Is *Helicobacter pylori* prevalence associated with the family population?

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**ABSTRACT**

**Aim:** To investigate the incidence of *Helicobacter pylori* (*H. pylori*) and related lesions in patients who applied to the gastroenterology outpatient clinic of our institution and its relationship with the family population.

**Methods:** The data of 701 patients who underwent elective esophagogastroduodenoscopy and were suitable for *H. pylori* research were analyzed. The patients were classified according to gender and age groups (under 40 years of age and above) and evaluated for *H. pylori*, gastric atrophy and intestinal metaplasia by histopathological method. In addition, the relationship between *H. pylori* and gastric atrophy, intestinal metaplasia, family population, total income of the family, and gender were investigated.

**Results:** Intestinal metaplasia was found in 3.1% of women and 6.6% of men (*p* = 0.03). *H. pylori* positivity was found as 65% in the group under 40 and 54% in the group over 40 years old (*p* = 0.003). Atrophy was detected as 2% in the group under 40 years of age and 13.1% in the group over 40 years old (*p* < 0.001). There was no difference between the *H. pylori* positive group and the *H. pylori* negative group in terms family populations (*p* > 0.05).

**Conclusion:** Our current findings from our study do not suggest that the prevalence of *H. pylori* is related to the number of family members, but we think that intestinal metaplasia is more common in males than females, and the eradication of *H. pylori* in this population should be detailed.

**Key words:** *Helicobacter Pylori*, gastric atrophy, intestinal metaplasia, family populations, gender.

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**Introduction**

*Helicobacter pylori* (*H. pylori*) is among the most common infections in the world [1]. It is a microorganism accused for chronic gastritis, gastric atrophy, intestinal metaplasia (IM), dysplasia and gastric carcinogenesis [2]. *H. pylori* is defined by the World Health Organization in the category of factor directly responsible for malignancy [3,4]. While it was recommended to be eradicated in special groups previously, it is stated in the latest consensus reports that it is more appropriate to eradicate it when detected [5-7]. It is claimed that its prevalence in the world increases towards the east from west. It has been suggested that poor sanitation conditions, low socioeconomic level and crowded familial environment increase the prevalence of the *H. pylori* infection [4]. Transmission of *H. pylori* remains inter-human and mainly intra-familial. Two separate Japanese studies used multisite sequence typing and fast amplified polymorphic DNA to demonstrate strain transmission in families [8]. In addition, allele
matches were found between index child and mother in 25 (60%) of 35 index pediatric patients, and between index child and mother and father in 9 (25.7%) patients [8,9]. In the study in which 19 isolates from five families were analyzed, mother-to-child in four families, father-children in two families, and sibling-to-sibling transitions in one family were documented [10]. In the study of Carreira et al., it was suggested that transmission can be prevented by breastfeeding in families with less economic development [11]. In addition, it is also known that antibiotic therapy affects the prevalence of _H. pylori_ infection later in life [12].

In this study, _H. pylori_ prevalence data from Hakkari province, located in the Eastern Anatolia Region of our country, was reported for the first time. In addition to demographical information, the frequency of _H. pylori_, gastric atrophy and intestinal metaplasia were also investigated by histopathological method in patients who applied to the gastroenterology clinic.

**Materials and methods**

The data of 874 cases who applied to the gastroenterology outpatient clinic between November 2013 and August 2014 and underwent oesophago-gastro-duodenoscopy (OGD) for different indications were evaluated in present retrospective study. Approval was obtained from Bolu Abant Izzet Baysal University Clinical Research Ethic Committee (Date and number: 2021-97). OGD procedures were performed using Pentax EPK i-5000 system and EG-2990i model gastroscopy device. Patients who used drugs such as antibiotics, proton pump inhibitors, bismuth sub-citrate within 30 days, patients who could not give a clear anamnesis about drug use and whose medical records could not be reached, as well as inpatients and emergency patients were excluded from the study. Patients’ information on age, gender, household population, the number of children women have and the declared income level were recorded. Histopathological reports of biopsy specimens taken from the antrum and two walls of the corpus of 701 cases who met the inclusion criteria were evaluated. The patients were classified according to gender and age groups (under 40 years of age and above) and evaluated for _H. pylori_, gastric atrophy and intestinal metaplasia by histopathological method. In addition, the relationship between _H. pylori_ and gastric atrophy, intestinal metaplasia, family population, total income of the family, and gender were investigated.

