Epidemiology of *Helicobacter pylori* Infection in Tay Children in Vietnam

Thi Viet Ha Nguyen¹, Van Bang Nguyen¹, Thi Thanh Binh Phan², Thi Thu Hoang Ha³, Thi Lan Anh Le¹ and Dac Cam Phung³

¹Department of Pediatrics, Hanoi Medical University, National Hospital of Pediatrics, Hanoi, Vietnam  
²Department of Pediatrics, Ducgiang Hospital, Hanoi, Vietnam  
³National Institute of Hygiene and Epidemiology, Hanoi, Vietnam

**Correspondence author:** Thi Viet Ha Nguyen, Department of Pediatrics, Hanoi Medical University, 879 Lathanh Street, Dongda District, Hanoi, Vietnam, Tel: 84913555187; E-mail: vietha@hmu.edu.vn

**Received:** 22 September 2016; **Accepted:** 10 October 2016; **Published:** 14 October 2016

**Citation:** Nguyen TVH, Nguyen VB, Phan TTB, et al. Epidemiology of *Helicobacter pylori* Infection in Tay children in Vietnam. Ann Clin Lab Res. 2016, 4: 4.

**Keywords:** *Helicobacter pylori*; Children; Serology; Ethnic; Seroprevalence;

---

**Abstract**

**Background:** Differences in prevalence of *H. pylori* among racial and ethnic groups have been described worldwide. The aim of the present study was to evaluate the seroprevalence of *H. pylori* infection and factors associated with the *H. pylori* infection among Tay children and adults living in Vietnam.

**Materials and methods:** In a cross-sectional study 1,094 healthy individuals of all generations living together in the same home were selected from 278 households, based on cluster sampling of residential location between September and December 2013 in Langson province. *H. pylori* infection status was determined by serology test on samples obtained at each visit. A questionnaire was filled out at the start of the study. *H. pylori* serology data were analyzed using χ² test and logistic regression models.

**Results:** An overall *H. pylori* seroprevalence was 46.8%. *H. pylori* seroprevalence was 51.4% in adults versus 41.4% in children ≤18 years old (p<0.05). Regular hand washing after defecation and breastfeeding over 12 months were protective factors for *H. pylori* infection [OR (95%CI): 0.73, (0.39-0.92); 0.71 (0.35-0.94); respectively]. *H. pylori* infected mothers, first siblings and grandparents were found as risk factors for *H. pylori* infection in children [OR (95%CI): 2.98 (1.13-3.14), 1.4 (1.0-3.12), 1.41 (1.11-4.64); respectively]. No other factors such as size of household or sibling, infected fathers, regular sharing bed, collective life initiation and antibiotic use were found to be significant risk factors for infection.

**Conclusion:** The first community-based study in Tay population showed moderate and indifferent rates of *H. pylori* infection. Data from the present study are consistent with intra-familial *H. pylori* transmission and suggest that improvement of living conditions should be protective against infection.

---

**Introduction**

*Helicobacter pylori* (*H. pylori*) is one of the most common infections in humans, with an estimated 50% of the world’s population being infected [1]. This organism has been implicated in the pathogenesis of active and chronic gastritis, peptic ulcer and gastric cancer [1,2]. Childhood has been identified as the critical time for acquisition of *H. pylori* infection [3-6]. The exact route of transmission remains unclear but the infection clusters in families and familial spread is thought to be the major mode of transmission, both in developed and developing countries [7]. The prevalence of *H. pylori* infection in children all over the world is diverse and dependent on many factors. The highest rates *H. pylori* infection are reported in developing countries with lower socioeconomic and hygiene conditions as well as a high density of people in the household, while the lower percentage of infected children is observed in high income countries. Differences in prevalence among racial and ethnic groups have been described worldwide, but it is unclear to what extent such differences can be ascribed to socioeconomic factors and other possible risk factors [4,8]. Vietnam is the easternmost country in Southeast Asia with an estimated 90.5 million inhabitants. Vietnam is also home to 54 ethnic groups with different cultures, of which 75% to 80% living in rural or remote areas [9,10]. High rates of *H. pylori* infection was reported in both hospital-based and population-based studies. Prevalence of infection with *H. pylori* has been reported between 50% and 80% in several studies conducted in adults and around 26.0% to 71.4% in children [11-13]. Different risk factors for has been identified *H. pylori* infection in Kinh ethnic majority [11-13] and in some among other ethnic minorities [14,15]. As the socioeconomic level and lifestyle vary considerably among ethnic groups of the country, the prevalence of *H. pylori* infection and particularly risk factors for this infection were different in previous studies [11-15]. According to the general statistics office of the...
government of Vietnam 2009 [10], the Tay is the second largest ethnic group in Vietnam after the majority Kinh ethnic group with an estimated 1.7 million people. Tay people have their own habits, customs, cultural practices and economic condition that differ from those of Kinh people [10]. No data is available about prevalence of H. pylori infection and risk factors for this infection in Tay people to develop appropriate preventative measures for managing the H. pylori infection in this subpopulation in the future. The aim of the present study was to evaluate the seroprevalence of H. pylori infection and factors associated with the H. pylori infection among Tay children and adults living in the Langson province.

