Prevalence of *Helicobacter pylori* Infection in Children in the Bahamas

F Carter[1], T Seaton[2], Y Yuan, D Armstrong[2]

ABSTRACT

Objective: *Helicobacter pylori* (*H pylori*) is a common bacterial infection that is associated with significant morbidity and mortality worldwide. This bacterium causes a chronic infection that is causally related to illnesses ranging from gastritis, peptic ulcer disease to gastric cancer. It is generally considered that it is acquired in childhood but the prevalence varies considerably between countries and communities. There are few data on the prevalence of *H pylori* in the Caribbean and none on the prevalence of *H pylori* in children in the Bahamas. The aim of this pilot study was to determine the prevalence of *H pylori* infection in a cohort of school children in the Bahamas.

Methods: One hundred and sixty-one children attending a public primary school in the Bahamas were invited to participate in this study. Consent was obtained for 107 children and each participant completed a brief questionnaire. Valid data were available for 96 of these children. Active *H pylori* infection was determined using the 13C urea breath test (UBT).

Keywords: Children, Bahamas, helicobacter pylori, prevalence, urea breath test

[1]The University of the West Indies, Nassau, Bahamas and [2]Division of Gastroenterology, McMaster University, Hamilton, Ontario, Canada.

Correspondence: Dr FP Carter, Apt 404, #35 Brock St Hamilton, Ontario, Canada L8l 4L7
E-mail: flloydcarter@hotmail.com

Results: Fifty-two children tested positive for *H pylori,* yielding a prevalence of 54.2%. The median age in the study was nine years with 46.9% male and 53.1% female. No significant relationship was found between gender, breastfeeding, pets and *H pylori* status.

Conclusion: The prevalence reported in this study is the highest reported in asymptomatic children in the Caribbean. Further studies are required to determine risk factors for acquisition of *H pylori* infection in this population.

INTRODUCTION

The Bahamas is an archipelago of over 700 islands and keys. Population estimate for the Bahamas in 2007 was 305 655 and almost 60% of the population lives on New Providence, which includes the capital city of Nassau. Bahamians are mainly of African descent; 85% of the population is black and 15% white or mixed. There is a paucity of medical literature for this population in the Caribbean. The prevalence of *H pylori* varies depending on age, socio-economic status and country of origin (1). Acquisition of this organism occurs most commonly during the childhood years; it is therefore essential that studies in this population be done.

Understanding the risk factors for acquisition of this infection is essential to reducing the prevalence and complications of the infection. There is no doubt that a low socio-economic status is a strong risk factor for *H pylori* acquisition. This may explain the higher rates seen in the developing countries. Low socio-economic status, with its attendant household overcrowding and poor sanitation, create an environment that favours *H pylori* transmission (2−6).

H pylori is transmitted by person-to- person; transmission is widely believed to be faeco-oral, but other mechanisms may also be involved (5−7). *H pylori* transmission may be water-borne (8) so the source of water supply may be an important risk factor for *H pylori* infection in certain regions such as Peru (9). Indeed, the water source was noted to be more important than socio-economic status for *H pylori* acquisition in a Peruvian population (10). Data from Canadian First Nations peoples in Manitoba and Northern Alberta also suggest water-borne transmission (11).

A review, in 2009, of 14 epidemiological studies evaluating this relationship, reported an odds ratio (OR) < one in nine studies and an OR > one in only one study (12). The weight of evidence, therefore, suggests a protective effect from breastfeeding although this is not conclusive.

Prevalence rates in adult populations range from 8% to 80% (13, 14), whilst those in children range from 5.4% to 62% (15). The prevalence of *H pylori* in the Caribbean is quite varied but there are no data for the Bahamas. A questionnaire was completed by participants in the present study to identify possible risk factors for *H pylori* infection in the Bahamas.

Helicobacter pylori infection and its complications pose a significant healthcare problem in developing countries. The present study is the first to evaluate the prevalence of *H pylori* in Bahamian children.

