Effect of maternal *Helicobacter Pylori* infection on gestational weight gain in an urban community of Uganda

Ronald Wanyama¹², Gerald Obai², Pancras Odongo³, Michael Kagawa⁴, Rhona Baingana⁵

¹Department of Biochemistry, Faculty of Medicine, Gulu University, Gulu, Uganda, ²Department of Physiology, Faculty of Medicine, Gulu University, Gulu, Uganda, ³Department of Internal Medicine, Faculty of Medicine, Gulu University, Gulu, Uganda, ⁴Department of Obstetrics & Gynecology, College of Health Sciences, Makerere University, Kampala, Uganda, ⁵Department of Biochemistry and Sports Science, School of Biosciences, Makerere University, Kampala, Uganda

Corresponding author: Ronald Wanyama, Department of Biochemistry, Faculty of Medicine, Gulu University, Gulu, Uganda

Key words: H. pylori infection, pregnancy, gestational weight gain, parity, Kampala, Uganda

Received: 11/06/2016 - Accepted: 14/08/2017 - Published: 16/10/2017

Abstract

**Introduction:** Maternal *Helicobacter pylori* (*H. pylori*) infection has been associated with undesirable effects during pregnancy such as; hyperemesis gravidarum, anemia, intrauterine fetal growth restriction and miscarriage. Our aim was to document the effect of *H. pylori* infection on gestational weight gain (GWG) in a low-income urban setting in Uganda. **Methods:** This was a prospective cohort study conducted in Kampala between May 2012 and May 2013. The participants were HIV negative, *H. pylori* positive and *H. pylori* negative primigravidae and secundigravidae. Recruitment was at gestation age of eighteen or less weeks and follow up assessments were carried out at 26 and 36 weeks gestation age. *H. pylori* infection was determined using *H. pylori* stool antigen test. Maternal weight and height were measured, and body mass index (BMI) and rates of GWG were calculated. **Results:** The participants’ mean±standard deviation (sd) age was 20.9±2.7 years. Primigravidae were 68.8% (n = 132) and 57.3% (n = 110) of the participants were positive for *H. pylori* infection. Low pre-pregnancy BMI (< 18.5 kg/m²) was recorded in 14.6% (n = 28). The mean±sd rate of GWG during second and third trimesters was 300.5±79.7 grams/week. The mean±sd weight gained by 36 weeks of gestation was 9.6±2.2 kg while gestation age at delivery was 39.4±1.0 weeks. Factors independently associated with the rates of GWG during the second and third trimesters were parity (P = 0.023), *H. pylori* infection (P = 0.006), pre-pregnancy BMI (P = 0.037), height (P = 0.022) and household income (P = 0.003). **Conclusion:** *H. pylori* infection is associated with low rates of GWG among primigravidae and secundigravidae.
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 [3]. However, pregnancy increases the susceptibility to *H. pylori* infection [4] probably due to decreased cell-mediated cytotoxic immune response [5]. Although many infected individuals are asymptomatic, *H. pylori* is an important health problem. *H. pylori* infection has been recognized as a major cause of various gastroduodenal diseases, such as chronic gastritis, peptic ulcer disease, and gastric cancer [3]. In Uganda the prevalence of *H. pylori* infection in dyspepsia patients who underwent endoscopy was 74% and 86% in patients with cancer and benign tumors [6, 7]. Recently, Baingana et al., found the prevalence of *H. pylori* infection of 60.5% among pregnant women attending an antenatal clinic in Kampala [8]. Pregnancy is a physiological condition in which a marked increase in body weight occurs over a short period of time. An optimum weight gain over the course of pregnancy, as recommended by the Institute of Medicine (IOM), is one that produces a healthy newborn [9]. Optimum weight gain also provides sufficient postpartum maternal fat stores to support lactation without increasing obesity risk [9]. Furthermore, there is evidence to show that maternal pre-pregnancy weight and the weight gained during pregnancy influence birth weight [10]. However, gestational weight gains below the IOM recommendation are common in developing countries [11]. Inadequate gestational weight gain increases the risk of preterm delivery and low birth weight infants [12, 13]. Current evidence shows that total gestational weight gain and rate of weight gain decreases with increasing pre-pregnancy body mass index (BMI) [14] and this is in agreement with 2009 IOM recommendations [9].