**Population and Methods**

**Study population**

Langson is a province in far northern Vietnam, covers an area of 8327.6 square kilometers and a population of 759,000 people. Tay people comprised 35.92% of the population [9,10]. Bacson is a district of Langson with population of 65,073 people over a total area of 698 square kilometers where we implemented the study, enrolling 1,094 members of all generations living together in the same home from 278 households.

To avoid a selection bias, children aged less than 6 months were excluded due to the likelihood of residual maternal H. pylori antibody, as well as those with severe diseases or immuno-compromised status due to the possibility of an altered immune response.

Data were collected after obtaining written consents from local administration and health authorities. Informed consent was obtained from each household member. The study was granted ethical clearance in Vietnam by the ethics committee of the Hanoi Medical University.

**Data collection**

In this cross-sectional study, door-to-door sampling method was adopted. We used a structured questionnaire, as in previous studies [12-15] for data collection on socio-demographic, health status and potential exposure. The questionnaires were completed by the study investigators and information was collected from the head of the households. Blood samples (5 mL) were obtained from each subject by aseptic venipuncture under aseptic conditions and were immediately centrifuged. Sera were separated and preserved in vaccine thermos, then sent to the reference laboratory (Microbiology Division of Digestive Diseases, National Institute of Epidemiology and Hygiene) on the same day where sera were stored at -20°C and processed as previously described [16].

**Variable definitions**

H. pylori infection was determined by in-house ELISA for dosage of H. pylori IgG an body against specific H. pylori antigen. The ELISA was carried out in the reference laboratory (Microbiology Division of Digestive Diseases, National Institute of Epidemiology and Hygiene) using sonicated Swedish and Vietnamese H. pylori strains as antigens prepared and validated in the microbiology department, Karolinska Institute, Stockholm for use in Vietnamese adults and children (sensitivity of 99.6%, and specificity of 97.8%) [16]. Children's sera with a value equal to or over 0.18 optical density (OD) unit were classified positive.

Socio-demographic variables consisted of age and sex of all children and adults in every household, monthly family income classified into three categories (i.e. <500.103 VND, actual national minimal monthly income, 500 to 1,000 or >1,000.103 VND), household space (in 3 categories, i.e. up to 10 m²/capita according to national mean standard, 10 to 20 or >20 m²/capita), parents’ occupation and parents’ education level [10].

Potential exposure variables were divided into three major subgroups

Environmental, individual hygiene and life-style including water sources classified in 2 main sources (family well and others such as streams, raining or collective wellness), latrine in 2 main types (existence or not), pet (dog or cat) in the house in 2 categories (presence or not), behavior of children on hand washing before meal and after toilet in 2 categories (not regular for those who practiced less frequently than one per twice, and regular for those who practiced every time or every other time), mouth-to-mouth feeding from mothers or caregivers to child in 2 categories (regular or not), breast feeding duration in 2 categories (shorter or longer than 12 months of age).

Promiscuity including size of household in 2 categories (≤4 or ≥5), sibling size in 2 categories (≤2 or ≥3), sharing a bed in 2 categories (with ≤3 and with ≥4 persons), collective life initiation in 2 categories (<3 years and ≥3 years of age).

Health status variables consisted of H. pylori infection status in parents and sibling (H. pylori seropositivity or seronegativity), gastro-duodenal history of child and parents (defined as having had a gastro-duodenal disease if this disease was diagnosed and treated by health-givers from district health center or higher levels) in 2 categories (presence or absence), and history of child’s antibiotic use during the previous 6, 12 months determined from individual health booklets or in-depth interviews of the main caregivers, in 2 categories (absence or presence).

**Statistical analysis**

First, study population characteristics were compared according to their H. pylori infection status using the Chi square (χ²) test. As one of the major aims of our study was to detect the factors potentially influencing H. pylori infection in this subpopulation, the appropriate strategy of analysis must be able to control for mutual confounding, point out the risk factors and avoid overlooking important associated variables. We then analyzed separately the associations between H. pylori infection status with the demographic and socio-
economic variables and with variables related to potential exposure. Analysis was performed firstly by univariate technique, by adjusting in each group on every variable, and finally by using backward stepwise conditional logistic regression to select variables importantly associated with H. pylori infection within each group, including all variables significantly associated with H. pylori seropositivity after adjustment and those with p values less than 0.20 by Chi square test. Associations were expressed as odds ratio (OR) and their confidence intervals (95% CI). Finally, backward stepwise procedures were used again to include in the final model not only variables independently associated with H. pylori serological status in each group, but also those known to be important for transmission pathways. Statistical significance was set up at the 0.05 level. All p values were 2-tailed. Data were analyzed using SPSS software (SPSS® for Windows™ version 16.0 Copyright SPSS Inc.).

