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Prevalence and Risk Factors of *Helicobacter pylori* Infection in Muong Children in Vietnam

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Abstract

Aim: *H. pylori* infection is predominantly acquired in early childhood. The aim of the present study was to determine the prevalence of *H. pylori* infection and to identify factors associated with the infection in Muong children living in Vietnam.

Materials and methods: In a cross-sectional study 918 asymptomatic individuals of all generations living together in the same home were selected from 219 households, based on cluster sampling of residential location in Hoabinh Province, Vietnam. *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 χ^2 test and logistic regression models.

Results: An overall *H. pylori* seroprevalence was 48.8%. *H. pylori* seroprevalence was 52.1% in adults versus 48.2% in children ≤18 years old (p=0.05). Monthly income more than 50 US/capita, no regularly receiving chewed food and breastfeeding over 12 months were protective factors for *H. pylori* infection (OR: 0.62, 95% CI: 0.39-0.99); 0.58, (0.34-0.80); 0.58 (0.35-0.94); respectively). Risk factors for *H. pylori* infection in children were no regularly hand washing after defecation [OR, 38.6; 95% CI: 11.8-126.3), *H. pylori* infected mothers (OR, 1.34; 95% CI: 1.01-2.32] and infected grandfather (OR, 1.73; 95% CI: 1.01-4.34); father's occupation (OR: 1.38, 95%CI: 1.01-1.98), mother's education (OR: 2.61, 95%CI: 1.61-4.31) and size of households (OR, 1.46; 95% CI: 1.08-2.68). No other factors such as size of 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 Muong population showed familial clustering in multi-generation population and supported the hypothesis of person-to-person transmission in *H. pylori* infection

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

Introduction

Helicobacter pylori (*H. pylori*) is now recognized as a worldwide problem, with an estimated of about half of the world's population being infected [1]. It is the most common cause of chronic gastritis, peptic ulcer and is strongly linked to gastric cancer [1,2]. The sources of *H. pylori* and the mechanisms of acquisition remain poorly understood [1-3]. Childhood has been identified as the critical time for acquisition of *H. pylori* infection [3-5]. The prevalence of *H. pylori* infection varies by country from 20% to 90%, with rates of over 60% in developing countries [1-6]. Person-to-person transmission within the family have been considered as major modes of transmission by epidemiological and microbiological studies, both in developed and developing countries [1,6]. 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 [4,7]. 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% are living in rural or remote areas [8,9]. High rates of *H. pylori* infection were reported in both hospital-based and community-based studies from Vietnam [10-13]. Different risk factors for *H. pylori* infection have been identified in the Kinh ethnic majority [10-12] and among ethnic minorities [13-15]. As the socioeconomic level and the lifestyle vary considerably among ethnic groups in the country, the prevalence of *H. pylori* infection and particularly the risk factors for this infection differed in previous studies [10-15]. According to the General Statistics Office of the Government of Vietnam 2009 [9], the Muong is the thirst largest ethnic group in Vietnam after the majority Kinh and Tay ethnic groups with an estimated more than one million people. No data is available about prevalence of *H. pylori* infection and risk factors for this infection in

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Muong people. Further knowledge about the mode of transmission could be valuable for primary prevention of the *H. pylori* infection in this subpopulation. The aim of the present study was to determine the prevalence of *H. pylori* infection and to identify factors associated with the infection in Muong children living in Vietnam.

Population and Methods

Study population

Muong people inhabit the mountainous slopes of north central Vietnam, from the lower reaches of the Da river to the upper reaches of the Ma river. Each village generally consists of 10 to 50 households. This native ethnic group shares some distant roots with the Kinh (majority people) after the split into two groups (in the 10th century) but has all the elements of an ethnic group, having preserved and developed their own habits, customs, cultural practices and economic condition that differ from those of the Kinh people [9]. The Muong people live in traditional stilt houses where they also worship their ancestors and are involved in tasks like weaving and embroidery. A traditional stilt house is made from wood, bamboo, cane, or rattan and set one or two steps below the main level. The area under the house is either unused or used as a pen for livestock [9].

