correlation between *H. pylori* infection and pregnancy-related disorders were cross-sectional investigations where *H. pylori* positivity was detected during pregnancy or soon after delivery. *H. pylori* infection was tested before conception only in one prospective study, where early pregnancy loss was associated with maternal *H. pylori* CagA-strains seropositivity before intracytoplasmic sperm injection[17]. Indeed, it is not possible to definitely conclude whether pregnancy-related complications are correlated to *H. pylori* infection acquired before or during pregnancy. The prevalence of *H. pylori* infection in pregnant women varies according to geographic area, socioeconomic conditions and method used to detect *H. pylori* infection. For example, the prevalence of *H. pylori* infection among pregnant women is about 20%-30% in most European countries[18-20], Japan[21] and Australia[22], while it is 50%-70% in Turkey[23,24], Mexico and in Texas, United States[25,26], more than 80% in Egypt[27] and Gambia[28]. Furthermore, inadequate sanitation practices, low social class and crowded or high-density living conditions seem to be related to a higher prevalence of *H. pylori* infection. These observations suggest that poor

hygiene and crowded conditions may facilitate transmission of infection among family

members and they are consistent with data on intra-familial and institutional

Gastrointestinal Disorders In Pregnancy

Mild to moderate dyspepsia is commonly associated with nausea and vomiting and complicates about 50% of all pregnancies and it diminishes women's life quality and social functions during early pregnancy. In most women, these symptoms resolve by fluid and vitamin supplementation as well as dietary modification. About 0.3%-2% of pregnant women suffer from Hyperemesis Gravidarum (HG) characterized by severe and protracted vomiting that often results in dehydration, electrolyte imbalance, ketonemia, ketonuria, and weight loss. Dehydration and acid base disturbances may lead to renal and hepatic injury. Patients who manifest continuous weight loss and electrolyte disturbances may be at

risk for growth restriction, fetal anomalies and decreased neonatal birth weight. The onset of gastrointestinal symptoms is always during the first trimester, but HG may persist throughout gestation. The etiology of HG, which still remains unknown, seems to be multifactorial and may be the final result of various unrelated conditions. Indeed, treatment is performed on a symptomatic basis. In particular, psychological causes, gastrointestinal tract dysfunctions, endocrine factors (*i.e.*, elevated human chorionic gonadotropin and estrogen), genetic incompatibility, immunological factors and nutritional deficiencies have been considered part of the pathologic mechanism underlying HG. However, no single theory seems to provide an adequate explanation for HG.

Significant positive association between HG and *H. pylori* infection has been demonstrated by several case-control studies and in a systematic review of 14 case-control studies, Golberg *et al* (2007) found higher prevalence of HG in *H. pylori*-infected pregnant women than uninfected ones (pooled OR = 4.45; 95%CI: 2.31-8.54). In contrast, most of the studies aimed to determine the link between *H.*

pylori and dyspepsia failed to show a significant correlation between the clinical symptoms of the disease and *H. pylori* infection. Only

two studies investigated the relationship between CagA-positive *H. pylori* strains and gastrointestinal problems in pregnancy. Noyan *et al* (2004) found a significant association between CagA-seropositivity and dyspepsia in pregnancy, though *H. pylori*-seroprevalence resulted slightly but not significantly higher in pregnant women with dyspeptic complaints (74.6%) compared to the controls (63.8%). Xia *et al* (2004) demonstrated that the infection rates of both *H. pylori* and CagA-positive

strains are significantly higher in HG patients (88.9% and 78.1%, respectively) than in asymptomatic pregnant women (45.0% and 31.3%, respectively) ($P < 0.01$ for both). Despite a high seropositive rate in pregnant women with severe gastrointestinal symptoms during early pregnancy, no correlation was found between seropositivity and clinical symptoms or their duration. Shirin and colleagues (2004) reported an association between *H. pylori* and mild vomiting during early pregnancy but not with gastrointestinal symptoms later in pregnancy. Studies performed on endoscopic biopsies of gastric

mucosa demonstrated that the severity of gastrointestinal symptoms in early pregnancy may be associated with the density of *H. pylori* in the gastric epithelium. Additionally, two case reports showed that *H. pylori* eradication treatment reduces the severity of HG. In contrast, several studies found no relationship between HG and *H. pylori*. These contradictory

findings are probably due to the fact that a universally accepted HG definition does not exist, thus indicating a high heterogeneity of the study population. It has been proposed that a reduction of gastric acid production during early pregnancy as a result of increased accumulation of woman's body fluid, steroid hormone changes, and immunologic tolerance could lead the activation of latent *H. pylori* infection, which can

exacerbate nausea and vomiting symptoms.