**Results**

Among 1,094 individuals enrolled in the study, 512 (46.8%) were seropositive for H. pylori infection. H. pylori seroprevalence was 51.4% (304/591) in adults and 41.4% (197/476) in children ≤18 years old (p<0.05). Rates of H. pylori infection is associated with socio-demographic variables in Tay children aged 6 months to 18 years is presented in Table 1. There were no significant differences in H. pylori seropositivity based on sex (p>0.05). The prevalence of H. pylori infection was 30.9% for children under 3 years of age, rising to 53.1% for those older than 15 years. When analyzed by age strata, the odds of infection were higher for children aged over 15 years compared with those aged less than 3 years (odds ratio [OR], 1.59; 95% CI, 1.09-2.32). No significant relationship was found between H. pylori seropositivity and variables related to socioeconomic status of their household.

### Table 1 Socio-demographic associated with H. pylori seropositive in Tay children aged 6 months to 18 years

| Study variables | Categories | n/N   | Positive (%) | p-value | OR    | 95% CI |
|----------------|------------|-------|--------------|---------|-------|--------|
| Gender         | Female     | 103/240 | 42.9         | NS      | 1.19  | (0.82-1.71) |
|                | Male       | 94/236  | 39.8         |         | 1     |         |
| Age group (years) | <3         | 17/55  | 30.9         |         | 1     |         |
|                | 3-6        | 31/83   | 37.3         | NS      | 1.33  | (0.65-2.75) |
|                | 6-10       | 65/153  | 42.5         | NS      | 1.65  | (0.86-3.18) |
|                | 10-15      | 50/121  | 41.3         | NS      | 1.57  | (0.8-3.1)  |
|                | 15-18      | 34/64   | 53.1         | 0.016   | 1.59  | (1.1-2.32) |
| Monthly income (Thousands VND/capita) | <500       | 16/42   | 38.1         | 1       |       |         |
|                | 500-1,000  | 83/193  | 43.0         | NS      | 1.08  | (0.76-1.53) |
|                | >1,000     | 86/209  | 41.1         | NS      | 1.09  | (0.54-2.19) |
| House space (m²/person) | <10        | 14/26   | 53.8         | 1       |       |         |
|                | 10-20      | 74/196  | 37.8         | 0.05    | 0.72  | (0.47-0.99) |
|                | >20        | 86/205  | 41.9         | NS      | 0.58  | (0.25-1.4)  |
| Mother occupation | Peasant    | 117/277 | 42.2         | NS      | 1.05  | (0.72-1.85) |
|                | Others     | 80/199  | 40.2         |         | 1     |         |
| Father occupation | Peasant    | 100/244 | 40.9         | NS      | 0.98  | (0.83-2.79) |
|                | Others     | 97/232  | 41.8         |         | 1     |         |
| Mother education level | ≥ secondary | 86/208 | 41.3         | NS      | 0.99  | (0.76-3.94) |
|                | <secondary | 111/268 | 41.4         |         | 1     |         |
| Father education level | ≥ secondary | 88/188 | 46.8         | NS      | 1.24  | (0.65-3.98) |
|                | <secondary | 109/288 | 37.8         |         | 1     |         |

H. pylori seropositive in relation to environment, individual hygienic status and children lifestyle are presented in Table 2. No significant differences in rates of H. pylori infection based on water source, latrine presence, regular hand washing before meal and having pets. Children doing hand washing after defecation was less likely to H. pylori seropositivity (OR: 0.63, 95% CI 0.49 to 0.96, p=0.023). Breastfeeding more than 12 months was negatively and independently associated with...
**H. pylori** seropositivity (adjusted OR (95% CI): 0.71 (0.35 to 0.94), p=0.036)