Pathology samples of the cases were delivered to the laboratory in 10% formol containers. Histopathological analysis was performed by staining with Giemsa and Arcien Blue by the same pathologist. Gastritis type, activation severity, inflammation, atrophy, metaplasia, lymphoid hyperplasia, and _H. pylori_ were investigated in gastric tissue according to the modified Sydney classification [13].

**Statistical analysis**

SPSS (Statistical Package for Social Sciences) for Windows 16.0 program was used for the statistical analysis of the findings obtained in the study. _P_ values lower than 0.05 are considered as statistically significant. The normality of data distribution was checked with the Kolmogorov-Smirnov test. Independent samples _t_-test was used for comparison of normally distributed data, and Mann-Whitney _U_ test was used to compare data that did not conform to normal distribution. Categorical data were compared using the Chi-square test. The Pearson correlation test was used for the correlation of the data conforming to the normal distribution, and the Spearman correlation test.
was used for the correlation of the data that did not fit the normal distribution.

**Results**
A total of 701 patients; 354 (50.5%) female, 347 (49.5%) male subjects were included to the study. The median ages of women and men were 38.5 (17-85) years and 41 (17-88) years, respectively (p=0.21). Table 1 shows \textit{H. pylori}, the family population, and the number of children women have, by gender and age.

\textit{H. pylori} was positive in 59.6% of women and 58.8% of men (p=0.83). The average number of children among married women was found to be 5.13 ±3.6 (min: 0 - max: 12). There was no difference between the \textit{H. pylori} positive group and the \textit{H. pylori} negative group in terms of the number of children (p>0.05). There was no difference between the \textit{H. pylori} positive group and the \textit{H. pylori} negative group in terms of the family population (mean family population 5.8). The income statements of the families

### Table 1. \textit{Helicobacter pylori} prevalence by gender and age group.

| Parameters          | \textit{H. pylori} positive (N, %) | \textit{H. pylori} negative (N, %) | Total | p    |
|---------------------|-----------------------------------|-----------------------------------|-------|------|
| Female              | 211 (59)                          | 143 (41)                          | 354   | 0.826|
| Male                | 204 (58)                          | 143 (42)                          | 347   |      |
| Total               | 415 (59)                          | 286 (41)                          | 701   |      |
| <40 year Female     | 123 (66)                          | 63 (33)                           | 186   | 0.008|
| >40 year Female     | 88 (52)                           | 80 (48)                           | 168   |      |
| Have child          | 5.1                               | 5.2                               |       | ns   |
| Family population   | 5.2                               | 5.3                               |       |      |
| <40 year Male       | 104 (63)                          | 61 (37)                           | 165   | 0.126|
| >40 year Male       | 100 (54)                          | 82 (46)                           | 182   |      |
| Total<40 year       | 227 (64)                          | 124 (36)                          | 351   | 0.003|
| Total>40 year       | 188 (53)                          | 162 (47)                          | 350   |      |

\textit{ns}: non-significant.