Cardaropoli S et al. *H. pylori* and pregnancy

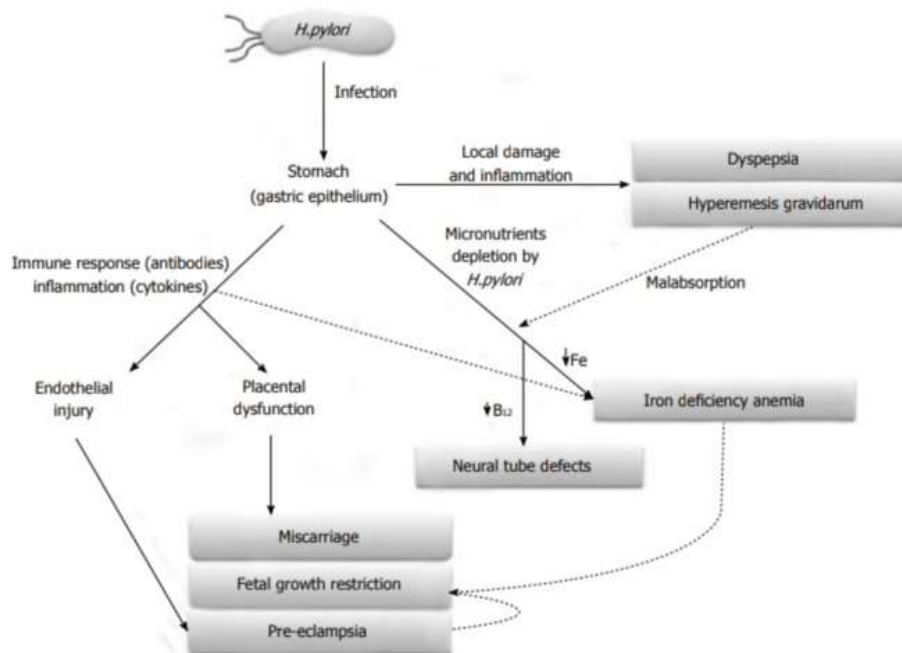


Figure 1 *Helicobacter pylori* infection correlation with pregnancy-related disorders. *Helicobacter pylori* (*H. pylori*) infection can cause local damage and inflammation, leading to gastrointestinal disorders such as dyspepsia in pregnancy and hyperemesis gravidarum. *H. pylori* sequesters essential micronutrients from the host organism. In particular, iron depletion may lead to iron deficiency anemia (IDA), while reduction of vitamin B₁₂ and folate may result in fetal neural tube defects. Lack of these micronutrients may also be favored by gastric malabsorption in case of the above mentioned gastrointestinal problems. Furthermore, IDA could indirectly be the consequence of local and systemic inflammation induced by *H. pylori* infection. Finally, the immune and inflammatory responses caused by this infection lead to endothelial and placental injury, through the cross-reaction of anti-*H. pylori* and tissue antigens and through the production of pro-inflammatory cytokines. Placental dysfunction characterizes important diseases of pregnancy, such as miscarriage, fetal growth restriction (FGR) and pre-eclampsia that it is also characterized by endothelial damage and it is often associated with FGR. Furthermore, IDA could be a risk factor for FGR.

H. Pylori Infection Diagnosis During Pregnancy

The current diagnostic methods include invasive and non-invasive tests. Invasive tests involve an upper gastrointestinal endoscopy with gastric mucosal biopsy and rapid urease activity detection, histology, microbiological culture, or polymerase chain reaction assays. Although mucosal biopsy and histopathologic examination of specimens for the presence of *H. pylori* and/or gastritis is considered the gold standard for the diagnosis of *H. pylori* infection, invasive tests are not well tolerated by patients

and may be a source of ethical problems. Gastroscopy can be performed in pregnant patients, but only when it is strictly necessary. The non invasive methods are more widely accepted in the prenatal period and include serum antibody detection, carbon-labeled urea breath tests, and stool antigen detection. Serologic and stool antigen tests are the first choice for *H. pylori* infection diagnosis in pregnancy, since they are easy to perform and low-cost non invasive diagnostic tests. Serologic tests are usually based on the detection of specific anti-*H. pylori*

IgG antibodies in the patients' sera by immunoenzymatic assay. Measurement of IgG antibodies against *H. pylori* reveals an immune response that could represent either a current infection or a previous exposure, since IgG antibodies disappear only several months after eradication of the microorganism. The stool antigen test is an enzymatic immunoassay that detects the active presence of *H. pylori* antigen in human feces. Stool antigen test is preferred to determine the *H. pylori* status after eradication.