**Table 2** H. pylori seropositive in relation to environment, individual hygienic status and children lifestyle

| Study variables                  | n/N     | Positive (%) | p-value | OR 95%CI |
|---------------------------------|---------|--------------|---------|----------|
| Variables                        |         |              |         |          |
| Water sources                    |         |              |         |          |
| Family well                      | 99/208  | 47.6         | NS      | 0.79 (0.54-1.16) |
| Others                           | 98/262  | 37.4         |         | 1        |
| Latrine presence                 |         |              |         |          |
| Yes                              | 29/59   | 49.2         | NS      | 1.17 (0.51-1.57) |
| No                               | 168/407 | 41.3         |         | 1        |
| Eating with the fingers          |         |              |         |          |
| Yes                              | 106/245 | 43.2         |         | 1.1 (0.54-1.64) |
| No                               | 91/231  | 39.4         |         | 1        |
| Regular hand washing before meal |         |              |         |          |
| Yes                              | 181/438 | 41.3         | NS      | 1.11 (0.56-2.19) |
| No                               | 16/38   | 42.1         |         | 1        |
| Regular hand washing after defecation |       |              |         |          |
| Yes                              | 188/462 | 40.7         | 0.023   | 0.63 (0.49-0.96) |
| No                               | 9/14    | 64.2         |         | 1        |
| Regular receiving chewed food    |         |              |         |          |
| Yes                              | 115/272 | 42.2         | NS      | 1.05 (0.51-1.35) |
| No                               | 82/204  | 40.2         |         | 1        |
| Dog in house                     |         |              |         |          |
| Yes                              | 127/317 | 40.0         | NS      | 0.91 (0.75-1.69) |
| No                               | 70/159  | 44.0         |         | 1        |
| Cat in house                     |         |              |         |          |
| Yes                              | 118/293 | 40.3         | NS      | 0.94 (0.67-1.68) |
| No                               | 79/183  | 43.2         |         | 1        |
| Breast feeding duration          |         |              |         |          |
| <12 months                       | 59/110  | 53.6         | 0.036   | 1.42 (1.07-2.68) |
| ≥12 months                       | 138/366 | 37.7         |         | 1        |

**H. pylori** seropositive in Tay children in relation to variables related to promiscuity and health status of study population as well as antibiotic use in children are presented in **Table 3**. The prevalence of **H. pylori** was higher among children with a family member infected with the bacterium than among children without any infected family member. The differences were significant for mothers, siblings, and also grandparents (p<0.001). We did not find any relationship between **H. pylori** seropositivity in children and variables related to promiscuity (i.e. size of household or sibling, **H. pylori** infection in fathers, regular sharing bed, collective life initiation and history antibiotic use within 6 or 12 months in children).

**Table 3** **H. pylori** seropositive in Tay children in relation to variables related to promiscuity and health status of study population

| Study variables                  | n/N     | Positive (%) | p-value | OR 95%CI |
|---------------------------------|---------|--------------|---------|----------|
| Variables                        |         |              |         |          |
| Size of household                |         |              |         |          |
| ≤4                               | 98/241  | 40.7         | NS      | 1        |
| >5                               | 99/235  | 42.2         |         | 1.13 (0.78-1.64) |
| Size of sibling                  |         |              |         |          |
| ≤2                               | 111/278 | 39.9         | NS      | 0.92 (0.61-1.87) |
| >3                               | 86/198  | 43.4         |         | 1        |
| Regular sharing bed              |         |              |         |          |
| ≤3                               | 54/132  | 40.9         | NS      | 0.98 (0.67-1.64) |
| >4                               | 143/344 | 41.6         |         | 1        |
In the final model of multivariate analysis (data not shown), low risk for *H. pylori* infection in children was independently associated with regular washing hand after defecation (OR 0.73 (0.39-0.92). In contrast, *H. pylori* infection in mothers, first sibling and grandparents were found associated with higher risk factors for *H. pylori* infection in children (OR (95%CI): 2.98 (1.13-3.14); 1.4 (1.0-3.12); 1.41 (1.11-4.64); respectively).

**Discussion**

Our study showed an overall prevalence of *H. pylori* infection in Tay people was 46.8%. *H. pylori* seroprevalence was 51.4% in adults and 41.4% in children ≤18 years old. The seroprevalence of *H. pylori* infection of the present study was comparable to those reported in other community-based studies in Vietnam [11-15]. Hoang et al. conducted a community-based study in Kinh majority ethnic in Hanoi showed an overall seroprevalence of *H. pylori* of 58.2% with 47.3% in children [11]. In another community-based study in Kinh of a rural area from Nghean province, Nguyen et al. reported an overall rate of *H. pylori* infection of 61.5% and 55.5% prevalence in children [17]. Recently, Le et al. reported an overall rate of 45.2% with 40.0% in children under 15 in their community-based study carried out in a population of Kinh and 4 other minority ethnics in central highland [15]. However, the overall *H. pylori* infection rate and rate of infection in children in the present study was comparable to the higher than those of the previous study carried out in a population of Kinh and 3 other minority ethnics in a mountainous area conducted by Trinh et al. [14].