*H. pylori* infection in pregnancy is associated with many adverse effects, such as extreme, persistent nausea and vomiting (hyperemesis gravidarum) [15, 16], neural tube defects in newborns, pre-eclampsia, intrauterine fetal growth restriction and miscarriage, and thrombocytopenia [17-20]. Conditions such as nausea and vomiting reduce appetite. This can lead to reduced food intake and in due course, inadequate supply of nutrients to the body. Furthermore, *H. pylori* infection has been associated with reduced production of ghrelin and increased levels of gastric leptin [21, 22]. Ghrelin increases appetite, facilitates fat storage, and may influence energy homeostasis [23-25]. Increased expressions of gastric leptin make the affected individuals to experience decreased appetites and subsequently weight loss [26]. An association between *H. pylori* and weight loss has been suggested [25, 26]. However, there is limited data on association between *H. pylori* and gestation weight gain especially in developing countries where inadequate GWG is already common. The objective of this study was to establish the association between *H. pylori* infection and maternal weight gain during pregnancy.

Methods

The study protocol was reviewed and approved by the Research and Ethics Committee of the School of Medicine, Makerere University. Clearance to conduct this study was obtained from Uganda National Council for Science and Technology. Permission to conduct the study at Kawempe Health Centre IV was granted by the Kampala Capital City Authority, Health Department. Participation in the study was voluntary and each participant signed a consent form.

**Study design, site and population**

This was a prospective cohort study conducted between May 2012 and May 2013. Pregnant women were followed from early second trimester to late third trimester. The study was conducted at the antenatal clinic of Kawempe Health Centre IV. The Health Centre is supported by the Ministry of Health, Uganda and the services in the antenatal clinic are free to the public. This clinic serves a densely-populated, low-income area in Kawempe Division, one of the five divisions forming Kampala District in Uganda. The division is located in the Northern part of Kampala District. The study targeted HIV negative primigravidae and secundigravidae.

**Sample size**

We used the online openEpi software, based on Kelsey Lesley formula (1996) to calculate the sample size. In the formula we used a confidence level of 95%, power of 80%, ratio of *H. pylori* positive to *H. pylori* negative of one. Furthermore, in the formula we used 18 and 35 as the percentages of unexposed and exposed participants with outcome of interest according to Elsick [21]. The exposed group comprised of those who tested positive for *H.
pylori infection while the unexposed group comprised those who tested negative for *H. pylori* infection.

**Recruitment and follow up**

A consecutive sampling procedure was used to select participants who met the selection criteria until the sample size was achieved. The participants were chosen as they got registered at the antenatal clinic. Written informed consent for each eligible participant was sought after clear information being given about the study objectives, procedures and benefits. In Uganda HIV testing for pregnant women is recommended and is always done on the day of the first visit to the antenatal clinic. The study participants were recruited as informed volunteers at 12-18 weeks of gestation based on the reported last menstrual period and the experienced midwife’s examination. Follow up assessments were carried out at 26 and 36 weeks of gestation. The study participants were included in our cohort based on the following criteria; between 18-35 years of age, pregnant for the first or second time, HIV negative, carrying a singleton pregnancy, free of any systemic illness such as hypertension, active peptic ulcers, diabetes mellitus or genetic abnormality like sickle cell disease, and between gestation weeks 12-18 at the time of recruitment. However, some of the pregnant women were excluded from this study based on the following criteria; not able to recall their pre-pregnancy weight, not able to schedule their return visits, not able to speak and/or hear, mentally ill, history of drug or alcohol abuse Based on the set exclusion criteria, a total of 56 women were excluded from this study. Fourteen of them could not adhere to the scheduled return visits, two had sickle cell disease, four had alcohol related problems, twenty-eight could not recall their pre-pregnancy weight, six had active peptic ulcers and two were carrying twins.

**Data collection and determination of nutritional status**

During the participant’s interview, demographic data including social, behavioral and medical history were collected in researcher-administered structured questionnaires. Nutritional status of each participant was assessed using anthropometric parameters. Anthropometric measurements were carried out with the help of a midwife in a closed room when the participant was barefoot and wearing light clothing with the help of a midwife. Body weight was measured using an adult portable beam scale with 150 kg capacity divided into 0.5kg increments (GmbH & co.kg, Germany model 7621019009). Height was determined with the individual barefoot and in an orthostatic position with the aid of a portable stadiometer consisting of a non-extendable 2 meter measuring tape divided into 0.1cm increments. Body weight and height were measured twice for every participant and the average of the readings was considered as the participant’s weight and height respectively. Each participant’s BMI was calculated using the following formula: BMI = pre-pregnancy body weight (kg) divided by height (m) squared. The BMI was categorized using the World Health Organization criteria as follows; underweight (< 18.5 kg/m²), normal weight (18.5-24.9 kg/m²), overweight (25.0-29.9 kg/m²), obese (≥ 30.0 kg/m²) [27]. Pre-pregnancy weight in kg (Wp) considered in this study was that reported by the participant at recruitment. The measured gestation weight at each time point (GWR = weight at recruitment, GW26 = weight at 26 weeks of gestation and GW36 = weight at 36 weeks of gestation) were recorded. Rate of GWG during second trimester was calculated as \([(GW26 - GWR)/(26 - gestation age at recruitment)]kg/week. Similarly, rate of GWG during third trimester was calculated as \([(GW36 - GW26)/10]kg/week. GWG by 36 weeks was got by subtracting Wp from W36.

