Presence of oral *Helicobacter pylori* DNA and its association with dental hygiene in older adults

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Abstract. The aim of the present study was to clarify the association between the presence of oral *Helicobacter pylori* (*H. pylori*) DNA and dental hygiene status in older adults. For this purpose, 98 patients aged ≥65 years (28 males, 70 females; median age, 75 years) were examined in the present study. Quantitative polymerase chain reaction analysis was performed to detect *H. pylori* DNA in the samples collected from the tongue surface. The accumulation of dental plaque was examined using the modified O'Leary Plaque Control Record to assess dental hygiene condition. Additionally, bleeding on probing and periodontal pocket depth were examined. *H. pylori* DNA was determined as positive in 7 of the 98 participants (7.1%). Participants in their 70s exhibited a greater positive rate of oral *H. pylori* DNA (12.5%) than the other age groups. The participants who were positive for oral *H. pylori* exhibited higher median plaque control record scores (37%) than those who were negative for oral *H. pylori* (16%). A significant difference in plaque control record scores was found between the oral *H. pylori*-negative and -positive participants (P=0.04). The oral *H. pylori*-positive participants exhibited a higher rate of ≥4 mm deep periodontal pockets (85.7%) than the oral *H. pylori*-negative participants (49.5%). However, no significant association between oral *H. pylori* and deep periodontal pockets was found. Logistic regression analysis was performed as independent variables in univariate analysis (i.e., ≥4 mm deep periodontal pockets and plaque control record score) and with oral *H. pylori* as the dependent variable. There was no significant association between oral *H. pylori* and ≥4 mm deep periodontal pockets and plaque control record score. On the whole, these findings suggest that the presence of oral *H. pylori* DNA is associated with poor dental hygiene in older adults. The maintenance of good oral health by daily oral care may help to reduce the risk of oral *H. pylori* infection.

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Introduction

Helicobacter pylori (*H. pylori*) is a Gram-negative bacteria that normally inhabits the human stomach (1). It is known that *H. pylori* augments the risk of developing gastric diseases, such as gastric ulcers, gastric cancers and gastric mucosa-assisted lymphoid tissue lymphoma by infecting the gastric mucosa epithelial cells (2). It has been reported that approximately half of the population worldwide is infected with *H. pylori*, with a particularly high prevalence rate in African regions (3). The prevalence rate of *H. pylori* is 30-40% in developed countries and ≥80% in developing countries, suggesting that socio-economic conditions are markedly associated with *H. pylori* infection (4). *H. pylori* infection occurs during childhood via oral transmission as an asymptomatic infection (5).

It may be challenging to demonstrate the colonization of *H. pylori* in the oral cavity (6). It has been reported that *H. pylori* DNA can be detected in inflamed dental pulp and subgingival dental plaque in the oral cavity, as well as in the gastric mucosa (6-8). A recent meta-analysis revealed that gastric *H. pylori* infection is more frequently found in patients with current oral *H. pylori* infection than in patients without oral *H. pylori* infection (9). Additionally, it has been reported that treatment for oral *H. pylori* infection is effective for the successful eradication of gastric *H. pylori* (10), indicating that there is a significant relationship between oral *H. pylori* and gastric *H. pylori* infection. Therefore, the oral cavity may be an important reservoir for gastric *H. pylori*. It is speculated that poor oral hygiene and periodontal inflammation may be

linked to the prevalence of oral *H. pylori* and gastric *H. pylori* infection. However, the association between the prevalence of oral *H. pylori* and oral health has not yet been fully investigated in Japanese older adults. Therefore, the aim of the present study was to investigate the association between the presence of oral *H. pylori* DNA and dental hygiene condition in older adults.

Patients and methods

Study participants. The present study targeted patients aged ≥65 years who visited the Oral Health Department at Hiroshima University Hospital from April, 2021 to February, 2023. Patients with immunodeficiency (i.e., post-operative inpatients, cancer patients receiving chemotherapy and patients with immune deficiency disorders) were excluded (n=0). Finally, 98 older patients (28 males, 70 females; median age, 75 years; range, 65-91 years) were included in the present study. The present cross-sectional study was a part of a general research project on the association between oral microbiome and oral health approved by the Ethics Committee of Hiroshima University (Approval no. E-1115). Patients who agreed to participate in this study signed an informed consent form. Clinical variables (i.e., participants' age, gender, lifestyle-related diseases, number of remaining teeth and denture use) were obtained from medical records of the participants. Periodontal pocket depth and bleeding on probing (BOP) were investigated at six sites (mesio-buccal, mid-buccal, disto-buccal, disto-lingual, mid-lingual and mesio-lingual sites) for each tooth. The accumulation of dental plaque was evaluated using a modified O'Leary Plaque Control Record by assessing six surfaces (mesio-buccal, mid-buccal, disto-buccal, disto-lingual, mid-lingual and mesio-lingual surfaces) of each tooth, as previously described (11).