### Table 2. Distribution of gastric atrophy and intestinal metaplasia by gender and age.

| Parameters          | Gastric atrophy (%) | p    | Intestinal metaplasia (%) | p    |
|---------------------|---------------------|------|---------------------------|------|
| Female              | 7.6                 | \textit{ns} | 3.1                       |      |
| Male                | 7.5                 | \textit{ns} | 4.7                       |      |
| All cases           | 7.5                 | \textit{ns} | 4.7                       |      |
| \textit{H. pylori} (+) | 6.5                 | \textit{ns} | 4.5                       |      |
| \textit{H. pylori} (-) | 9.1                 | \textit{ns} | 5.2                       | \textit{ns} |
| <40 year Female     | 2.7                 | \textit{P}<0.001 | 2.2                       | \textit{ns} |
| >40 year Female     | 13.1                | \textit{P}<0.001 | 4.2                       | \textit{ns} |
| <40 year Male       | 1.2                 | \textit{P}<0.001 | 0                         | \textit{P}<0.001 |
| >40 year Male       | 13.2                | \textit{P}<0.001 | 12.6                      |      |
| <40 year \textit{H. pylori} (+) | 1.3                 | \textit{ns} | 0.8                       |      |
| <40 year \textit{H. pylori} (-) | 3.2                 | \textit{ns} | 1.6                       | \textit{ns} |
| >40 year \textit{H. pylori} (+) | 12                  | \textit{ns} | 9                         |      |
| >40 year \textit{H. pylori} (-) | 13.5                | \textit{ns} | 8                         | \textit{ns} |

\textit{ns}: non-significant.
were excluded from the evaluation by local employees who knew the local people on the grounds that they were unsafe/unrealistic.

Gastric atrophy was found in 7.6% of women and 7.5% of men (p = 0.95). There is no difference between the sexes in terms of gastric atrophy.

Gastric atrophy 6.5%, intestinal metaplasia 4.6% in the H. pylori positive group; In the H. pylori negative group, the rate of atrophy was 9.1% and the rate of metaplasia was 5.2%. No correlation was found between the presence and absence of H. pylori and atrophy/metaplasia. Intestinal metaplasia was found in 3.1% of women and 6.6% of men (p = 0.03). The rate of metaplasia in women is significantly lower than in men. Table 2 shows the distribution of gastric atrophy and metaplasia by gender and age group.

The cases were evaluated in terms of the presence of H. pylori positivity, atrophy and metaplasia by taking the age limit of 40. Table 2. H. pylori positivity in women under 40 years of age was 66%, and H. pylori positivity was 52% above the age of 40 (p = 0.008). Atrophy was found in 2.7% of women under the age of 40 and in 13.1% of women over the age of 40 (p <0.001). Intestinal metaplasia was found 2.2% of women under the age of 40, and in 4.2% of women over the age of 40 (p = 0.28). H. pylori positivity was found in 63% of men under 40 and 55% of men over 40 (p = 0.13). Atrophy was found in 1.2% of men under the age of 40 and in 13.2% of men over the age of 40 (p <0.001). While metaplasia was not found in men under 40 years of age, metaplasia was found in 12.6% of men over 40 years old (p <0.001).

When H. pylori, atrophy and metaplasia were examined by taking the age limit of 40 in the whole study population (Table 3); H. pylori positivity was found as 65% in the group under 40 and 54% in the group over 40 years old (p = 0.003). Atrophy was detected as 2% in the group under 40 years of age and 13.1% in the group over 40 years old (p <0.001). 1.1% in the under 40 age group; Intestinal metaplasia was detected in 8.6% of the group over 40 years old (p <0.001). After the age of 40, the frequency of H pylori decreases, while the incidence of gastric atrophy and intestinal metaplasia increases significantly.

Discussion

Tremendous results of the present work are; (a) the classic information regarding the prevalence of H. pylori and the family population show a positive correlation may not be valid in all regions, since H. pylori prevalence is not associated with the number of family members in present study, (b) significantly higher rates of intestinal metaplasia in male subjects compared to women, (c) lack of association between H. pylori positivity and intestinal metaplasia.