It is believed that *H. pylori* is mainly acquired during childhood and little is known about its age of onset, rate, or mode of colonization [3-5]. Okuda et al. followed-up 108 out of 237 Japanese infants until 24 months by a fecal antigen test found that 16 infants turned to be HpSA positive within 12 months. Among them four infants remained positive by the consecutive tests demonstrated infants seemed to acquire *H. pylori* infection in the first year of life [18]. The proportion of infected children in the present study increases with age. *H. pylori* infection rate was 30.9% for children under 3 years of age.
age, rising to 53.1% for those older than 15 years. The odd of infection was 1.59 fold for children aged over 15 years compared with those under 3 years old (OR: 1.59; 95%CI: 1.09-2.32) confirming that children from developing countries are at greater risk of infection. Several studies supported the observation that early childhood is the main period of acquisition of *H. pylori* infection in high prevalence population. Vanderpas et al. [19] reported the prevalence of *H. pylori* was ranged from 18.2% in children less than 6 years old to 49.3% among group aged 12-17 years. *H. pylori* infection rates in children aged 3 to 7-year old, 8 to 12-year old and 13 to 16-year-old children were 39.5%, 41.0% and 54.5%, respectively were showed by Zhang [20]. Rothenbacher et al. [3] also found seroprevalence of *H. pylori* of 8.9% in the 1-year-old children, 36.4% in the 2-year-old children, and 31.9% in the 4-year-old children. Among 603 subjects of *H. pylori* negative, 38.7% became infected within 12 years. Nguyen et al. reported an increase of *H. pylori* seroprevalence from 22.6% in children under 3 to 39.3% in 10-15 years’ group (OR: 2.1; 95%CI: 1.4-3.1) in a study population of 824 children [12]. Our data is thus in line with the finding of others studies presently.

A significant association of *H. pylori* with male was showed in some previous studies [21]. The seroprevalence of *H. pylori* was significantly higher in boys (p<0.01) in two studies conducted in Spain and Japan. In contrast, the rate of *H. pylori* infection was not found to be related to gender in Taiwan, Korea, and Mexico [22-24]. There was no significant difference between the prevalence of *H. pylori* infection and gender in our study.

Low socioeconomic status and poor sanitary standards have been described as risk factors for the acquisition and transmission of *H. pylori* [25]. No significant relationship was found between *H. pylori* seropositivity and variables related to socio-economic status of their household in our study. Data from some previous studies have revealed that socioeconomic level is a risk factor for *H. pylori* infection. The prevalence by age decreasing along with socioeconomic development was reported in a multi-center study from Czech Republic [26]. The rate of *H. pylori* infection was significant decreased from 41.7% in 2001 to 23.5% in 2011. Decrease in the rate of *H. pylori* infection in children was also reported in Estonia [27] and Russia [28]. It has been explained by improving socioeconomic conditions and standards of living together. Mother’s educational levels rather than the fathers were associated with the rate of *H. pylori* infection in children has been shown in Argentina study [29]. One Korean study showed that the prevalence of *H. pylori* infection was lower in children from families of higher socioeconomic status [23]. The poor socioeconomic status of people in southwest Tehran is correlated with a higher incidence of infection (p<0.05) [30].

Potential exposure variables in relation to individual hygiene as well as exclusive habits and life-style have been always sought by investigators in epidemiological studies on *H. pylori* infection [22,23]. The present study showed no significant differences in rates of *H. pylori* infection based on water source, latrine presence, regular hand washing before meal and having pets. Children doing hand washing after defecation was less likely to *H. pylori* seropositivity (OR: 0.63, 95%CI: 0.49-0.96, p=0.023). In the present study, Nguyen et al. has shown that owing latrine in the family and regular washing hand before meal is significantly associated to lower *H. pylori* seroposivity [13,31]. In contrast, regular washing hand after defecation and no taking food by hand appeared as protective factors for *H. pylori* infection in ethnic people in Vietnam. Abebaw et al. also reported higher seroprevalence of *H. pylori* in people who used unprotected surface water and irregular washing hand before meal [32].

Breastfeeding more than 12 months was negatively and independently associated with *H. pylori* seropositivity in the present study (adjusted OR 0.71; 95% CI: 0.35-0.94, p=0.036). Some studies have shown breastfeeding’s protective effect against the acquisition of *H. pylori* infection [33,34], while other studies have reported that breastfeeding does not have a protective effect against the acquisition of *H. pylori* infection [35,36]. In a case-control study parents of children with and without *H. pylori* infection who had undergone endoscopic survey and gastric biopsy in the Tehran, Monajemzadeh et al. found that breastfeeding in the first 6 months after birth may decrease the degree of *H. pylori* colonization, postpone infection until older age, shorten the duration of symptoms, and be concomitant with milder gastritis [37]. Results from previous study showed that children breastfed longer than 12 months were likely to be more infected by *H. pylori* than those breastfed for a shorter duration (OR: 1.64; 95% CI: 1.09-2.71) [30]. A systematic review conducted by Chak showed that breast-feeding is protective against *H. pylori* infection (OR 0.78; 95% CI: 0.61-0.99; p=0.02) [34]. OR was 0.55 (95% CI: 0.33-0.93; p=0.01) in studies in which the subjects resided in developing countries compared with 0.93 (95% CI: 0.73-1.19; p=0.28) in those of developed nations. Thomas et al. found a positive relation between the age at acquisition of the infection and amount of anti-*H. pylori* IgA in human breast milk in Gambian children [38]. They concluded that specific human milk IgA may have a crucial role in delaying the onset of *H. pylori* infection. Gold et al. showed an association between breastfeeding and an increased risk for *H. pylori* IgG-seroconversion during 14 months of follow-up. The possible mechanisms enrolled in this protection may be the lactoferrin in human milk which binds to *H. pylori* liposaccharide inactivating the organism [39] and breast milk inhibited *H. pylori* adherence to gastric adenocarcinoma cell line in vitro [40].