Hoabinh is a mountainous province in the North of Vietnam that covers an area of 4,684.2 square kilometers and has a population of 793,500 people. The Muong people comprises 63.4% of the population [9]. Kyson is a rural district of Hoabinh with population of 34.800 people over a total area of 202 square kilometers where we implemented the study, enrolling 918 family members of all generations living together in the same home from 219 households. All family members were included in order to investigate whether the size and composition of the families and *H. pylori* infection rates in family members would represent a risk factor for the children under study.

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 immunocompromised 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 [12-14] for data collection on socio-demographic, health status and potential exposure. The information was collected from the head of the households while the study investigators completed the questionnaires. Blood samples (5 mL) were obtained from each subject by peripheral 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 antibody 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. The method had been established and evaluated previously, giving with a cut-off level of OD 0.22 a sensitivity of 93.9% to 99.6% and a specificity of 90.7%. [16]. Since this type of assay can have ±10% variability around the established cut-off level, we used the lower cut-off of 0.2 in order to ensure a high sensitivity in the assay.

Socio-demographic variables consisted of age and sex of all children and adults in every household, household monthly income per person (calculated by taking the total gross household monthly income divided by the total number of family members living together) classified into two categories (i.e. ≤ one million VND (<50 US/capita) and >one million VND (≥50 US/capita)), household space (in 2 categories, i.e. up to 20 m^2/capita and ≥20 m^2/capita), parents' occupation and parents' education level [9].

Potential exposure variables were divided into three major subgroups

Environmental, individual hygiene and life-style variables including water sources were classified in 2 main sources (family well and others, e.g. streams, collected rain or ponds), regular latrine in 2 main types (existence or not), pet ownership (dog, cat or pig) in the house in 2 categories (yes or no), behavior of children on hand washing before meal and after toilet in 2 categories (not regular, i.e. those who practiced hand washing only once or twice, and regular, i.e. those who practiced hand washing every time or most of the 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 until 12 months of age).

Living conditions were evaluated by dividing the size of the household in 2 categories (≤4 or ≥5 persons), number of sibling in 2 categories (≤2 or ≥3), sharing a bed in 2 categories (with ≤2 and with ≥3 persons), collective life initiation (age for a child to start daycare) 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 (previous gastro-duodenal disease, diagnosed and treated by health-givers from district health center or higher levels) in 2 categories (presence or absence).

Table 1 Socio-demographic variables associated with *H. pylori* seropositivity in Muong children aged 6 months to 18 years.

Study variables		*H. pylori* **infected**		**p-value**	**Adjusted OR 95%CI**
		n/N	(%)		
Gender	Female	71/177	40.1	NS	1.27 (0.82-1.97)
	Male	70/152	46.1		1
Age group (years)	15-18	28/61	45.9	0.046	1.74 (1.01-3.76)
	10-15	36/80	45.0	NS	1.68 (0.81-3.49)
	6-10	36/82	43.9	NS	1.77 (0.81-3.48)
	3-6	22/54	40.7	NS	1.45 (0.64-3.13)
	<3	17/52	32.7		1
Monthly income (US/capita)	>50	57/151	37.7	0.04	0.62 (0.39-0.99)
	≤50	68/138	49.3		1
House space (m2/person)	<20	51/127	40.2	0.055	1.06 (0.49-1.73)
	≥ 20	67/148	37.8		1
Mother occupation	Peasant	51/112	45.5	NS	1.14 (0.69-1.86)
	Others	78/189	41.3		1
Father occupation	Peasant	51/112	45.5	0.04	1.38 (1.01-2.98)
	Others	57/173	32.9		1
Mother education level	<secondary	78/142	54.9	0.001	2.61 (1.61-4.31)
	≥secondary	57/173	32.9		1
Father education level	<secondary	56/135	41.5	0.052	1.30 (0.46-2.07)
	≥secondary	47/148	31.8		1
OR: Odds Ratio; CI: Confidence Interval; NS: Non-significant Difference. *Adjusted for age, sex, house space, number of people in the same household, education level of mother and father, water source, latrine and hygienic status and children lifestyle					

Statistical analysis

First, study population characteristics were compared according to their *H. pylori* infection status using the Chi square (χ^2) 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 0.05 by Chi square test. Associations were expressed as odds ratio (OR) and their confidence intervals (95% CI). Finally, backward stepwise conditional 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 so ware (SPSS® for WindowsTM version 16.0 Copyright SPSS Inc.).