SUBJECTS AND METHODS

The study population was a public primary school in Nassau, Bahamas. This school was chosen at random from a list of all public primary schools in Nassau. Children attending this school were between five and 12 years old (Grades 1−6). They were all of African descent, matching the general school population. Study sampling was not random, but depended on parents or guardians giving written consent and answering a brief questionnaire (Appendix 1). All students in grades 4−6 were approached to participate in the study. One hundred and sixty-one children were invited to participate in the study, of whom 107 (66%) returned consent forms signed by a parent or legal guardian. The study protocol was approved by the Ethics committee of the Public Hospitals Authority, Nassau, Bahamas. The Ministry of Education gave their permission to allow the study to be conducted in one of their schools.

The ^{13}C urea breath test (UBT) [Helikit, Isotechnica Diagnostics, Canada] was administered to assess for infection with *H pylori*. All children were tested, on the same day, in their school, after an overnight fast. Two breath samples were collected, one before and the second 30 minutes after the urea test drink. The ^{13}C UBT involved the ingesting of 100 ml of an odourless, colourless citrus-flavoured liquid that contained 75 mg of ^{13}C-labelled urea. The samples were obtained by having the children gently exhale through a plastic straw, with its distal tip placed in the bottom of a 10 ml glass tube. The tube was sealed with a stopper immediately after exhalation. The breath samples were stored in collection tubes at room temperature. The samples were packaged and shipped to McMaster University Medical Centre where the analysis was performed. The samples were analysed by isotopic ratio mass spectrometry (BreathMAT, Finnigan MAT Gmbh, Germany). Based on prior validation, the cutoff for a positive result was a delta over baseline (DOB) of ≥ 3.0 (16, 17).

Categorical variables were compared using the Chi-square test or the Fisher's exact test when expected cell sizes were less than five, and continuous variables were compared using the *t*-test, between the *H pylori* positive and *H pylori* negative group. Seven clinically relevant variables (age, gender, number of other children, number of adults, number of pets, drinking water source and breastfeeding) were selected into the multivariable logistic regression model to estimate the odds ratio associated with each variable for the outcome (*H pylori* positivity) adjusting for other confounders. Adjusted ORs (AOR) and 95% confidence interval (CI) were calculated for each study variable. Missing values were not imputed but excluded in the corresponding analysis. Statistical analysis was performed by using the SPSS version 18 (IBM, New York, USA). All tests of significance were two-tailed, and a *p*-value of 0.05 was considered to indicate statistical significance.

RESULTS

Of the 107 subjects who were eligible, nine were excluded because of recent confirmed or possible antibiotic use and two were excluded because the ^{13}C UBT breath samples were insufficient for analysis. Overall, valid data were available for 96 subjects (51 [53.1%] females, 45 [46.8%] males) with a median age of nine years (min–max: 6–12 years) and a mean age of 9.3 years with a normal distribution (Table 1).

Overall, 52 (54.2%) subjects tested positive for *H pylori* (Figure); there was no relationship between *H pylori* positivity and age (AOR 0.8, 95% CI 0.5, 1.2) and gender (AOR 0.8, 95% CI 0.3, 2.1). Breastfeeding, reported by 61 (67.8%) subjects, conferred no protection against *H pylori* infection in this study (AOR 1.3, 95% CI 0.5, 3.4).

No statistically significant relationships were noted between *H pylori* infection and the presence of pets at home (AOR 0.8, 95% CI 0.3, 2.2) or the number of other children (estimated average 3.6 ± 1.2; median 4) or adults living in the household. (AOR 0.9, 95% CI 0.6, 1.4; AOR 1.1, 95% CI 0.8, 1.7, respectively).

Over 86% of participants reported using bottled water as their main source of drinking water. Additional sources of drinking water were only reported by 11 children in total. Compared to bottled water, none of the other three drinking sources (well-water, city-water, bottled and city water) was significantly associated with *H pylori* seropositivity (Table 2).