The average number of children of female patients who applied to our clinic is 5.1. There is no difference between the H. pylori positive group and the negative group in terms of household population. The declared economic

Table 3. Distribution of gastric atrophy, intestinal metaplasia and H. pylori positivity by age.

| Parameters      | All cases | <40 year | >40 year | p     |
|-----------------|-----------|----------|----------|-------|
| Gastric Atrophy | %7.5      | %1.9     | %13.1    | P<0.001 |
| Intestinal metaplasia | %4.7 | %1.1 | %8.5 | P<0.001 |
| H. pylori (+)   | %59       | %65      | %53      | P=0.003 |
revenues were excluded by local employees on the grounds that they did not reflect the truth. No difference was found between the *H. pylori* positive group and the negative group in terms of the family population. Therefore, the number of family population alone does not lead to a high *H. pylori* prevalence. Our observation may be that the local people pay great attention to their personal hygiene and that the clean water resources are abundant and the infrastructure problems are relatively minimal. In the TURHEP study, as the household population increases, *H. pylori* prevalence increases, the highest rate is reached in those with more than 6 households [14]. In this context, the household average of our study group did not contribute to *H. pylori* positivity.

It has been reported that intra-familial transmission is important in the spread of *H. pylori*, and the risk of infection increases as the number of children increases [15,16]. In our study, no significant difference was found between the number of children and family population of the *H. pylori* positive group and the negative group. Families’ sanitation conditions, hand washing habits before and after meals, and differences in family behavior among cultures may be factors in this. In addition, eating habits and breastfeeding times of the baby may also be factors in this difference.

*H. pylori* infection is the strongest known risk factor for malignancies occurring in the stomach, and epidemiological studies have determined that the attributable risk for stomach cancer is approximately %89 [17]. It has been shown that there is a reduction in the risk of malignancy with *H. pylori* eradication [18]. We investigated the incidence of this infection, which is a public health problem for our country, among the patients who applied our gastroenterology outpatient clinic. Among the patients who underwent OGD, the frequency of *H. pylori* was 58%, atrophic gastric 7.5%, and intestinal metaplasia 4.7% by histopathological method in 701 patients who met the inclusion criteria.

In the Turkish *H. pylori* Prevalence (TURHEP) study, the frequency of *H. pylori* was reported to be 83% by the urea breath test (UBT) method [14]. According to this study, the country is divided into five regions; *H. pylori* positivity was found at a higher rate in those living in the Central and Eastern Anatolian regions than in those living in the western and southern regions. In this study, in which data from 55 of 81 provinces were collected, there is no data indicating that samples were taken from our center. The *H. pylori* positivity rate (88.1%) reported in the Eastern region is considerably higher than the data of our center (58%). Although the urea breath test is a method used in epidemiological studies, it is weaker than the histopathological method in terms of providing data such as gastritis type, metaplasia, and atrophy [6]. The histopathological method has the disadvantage that *H. pylori* colonization areas are not homogeneous [19]. In addition, the 10-year time interval between the TURHEP date and our study may be a factor in explaining the difference in *H. pylori* prevalence.

In the years of our study, the prevalence of *H. pylori* reported from different provinces of the western region of the country varies between 50-75% [19-22]. The prevalence of *H. pylori* is reported as 59% in Iran, which is close to the center where the study was conducted. Globally, Oceania and northern European countries are among the countries with the lowest prevalence of *H. pylori*, while Portugal has the highest prevalence in Europe and Switzerland the lowest. Although in the same geography However, there is a high prevalence among the
natives in Australia and among the Alaskan natives in the North American continent compared to the continent they are in. These differences suggest that nutritional habits, sanitation conditions, socioeconomic development level are also important factors in the prevalence of *H. pylori* [23].

Two methods, histopathological and rapid urease test, were planned in our study. However, since all results of the urease test were positive, it was understood that the kits gave wrong results because they did not comply with the cold chain conditions in our study. Therefore, our data were analyzed according to the histopathological method. In addition, the fact that the study was conducted among patients who applied to the polyclinic and the sampling method that would represent the entire population of the province was not used can be considered as a deficiency. However, given the limited opportunities due to the location of the region, our data serve as a reference for further studies.

Intestinal metaplasia is one of the important steps in gastric carcinogenesis. In different studies conducted in Turkey between 2008 and 2013, rates of intestinal metaplasia ranging from 11.5% to 13.5% are reported [24-28]. The association of *H. pylori* with gastric cancer has been defined, and it has been shown in different studies that a decrease in gastric cancer prevalence may occur with the eradication of *H. pylori* [29-31].