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

Study variables		*H. pylori* infected		p-value	Adjusted OR 95% CI
		n/N	(%)		
Water sources	Family well	44/115	38.3	NS	1
	Others	97/214	45.3		1.34 (0.84-2.14)
Regular latrine Presence	Yes	76/177	42.9	NS	1
	No	53/123	43.1		0.99 (0.62-1.59)
Eating with the fingers	Yes	91/208	43.8	NS	1
	No	44/114	38.6		0.87 (0.47-1.62)
Regular hand washing before meal	Yes	120/292	41.1	NS	1
	No	13/29	44.8		1.21 (0.31-4.66)
Regular hand washing after defecation	Yes	3/86	3.49	0.021	1
	No	138/242	57.0		38.6 (11.8-126.3)
Regular receiving chewed food	Yes	61/114	53.5	0.04	1
	No	80/214	37.4		0.58 (0.31-0.80)
Dog in house	Yes	65/173	37.6	NS	1
	No	41/96	42.7		1.12 (0.75-1.69)
Cat in house	Yes	65/163	39.8	NS	1
	No	65/153	42.4		1.01 (0.67-1.59)
Pig in house	Yes	70/151	44.4		1
	No	68/164	41.5		0.91 (0.57-1.45)
Breast feeding duration	<12 months	71/120	59.2	0.036	1
	≥12 months	70/209	33.5		0.58 (0.35-0.94)

OR: Odds Ratio; CI: Confidence Interval; NS: Non-Significant difference. ∗Adjusted for age, sex, house space, number of people in the same household, education level of mother and father, water source, latrine and hygienic status and children lifestyle

Results

Overall 918 healthy individuals of all generations living together in the same home from 219 families were enrolled in the study. The proportion of *H. pylori* infected adults and children ≤18 years old was 52.1% (307/589) and 42.8% (141/329), (p=0.05) respectively (data not shown). Socio-demographic variables associated with the prevalence of *H. pylori* infection in Muong children aged 6 months to 18 years are presented in **Table 1**. There was no significant difference in *H. pylori* seropositivity based on gender (p>0.05). The prevalence of *H. pylori* infection was 32.7% in children under 3 years of age, rising to 45.9% for those older than 15 years. Monthly income was significant determination for seropositive of children in the multivariate models. The adjusted OR for infection in children was 0.62 (95%CI: 0.39-0.99, p=0.004) among family having monthly income more than 50 US/capita compared to family having monthly income less than 50 US/capita. A significant relationship was found between *H. pylori* seropositivity and some variables related to socioeconomic status of the household such as father's occupation and mother’s education **(Table 1)**.

H. pylori seropositivity in relation to environment, individual hygienic status and the children’s lifestyle are presented in **Table 2**. No significant differences were found in rates of *H. pylori* infection based on water source, regular latrine presence, regular hand washing before meal, eating with fingers and having pets. Prevalence of *H. pylori* infection in children who do not regularly wash their hands after defecation was significant higher than in those regularly washing their hands (adjusted OR: 38.6, 95%CI: 11.8-126.3, p=0.021). Children who did not regularly receive chewed food were less likely to be *H. pylori* seropositive (adjusted OR: 0.58, 95%CI 0.31-0.80, p=0.04). Breastfeeding more than 12 months was negatively and independently associated with *H. pylori* seropositivity (adjusted OR (95% CI): 0.58 (0.35-0.94), p=0.036).