Urea breath tests are not commonly used during pregnancy, despite they are reliable and noninvasive diagnostic test. In fact, it is demonstrated that ^{13}C -urea breath test, using the stable isotope ^{13}C as tracer, is not radioactive and safe also in children and pregnancy.

Therefore, it could be used as a valuable non-invasive semi-quantitative diagnostic tool for the assessment of gastric bacterial *H. pylori* infection. The urea breath test is recommended for test-and-treat strategies and suitable for control after eradication therapy and in epidemiological or pharmacological studies. Despite the excellent sensitivity and specificity of these tests, they are expensive and require specific instrumentation and specialized staff. Furthermore, it was stated that ionizing radiation dose involved in ^{14}C -urea breath test is extremely low, much lower than the radiation dose adsorbed from natural sources, a thousand times lower than the amount of fetal radiation considered to be teratogenic, therefore in the event of inadvertent exposure during pregnancy, the pregnant women should be reassured.

H. Pylori Infection Treatment During Pregnancy

There are multiple options for *H. pylori* infection treatment. The association of a proton-pump inhibitor and two antibiotics for 1 or 2 wk gives the best eradication rates in non pregnant subjects. Currently, there are no guidelines to treat *H. pylori* infection during pregnancy and the optimal therapy in pregnancy remains uncertain[Hayakawa *et al* treated four women with hyperemesis

gravidarum by a combination of penicillin and erythromycin, leading to alleviation of symptoms thus demonstrating the possible effectiveness of this specific *H. pylori* treatment. This hypothesis is supported in four additional case reports that showed similar symptom relief after antibiotic treatment. Several investigators have evaluated the safety of individual drugs, including proton pump inhibitors used in the anti *H. pylori* drug therapy in pregnant women. A recent meta-analysis reported that the use of proton pump inhibitors during first-trimester does not seem to be associated with increased risk of spontaneous abortion, preterm delivery or major congenital birth defects.

Nevertheless, some experts recommend that *H. pylori* eradication should be deferred until after pregnancy and lactation. It must be considered that treatment of *H. pylori*

infection has a low successful rate, with 35%-85% of infections being cleared, reaching the lowest values in some European countries. The gradual but steady occurrence

of antibiotic-resistant strains represents a major obstacle in the treatment of *H. pylori* infection. Pharmacogenomics-based approaches seem to increase the cure

rates, but re-infection also remains problematic. In fact, it is well known that eradication of *H. pylori* infections with antimicrobial agents in adults does not induce immunity against re-infection. In general, low annual recurrence rates were observed in developed countries (up to 2% for both adults and children), but high recurrence rates (> 10%) were observed in developing countries. There is no clear evidence that pregnancy predisposes to

de novo H. pylori infection. In view of these evidences, new approaches need to be considered for treatment of this disease, such as design of effective vaccines. Especially in case of pregnancy related diseases, it would be preferable to prevent *H. pylori* infection consequences, thus avoiding pharmacologic therapies during pregnancy. Recently, several clinical trials and animal studies have been focused on generating *H. pylori* recombinant

vaccines useful to eradicate and protect against the infection; however a safe and effective *H. pylori* vaccine has not yet been developed for use in humans.

Therefore, if *H. pylori* infection will be confirmed as an important risk factor for pregnancy complications, we suggest the conventional *H. pylori* eradication, namely triple therapy, should ideally be obtained several months before conception in order to reach seronegativity. This approach would avoid cross-reaction between anti-*H. pylori* antibodies and host tissue antigens, waiting for the discovery of novel effective vaccines.

Methods

Location, and Schematics of the Experiment

Pregnant women were followed over time in a prospective cohort study. The ladies were tracked from the point of recruiting all the way through shipment to Iraq between July 2020 and May 2021.

Participants in a Research Expectant mothers at three Diyala hospitals for prenatal care. governorate formed the subject population.

Size of the sample 96 patients When developing the formula, we settled on a 95% certainty threshold, *H. pylori* positive/negative, 80% power of one. In addition, we utilised 18 and to complete the calculation, 35 for the combined proportion of those who were not exposed and those who were with a desirable result, per Eslick [14].

There were two sets of data collected (exposed and unexposed). Those who were out in the open were the exposed group (n=96). *H. pylori* infection was confirmed by those who were tested whereas those who had not been exposed Ninety people made up the group that did not have *H. pylori* infection. We expected a 5% drop-off due to lack of contact. Getting people on board and keeping tabs on them Methods included a sequential sampling of who were eligible to participate till the sample size The desired size was attained. These individuals were selected because: They went ahead and signed up for prenatal care. Approval in Writing each participant who met the requirements explanations of the study's aims and methods and advantages.