The prevalence of *H. pylori* was higher among children with a family member infected with the bacterium than among children without any infected family member. Our study showed the presence of infected siblings, mother and grandparents as a risk factor for the infection in children. We did not find any relationship between *H. pylori* seropositivity in children and variables related to promiscuity such as size of household or sibling, *H. pylori* infection in fathers, regular bed sharing, collective life initiation and history of antibiotic use within 6 or 12 months in children. Some authors have demonstrated the mode of *H. pylori* transmission was infection clusters in families and familial spread [35,41]. Data from those studies provided an evidence that having infected family
members is highly associated with the infection in children. Drumm et al. reported seroprevalence of 80% in children whose has infected siblings compared to 13% of age-matched controls [41]. Malaty et al. also provided data demonstrating an increased prevalence of colonized children of infected parents [42,43]. The higher prevalence of infection due to \( H. pylori \) in parents of infected children suggests person-to-person transmission within the family [22,35]. The \( H. pylori \) status of the mother was found to be a strong determinant for childhood infection and more predictive than the status of the father. Kivi et al. showed that having an infected mother (OR: 11.6; 95% CI: 2.0-67.9) or at least one infected sibling (OR: 8.1, 95% CI: 1.8-37.3) was a major risk factor for \( H. pylori \) infection in Swedish children [41]. The mother is likely to have introduced the infection to her offspring, because daycare attendance and \( H. pylori \) prevalence amongst classmates have been refuted as risk factors for the infection in this child population, thus rendering child-to-child transmission outside the family improbable in Sweden [44]. Goodman et al. showed that having infected siblings was a predictor of the infection in children, although that study did not control for parental infection status [45]. The importance of both infected siblings and mothers was recently corroborated in a Brazilian high-prevalence community [46]. The findings are substantiated by a previous report of \( H. pylori \) strain concordance between mothers and offspring and amongst siblings, demonstrated by using bacterial molecular typing in a subset of the currently studied families [47]. Aguemon et al. also reported the higher infection rate in children whose parents were both infected or only infected mother. By using logistic regression analysis, sharing bed (OR: 3.85; 95% CI: 1.53-9.67, p<0.003) and infected mothers (OR: 9.82, 95% CI: 4.13-23.31, p<0.001) were independent predictors for \( H. pylori \) infection. Family contact with infected persons and crowded living conditions were associated with increased risk of infection [4,22,43]. In the present study, we observed rates of higher positivity among children of \( H. pylori \) positive mothers or grandparents compared to those whose family members were negative. Result from our study is as same as those from another study conducted in Mekong delta by Nguyen et al. \( H. pylori \) infection in mothers (OR: 4.04; 95% CI: 2.72-6.75; p<0.001), in the first and third siblings (OR (95% CI): 1.63 (1.26-2.14), p=0.001 and 1.6 (1.07-14.2), p=0.013) and grandparent (OR (95% CI) 2.6 (1.82-5.1) and 1.98 (1.1-3.3), p=0.001) were identified as risk factors for infection in \( H. pylori \) children [30]. The fact that \( H. pylori \) infection in siblings influenced more on the infection of other children than the infection in the mother might be explained by longer and closer contact between siblings than with mother, because in Vietnam, like in most developing countries, after the first year of age, children spent more time to play and to sleep together with siblings than with mother or father. Mouth secretions of mother could be contaminated with \( H. pylori \) may be transmitted to the infant and child. Transmission may occur also for sharing common cups, spoons, chopsticks, teats of feeding bottles, or for chewing or tasting children’s food.