Oral sample collection method and DNA extraction. Swab samples were collected from the tongue surface using a sterile disposable Orcellex® Brush (Rovers Medical Devices). After the collected samples were dissolved in cell lysis buffer, DNA was extracted and purified using a PureLink™ Microbiome DNA Purification kit (Invitrogen; Thermo Fisher Scientific Inc.) in accordance with the manufacturer's instructions.

Quantitative polymerase chain reaction (qPCR). qPCR was performed in the Thermal Cycler Dice® Real Time System III (Takara Bio, Inc.) using THUNDERBIRD SYBR qPCR mix (Toyobo Co., Ltd.). The amplification cycle consisted of 95˚C for 2 min, followed by 50 cycles of 95˚C for 1 min, 55˚C for 1 min and 72˚C for 1 min, and 72˚C for 2 min. A primer pair targeting the *H. pylori ureA* gene was used in the present study, as it has high specificity for detecting *H. pylori* DNA, as described in a previous study (8). The sequence of primers for *H. pylori* was as follows: forward, 5'-ATGAAACTCACCCCAAAAGA-3' and reverse, 5'-TTCA CTTCAAAGAAATGGAAGTGTGA-3' (8). A standard curve for *H. pylori* was generated using 10-fold serially diluted samples of the AMPLIRUN® HELICOBACTER PYLORI DNA CONTROL (10,000-20,000 *H. pylori* DNA copies/µl; Vircell) (Fig. 1). A no-template control was used as the negative control in qPCR analysis.

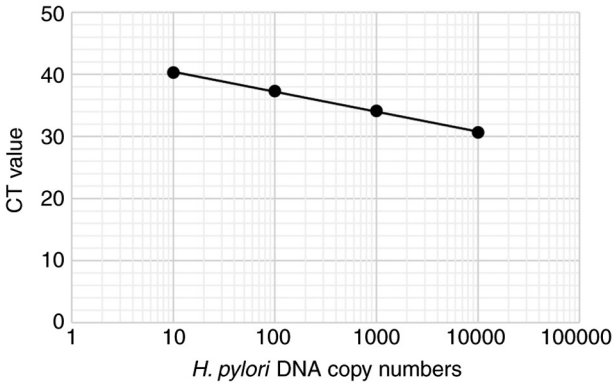

Figure 1. Standard curve indicating *H. pylori* DNA copy number vs. CT value. Serial 10-fold dilutions of *H. pylori* DNA (ranging from 10^1 to 10^4 copies/µl) were employed to make a standard curve. *H. pylori, Helicobacter pylori*; CT, threshold cycle.

Statistical analysis. Statistical analysis was conducted using SPSS software, version 24.0 (IBM Corp.). Logistic regression analysis was performed to examine the association between *H. pylori* as a dependent variable and clinical variables as independent variables. Clinical parameters with a P-value of <0.2 through univariate analysis were used as independent variables for logistic regression analysis. Continuous variables are expressed as the median and interquartile range (IQR). P-values <0.05 were considered to indicate statistically significant differences.

Results

Association between oral H. pylori and clinical variables. The association between the presence of oral *H. pylori* DNA and clinical variables is presented in Table I. Among the 98 participants, 7 participants (7.1%) were positive for oral *H. pylori* DNA. Participants in their 70s exhibited a greater positive rate of oral *H. pylori* DNA (12.5%) than the other age groups. Additionally, female participants exhibited a higher positive rate of oral *H. pylori* DNA (8.6%) than male participants (3.6%). There was no significant association between the presence of oral *H. pylori* DNA and clinical variables such as age, sex, lifestyle-related diseases, number of remaining teeth, or denture use.