**Stool collection and testing for *H. pylori* infection**

After clear instructions on how to collect the stool sample, each participant was given clean tissue paper on which to deposit the stool. After, she had to immediately transfer a stool sample into stool collection bottle using the scooper which was part of the bottle top. This was done in the antenatal clinic toilet. Stool samples were immediately placed in a cool box with ice packs. The samples were transported everyday from Kawempe Health Centre to the laboratory (~3 km) and stored in a -20°C freezer until analysis was carried out. *H. pylori* stool antigen test, i-Chek cassettes (Chem-Labs Limited, Nairobi, Kenya) were used to analyze the stool samples. It is a rapid one-step chromatographic immunoassay that utilizes a combination of anti-*H. pylori* antibodies and anti-mouse IgG. Instructions given by the manufacturer were followed. Approximately 100 µl of stool of completely thawed stool was brought into the sample diluent tube and vortexed for fifteen seconds. Three drops of the diluted sample were applied to the test and the result was read after fifteen minutes. The results were reported as positive or negative on the basis of the manufacturer’s guidelines. A procedural control was included with each test.
Data analysis

Data were analyzed using Statistical Package for Social Sciences (SPSS) V.15.0 (SPSS Inc., Chicago, IL, USA). Social, demographic and measurement parameters were summarized into frequencies and mean ± standard deviation (sd). The outcome variable was rates of GWG while the independent variables were H. pylori infection, pre-pregnancy weight, pre-pregnancy BMI, parity and maternal height. Continuous data were checked for normality. Tests for the significance of association were made using the Pearson chi-square ($\chi^2$) test for categorical variables and independent sample t test for continuous variables. Factors associated with GWG were determined with linear regression. Factors associated with rates of GWG with P values < 0.2 during bivariate analysis were considered for multivariate analysis using linear regression to determine factors independently associated with rates of GWG. At multivariate analysis, statistical significance was determined if p < 0.05.

Results

Table 1 summarizes the overall socio-demographic characteristics of the study participants. Two hundred twenty one HIV-negative pregnant women were enrolled into the study. Twenty six were lost to follow up and of the 26, 20 were negative for H. pylori infection. However, data from only 192 participants was used to perform all the analyses because two of the participants lost their pregnancies at 22 and 25 weeks of gestation, one delivered at 35 weeks of gestation. Primigravidae were 68.8% (132/192) while 87.5% (168/192) of the participants were married, 9.9% (19/192) were single and the rest were either divorced/separated or widowed. Only 1.0% (2/192) of the participants were smokers while those who took alcohol were 5.2% (10/192). Nearly two thirds of the participants, 64.6% (124/192), had acquired secondary education. The majority of the participants, 78.1% (144/192), were housewives and only 19.8% (38/192) were employed. Underweight (BMI < 18.5 kg/m$^2$) was recorded in 14.6% (28/192) of the participants. Over 99% of the participants were using clean water in their households. We observed no differences in the socio-demographic variables between the participants with H. pylori infection and those without H. pylori infection. Table 2 shows the means ± standard deviations (sd) and ranges for the selected variables. The mean±sd gestational age at recruitment was 16.9±1.5 weeks with range of 12-18. The mean±sd (range) pre-pregnancy weight, weight at recruitment and age was 53.1±7.6 (37–76) kg, 56.9±8.1 (38–82) kg and 20.9±2.7 (18–35) years respectively. The mean±sd (range) maternal height and maternal pre-pregnancy BMI of participants was 157.4±5.7 (142.0–173.1) cm and 21.3±2.7 (15.0–29.4) kg/m$^2$ respectively. The mean rate of weight gain during the second and third trimesters was 300.5±79.7 grams/week with a range of 90–630 grams/week. The mean±sd weight gained by 36 weeks of gestational was 9.6±2.2 kg with a range of 5-16. Table 3 shows the means±sd and ranges for the selected variables and the differences between H. pylori positive and H. pylori negative participants. The mean pre-pregnancy weight, maternal pre-pregnancy BMI, maternal height and gestation age at delivery of H. pylori positive participants were not significantly different from those of H. pylori negative participants. Furthermore, the rates of weight gain during second trimester for H. pylori negative of 322.3±97.4 grams/week was higher than 294.9±102.4 grams/week for H. pylori positive but was not significantly different (P=0.063). However, the mean±sd rate of weight gain during second and third trimesters of 317.0±74.1 grams/week for H. pylori negative participants was significantly higher than that of H. pylori positive participants (288.2±81.8 grams/week) and significantly different (P = 0.013). The mean±sd gestational weight gained by H. pylori negative participants (10.1±2.3kg) by 36 weeks of gestation was higher than that gained by H. pylori positive participants (9.2±2.1kg). This difference was significantly different (P = 0.002).