Strength of our study lies in this cross-sectional community-based study. We recruited a large population who were all members of all generations living together in the same household. This method of sampling facilitated the data analysis and interpretation, provided a more comprehensive understanding about the interaction or interrelation between studied variables and \( H. pylori \) infection. Therefore, it rendered more feasible and more reliable for risk factors to be identified among studied socio-demographic and potential exposure variables. Because of the possible limitations of our study in our study are issued from the limitation of structured questionnaire reluctantly adopted according to human and financial conditions of the study. The first limitation resides in recall biases inevitably committed by respondents during interview. One more limitation is ambiguity and difficulty to calculate exact income per capita per month given diversity in homemade and self-serving products, very popular in this population living in remote area of mountain province. Another point of limitation issued from limited knowledge of participants in recognizing history of gastro duodenal disorders and loss of health booklet or medical prescription in many cases, sources of inaccuracy in deciding whether someone had suffered from digestive disease in relation to \( H. pylori \) infection.

Conclusion

This first community-based study of epidemiology of \( H. pylori \) infection in the Tay population settled in mountain province showed moderate and indifferent rates of seropositivity of \( H. pylori \) infection. Data from the present study are consistent with intrafamilial \( H. pylori \) transmission and suggest that improvement of living conditions should be protective against infection.

Acknowledgement

We sincerely thank

1. National Fund of Scientific and Technological Development (NAFOSTED) for sponsoring this study.

2. Hanoi Medical University and the National Institute of Epidemiology and Hygiene for material and technical supports indispensable for the study.

3. The personnel of the Provincial Department Health of Bacson District, whose invaluable and primordial assistance and involvement in organization and participation make these funding possible.

4. And all household members participating to the study for their precisely collaborative spirit.

Funding

This study was funded by National Fund of Scientific and Technological Development (NAFOSTED) of Vietnam
References

1. Torres J (2000) A comprehensive review of the natural history of Helicobacter pylori infection in children. Arch Med Res 31: 431-469.

2. Kandulska A, Selgrad M, and Malferttheiner P (2008) Helicobacter pylori infection: a clinical overview. Dig Liver Dis 40: 619-626.

3. Rothenbacher D (2000) Acquisition of Helicobacter pylori infection in a high-risk population occurs within the first 2 years of life. J Pediatr 136: 744-748.

4. Malaty HM (2002) Age at acquisition of Helicobacter pylori infection: a follow-up study from infancy to adulthood. Lancet 359: 931-935.

5. Rowland M (2006) Age-specific incidence of Helicobacter pylori. Gastroenterology 130: 65-72.

6. Sabb T, De Angelis P, Dall’Oglio L (2008) Helicobacter pylori infection in children: management and pharmacotherapy. Expert Opin Pharmacother 9: 577-585.

7. Delport W, van der Merwe SW (2007) The transmission of Helicobacter pylori: the effects of analysis method and study population on inference. Best Pract Res Clin Gastroenterol 21: 215-236.

8. Replogle ML (1995) Biologic sex as a risk factor for Helicobacter pylori infection in healthy young adults. Am J Epidemiol 142: 856-863.

9. Rheinlander T (2010) Hygiene and sanitation among ethnic minorities in Northern Vietnam: does government promotion match community priorities? Soc Sci Med 71: 994-1001.

10. Central Popula on and Housing Census Steering Committee (2010) The 2009 Viet Nam Popula on and Housing Census: Major Findings.

11. Hoang TT (2005) Seroprevalence of Helicobacter pylori infection in urban and rural Vietnam. Clin Diagn Lab Immunol 12: 81-85.

12. Nguyen VB (2006) Intra-familial transmission of Helicobacter pylori infection in children of households with multiple generations in Vietnam. Eur J Epidemiol 21: 459-463.

13. Nguyen BV (2006) Prevalence of and factors associated with Helicobacter pylori infection in children in the north of Vietnam. Am J Trop Med Hyg 74: 536-539.

14. Xuan T, Long, Minh LT, Nguyen VB (2007) Epidemiological features of Helicobacter pylori infec on in children of ve di erent minority ethnicities in a mountainous village. J Med Res 55: 146-153.

15. Le T, Nguyen VB, Hoang MH, Ngo VT, Hoang TH (2012) Risk factors for Helicobacter pylori infec on among children of 3 minority ethnicities in a rural village. J Med Res 79: 171-178.

16. Hoang TT (2004) Enzyme-linked immunosorbent assay for Helicobacter pylori needs adjustment for the population investigated. J Clin Microbiol 42: 627-630.

17. Nguyen TAX, Nguyen VB (2007) Epidemiological features of Helicobacter pylori infec on in a rural central village. J Med Res 332: 621-629.

18. Okuda M (2007) Helicobacter pylori colonization in the first 3 years of life in Japanese children. Helicobacter 12: 324-327.

19. Vanderpas J (2014) Follow-up of Helicobacter pylori infection in children over two decades (1988-2007): persistence, relapse and acquisition rates. Epidemiol Infect 142: 767-775.