Subjects in the research between the ages of 12 and 18 as educated volunteers, the presence of a foetus depending on the date of the previous menstrual cycle

and a thorough check by the seasoned midwife. After the fact Two prenatal checks were performed, at weeks 26 and 36. Preterm infants were excluded from this analysis. infants who were born during or after the 37th week of pregnancy. Our cohort consisted of the people who took part in the research. defined as those who fall between the ages of 18 and 35 who who are HIV-negative, first-time mothers, and women of a certain age be in the midst of a healthy, single-pregnancy pregnancy with no underlying medical conditions such is high blood pressure, active ulcers, and diabetes Having a genetic defect like sickle cell disease, 12-18 weeks of pregnancy when recruiting begins. However, not all pregnant women were allowed to participate. depending on how you cut it, this research should be disregarded; not possible remember what they weighed before they were pregnant, and are unable to their subsequent appointments when they were either physically or cognitively

sickness, prior substance abuse On the grounds of established exclusion using these standards, 56 women were deemed ineligible. study. They were unable to keep to the timetable in 14 cases. Among the 6 patients who came back for follow-up care, 2 had sickle cell, and 4 had alcohol-related issues. the abusers, 28 did not know their weight before becoming pregnant, 6 had a history of peptic ulcers and two were expecting multiples.

Study of Data Statistical Analysis Software for the Social Sciences was used for the data collection and analysis. (SPSS) V.16.0 (SPSS Inc., Chicago, IL, USA). Social,

Summary statistics for demographic and quantitative variables into frequency distributions and averages and deviations (SD). The birth weight of a baby was the dependent variable, and the number of *H. pylori* infection rates, birth weight, and other factors were examined. weight, height, and BMI back to pre-pregnancy levels; reaches and maintains parity motherly stature, also. Verification of continuous data was performed. normality.

Significance tests of associations were performed with the use of the Pearson chi-square (2) test for categorical data tests for categorical variables and the t-test for continuous variables with independent samples. Birth weight and its related factors the means of a logistic regression. Aspects linked to univariate analysis birth weights with P values less than 0.05 Multivariate analysis using logistic regression was performed on the

data. in order to identify causal elements that may while considering the infant's birth weight. Comparison of the odds using a 95% confidence interval and odds ratio were reported with a CI. Statistical multivariate analysis If the probability level was less than 0.05, then it was considered significant.

Results

Table 1 Comparison of categorical socio-demographic characteristics by H. pylori status.

Variable (n = 96)	H. pylori positive	H. pylori negative	P value
Parity. Primigravidae	35	24	
Parity , Secundigravidae	20	17	0.473*
<u>Maternal pre-pregnancy BMI (kg/m2)</u>			
Underweight (<18.5)	7	7	
Normal weight (18.5-24.9)	40	31	
Overweight (25.0-29.9)	8	3	0.372*
<u>Sex of the baby</u>			
Male	28	22	
Female	27	19	0.706
<u>Birth weight (grams)a</u>			
<2500	11	2	
≥2500	43	39	0.002*
<u>Maternal pre-pregnancy height (cm)</u>			
<156	21	16	

≥156	34	25	0.958*
<u>Maternal age (years)</u>			
<21	35	28	
≥21	20	13	0.502*
<u>Smoking</u>			
Yes	5	4	
No	50	38	0.100*
<u>Education level</u>			
Low (No education to primary 7)	11	10	
Medium (Secondary level)	36	26	
High (tertiary education)	8	5	0.114*

Table 2 Study results evaluating markers of *H. pylori* infection in patients with micronutrients deficiency

Study (Year)	<i>H. pylori</i> diagnosis		Description of the Indicators	Outcomes	Limitations/strength
	Antibody/targeted molecule	Method			
Ali <i>et al</i> (2018) ⁴⁰	Anti-Helicobacter pylori IgA	ELISA	Iron	Significant positive correlation between iron deficiency and <i>H. pylori</i> -positive cases.	Smaller sample size, limited access data, study purpose was highly focused.
Felkner <i>et al</i> (2007) ³⁸	Anti-Helicobacter pylori IgG	ELISA	Ferritin, folate, vitamin B ₁₂	No association was found between micronutrients and <i>H. pylori</i> -positive cases.	Larger sample size, well-validated study, sample was collected in just after post partum.
Golalipour <i>et al</i> (2012) ³⁷	Anti-Helicobacter pylori IgG	ELISA	Ferritin, folate, vitamin B ₁₂	No association was found between micronutrients and <i>H. pylori</i> -positive cases.	Smaller sample size, sample was collected in just after post partum.
Mulayim <i>et al</i> (2008) ³⁹	Urease	¹⁴ C-urea breath test	Iron	Significant positive correlation between iron deficiency and <i>H. pylori</i> -positive cases.	Data were not normally distributed when calculated for iron deficiency.
Mubarak <i>et al</i> (2014) ³⁶	Anti-Helicobacter pylori IgA	ELISA	Ferritin	There is a no link between iron deficiency, anaemia, and thrombocytopenia with <i>H. pylori</i> -positive cases.	Larger sample size, well-validated study, limited biasness
Ugwuja <i>et al</i> (2010) ³⁵	Anti-Helicobacter pylori IgG	ELISA	Copper, iron and zinc	Trace elements (Cu, Fe, and Zn) are not significantly associated with <i>H. pylori</i> positive cases	Larger sample size, limited access data, highly biased.