Factors associated with gestational weight gain

For linear regression, the outcome variable was rates of GWG during second and third trimesters. Factors independently associated with GWG were; parity (95% confidence interval (CI) (-0.051 – -0.004; P = 0.023), H. pylori infection status (95% CI -0.053 – -0.009; P=0.006), maternal pre-pregnancy BMI (95% CI -0.008 – -0.000; P = 0.037), maternal height (95% CI -0.000 – -0.004; P = 0.022) and household monthly income (-0.023 – -0.005; P = 0.003) (Table 4).

Discussion

The pattern of maternal weight gain during pregnancy is an important determinant of fetal growth [9]. Although several studies have reported how differences in the timing of maternal weight gain
may be related to fetal growth outcomes [28, 29], none addresses the effect of maternal *H. pylori* infection on GWG especially in a developing country. In this paper, we investigated the relationship between maternal *H. pylori* infection and rates of GWG during the second and third trimesters of pregnancy because these trimesters greatly influence birth outcome [9]. Although effects of *H. pylori* infection on fetal growth and birth outcome are known [17-20], our study is among the first to evaluate the relationship between *H. pylori* infection and rates of GWG during the second and third trimesters among primigravidae and secundigravidae. We found that the presence of *H. pylori* infection significantly affects the rate of GWG in this population.

In this current study we found the mean±sd rate of GWG of *H. pylori* positive pregnant women (288.2±81.8 grams/week) to be significantly lower than that of *H. pylori* negative pregnant women (317.0±74.1 grams/week), P = 0.013. We did not come across any published information relating *H. pylori* infection to GWG but studies involving non-pregnant have associated *H. pylori* infection with reduced appetite and weight loss [25, 26]. Our study found no differences in pre-pregnancy weight and pre-pregnancy BMI between *H. pylori* positive and *H. pylori* negative participants. This can be explained by the fact that the participants were more of a homogenous and apparently healthy population. This same study also found out that the mean rate of weight gain in primi-gravidae was higher than in secudi-gravidae. This finding is in agreement with recent findings of other studies [30-33] which found that primigravidae are more likely to gain a greater amount of gestational weight and experience excessive GWG than their multigravidae counterparts. This present study further found a positive correlation between rates of GWG during the second and third trimesters and birth weight (P < 0.001). Our finding agrees with several other studies [34-36].

*Helicobacter pylori* infection was found to be independently associated with low rates of gestation weight gain (P = 0.006). Studies have associated *H. pylori* infection with weight loss [25] and weight gain after eradication [21]. One of the mechanisms through *H. pylori* infection may lead to low GWG is by reducing the production of ghrelin and increasing the production of gastric leptin [21, 22]. Ghrelin increases appetite and facilitates fat storage [23] whereas leptin reduces appetite and leads to weight loss [25]. Maternal pre-pregnancy BMI was also found to be independently associated with the rate of GWG (P = 0.037). This is in agreement with other studies that have showed that low pre-pregnancy BMI increases rates of maternal weight gain [37]. Total gestational weight gain and rate of weight gain decreases with increasing pre-pregnancy body mass index [9,14]. Parity was another factor independently associated with rate of GWG (P = 0.023) in this study as seen in Table 4. This finding is in agreement with the findings of other studies which found that primigravidae are more likely to gain a greater amount of gestational weight and experience excessive GWG than their multigravidae counterparts [30-33]. Furthermore, household monthly income was also found to be associated with GWG during the 2nd and 3rd trimesters. In sub Saharan Africa, increased income is associated with lifestyle factors including increased food intake especially calories and reduced physical activity [38]. These factors have been associated with increased total GWG [39, 40]. Although Pickett and colleagues [41] found no interaction between maternal height and net pregnancy weight gain, our present findings show that there is a significant relationship between maternal height and rates of GWG during the second and third trimesters. This is in agreement with the findings of several authors [42, 43]. The strength of our study lies in the fact it was a prospective cohort and we were able to control for some of the known risk factors for GWG such as chronic and genetic diseases. We also included a homogenous population and we are able to attribute the rates of GWG to *H. pylori* infection. However, this current study had some limitations. We did not collect data of all the risk factors for low GWG, for example, level of physical activity during pregnancy, number of antenatal visits, previous poor pregnancy outcome for secundigravidae, neither did we consider other infections, such as malaria and helminth infestations, which are endemic in the study area and have been associated with low rates of GWG [44, 45].