20. Zhang Y, Li JX (2012) Investigation of current infection with Helicobacter pylori in children with gastrointestinal symptoms. Zhongguo Dai Dan Ke Za Zhi 14(9): 675-677.

21. de Martel C, Parsonnet J (2006) Helicobacter pylori infection and gender: a meta-analysis of population-based prevalence surveys. Dig Dis Sci 51: 2292-2301.

22. Lin DB (2007) Seroprevalence of Helicobacter pylori infection among schoolchildren and teachers in Taiwan. Helicobacter 12: 258-264.

23. Malaty HM (1996) Prevalence of Helicobacter pylori infection in Korean children: inverse relation to socioeconomic status despite a uniformly high prevalence in adults. Am J Epidemiol 143: 257-262.

24. Jimenez-Guerra F, Shetty P, Kurpad A (2000) Prevalence of and risk factors for helicobacter pylori infection in school children in Mexico. Ann Epidemiol 10: 474.

25. Yucel O (2014) Prevention of Helicobacter pylori infection in childhood. World J Gastroenterol 20: 10348-10354.

26. Bures J (2012) Significant decrease in prevalence of Helicobacter pylori in the Czech Republic. World J Gastroenterol 18: 4412-4418.

27. Oona M (2004) Helicobacter pylori infection in children in Estonia: decreasing seroprevalence during the 11-year period of profound socioeconomic changes. Helicobacter 9: 233-241.

28. Tkachenko MA (2007) Dramatic changes in the prevalence of Helicobacter pylori infection during childhood: a 10-year follow-up study in Russia. J Pediatr Gastroenterol Nutr 45: 428-432.

29. Olmos JA, Rios H, Higa R (2000) Prevalence of Helicobacter pylori infection in Argentina: results of a nationwide epidemiologic study. Argentinean Hp Epidemiologic Study Group. J Clin Gastroenterol 31: 33-37.

30. Alborti A (2006) Prevalence of Helicobacter pylori infection in children (south of Iran). Diagn Microbiol Infect Dis 54: 259-261.

31. Nguyen BV (2015) Epidemiology of Helicobacter pylori infection in Kinh and Khmer children in Mekong Delta, Vietnam. Ann Clin Lab Res 1-8.

32. Abebaw W, Kibret M, Abera B (2014) Prevalence and risk factors of H. pylori from dyspeptic patients in northwest Ethiopia: a hospital based cross-sectional study. Asian Pac J Cancer Prev 15: 4459-4463.

33. Ertem D, Harmanci H, Pehlivanoglu E (2003) Helicobacter pylori infection in Turkish preschool and school children: role of socioeconomic factors and breast feeding. Turk J Pediatr 45: 114-122.

34. Chak E, Rutherford GW, Steinmaus C (2009) The role of breastfeeding in the prevention of Helicobacter pylori infection: a systematic review. Clin Infect Dis 48: 430-437.

35. Kiwi M (2005) Helicobacter pylori status in family members as risk factors for infection in children. Epidemiol Infect 133: 645-652.

36. Jafar S (2013) Prevalence of helicobacter pylori infection in children, a population-based cross-sectional study in west Iran. Iran J Pediatr 23: 13-18.
37. Monajemzadeh M (2010) Breastfeeding and helicobacter pylori infection in children with digestive symptoms. Iran J Pediatr 20: 330-334.
38. Thomas JE (1999) Helicobacter pylori colonization in early life. Pediatr Res 45: 218-223.
39. Thomas JE (2004) Early Helicobacter pylori colonisation: the association with growth faltering in The Gambia. Arch Dis Child 89: 1149-1154.
40. Clyne M (1997) In vitro evaluation of the role of antibodies against Helicobacter pylori in inhibiting adherence of the organism to gastric cells. Gut 40: 731-738.
41. Kivi M (2005) Assessment of the cag pathogenicity island status of Helicobacter pylori infections with serology and PCR. Clin Microbiol Infect 11: 66-68.
42. Drumm B, Rowland M (2003) The epidemiology of Helicobacter pylori: where to from here? J Pediatr Gastroenterol Nutr 36: 7-8.
43. Malaty HM (2002) Helicobacter pylori infection in asymptomatic children: impact of epidemiologic factors on accuracy of diagnostic tests. J Pediatr Gastroenterol Nutr 35: 59-63.
44. Tindberg Y (2002) Intrafamilial transmission of Helicobacter pylori infection. Lakartidningen 99: 2848-2849.
45. Goodman KJ, Correa P (2000) Transmission of Helicobacter pylori among siblings. Lancet 355: 358-362.
46. Rocha GA (2003) Transmission of Helicobacter pylori infection in families of preschool-aged children from Minas Gerais, Brazil. Trop Med Int Health 8: 987-991.
47. Kivi M (2003) Concordance of Helicobacter pylori strains within families. J Clin Microbiol 41: 5604-5608.