Table 3 Comparison of continuous socio-demographic characteristics by *H. pylori* status.

Variable (n =96)	<i>H. pylori</i> positive	<i>H. pylori</i> negative	P value
Maternal age (years) 21.1 20.8 0.481**	22.1	20.3	0.481**
Mean household size	3.2	2.7	0.462**
Mean maternal recruitment BMI (kg/m2)*	21.5	21.1	0.352**
Mean maternal height (cm)	157.5	155.2	0.727**

**P valve for the independent sample t test and n is number

Table 4 Mean ± SD values selected variables in relation to *H. pylori* infection status

Variable	Mean ± SD by <i>H. Pylori</i> Status	Mean ± SD by <i>H. Pylori</i> Status	Range	P valve
	<i>H. pylori</i> -ve (n = 41)	<i>H. pylori</i> + ve (n = 55)		
Rate of weight gain (kg/week)	0.32 ± 0.11	0.25 ± 0.10	0.08–0.75	<0.001
Gestational age (weeks)	39.2 ± 1.1	39.1 ± 1.0	37.0–42.0	0.494
Birth weight (g)	3242 ± 407	2682 ± 370	1700–4400	<0.001
Pre-pregnancy BMI (kg/m2)	21.1 ± 2.5	21.2± 2.9	15.0–29.4	0.35

Pre-pregnancy weight (kg)	52.1 ± 7.0	53.4 ± 8.1	37-76	0.392
Maternal height (cm)	157.1 ± 5.9	157.8 ± 5.9	142.0-173.1	0.727

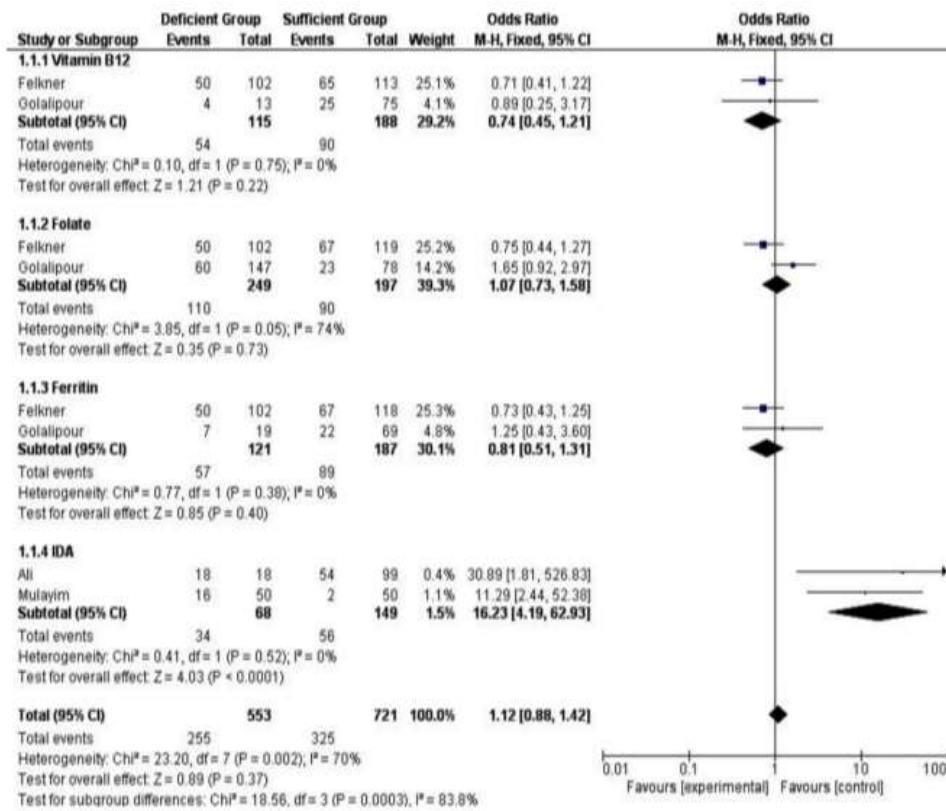


Figure 2 Forest plot of the association between *H. pylori* infection and micronutrient deficiency.

