# 主論文の要約

# Impact of Socioeconomic Status and Sibling Number on Prevalence of *Helicobacter pylori* Infection: a Cross-Sectional Study in a Japanese population

ヘリコバクター・ピロリ感染の有病率に対する 社会経済的地位と兄弟姉妹の人数の影響: 日本人集団における横断研究

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#### [Introduction]

H. pylori infects almost 50% of people all over the world. However, there is a substantial difference in the prevalence of H. pylori infection between developing and developed countries. In developing countries, the prevalence is very high and reach 70% among children, whereas in developed countries the prevalence is generally less than 40% in the general population. In Japan, the prevalence of H. pylori infection was historically very high, but has been decreasing by birth cohorts from 80-90% in the older population born before 1950 to less than 2% in children born after 2000. This wide difference in H. pylori prevalence between developing and developed countries and across birth cohorts may be attributable to geography, ethnicity, living conditions and socioeconomic factors.

Lower socioeconomic status (SES) is thought to be associated with higher prevalence of H. pylori infection because low SES is associated with poor hygiene and bad sanitary conditions, which are considered important risk factors for H. pylori infection. On the other hand, high sibling number (SN) can be positively correlated with higher prevalence of H. pylori infection. Close interpersonal contact (sharing cups, sharing a bed and close playing) between siblings might facilitate H. pylori transmission. Here, we conducted a cross-sectional study to investigate the impact of SES and SN on the prevalence of H. pylori infection after adjustment for confounding factors.

# [Material and methods]

We conducted a cross-sectional study to evaluate the impact of socioeconomic status, represented by education level, and sibling number on the prevalence of *Helicobacter pylori* infection among 3,423 non-cancer subjects who visited Aichi Cancer Center between 2005 to 2013.

H.~pylori infection was defined as an anti-H.~pylori IgG > 10 U/ml in serum. The sensitivity and specificity of this cut-off value are 90.7% and 91.5%, respectively, on confirmation against the 13C urea breath test. Serum pepsinogen (PG) levels were measured by chemiluminescence enzyme immunoassay, and atrophic gastritis was defined by PG I  $\leq$  70 ng/ml and PG I/PGII  $\leq$  3.

Subjects who had atrophic gastritis (defined as PG I  $\leq$  70 ng/ml and PG I/PG II  $\leq$  3) but were anti-H. pylori IgG negative on testing were considered to be H. pylori positive in this study, with reference to the natural history of H. pylori infection (negative seroconversion of H. pylori antibodies).

We estimated ORs using the following four models: Model 1 (crude analyses); Model 2, multivariable model which adjusted for age category (<40, 40-49, 50-59,60-70, >70) and sex; Model 3, which adjusted for birth year (<1950, 1950-1960, >1960), current BMI (<18.5, 18.5-23, 23-27.5, ≥27.5), BMI at age 20, age category and sex; and Model 4, which further adjusted for drinking category (never, light, moderate and heavy) and smoking

#### [Results]

We found a statistically significant dose-dependent negative association between SES and  $H.\ pylori$  infection in the crude analysis (Low: reference, Moderate: OR = 0.67, 0.53-0.84, High: OR = 0.43, 0.34-0.54; P for trend < 0.001). To control for potential confounders, we adjusted for age, sex, birth year, current BMI, BMI at age 20, drinking and smoking in Models 2, 3 and 4. The association between SES and  $H.\ pylori$  infection remained statistically significant in all the models (Low: reference, Moderate: OR = 0.86, 0.68-1.08, High: OR = 0.67, 0.52-0.84; P for trend < 0.001, Model 4).

In contrast, we observed a statistically significant dose-dependent positive association between SN and *H. pylori* infection in the crude analysis ( $\leq$ 2: reference, 3-4: OR = 1.74, 1.47–2.06,  $\geq$ 5: OR = 2.54, 2.12-3.04; *P* for trend= P < 0.001). After adjustment for potential confounders (age, sex, birth year, current BMI, BMI at age 20, drinking categories and smoking categories), the association was attenuated, but was still statistically significant in Model 2, 3 and 4 ( $\leq$ 2: reference, 3-4: OR =1.31, 1.10–1.57,  $\geq$ 5: OR = 1.29, 1.04-1.59; *P* for trend= 0.022, Model 4).

We didn't observe a significant interaction between SES and SN for developing *H. pylori* infection.

### [Discussion]

In this cross-sectional study, we observed a negative association between high SES and the prevalence of *H. pylori* infection after controlling for confounding variables. In addition, we found a positive association between higher SN and the prevalence of *H. pylori* infection. SES and SN were independently associated with *H. pylori* infection, and no obvious interaction between these two factors was observed. This is the first study to show a statistically significant association of SES and SN with the prevalence of *H. pylori* infection in Japan.

Our study is consistent with several previous studies, which reported that the prevalence of *H. pylori* was high among individuals with low SES, albeit that SES surrogates among these studies differed. Among several SES measures, educational level is thought to be strongly correlated with personal hygiene measures and child care. Accordingly, a higher educational level is associated with a greater knowledge of sanitation and mitigation of unsanitary conditions, which consequently acts to reduce the risk of *H. pylori* infection.

Our results showed that SN has a significant positive correlation with the prevalence of *H. pylori* infection. This finding is in agreement with the results of several previous studies demonstrating that high SN was a risk factor for *H. pylori* 

infection. Domestic overcrowding during childhood seems to be strongly associated with *H. pylori* infection.

## [Conclusion]

This study showed that the prevalence of H. *pylori* infection is high among individuals with low SES and among those with a high SN. Our findings indicate that close personto-person transmission and unfavorable sanitary conditions are the main mechanisms of H. *pylori* infection spread. Our results warrant further investigation to clarify natural history of *H. pylori* transmission over lifetime