As a result of this study, we can say that the information that the prevalence of *H. pylori* and the family population show a positive correlation may not be valid in all regions. We suggest that, *H. pylori* prevalence is not associated with the number of family members, however, intestinal metaplasia is more common in men compared to women, which warrant elaboration of *H. pylori* eradication in this population.

Limitations of present study are single center nature of the study and the establishment of *H. pylori* positivity with only one method. However, our study is the first data from eastern border of Turkey, which suggested comparable *H. pylori* positivity, atrophy and metaplasia rates with the average of the country.

**Conclusion**

In conclusion, family member count is not associated with *H. pylori* infection. In addition we also conclude that male subjects are more prone to develop intestinal metaplasia compared to women which warrant more aggressive evaluation of this gender.

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**Conflict of Interest:** The authors declare that they have no conflict of interest.

**Ethical statement:** Bolu Abant İzzet Baysal University ethics committee approved the study protocol (Approval ID: 2021/97).

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References

[1] Brown LM. Helicobacter pylori: epidemiology and routes of transmission. Epidemiol Rev. 2000; 22 (2): 283-97.

[2] Correa P. Human gastric carcinogenesis: a multistep and multifactorial process—first American Cancer Society award lecture on cancer epidemiology and prevention. Cancer Res. 1992; 52 (24): 6735-40.

[3] Schistosomes, liver flukes and Helicobacter pylori. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. Lyon, 7-14 June 1994. IARC Monogr Eval Carcinog Risks Hum. 1994;61:1-241.

[4] Kikuchi S. Epidemiology of Helicobacter pylori and gastric cancer. Gastric Cancer. 2002;5(1):6-15.

[5] Malferttheiner P, Megraud F, O'Morain CA et al. Management of Helicobacter pylori infection—the Maastricht IV/Florence consensus report. Gut. 2012; 61 (5): 646-64.

[6] Malferttheiner P, Megraud F, O'morain C et al. Management of Helicobacter pylori infection—the Maastricht V/Florence consensus report. Gut. 2017; 66 (1): 6-30.

[7] Sugano K, Tack J, Kuipers EJ et al. Kyoto global consensus report on Helicobacter pylori gastritis. Gut. 2015; 64 (9): 1353-67.

[8] Mentis A, Lehours P, Mégraud F. Epidemiology and Diagnosis of Helicobacter pylori infection. Helicobacter. 2015; 20: 1-7.

[9] Yokota Si, Konno M, Fujiwara Si et al. Intrafamilial, preferentially mother-to-child and intraspousal, Helicobacter pylori infection in Japan determined by multilocus sequence typing and random amplified polymorphic DNA fingerprinting. Helicobacter. 2015; 20 (5): 334-42.

[10] Osaki T, Konno M, Yonezawa H et al. Analysis of intra-familial transmission of Helicobacter pylori in Japanese families. J Med Microbiol. 2015; 64 (1): 67-73.

[11] Carreira H, Bastos A, Peleteiro B et al. Breast-feeding and Helicobacter pylori infection: systematic review and meta-analysis. Public Health Nutr. 2015; 18 (3): 500-20.

[12] Hoffmann A, Krumbiegel P, Richter T et al. Helicobacter pylori prevalence in children influenced by non-specific antibiotic treatments. Cent Eur J Public Health. 2014; 22 (1): 48-53.

[13] Stolte M, Meining A. The updated Sydney system: classification and grading of gastritis as the basis of diagnosis and treatment. Can J Gast. 2001; 15 (9): 591-98.

[14] Ozaydin N, Turkyilmaz SA, Cali S. Prevalence and risk factors of Helicobacter pylori in Turkey: a nationally-representative, cross-sectional, screening with the 13C-Urea breath test. BMC Public Health. 2013; 13: 1215.