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

Study variables		*H. pylori* infected		p-value	Adjusted OR 95%CI
		n/N	%		
Number of people in household	≤ 4	85/227	37.4	0.043	1
	≥ 5	52/95	54.7		1.46 (1.08-2.68)
Number of sibling in household	≤ 2	81/198	40.9	NS	1
	≥ 3	60/131	45.8		1.2 (0.71-1.67)
Regularly sharing bed	≤ 2	69/180	38.3	NS	1
	≥ 3	72/130	55.4		1.43 (0.87-2.64)
Collective life initiation	≥ 3 years	36/92	39.1	NS	1
	1-3 years	61/145	42.1		1.05 (0.53-1.56)
Mother's infection status	Positive	80/141	56.7	0.001	1.34 (1.01-2.32)
	Negative	61/144	42.4		1
Father's infection status	Positive	74/121	61.2	NS	1.11 (0.39-2.13)
	Negative	67/121	55.3		1
Parent's infection status	All positive	36/65	55.4	NS	1.40 (0.89-2.5)
	1 positive + 1 negative	44/104	42.3	NS	1.08 (0.35-1.91)
	2 negative	22/56	39.3		1
Sibling's infection status	All positive	45/91	49.5	0.045	1.37 (0.99-1.98)
	All negative	44/122	36.1		1
Grandmother's	Positive	68/141	48.2	NS	1.24 (0.72-1.53)
infection status	Negative	56/144	38.9		1
Grandfather's infection status	Positive	49/129	37.9	NS	0.97 (0.62-1.63)
	Negative	44/113	38.9		1
Mother's GI disease status	Yes	19/34	55.9	NS	1.05 (0.38-2.95)
	No	16/30	53.3		1
Father's GI disease status	Yes	19/31	61.3	0.003	1.73 (1.01-4.34)
	No	23/65	35.4		1
OR: Odds Ratio; CI: Confidence Interval; NS: Non-Significant difference; GI: Gastrointestinal					
Adjusted for age, sex, house space, number of people in the same household, education level of mother and father, water source, latrine and hygienic status and children lifestyle					

H. pylori seropositivity in Muong children in relation to variables related to living conditions and health status of study population as well as antibiotic use are presented in **Table 3**. An association was found between *H. pylori* seropositivity and the size of the household (adjusted OR: 1.46; 95% CI, 1.08-2.68, p=0.043). A family clustering of *H. pylori* infection was found. The adjusted OR for the risk of infection in the children when the mothers were infected was 1.34 (95% CI 1.01-2.32, p=0.001); when the siblings were infected it was 1.37 (95% CI 0.99-1.98, p=0.045) and it was 1.73 (95% CI 1.01-4.43, p=0.003) when grandfathers were infected compared to children without any infected family members. We did not find any relationship between *H. pylori* seropositivity in children and variables related to other living conditions (i.e. number of sibling, H. pylori infection status of the father, regularly sharing bed, collective life initiation.

Discussion

This is the first study describing the prevalence of *H. pylori* infection in Muong people in Vietnam. Our study showed an overall prevalence of *H. pylori* infection in Muong people was 48.8%. *H. pylori* seroprevalence was 52.1% in adults and 42.8% in children ≤18 years old. Our findings are comparable to those reported in other community-based studies in Vietnam [10-15]. In one community-based study carried out in Kinh majority ethnic, using the same serological test, Hoang et al. found an overall seroprevalence of *H. pylori* of 58.2% with 47.3% in children [10]. Data from one community-based study carried out in a population of Kinh and four other minority ethnic groups in the central highland previously showed an overall rate of 45.2% with 40.0% in children under 15 years [15]. Nguyen et al. conducted another community-based study in a Tay population in a rural area of the Langson province, found an overall rate of *H. pylori* infection of 46.8% and 41.4% in children [13].