**Conclusion**

*Helicobacter pylori* infection has a negative effect on GWG during second and third trimesters. Other factors which independently affect GWG are parity, household monthly income, maternal height and pre-pregnancy BMI. We recommend that women of child bearing age be screened for *H. pylori* infection. Those found positive for *H. pylori* infection should be treated before they become pregnant since drugs used in the treatment of *H. pylori* infection are not safe in pregnancy.
What is known about this topic

- Parity has an effect on the rate of gestational weight gain;
- Pre-pregnancy body mass index and height also have an effect on gestational weight gain;
- Level of household income significantly affects the rate of gestational weight gain.

What this study adds

- Maternal H. pylori infection negatively affects the rates of gestational weight gain during the second and third trimesters of pregnancy;
- Maternal H. pylori infection has no effect on pre-pregnancy weight and height.

Competing interests

The authors declare no competing interests.

Authors’ contributions

Wanyama R conceived the idea and developed the study concept; oversaw the process of data collection and entry. Obai G designed the analysis plan, analyzed and interpreted the data. Both Wanyama R and Obai G wrote the draft manuscript. Kagawa M and Odongo P were involved in the development and writing of the study proposal and provided critical revision of the manuscript for intellectual content. Baingana RK provided laboratory assistance and critical revision of the manuscript for intellectual content. All authors have read and agreed to the final version of this manuscript.

Acknowledgments

We are thankful to the pregnant women who participated in this study. The authors would like to thank the administration and staff of Kawempe Health Centre antenatal clinic. This study was supported by the International Atomic Energy Agency (IAEA). The content is solely our responsibility as authors and does not necessarily represent the official views of the IAEA.

Tables

| Table 1: Socio-demographics characteristics of participants |
| Table 2: Mean±sd values of participants’ biological characteristics |
| Table 3: Mean±sd values of selected variables in relation to H. pylori infection status |
| Table 4: Factor independently associated with GWG during the second and third trimesters |

References

1. Ford AC, Axon AT. Epidemiology of Helicobacter pylori infection and public health implications. Helicobacter. 2010 Sep; 15 Suppl 1: 1-6. PubMed | Google Scholar
2. Hunt RH, Xiao SD, Megraud F, Leon-Barua R, Bazzoli F, van der Merwe S et al. Helicobacter pylori in developing countries. World Gastroenterology Organisation Global Guidelines. August 2010. Google Scholar
3. Suerbaum S, Michetti P. Helicobacter pylori infection. N Engl J Med. 2002 Oct 10; 347(15): 1175-86. PubMed | Google Scholar
4. Lanciers S, Despinasse B, Mehta DI, and Blecker U. Increased Susceptibility to Helicobacter pylori Infection in Pregnancy. Infect Dis Obstet Gynecol. 1999; 7(4): 195-8. PubMed | Google Scholar
5. Chang J, Streitman D. Physiologic adaptations to pregnancy. Neurol Clin. 2012 Aug; 30(3): 781-9. PubMed | Google Scholar
6. Ochama P. Testing for Helicobacter pylori status in patients undergoing diagnostic endoscopy in Mulago and Nsambya Hospitals, Dissertation. Kampala: Makerere University. 2001. Google Scholar
7. Newton R, Ziegler JL, Casabonne D, Carpenter L, Gold BD, Owens M, Beral V, Mbidde E, Parkin DM, Wabinga H, Mbulaiteye S, Jaffe H. Helicobacter pylori and cancer among adults in Uganda. Infect Agent Cancer. 2006 Nov 7; 1: 5. PubMed | Google Scholar

8. Baingana RK, Enyaru JK and Davidsson L. Helicobacter pylori infection in pregnant women in four districts of Uganda: role of geographic location, education and water sources. BMC Public Health. 2014 Sep 4; 14: 915. PubMed | Google Scholar

9. Institute of Medicine and National Research Council Committee to Reexamine IOMPWG. The National Academies Collection: reports funded by National Institutes of Health. In: Rasmussen KM, Yaktine AL, eds. Weight gain during pregnancy: reexamining the guidelines. Washington (DC): National Academies Press (US) National Academy of Sciences. 2009; 241-80. Google Scholar

10. Forsum E, Lof M, Olausson H, Olhager E. Maternal body composition in relation to infant birth weight and subcutaneous adipose tissue. Br J Nutr. 2006 Aug; 96(2): 408-14. PubMed | Google Scholar

11. Maddah M, Karandish M, Mohammadpour-Ahranjani B et al. Social factors and pregnancy weight gain in relation to infant birth weight: a study in public health centers in Rasht, Iran. Eur J Clin Nutr. 2005 Oct; 59(10): 1208-12. PubMed | Google Scholar