[15] Kikuchi S, Kurosawa M, Sakiyama T. Helicobacter pylori risk associated with sibship size and family history of gastric diseases in Japanese adults. Jpn J Cancer Res. 1998; 89 (11): 1109-12.

[16] Webb P, Knight T, Greaves S et al. Relation between infection with Helicobacter pylori and living conditions in childhood: evidence for person to person transmission in early life. BMJ. 1994; 308 (6931): 750-53.

[17] Lu B, Li M. Helicobacter pylori eradication for preventing gastric cancer. World J Gastroenterol. 2014; 20 (19): 5660-65.

[18] Wong BC, Lam SK, Wong WM et al. Helicobacter pylori eradication to prevent gastric cancer in a high-risk region of China: a randomized controlled trial. JAMA. 2004; 291 (2): 187-94.

[19] Bor S, Kitapcioglu G, Kasap E. Prevalence of gastroesophageal reflux disease in a
country with a high occurrence of Helicobacter pylori. World J Gastroenterol. 2017; 23 (3): 525-32.

[20] Korkut Y, Kilit T, İşik İ et al. Comparison of endoscopy and C-14 urea breath test in terms of helicobacter pylori positivity in patients admitted with complaints of dyspepsia. Family Prac and Pall Care. 2016; 1 (1): 9-12.

[21] Kosekli MA. Prevalence of Helicobacter pylori, gastric atrophy and intestinal metaplasia in gastric biopsy specimens: A retrospective evaluation of 1605 patients. Exp Biomed Res. 2021; 4 (4): 270-75.

[22] Konakci N, Gülten M, İbanoğlu MS et al. Kronik aktif gastritli olgularda Helicobacter pylori sıklığı. Uludağ Üni Tıp Fak Derg. 2010; 36 (1): 7-10.

[23] Hooi JK, Lai WY, Ng WK et al. Global prevalence of Helicobacter pylori infection: systematic review and meta-analysis. Gastroenterology. 2017; 153 (2): 420-29.

[24] Adim ŞB, Filiz G, Gürel S et al. Kronik Gastrit Olgularında İntestinal Metaplazı Sıklığı ve İntestinal Metaplazı İle Helicobacter Pylori İlişkisi. Uludağ Üni Tıp Fak Derg. 2008;34 (1): 1-4.

[25] Emre E, Ahishali E, Dolapcióğlu C et al. Peptik Ülser ve Gastrit Saptanan Hastalarda Helicobacter Pylori Sıklığı. J Kartal TR. 2013; 24(2): 87-92.

[26] Ümit HC, Ünsal G, Tezel A et al. Helicobacter pylori infection and benign gastroduodenal diseases, data from the Trakya Region. Trakya Üniversitesi Tıp Fakültesi Derg. YA Balkan Med J. 2010; 27 (4): 400-3.

[27] Mete R, Oran M, Güneş H et al. Tekirdağ bölgesinde Helicobacter pylori prevalansı ve patolojik parametrelerin çok yönlü analizi; literatür ile güncelleme. Genel Tıp Derg. 2014; 24: 1-6.

[28] Ozdil K, Sahin A, Kahraman R et al. Current prevalence of intestinal metaplasia and Helicobacter pylori infection in dyspeptic adult patients from Turkey. Hepatogastroenterol. 2010; 57 (104): 1563-66.

[29] Gonzalez C, Megraud F, Buissonniere A et al. Helicobacter pylori infection assessed by ELISA and by immunoblot and noncardia gastric cancer risk in a prospective study: the Eurgast-EPIC project. Ann Oncol. 2012; 23 (5): 1320-24.

[30] Liou J-M, Malfertheiner P, Lee Y-C et al. Screening and eradication of Helicobacter pylori for gastric cancer prevention: the Taipei global consensus. Gut. 2020; 69 (12): 2093-12.

[31] Piazuelo MB, Bravo LE, Mera RM et al. The Colombian chemoprevention trial: 20-year follow-up of a cohort of patients with gastric precancerous lesions. Gastroenterology 2021; 160 (4): 1106-17.