It is believed that *H. pylori* is mainly acquired during childhood and little is known about its age of onset, rate, or mode of infection [3-5]. We found an increasing prevalence with age. *H. pylori* infection rate was 32.7% for children under 3 years of age, rising to 45.9% for those older than 15 years. The odd of infection was 1.74-fold for children aged over 15 years compared with those under 3 years old (OR, 1.74; 95% CI, 1.01-3.76) 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. One study conducted in Cameroon, using an enzyme-linked immunosorbent assay, the *H. pylori* stool antigen (HpSA) test reported a similar prevalence of 37.5% in children younger than 3 years [17]. Ceylan et al. [18] reported that the prevalence of *H. pylori* ranged from 4.62% in children less than 5 years old to 70.76% among group aged 11-15 years, in Turkey. In another study conducted in China, Zhang et al. showed a significant relationship between the *H. pylori* seropositivity and age group. A higher rate of *H. pylori* infection in children 13 to 16-year-old compared to those aged 3 to 7-year old (54.4% versus 39.5%, $p<0.05$) [19]. Nguyen et al. reported an increase of *H. pylori* seroprevalence from 30.9% in children under 3% to 53.1% in 15-18 year group (OR: 1.59; 95% CI: 1.1-2.32) in a study population of 476 children [13]. Our results are comparable with some other findings [13,16,20,21].

Low socioeconomic status and poor sanitary standards have been considered as risk factors for the acquisition and transmission of *H. pylori* [21,22]. Our findings showed a significant relationship between *H. pylori* seropositivity and some variables related to socioeconomic status of their household such as monthly income, father's occupation and mother’s education. The *H. pylori* infection rate decreasing along with socioeconomic development was shown in a multi-center study from Czech Republic [23]. The seroprevalence of *H. pylori* was significantly decreased from 41.7% in 2001 to 23.5% in 2011. Decrease in the prevalence of *H. pylori* infection in children was also shown in Estonia [24] and Russia [25]. It has been explained by improving socio-economic conditions and standards of living together. Hasosah et al. found that an income of <5000 Saudi riyal was positively associated with the presence of *H. pylori* in multivariable analyses (OR=2.06, 95% CI=1.07-3.95) [21]. The finding that it was the mother’s educational levels rather than the father’s that were associated with the rate of *H. pylori* infection in children has been reported in Turkish study [26]. One Nigerian study showed that the prevalence of *H. pylori* infection was lower in children from families of higher socioeconomic status [27]. The poor socioeconomic status of people in the south of Iran was correlated with a higher incidence of infection ($p<0.05$) [28]. Our data is thus in line with these findings of others previous studies [26-28].

Epidemiological studies of *H. pylori* infection have always looked to find potential exposure variables in relation to individual hygiene as well as habits and life-style [22,23,27]. Our data showed no significant differences in *H. pylori* infection rates based on water source, regular latrine presence, regular hand washing before meal and having pets. Prevalence of *H. pylori* infection in children without regular hand washing after defecation was significantly higher than those doing regular hand washing (OR: 38.6, 95% CI 11.8-126.3, p=0.021). In some previous studies, Nguyen et al. has shown that owing latrine in the family and regular washing hands before meal is significantly associated to lower *H. pylori* seropositivity [29]. Regular washing hand after defecation and no taking food by hand appeared as protective factors for *H. pylori* infection in ethnic people in Vietnam [12,29]. Abebaw et al. also reported higher rate of *H. pylori* infection in people who used unprotected surface water and irregular washing hand before meal [30]. Children who did not regularly receive chewed food were less likely to be *H. pylori* seropositive (adjusted OR: 0.58, 95% CI 0.31-0.80, p=0.04). In our previous study, we found no association between a rate of *H. pylori* infection and behavior of regularly chewed food in Tay ethnic people [13]. Using multivariate logistic regression analysis, mouth-to-mouth feeding from mothers or caregivers to a child was a risk factor for *H. pylori* infection in Thai and Kinh population in Vietnam (OR: 1.81, 95%CI 1.08-3.03) [14].

Some studies have demonstrated that breastfeeding is protective against the acquisition of *H. pylori* infection [31-33], while other studies have reported no protective effect of breastfeeding [3,5,34]. Prevalence of *H. pylori* infection in children breastfed longer than 12 months was lower than those breastfed for a shorter duration [OR (95% CI): 0.58 (0.35-0.94)] **(Table 2)**. In one study conducted in Germany, Rothenbacher et al. [3] found that breastfeeding longer than 6 months after birth may increase the rate of *H. pylori* infection [OR (95% CI): 2.57 (1.19-5.55)]. Results from a previous study in Tay children 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 (95% CI): 1.42 (1.07-2.68)] [13]. 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) [32]. 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. Our data is thus in line with findings from this systematic review [32].