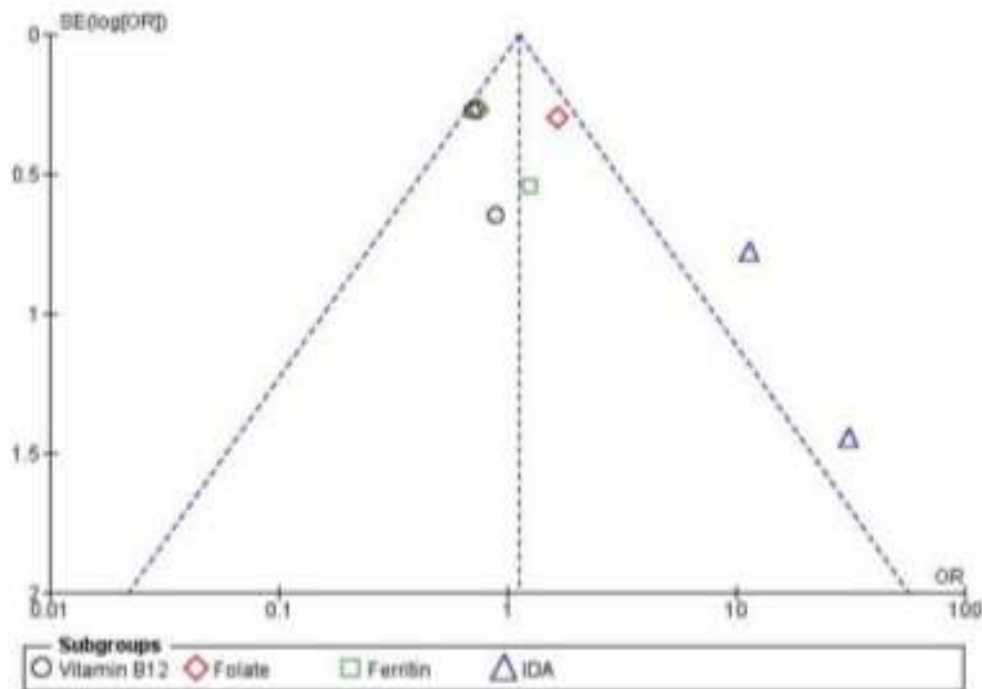


Figure 3 Funnel plot with estimated 95% CIs for meta-analysis of the effect of *H. pylori* on micronutrient status. ID iron-deficiency anaemia.

Table 5 Subgroup metaanalysis of indicator of micronutrients

Subgroup	OR (95% CI)	Heterogenicity (χ^2)	P value
Vitamin B12	0.74 (0.45 to 1.21)	0.1	0.22
Folate	1.07 (0.73 to 1.58)	3.85	0.73
Ferritin	0.81 (0.51 to 1.31)	0.77	0.40
IDA	16.23 (4.19 to 62.93)	0.41	<0.001

Discussion

The frequency with which *H. pylori* is based on a *pylori* infection, *H. pylori* antigens in the stool of this group 52.9% of pregnancies were to women who were neither primigravidae or secundigravidae. Here we have consistent with what would be anticipated given the current state of society in our nation. severity and frequency of illness among ladies who are pregnant and are receiving prenatal treatment in the health facility [2, 6]. An alarmingly high percentage of *H. pylori*. This study's findings on *H.*

pylori infection are consistent with those rates of 69.8%, 68.5%, and 52.2% for *H. pylori*. Peptic ulcer disease among expecting mothers in Chile and neighbouring Sudan as well as rural Mexico [7–9]. Enhanced frequency, Both 74% and 88% of *H. pylori* documented cases of *pylori* infection among border-crossing expectant mothers and Egypt, two different phrases [26, 27]. Our research included Mean Standard Deviation for Primigravidae and Secundigravidae the mean age of 20.9 2.7 years, and there is evidence that prevalence increase of *H. pylori* infections are

more common as people become older. together with the total number of births [9]. Newborns infected with H. female *Helicobacter pylori* carriers were at a much higher risk for developing stomach cancer. The average birth weight was lower (2681 370 g). compared to individuals that didn't have H in their genes. women without p. pylori (n = 3245) 407 g) P < 0.001. As so, this is in complete agreement with the results. based on the research conducted in Turkey [16] by Mulayim et al. Similarly, babies delivered to first-time mothers had a much Birth weights that are on the lower end of the spectrum (2791 422 g) are more common than newborns of the secundigravidae family (3210 465 g, P 0.001).