DISCUSSION

H pylori is a common pathogen worldwide and is thought to be one of the most common chronic bacterial infections (18, 19). The prevalence of *H pylori* infection in Bahamian children in this study was 54.2%. Seroprevalence rates for children in the Caribbean range from 14% in Guyana (17), 22% in Barbados (21) and 27% in Jamaica (22). The prevalence reported in our study is the highest reported in the Caribbean in asymptomatic children. Sharon *et al* reported 62% prevalence in symptomatic Haitian children (23). Urea breath test in a Chinese population revealed a prevalence between 41 and 54% in their children (24). A Canadian study reported a low prevalence of 7.1% in children with upper gastrointestinal symptoms (25). A prevalence of 32% was reported in asymptomatic children in Texas in 2002 (26). The prevalence reported in this study is much higher than that in other Caribbean territories despite the fact that Nassau, Bahamas, has one of the highest standards of living in the region (27).

Although prevalence rates have been shown to be inversely related to socio-economic status, other factors may play a role. Genetic susceptibility may influence prevalence rates. Malaty *et al* in 1994 demonstrated a high concordance in monozygotic twins reared apart. This suggests that genetics played a role in determining who will be infected and this may, ultimately, affect prevalence rates (28).

In addition to genetics, the extent of antibiotic use in children may differ between territories and this can influence the rate of clearance of *H pylori* infection from a population (29). Unfortunately no data exist on antibiotic prescribing practices in the Caribbean territories that would allow us to test this postulate.

The present study was completed in October 2008, almost a decade after the other Caribbean prevalence studies. It is possible that the higher prevalence rate reported in this study represents a shift in prevalence rate in the Caribbean.

A comparison between *H pylori* prevalence in children and adults points to an alternative explanation. Reported prevalence rates in children (C) and adults (A): Jamaica [27%C/70%A] (22), Barbados [22%C/72%A] (21) and Bahamas (54%C/58%A). Jamaica and Barbados demonstrated the usual cohort effect seen in *H pylori* prevalence studies. However, no cohort effect was demonstrated in the available data from the Bahamas. It is also possible that this particular school or neighbourhood has a higher prevalence than the general population due to factors already discussed (genetic susceptibility, antibiotic use and environmental factors). Additional studies are needed to confirm the high prevalence in other settings and in the adult population in the Bahamas.

There was no difference between genders with respect to the prevalence of *H pylori* infection. This finding is consistent with present consensus that no significant gender differences exist. The prevalence of *H pylori* was not associated with having pets at home, confirming that there is little evidence to support zoonotic transmission in *H pylori* infection (30, 31). Therefore, it appears that humans are the primary reservoir of *H pylori.* A new postulate to be considered is that human to animal spread (reverse zoonosis/anthroponosis) may be more likely than the reverse (32).

One unexpected result in the present study was the absence of a significant relationship between the number of adults or children in the household and *H pylori* infection. Overcrowding indicators such as number of persons or children in the home, number of persons per room, sharing a room or bed have been consistently related to *H pylori* positivity (2−5). It is likely that the present study was underpowered to detect a significant relationship with these factors.

It is possible that the small sample size might have prevented the detection of a significant benefit in breastfeeding (12, 32). However, in the authors' opinion, the relationship between breastfeeding and *H pylori* may be related to feeding practices associated with breastfeeding rather than the actual breast milk constituent. Feeding practices such as personal hygiene and environmental sanitation are established risk factors to *H pylori* acquisition.

The study has several limitations. It was a non-randomized study from one school. This small pilot study was not powered to confidently identify risk factors associated with *H pylori* infection. The population studied in this school is quite similar to other public primary schools in Nassau in terms of ethnicity and economic status. Unfortunately, no economic data were collected in this study. However, the public schools in Nassau are composed of children mainly from low or middle income homes. A small but important subset of children, likely of higher income families, is enrolled in private schools. It is therefore possible that the results may not represent the entire school-aged population in Nassau, Bahamas.