12. Dietz P, Callaghan W, Cogswell M, Morrow B, Ferre C, Schieve L. Combined effects of prepregnancy body mass index and weight gain during pregnancy on the risk of preterm delivery. Epidemiology. 2006 Mar; 17(2): 170-7. PubMed | Google Scholar

13. Stotland NE, Cheng YW, Hopkins LM, Caughey AB. Gestational weight gain and adverse neonatal outcome among term infants. Obstet Gynecol. 2006 Sep; 108(3 Pt 1): 635-43. PubMed | Google Scholar

14. Johansson K, Hutcheon AJ, Stephansson O, Cnattingius S. Pregnancy weight gain by gestational age and BMI in Sweden: a population-based cohort study. Am J Clin Nutr. 2016 May; 103(5): 1278-84. PubMed | Google Scholar

15. Poveda GF, Carrillo KS, Monje ME, Cruz CA, Cancino AG. Helicobacter pylori infection and gastrointestinal symptoms on Chilean pregnant women. Rev Assoc Med Bras (1992). 2014 Jul; 60(4): 306-10. PubMed | Google Scholar

16. Mansour GM, Nasaht EH. Role of Helicobacter pylori in the pathogenesis of hyperemesis gravidarum. Arch Gynecol Obstet. 2011 Oct; 284(4): 843-7. PubMed | Google Scholar

17. Nanbaksh F, Mohaddes H, Bahadory F, Amirfakhrian J, Mazloomi P. Comparison of Helicobacter Pylori Infection between Pregnant Women with Hyperemesis Gravidarum and Controls. World Applied Sci J. 2013; 28(12): 1918-1922. Google Scholar

18. Golalipour MJ, Sedehi M, Qorbani M. Does maternal Helicobacter pylori infection increase the risk of occurrence of neural tube defects in newborns in Northern Iran?. Neurosciences (Riyadh). 2012 Jul; 17(3): 219-25. PubMed | Google Scholar

19. Cardaropoli S, Rolo A, Piazzese A et al. Helicobacter pylori's virulence and infection persistence define pre-eclampsia complicated by fetal growth retardation. World J Gastroenterol. 2011 Dec 21; 17(47): 5156-65. PubMed | Google Scholar

20. Eslick GD, Yan P, Xia HH, Murray H, Spurrett B, Talley NJ. Foetal intrauterine growth restrictions with Helicobacter pylori infection. Aliment Pharmacol Ther. 2002 Sep; 16(9): 1677-82. PubMed | Google Scholar

21. Jang EJ, Park SW, Park JS, Park SJ, Hahm KB. The influence of the eradication of Helicobacter pylori on gastric ghrelin, appetite, and body mass index in patients with peptic ulcer disease. J Gastroenterol Hepatol. 2008 Dec; 23 Suppl 2: S278-85. PubMed | Google Scholar

22. Nwokolo CU, Freshwater DA, O'Hare P, Randeva HS. Plasma ghrelin following cure of Helicobacter pylori. Gut. 2003 May; 52(5): 637-40. PubMed | Google Scholar

23. Tschöp M, Smiley DL, Heiman ML. Ghrelin induces adiposity in rodents. Nature. 2000 Oct 19; 407(6806): 908-13. PubMed | Google Scholar
24. Murray CD, Kamm MA, Bloom SR, Emmanuel AV. Ghrelin for the gastroenterologist: history and potential. Gastroenterology. 2003 Nov; 125(5): 1492-502. PubMed | Google Scholar

25. Cummings DE. Helicobacter pylori and ghrelin: interrelated players in body-weight regulation?. Am J Med. 2004 Sep 15; 117(6): 436-9. PubMed | Google Scholar

26. Blaser MJ, Atherton JC. Helicobacter pylori persistence: biology and disease. J Clin Invest. 2004 Feb; 113(3): 321-33. PubMed | Google Scholar

27. World Health Organization. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. Geneva. 1995. Google Scholar

28. Abrams B, Selvin S. Maternal weight gain pattern and birth weight. Obstet Gynecol. 1995 Aug; 86(2): 163-9. PubMed | Google Scholar

29. Strauss RS, Dietz WH. Low maternal weight gain in the second or third trimester increases the risk for intrauterine growth retardation. J Nutr. 1999 May; 129(5): 988-93. PubMed | Google Scholar

30. Paulino DSM, Surita GF, Peres GB, Nascimento SL, and Morais SS. Association between parity, pre-pregnancy body mass index and gestational weight gain. J Matern Fetal Neonatal Med. 2016 Mar; 29(6): 880-4. Google Scholar