Having infected family members was a risk factor for *H. pylori* infection in children in the present study. The prevalence of *H. pylori* was higher among children with infected mothers as compared to children whose mothers were uninfected [OR (95% CI): 1.34 (1.01-2.32)]. Our data found that also the presence of infected siblings and grandfather were risk factors for *H. pylori* infection in children. The adjusted OR for infection was [OR (95% CI): 1.37 (0.99-1.98)] and [OR (95% CI): 1.73 (1.01-4.34)], respectively. The prevalence of *H. pylori* infection in children living with more than 5 people in the household was higher than in those having less than 4 people (OR: 1.46, 95% CI: 1.08-2.68, p=0.043). These results are similar to findings in other studies [27,33,34]. We did not find any relationship between *H. pylori* seropositivity in children and some other variables such as the number of siblings, *H. pylori* infection in fathers, regularly sharing bed, collective life initiation and history antibiotic use within 6 or 12 months in children. Infection clusters in families and familial spread were considered as the mode of *H. pylori* transmission [5,20,35,36]. Data from those studies provided an evidence of having infected family members is highly associated with the infection in children. 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 [37]. Urita et al. reported a significantly higher seroprevalence of *H. pylori* in children who had infected siblings compared to the control group [38]. Malaty et al. also provided data demonstrating an increased prevalence of infected children to infected parents [4,39]. The higher prevalence of infection due to *H. pylori* in parents of infected children suggests person-to-person transmission within the family [5,40]. 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 [13,14,36]. The importance of both infected siblings and mothers was recently corroborated in a Brazilian high-prevalence community [41]. 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 [40]. 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, 95% CI: 3.85, 1.53-9.67, p 0.003) and infected mothers (OR, 95% CI: 9.82, 4.13-23.31, p<0.001) were independent predictors for *H. pylori* infection [42]. Family contact with infected persons and crowded living conditions were associated with increased risk of infection [5,37,40]. In the present study, we found higher rates of seropositivity among children who had *H. pylori* positive mothers, siblings or grandfather compared to those whose family members were negative. Results from our study are similar to those from other studies in Vietnam [13,14,29]. These findings might be explained by longer and closer contact between siblings, mothers and grandfathers in Muong people.

Mouth secretions of mother or grandfather could be contaminated with *H. pylori* and 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. Vomitus has been suggested as an important vehicle for *H. pylori* transmission as this organism had been successfully cultured from gastric juice and vomitus [43]. The faecal-oral route is another potential route of transmission. Evidence for a faecal-oral transmission route of *H. pylori* has been reported in several studies using DNA to detect *H. pylori* in stool of infected patients [44] although it is difficult to detect *H. pylori* in faecal samples by DNA methods because of potential inhibitors. In addition, this bacterium has also been isolated by culture of faecal samples in several studies [43,44]. This mode of transmission has been proposed to commonly occur in developing countries because of limitations in hygienic conditions and high risk of diarrheal disease [1,2].

Strength of the present study is that all members of all generations living together in the same household recruited in this cross-sectional community based-study. 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. The possible limitations of our study emanate from the limitation of structured questionnaire reluctantly adopted according to human and financial conditions of the study. Since we had to rely on self-reported from householder, the first limitation resides in recall biases inevitably committed by respondents during interview. Another limitation may be the difficulty to calculate exact income per capita per month given the diversity of homemade and self-serving products, very popular in this population living in a remote area of a mountain province.

Conclusion

The present first community-based study in a Muong population found a moderate prevalence of *H. pylori* infection among Muong children at an early age and an increase with age. The findings showed a familial clustering in these multi-generation familial structures and supported the hypothesis of person-to-person transmission in *H. pylori* infection.

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