This result agrees with those found by Chiba et al. [28]. Nonetheless, the LBW was not different. ratio of babies born to first-time mothers to babies born to other mothers born into the family Secundigravidae (P = 0.70). As a result, this observation stands in stark contrast based on the research done by Chiba and coworkers who observed a dramatic increase in the LBW rate in primigravidae than in multigravidae females. This dissimilarity possibly be due to the fact that our sample was somewhat representative of the majority of the demographic and dietary factors finding a statistically significant correlation. In this population analysis, the rate of LBW was This number was somewhat higher than the national LBW average of 13.6%. prevalence (10.5%), although it's quite similar to the 14% that's said to have occurred in central Ugandan regions where this investigation was conducted undertaken [24]. We conclude that the LBW in this urban population was greater due to the composition of the population, which studied. Only pregnant women participated in this research. the first or second time, with a mean SD age of 20. Women from both urban and rural areas were Researchers analysed populations of all ages and stages of pregnancy in a countrywide poll [24]. Quite a few of our members Second-time mothers made for 38.5% of all pregnancies, with a mean Normalized Variance Age: 21.7 3.2 There's a pile of proof studies demonstrate that infants delivered to mothers who have already had one

Births with Low Birth Weight (LBW) are more common among teenagers [29]. It is also now believed, and supported by data, that those beyond the age of 20 are more likely to possibility of having a low birthweight baby [30]. As a result of our investigation, we identified H. infections caused by *Helicobacter pylori* to stand on their own indicators of preterm birth (OR = 3.6, 95% CI: 1.1 – 11.5; P = 0.031). Our results are consistent with those of the conclusion reached by Eslick et al. and Mulayim et al. Slick et al. et al. (2002) first noticed a correlation between H. pylori and preterm birth, and in particular they established that foetal growth restriction was a more typical of H. greater proportion of women with H. pylori (13.5%) than in mother disapproval (6.0%; OR = 2.41; 95% CI = 1.14-5.08) P = 0.018). Pregnant women, as noted by Mulayim et al. with H. new-born infants infected with *Helicobacter pylori* had a much higher risk of developing severe birth weights than moms who did not have it's infection. It has also been shown to be effective in animal models. had it reported to them that H. mice with H. pylori infection had a lower production of rate, and their infants had poor birth weights. the poundage [31]. However, in a separate mouse model used for experiments research, the findings were not replicated [32]. In this case, the mechanism(by which H. infections with *Helicobacter pylori* may impede a fetus's development. is not quite certain, however a number of other things have suggested. H. pylori is associated with a rise in the incidence of digestive distress, nauseousness, or vomiting [8] because caused by an underlying case of peptic ulcer disease that has not been identified impacting gastric absorption and lactation. thereby hinder development throughout pregnancy. Humans are susceptible to H. Peptic ulcer disease has been linked to maternal anaemia [33] and iron H. septicaemia-related anaemia. possible outcomes of pylori infection slow development in a foetus. According to the research of Jasem et al. [33], Possible causes of prenatal growth restriction include maternal disease, since all pregnant women with anaemia were housed in the H.

Group of People With Helicobacter Pylori. Reduced foetal growth might be caused by either the mother or the foetus or the placenta. Feto-placental infections and other placental diseases may contribute to this. Anti-CagA antibodies have been shown to be effective in preventing the spread of respond to placental tissue in vitro and inhibit its invasiveness via cross-reactivity the ability and the resulting aberrant placentation caused foetal growth restriction . However, Scientists Cardaropoli et al. discovered a robust correlation between H. pylori infection with Helicobacter pylori and the risk of preeclampsia pregnancy, but H. pylori was not linked to any. pylori and intrauterine growth retardation due to an unknown cause [12]. How a woman puts on weight when pregnant determines how much a foetus will grow [17]. This analysis revealed that first-time mothers had a higher average Gaining weight at a slower pace than the secundigravidae (0.33% 0.11 kg/week)

(27 10 grammes per week) P 0.001. This result fits well with the results found by Lumbanrajaa et al., [36]. Pregnancy rates were also discovered in the current investigation. Gains in weight of 0.30 kg/week or less in weeks 2 and 3 independent predictor of low birth weight throughout trimesters. size (OR = 3.80; 95% CI 1.01-14.10; P = 0.044). This result in agreement with the findings of a number of previous research shown that larger birth weights are correlated with birth weight [37-39] of the mother.