Association between oral H. pylori and the oral health condition. The participants who were positive for oral *H. pylori* exhibited higher median plaque control record scores (37%) than those who were negative for oral *H. pylori* (16%) (Fig. 2). A significant difference in plaque control record scores was found between the oral *H. pylori*-negative and -positive participants (P=0.04). As regards the association between oral *H. pylori* and clinical periodontal conditions, the oral *H. pylori*-positive participants exhibited a higher rate of ≥4 mm deep periodontal pockets with BOP (85.7%) than the participants who were negative for oral *H. pylori* (49.5%) (Table II). Additionally, the oral *H. pylori*-positive participants exhibited a higher rate of ≥6 mm deep periodontal pockets with BOP (28.6%) than the participants who were negative (15.4%) (Table II). However, no significant association between oral *H. pylori* and deep periodontal pockets with BOP was found. These results indicate

Table I. Clinical characteristics of the study participants and their association with the presence of oral *H. pylori* DNA.

Clinical variables (n)	*H. pylori*-negative (n=91)	*H. pylori*-positive (n=7)	P-value
Age, median (IQR)	75 (11.0)	75 (5.0)	0.86[a]
Age group, n (%)			
65-69	20 (22%)	0 (0%)	0.3[b]
70-79	42 (46.2%)	6 (85.7%)	
80-89	27 (29.7%)	1 (14.3%)	
90-99	2 (2.2%)	0 (0%)	
Sex, n (%)			
Male	27 (29.7%)	1 (14.3%)	0.67[b]
Female	64 (70.3%)	6 (85.7%)	
Hypertension, n (%)			
Yes	22 (24.2%)	3 (42.9%)	0.37[b]
No	69 (75.8%)	4 (57.1%)	
Diabetes, n (%)			
Yes	14 (15.4%)	0 (0%)	0.59[b]
No	77 (84.6%)	7 (100%)	
Dyslipidemia, n (%)			
Yes	20 (22%)	2 (28.6%)	0.65[b]
No	71 (78%)	5 (71.4%)	
Number of remaining teeth, median (IQR)	24 (7.0)	27 (8.0)	0.27[a]
Denture user, n (%)			
Yes	43 (47.3 %)	2 (28.6%)	0.45[b]
No	48 (52.7%)	5 (71.4%)	

Data were analyzed using the [a]Mann-Whitney U test or [b]Fisher's exact test. *H. pylori*, *Helicobacter pylori*; IQR, interquartile range.

that oral *H. pylori*-positive participants may have poorer dental hygiene than oral *H. pylori*-negative participants.

Logistic regression analysis with oral H. pylori as the dependent variable. Logistic regression analysis was conducted as independent variables in univariate analysis (i.e., variables with a P-value <0.2) and with oral *H. pylori* as the dependent variable. The results of logistic regression analysis are presented in Table III. There was no significant association between oral *H. pylori* and ≥4 mm deep periodontal pockets with BOP or plaque control record score.

Correlation between oral H. pylori DNA copy numbers and plaque control record scores. The present study then calculated the *H. pylori* copy numbers per each 1 µl DNA sample from the participants who were positive for oral *H. pylori*. The median *H. pylori* copy number was 56.8 (IQR, 39.2) copies/µl. The scatter plot illustrates correlation between oral *H. pylori* DNA copy numbers and plaque control record scores (Fig. 3). There was no significant correlation between the oral *H. pylori* copy numbers and plaque control record scores, as demonstrated in Table IV.

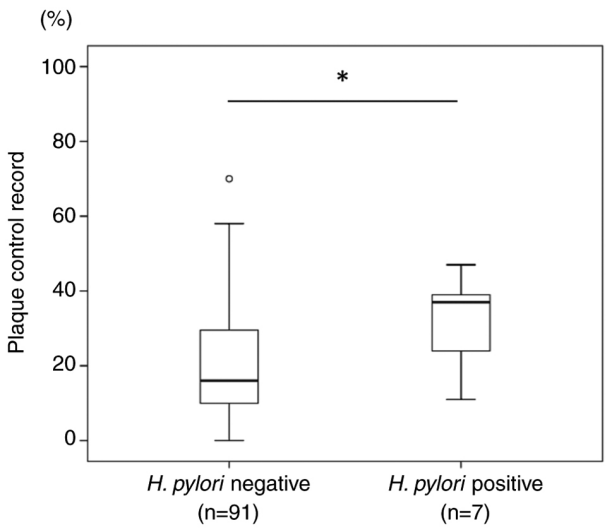

Figure 2. Plaque control record scores in *H. pylori*-positive and *H. pylori*-negative participants. *P=0.04 as indicated, according to the Mann-Whitney U test. *H. pylori, Helicobacter pylori*.