The study demonstrates the utility and acceptability of UBT testing in children. This form of testing is preferred over serological tests as it allows determination of active *H pylori* infection.

The high prevalence reported in Bahamian children in this study has significant healthcare implications. These children are at risk for peptic ulcer disease and gastric carcinoma. Working with the Bahamian government, additional populations need to be tested. The effectiveness of current eradication regimens in this region need to be assessed. Additional studies are required to determine the antibiotic sensitivity of *H pylori* in this region. The cost effectiveness of a population based *versus* an opportunistic test and treat programme needs to be determined.

In summary, the study reports a high prevalence of *H pylori* infection in Bahamian children. This is the highest reported prevalence in asymptomatic children in the Caribbean. It is imperative that we evaluate possible genetic and other risk factors in this population. A reassessment of current prevalence rates in other Caribbean territories is also warranted.

ACKNOWLEDGEMENTS

The authors thank Cassandra Forbes and her team of teachers at the TG Glover Primary School for their invaluable assistance in conducting this study. We also thank Melanie Wolfe for analysing the ^{13}C-UBT specimens. We also thank the medical students and DM residents at The University of the West Indies, Nassau for their invaluable assistance.

REFERENCES

1. Graham DY, Malaty HM, Evans DG, Evans DJ Jr, Klein PD, Adam E. Epidemiology of *Helicobacter pylori* in an asymptomatic population in the United States. Effect of age, race and socio-economic status. Gastroenterology 1991; **101:** 1495−1501.
2. Fiedorek SC, Malaty HM, Evans DL, Pumphrey CL, Casteel HB, Evan DJ Jr et al. Factors influencing the epidemiology of *Helicobacter pylori* infection in children. Pediatrics 1991; **88:** 578−82.
3. Malaty HM, Graham DY. Importance of childhood socio-economic status on current prevalence of *Helicobacter pylori* infection. Gut 1994; **35:** 742−5.
4. McCallion WA, Murray LJ, Bailie AG, Dalzell AM, O'Reilly DP, Bamford KB. *Helicobacter pylori* infection in children: relation with current household living conditions. Gut 1996; 39: 18−21.
5. Megraud F. Transmission of Helicobacter pylori: faecal-oral versus oral-oral route. Aliment Pharmacol Ther 1995; **9 (Suppl 2):** 85−91.
6. Cave DR. Transmission and epidemiology of Helicobacter pylori. Am J Med 1996; **100 (Suppl 5A)**, 12S−17S; discussion 17S−18S.
7. Osato MS, Ayub K, Le HH. Houseflies are an unlikely reservoir or vector of Helicobacter pylori. J Clin Microbiol 1998; **36:** 2786−8.
8. Travis PB, Goodman KJ, O'Rourke KM, Groves FD, Sinha D, Nicholas JS et al. The association of drinking water quality and sewage disposal with *Helicobacter pylori* incidence in infants: the potential role of water-borne transmission. J Water Health 2010; **8:** 192−203.
9. Hulten K, Han SW, Enroth H, Klein PD, Opekun AR, Gilman RH et al. *Helicobacter pylori* in the drinking water in Peru. Gastroenterology 1996; **110:** 1031−5.

10. Klein PD, Graham DY, Gaillour A, Opekun AR, Smith EO. Water source as risk factor for *Helicobacter pylori* infection in Peruvian children. Gastrointestinal Physiology Working Group. Lancet 1991; **337:** 1503–6.

11. McKeown I, Orr P, Macdonald S, Kabani A, Brown R, Coghlan G et al. *Helicobacter pylori* in the Canadian arctic: seroprevalence and detection in community water samples. Am J Gastroenterol 1999; **94:** 1823−9.