31. Lumbanraja S, Lutana D, Usmana I. Maternal weight gain and correlation with birth weight infants. Procedia - Social and Behavioral Sciences. 2013; (103): 647–656. Google Scholar

32. Haugen M, Brantsæter AL, Winkvist A, Lissner L, Alexander J, Oftedal B, Meltzer HM. Associations of pre-pregnancy body mass index and gestational weight gain with pregnancy outcome and postpartum weight retention: A prospective observational cohort study. BMC Pregnancy Childbirth. 2014 Jun 11; 14: 201. PubMed | Google Scholar

33. Ashley-Martin J & Woolcott C. Gestational weight gain and postpartum weight retention in a cohort of Nova Scotian women. Matern Child Health J. 2014 Oct; 18(8): 1927-35. PubMed | Google Scholar

34. Terada M, Matsuda Y, Ogawa M, Matsui H, Satoh S. Effects of maternal factors on birth weight in Japan. J Pregnancy. 2013; 2013: 172395. PubMed | Google Scholar

35. Chihara I, Hayes DK, Chock LR, Fuddy LJ, Rosenberg DL, Handler AS. Relationship between gestational weight gain and birth weight among clients enrolled in the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC), Hawaii, 2003-2005. Matern Child Health J. 2014 Jul; 18(5): 1123-31. Google Scholar

36. Shrestha I, Sunuwar L, Bhandary S, Sharma P. Correlation between gestational weight gain and birth weight of the infants. Nepal Med Coll J. 2010 Jun; 12(2): 106-9. PubMed | Google Scholar

37. Zeal C, Remington P, Ndiaye M et al. The epidemiology of maternal overweight in Dane County, Wisconsin. WMJ. 2014 Feb; 113(1): 24-7. PubMed | Google Scholar

38. Scott A, Ejikeme SC, Clotey NE and Thomas GJ. Obesity in sub-Saharan Africa: development of an ecological theoretical framework. Health Promot Int. 2013 Mar; 28(1): 4-16. PubMed | Google Scholar

39. Olson CM, Strawderman MS. Modifiable behavioral factors in a biopsychosocial model predict inadequate and excessive gestational weight gain. J Am Diet Assoc. 2003 Jan; 103(1): 48-54. PubMed | Google Scholar

40. Heery E, Kelleher CC, Wall PG, McAuliffe FM. Prediction of gestational weight gain – a biopsychosocial model. Public Health Nutr. 2015 Jun; 18(8): 1488-98. PubMed | Google Scholar

41. Pickett EK, Abrams B, Selvin S. Maternal Height, Pregnancy Weight Gain, and Birth weight. Am J Hum Biol. 2000 Sep;12(5):682-687. PubMed | Google Scholar
42. Nohr EA, Vaeth M, Baker JL, Sorensen TI, Olsen J, Rasmussen KM. Pregnancy outcomes related to gestational weight gain in women defined by their body mass index, parity, height, and smoking status. Am J Clin Nutr. 2009 Nov; 90(5): 1288-94. PubMed | Google Scholar

43. Bodnar LM, Hutcheon JA, Platt RW, Himes KP, Simhan HN, Abrams B. Should gestational weight gain recommendations be tailored by maternal characteristics?. Am J Epidemiol. 2011 Jul 15; 174(2): 136-46. PubMed | Google Scholar

44. Woodburn PW, Muhangi L, Hillier S, Ndibazza J, Namujju PB et al. Risk Factors for Helminth, Malaria, and HIV Infection in Pregnancy in Entebbe, Uganda. PLoS Negl Trop Dis. 2009 Jun 30; 3(6): e473. PubMed | Google Scholar

45. De Beaudrap P, Turyakira E, White LJ, Nabasumba C, Tumwebaze B et al. Impact of malaria during pregnancy on pregnancy outcomes in a Ugandan prospective cohort with intensive malaria screening and prompt treatment. Malar J. 2013 Apr 24; 12: 139. PubMed | Google Scholar
### Table 1: Socio-demographics characteristics of participants