Discussion

The prevalence rate of oral *H. pylori* infection varies widely from 1 to 87% in the population, including among children, young, middle-aged and older individuals, as previously reported since 2016 (7,12-21). The majority of studies investigated the positive rate of oral *H. pylori* DNA using PCR. Oral samples included dental plaque, dental pulp, saliva and swabs from the tongue dorsum. It is speculated that variation

Table II. Periodontal condition of the study participants and its association with the presence of oral *H. pylori* DNA.

Clinical periodontal variables	*H. pylori*-negative (n=91)	*H. pylori*-positive (n=7)	P-value
Probing pocket depth, n (%)			
<4 mm	18 (19.8%)	0 (0%)	0.6[a]
≥4 mm and <6 mm	44 (48.4%)	4 (57.1%)	
≥6 mm	29 (31.9%)	3 (42.9%)	
BOP (%) (IQR)	5.0 (10.0)	3.0 (12.0)	0.78[b]
≥4 mm periodontal pocket with BOP, n (%)			
Yes	45 (49.5%)	6 (85.7%)	0.11[a]
No	46 (50.5%)	1 (14.3%)	
≥6 mm periodontal pocket with BOP, n (%)			
Yes	14 (15.4%)	2 (28.6%)	0.32[a]
No	77 (84.6%)	5 (71.4%)	

Data were analyzed using the [a]Fisher's exact test or [b]Mann-Whitney U test. *H. pylori*, *Helicobacter pylori*; IQR, interquartile range; BOP, bleeding on probing.

Table III. Logistic regression analysis with oral *H. pylori* as the dependent variable.

Clinical variables	Odds ratio	95% confidence interval	P-value
≥4 mm periodontal pocket with BOP	5.92	0.66-53.2	0.11
Plaque control record	1.05	0.99-1.1	0.08

H. pylori, *Helicobacter pylori*; BOP, bleeding on probing.

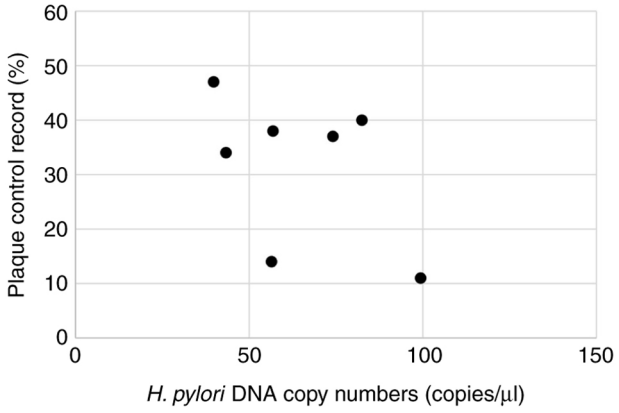

Figure 3. Scatter plot illustrating the correlation between *H. pylori* DNA copy numbers and plaque control record scores. *H. pylori*, *Helicobacter pylori*.

in the age and regional characteristics of the participants, and sample collection method may have affected the positive rate of oral *H. pylori*. Additionally, periodontal inflammation may be implicated in the high prevalence rate of oral *H. pylori* as oral *H. pylori* was more frequently detected in individuals with periodontitis than in those without periodontitis (22,23). Furthermore, the detection sensitivity of PCR and the specificity of PCR primers may have varied in each study.

It is considered that the chronic inflammation of periodontal tissues is involved in persistent infection with oral *H. pylori*. In the present study, the oral *H. pylori*-positive participants exhibited a higher percentage of ≥4 mm periodontal pockets with BOP than the oral *H. pylori*-negative participants. However, there was no significant association between oral *H. pylori* and deep periodontal pockets with BOP (i.e., active periodontitis). A number of participants had mild periodontal inflammation as the participants regularly received supportive periodontal therapy. Therefore, relatively mild periodontal inflammation may have been associated with a low positive rate of oral *H. pylori* in the present study. Therefore, additional studies are required to clarify the association between oral *H. pylori* and moderate to severe periodontitis.