12. Chak E, Rutherford G, Steinmaus C. The role of breastfeeding in the prevention of *Helicobacter pylori* infection: a systematic review. Clin Infect Dis 2009; **48:** 430−7.

13. Pounder RE, Ng D. The prevalence of *Helicobacter pylori* infection in different countries. Aliment Pharmacol Ther 1995; **9 (Suppl 2)**: 33−9.

14. Morris-Brown L. Helicobacter pylori: epidemiology and routes of transmission. Epidemiol Rev 2000; **22:** 283−97.

15. Neale KR, Logan RP. The epidemiology and transmission of *Helicobacter pylori* infection in children. Aliment Pharmacol Ther 1995; **9 (Suppl 2)**: 77−84.

16. Atherton J, Spiller RC. The urea breath test for helicobacter pylori. Gut 1994; **35:** 723−5.

17. Mauro M, Radovic V, Zhou P, Wolfe M, Kamath M, Bercik P et al. 13C urea breath test for Helicobacter pylori: determination of the optimal cut off point in a Canadian community population. Can J Gastroenterol 2006; **20:** 770−4.

18. Go MF, Vakil N. *Helicobacter pylori* infection. Clin Perspective Gastroenterology 1999; **2:** 141−53.

19. Peura DA. The report of the Digestive Health Initiative international update conference on Helicobacter pylori. Gastroenterology 1997; **113:** S4−S8.

20. Charles C. Personal Communication; 1999.

21. Edwards C, Douglin CN, Prussia CP. Epidemiology of *Helicobacter pylori* infection in Barbados. West Indian Med J 1997; **46:** 3−7.

22. Lindo JF, Lyn-Sue AE, Palmer CJ. Seroepidemiology of *Helicobacter pylori* infection in a Jamaican community. Tropical Med Int Health 1990; **4:** 862−6.

23. Sharon CK, Dana MG, Sepulved A. Chronic abdominal complaints and *Helicobacter pylori* in a Haitian population. Journal of Healthcare for the Poor and Underserved 2004; **15:** 183−92.

24. Cheng H, Hu F, Zhang L, Yang G. Prevalence of *Helicobacter pylori* infection and identification of risk factors in rural and urban Beijing. Helicobacter 2009; **14:** 128−33.

25. Segal I, Otley A, Issenman R, Armstrong D, Espinosa V. Low prevalence of *Helicobacter pylori* infection in Canadian children: a cross-sectional analysis. Can J Gastroenterol 2008; **22:** 485−9.

26. Malaty HM, Haveman T, Graham DY. *Helicobacter pylori* infection in asymptomatic children: impact of epidemiologic factors on accuracy of diagnostic tests. J Pediatr Gastroenterol Nutr 2002; **35:** 39−63.

27. United Nations Development Programme. Human Development report 2007/2008. Fighting climate change: human solidarity in a divided world. New York: United Nations Development Programme; 2001: 229.

28. Malaty HM, Engstrand L, Pedersen NL, Graham DY. *Helicobacter pylori* infection: genetic and environmental influences. A study of twins. Ann Internal Med 1994; **120:** 982−6.

29. Malaty HM, Graham DY, Wattigney WA, Srinivasan SR, Osato M, Berenson GS. Natural history of *Helicobacter pylori* infection in childhood: 12-year follow-up cohort study in a biracial community. Clin Infect Dis 1999; **28:** 279−82.

30. Neiger R, Simpson KW. Helicobacter infection in dogs and cats: facts and fiction. J Vet Intern Med 2000; **14:** 125−33.

31. El-Zaatari FA, Woo JS, Badr A. Failure to isolate *Helicobacter pylori* from stray cats may be an anthroponosis−an animal infection with a human pathogen. J Med Microbiol 1997; **46:** 372−6.