| Variable (N= 192)                  | Number (%)   |
|------------------------------------|--------------|
| **Parity**                         |              |
| Primigravidae                      | 132 (68.8)   |
| Secundigravidae                    | 60 (31.2)    |
| **Maternal H. pylori infection status** |          |
| H. pylori positive                 | 110 (57.3)   |
| H. pylori negative                 | 82 (42.7)    |
| **Occupation**                     |              |
| House wife                         | 144 (75)     |
| Peasant                            | 1 (0.5)      |
| Employee                           | 38 (19.8)    |
| Student                            | 9 (2.7)      |
| **Smoking**                        |              |
| Yes                                | 2 (1.0)      |
| No                                 | 190 (99.0)   |
| **Alcohol**                        |              |
| Yes                                | 10 (5.2)     |
| No                                 | 182 (94.8)   |
| **Marital status**                 |              |
| Married                            | 168 (87.5)   |
| Widowed                            | 2 (1.0)      |
| Divorced/Separated                 | 3 (1.6)      |
| Single                             | 19 (9.9)     |
| **Water source**                   |              |
| Tap/Borehole                       | 162 (84.4)   |
| Protected well                     | 28 (14.6)    |
| Tank (Harvested rain water)        | 1 (0.5)      |
| Unprotected well                   | 1 (0.5)      |
| **Building type**                  |              |
| Permanent                          | 190 (99.0)   |
| Temporary                          | 2 (1.0)      |
| **Household monthly income ($)**   |              |
| Low income (<100)                  | 94 (49.0)    |
| Medium income (101-250)            | 87 (45.3)    |
| High income (> 250)                | 11 (5.7)     |
| **Education level**                |              |
| Low (No education to primary 7)    | 42 (21.9)    |
| Medium (Secondary level)           | 124 (64.6)   |
| High (tertiary education)          | 26 (13.5)    |
| **Maternal pre-pregnancy (BMI kg/m²)** |          |
| Underweight (<18.5)               | 28 (14.6)    |
| Normal weight (18.5-24.9)          | 143 (74.5)   |
| Overweight (25.0-29.9)             | 21 (10.9)    |
## Table 2: Mean±sd values of some of the participants’ characteristics

| Variable                                      | Mean±sd | Min-Max  |
|-----------------------------------------------|---------|----------|
| Age (years)                                   | 20.9±2.7| 18–35    |
| Gestational age at recruitment (weeks)        | 16.9±1.5| 12–18    |
| Pre-pregnancy weight (kg)                     | 53.1±7.6| 37–76    |
| Weight at recruitment (kg)                    | 56.9±8.1| 38–82    |
| Maternal pre-pregnancy BMI (kg/m²)            | 21.3±2.7| 15.0–29.4|
| Maternal height (cm)                          | 157.4±5.7| 142.0–173.1|
| Rate of GWG during second trimester (grams/week) | 306.6±100.9 | 80–750 |
| Rate of GWG during third trimester (grams/week) | 294.3±101.4 | 100–600 |
| Average rate of GWG during 2nd & 3rd trimesters (grams/week) | 300.5±79.7 | 90–630 |
| Gestational weight gained by 36 weeks of gestation (kg) | 9.6±2.2 | 5.0–16 |
| Gestational age at delivery (weeks)           | 39.4±1.0 | 37–42 |

sd = standard deviation, Min = minimum, Max = maximum

## Table 3: Mean±sd values selected variables in relation to *H. pylori* infection status

| Variable                                      | Mean±sd by *H. Pylori*                                                                 | P-value |
|-----------------------------------------------|----------------------------------------------------------------------------------------|---------|
|                                              | *H. pylori*-ve (n=82)                                                                | *H. pylori*+ve (n=110) |         |
| Pre-pregnancy weight (kg)                     | 52.6±7.0                                                                              | 53.5±8.1 | 0.392   |
| Maternal pre-pregnancy BMI (kg/m²)            | 21.1±2.5                                                                              | 21.6±2.9 | 0.352   |
| Maternal height (cm)                          | 157.2±5.9                                                                              | 157.5±5.6 | 0.727   |
| Rate of GWG during second trimester           | 322.3±97.4                                                                             | 294.9±102.4 | 0.063   |
| Rate of GWG during third trimester            | 311.6±103.7                                                                            | 281.4±98.1 | 0.041   |
| Rate of GWG (second & third trimesters)       | 317.0±74.1                                                                             | 288.2±81.8 | 0.013   |
| (grams/week)                                  |                                                                                       |         |         |
| Gestation weight gained by 36 weeks (kg)      | 10.1±2.3                                                                               | 9.2±2.1  | 0.002   |
| Gestation age at delivery                     | 39.4±1.1                                                                               | 39.3±1.0 | 0.50    |

sd = standard deviation, n = number, -ve = negative, +ve = positive
Table 4: Factor independently associated with GWG during the second and third trimesters

| Variable                        | Standardized Coefficients (Beta) | P value | 95% confidence interval |
|---------------------------------|----------------------------------|---------|-------------------------|
| Parity                          | -0.158                           | 0.023   | -0.051 – -0.004         |
| H. pylori infection             | -0.192                           | 0.006   | -0.053 – -0.009         |
| Maternal pre-pregnancy BMI      | -0.142                           | 0.037   | -0.008 – -0.000         |
| Maternal height                 | 0.156                            | 0.022   | -0.000 – -0.004         |
| Household monthly income        | -0.207                           | 0.003   | -0.023 – -0.005         |