Our research is robust since it was a prospective cohort, and we were able to exert considerable risk factors for LBW such as premature birth, persistent conditions and inherited illnesses. And of course, we couldn't leave out a we may ascribe the results to the homogeneity of the population and the H. s birth weights. a pylori infection, for short. For completeness, we likewise only infants born late in pregnancy (after 37 weeks) newborns did not make up any of our sample population. Evidence linking H. pylori and vitamin a deficiency Insufficient research has been done on the topic of B12 levels during pregnancy. research. Prior research indicated that H. pylori-related 57% of macrocytic individuals

suffer from gastritis. vitamin b12 deficiency-induced anaemia 44 is linked to haptocorrin at the ideal pH for the stomach to separate the vitamin from the protein permeable, allowing for absorption.⁴⁵ Several investigations showed that H. Having a pylori infection has both short- and long-term impacts on stomach pH. The body's pH increases due to infection, a condition known as hypochlorhydria. Hyperchlorhydria or hypochlorhydria may be caused by persistent illnesses. depending on the location, hypochlorhydria (lower pH) infected person.^{46 47} However, the research we've included aren't powerful enough to H. has to be defined for each unique patient. Peptic ulcer disease statuses. No statistically significant differences were found, which is supported by two other research connectives between H. pylori and vitamin a deficiency Those cases of B12 insufficiency were among the elderly, and they populations. This time, another water-soluble Folate is a B vitamin. and a greater pH has a detrimental effect on its absorption. H's provocation. a pylori infection, for short. Both serums were analysed separately. , often known as a red blood cell (RBC), is a frequently-used unit of include folic acid. On the other hand, measuring serum folate is preferred over red blood cell folate by many doctors.⁵⁰ Specifically, red blood cell folate (RBC folate) is better than serum folate. during pregnancy because it increases folic acid levels⁵¹ meta-analysis, Approximately 50% of the studies assessed folate levels from half determined from the RBC sample and serum, respectively sample. This may be the reason for our findings, oppose the idea that H. test results for H. pylori and decreased folic acid. It should be noted that Rasool et al.⁵² came to similar conclusions. Insignificant effect of H. human vitamin B12 and folate serum pylori concentration of homocysteine Total body ferritin concentration correlates highly with serum ferritin concentration. serum ferritin concentrations and iron reserves to be the gold standard for IDA diagnosis.⁵³ H. was the cause of the problem, as determined by us. Having H. pylori was not linked to levels of ferritin. This disconnection might be due to confused because of how much H. A pylori infection is

present when the guts of it Researchers have discovered that H. testing positive for Helicobacter pylori A inverse association was seen in individuals with peptic ulcer disease. H. and I. individuals with Helicobacter pylori infection and low blood ferritin levels,⁵⁴ There is no difference for those with a healthy digestive system. changes in ferritin concentrations were detected as a result of the presence or absence of H. Interestingly, we found a correlation between H. pylori infections and a high and significant favourable association with H. Peptic ulcer disease combined with IDA (OR=16.23, 95% CI 4.19–62.93, p0.0001). Several plausible biological pathways exist by where H. iron stores are depleted in response to pylori. To start out, H. the bacterium pylori causes gastric adenocarcinoma, gastric haemorrhage, and and diseases like gastric ulcers, which are on the rise iron deficiency ⁵⁶ Secondly, H. pylori-related gastritis of the body. pylori may reduce stomach acid production by causing gland atrophy. the absorption of iron would be hampered, ^{57th} H. influenzae's CagA protein. pylori contributes to interstitial cystitis directly via holotransferrin ⁵⁸ Iron absorption Additional support for H. pylori is provided by the CagA protein. development of pylori and colonisation of the intestines.¹⁸ Our findings are corroborated by meta-analysis with respect to a case-control study statistical evidence supporting an H increases the possibility of a sever IDA. a pylori infection, for short.

Conclusion

What stands out as the study's most crucial result is revealed that pregnant women infected with H. pylori had a much an increased possibility of having a low birth weight baby. In Moreover, women who give birth first or second have smaller babies. Women who were shorter than 156 inches were more likely to get pregnant. kids born too early who are LBW. Without Regard to Maternal H. pylori Infection status, mother height, and birth weight distribution For LBW, gain was still the most critical consideration. Also Micronutrient deficits and H. pylori infections are linked. the

norm and pervasive among economically disadvantaged population, and particularly among pregnant women. According to the results of this meta-analysis demonstrates a correlation between having H. pylori and vitamin B12, folate, and iron all have been linked to a decreased risk of IDA, whereas vitamin B6 has been linked to ferritin when carrying a child. As a result, it is crucial that check for H. pylori infection, particularly for the sake of curative care incidence of IDA during pregnancy

Recommendations

Women should be screened for H. pylori infection, since it is common and potentially dangerous. able to have children People who tested positive for H. pylori

Pregnancy should not be attempted until the infection has been cured. because current H. pylori medication regimens are inadequate not safe during pregnancy. All pregnant women should also get pregnancy counselling and regular monitoring weight growth to guarantee sufficient weight, leading to birth weight that is considered normal.a

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