32. Rothenbacher D, Bode G, Brenner H. History of breastfeeding and *Helicobacter pylori* infection in pre-school children: results of a population based study in Germany. Int J Epidemiol 2002; **31:** 632−7.

Table 1: Summary of results

	Total n	***H pylori* +ve n (%)**	***H pylori* –ve n (%)**
All subjects	96	52 (54.2)	44 (44.8)
Gender			
Male	45	25 (48.1)	20 (45.5)
Female	51	27 (51.9)	24 (54.5)
Breastfed[a]			
Yes	61	36 (72.0)	25 (62.5)
No	29	14 (28.0)	15 (37.5)
Pet owners[b]			
Yes	31	17 (33.3)	14 (31.8)
No	64	34 (66.7)	30 (68.2)
# Other children living in the home[c]			
None	5	2 (3.8)	3 (7)
One	15	10 (19.2)	5 (11.6)
Two	22	11 (21.2)	11 (25.0)
Three	22	11 (21.2)	11 (25.0)
Four or more	30	17 (32.7)	13 (29.5)
# Adults living in the home[d]			
One	12	8 (15.4)	4 (9.1)
Two	46	22 (42.3)	24 (54.5)
Three	14	7 (13.5)	7 (15.9)
Four	13	7 (13.5)	6 (13.6)

Five or more	8	6 (11.5)	2 (4.5)
Source of drinking water[e]			
Bottled	83	42 (80.8)	41 (93.2)
Well-water	2	1 (1.9)	1 (2.3)
City-water	6	5 (9.6)	1 (2.3)
Bottled and city	3	3 (5.8)	0

a. 6 missing; b. 1 missing; c. 2 missing; d. 3 missing; e. 2 missing

Table 2: Logistic regression model with AOR estimates and 95% CIs for *Helicobacter pylori* seropositivity

Variable	**Adjusted OR (95% CI)**	***p*-value**
Age	0.8 (0.5, 1.2)	0.30
Gender	0.8 (0.3, 2.1)	0.70
Number of other children living in the home	0.9 (0.6, 1.4)	0.71
Number of adults living in the home	1.1 (0.8, 1.7)	0.53
Pet owner	0.8 (0.3, 2.2)	0.67
Breastfed	1.3 (0.5, 3.4)	0.65
Drinking well-water *	0.8 (0.04, 14.0)	0.86
Drinking city water*	5.2 (0.6, 49.0)	0.15
Drinking bottled and city water	1.0 (0.0, inf)	0.999

*Compared to bottled water. The effect of each variable was adjusted for those of the other independent predictors in the model.

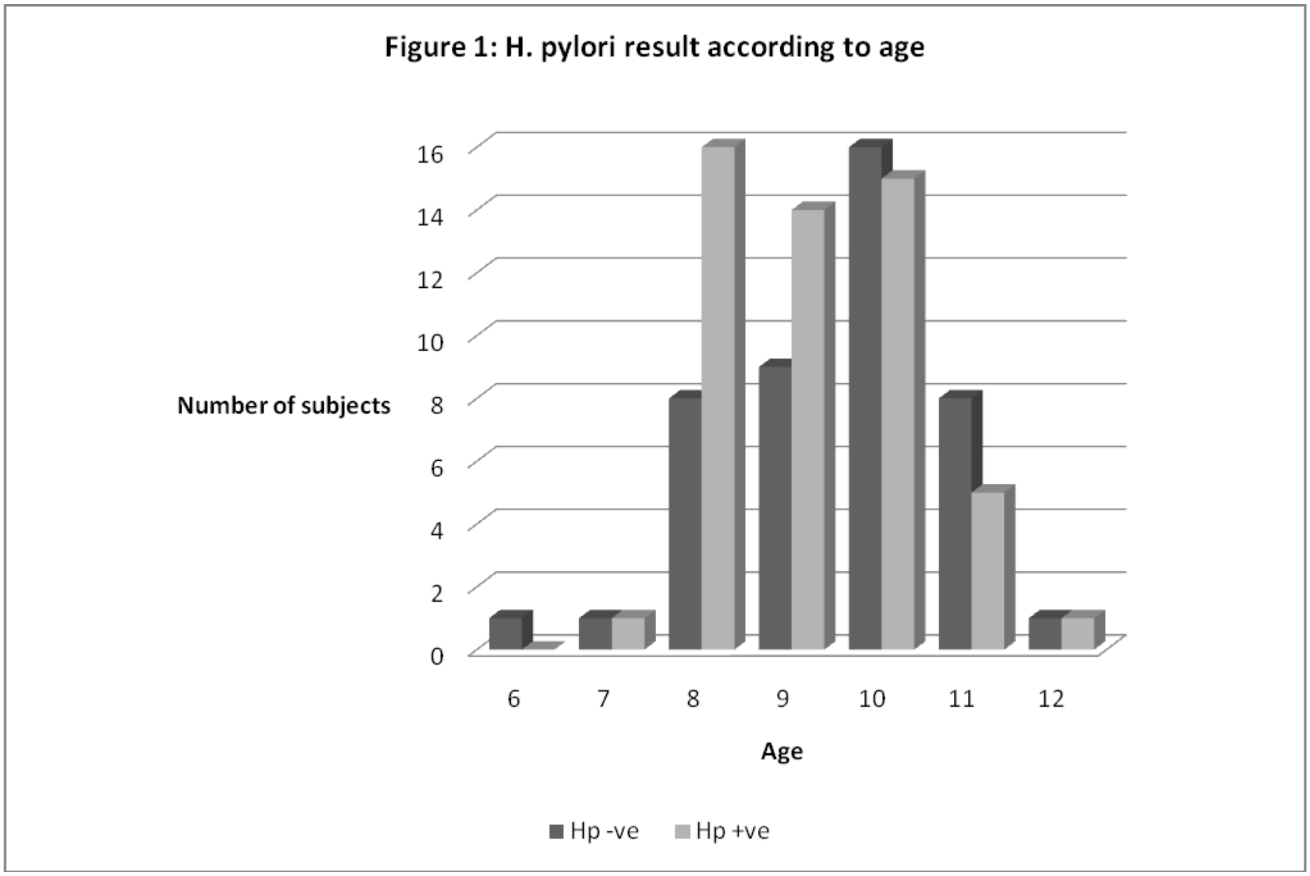

Figure 1: Distribution of *Helicobacter pylori* results according to age

Appendix 1: Questionnaire

***Helicobacter pylori* in School-Aged Children**

Surname: ……………..…… **Given Name:** …………………………..
Age: ………. **D.O.B.** (dd/mm/yy) ……/………/………..
Height: ………. cm **Weight:** …………….. kg
Gender: Male □ Female □
Address: ……………………………………………..
……………………………………………..
Home telephone number …………………………….
Parent's e-mail address: ……………………………….

Number of other children living in the home
0 □ 1 □ 2 □ 3 □ ≥4 □
Number of adults living in the home
1 □ 2 □ 3 □ 4 □ ≥5 □
Number of bedrooms in home
1 □ 2 □ 3 □ ≥4 □

Do you have any pets?
Yes □ No □ If Yes, please state type ……………………
Source of drinking water
Bottled □ Well- water □ City □
Did you breast feed your child?
Yes □ No □

Does your child complain of stomach pain?
Yes □ No □
If yes
Did your child have 3 or more episodes over a three month period that was severe enough to prevent normal activities? Yes ☐ No ☐

Has your child ever been diagnosed with stomach ulcers?
Yes □ No □
Has your child used any antibiotics in the last 2 weeks?
Yes □ No □
Have you or any of your child's family ever been diagnosed with stomach ulcers?
Yes □ No □

How would you describe the General Health of your Child?
Good Health ☐ Fair Health ☐ Poor Health ☐