Previous studies have reported that oral *H. pylori* can be detected in subgingival dental plaque (14,19,21). Thus, *H. pylori*, a component of the subgingival microbiome, may play a pathogenic role in the periodontium, as well as a carcinogenic role in the stomach. However, the biological mechanisms by which *H. pylori* is involved in periodontal inflammation have not yet been elucidated. *H. pylori* contributes to the induction of inflammatory cytokines, such as IL-17, which can facilitate chronic periodontal inflammation (24,25). *Porphyromonas gingivalis* (*P. gingivalis*), a Gram-negative oral anaerobe, is a major periodontopathic bacteria (26). *P. gingivalis* was detected in the oral cavity of ~50% of

Table IV. Correlation between oral *H. pylori* DNA copy numbers and plaque control record scores.

	Oral *H. pylori* DNA copy numbers	
Variables	Spearman's rank correlation coefficient	P-value
Plaque control record scores	-0.36	0.43

H. pylori, *Helicobacter pylori*.

middle-aged and older Japanese adults (27). *P. gingivalis* was more frequently found in *H. pylori*-positive dental plaque than in *H. pylori*-negative dental plaque (18). These results indicate that *H. pylori* may be involved in the acceleration of periodontal inflammation by inducing inflammatory cytokines. However, the potential role of *H. pylori* in periodontitis has not yet been elucidated. Additionally, it remains unclear whether *H. pylori* enhances cytokine induction in cooperation with *P. gingivalis* in periodontal tissues.

In the present study, the level of dental plaque accumulation was significantly higher in the oral *H. pylori*-positive participants than in the oral *H. pylori*-negative participants, suggesting that participants with oral *H. pylori* exhibit poor oral hygiene. Additionally, *H. pylori* may be detected more abundantly in dental plaque than on the tongue dorsum. Although the present study did not find a significant association between oral *H. pylori* DNA and plaque control record score in the logistic regression analysis, a significant association may be found using a larger study population with dental plaque sampling. These findings highlight the importance of daily oral hygiene practice and regular professional oral care to prevent oral *H. pylori* infection in older adults. However, the detection of oral *H. pylori* DNA is not necessarily associated with *H. pylori* colonization (i.e., active *H. pylori* infection) in the oral cavity. Additionally, it remains unknown whether the prevention of oral *H. pylori* infection contributes to the reduction of gastric *H. pylori* infection. Further research is required to investigate active oral *H. pylori* infection and its associations with gastric *H. pylori* infection.

Tongue coating samples are composed of food debris, oral bacteria, epithelial cells and blood cells. Tongue bacterial populations may be associated with oral health conditions. In a previous study, the authors detected periodontopathic bacteria DNA using swab samples collected from the tongue surface (27). Another group also reported that *H. pylori* DNA was abundantly detected from tongue coating samples (7). Therefore, the present study aimed to collect oral samples from the tongue surface. However, the presence of *H. pylori* DNA in periodontal pockets could not be determined in the present study. Therefore, further research to detect *H. pylori* DNA using subgingival dental plaque is required to clarify the association between *H. pylori* and periodontitis.

The present cross-sectional study had some limitations. First, the present study did not investigate the presence of *H. pylori* DNA in periodontal pockets. Therefore, it is necessary to prove the presence of *H. pylori* using subgingival dental plaque in future studies. Second, it remains unclear whether active *H. pylori* infection is associated with the oral hygiene condition as *H. pylori* colonization could not be examined in the present study. Third, the present study did not investigate gastric *H. pylori* infection or the history of eradication treatments for *H. pylori* infection (i.e., gastric lavage). Therefore, associations between oral *H. pylori* and gastric *H. pylori* remain unknown. Fourth, the presence of oral *H. pylori* in young individuals with healthy periodontal tissue remains unknown. Fifth, it was impossible to match *H. pylori*-positive and -negative cases by adjusting confounding factors such as age, sex and health conditions due to the small number of *H. pylori*-positive participants. Finally, the present study was performed at a single hospital.

In conclusion, the presence of oral *H. pylori* DNA may be associated with poor dental hygiene and periodontal inflammation in older adults. It is critical to maintain good oral health by practicing daily oral care to reduce the risk of oral *H. pylori* infection.

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Availability of data and materials

The data generated in the present study are included in the figures and/or tables of this article.

Authors' contributions

HY performed the experiments and analyzed the data. HS conceived the study, performed the experiments, analyzed the data, and wrote and reviewed the manuscript. HM, NH, HK, YK and YN performed the experiments. TT and MS interpreted the data and supervised the study. KO analyzed and interpreted the data and reviewed the manuscript. HS and KO confirm the authenticity of all the raw data. All authors have read and approved the final manuscript.

Ethics approval and informed consent statement

The Ethics Committee of Hiroshima University approved the study (No. E-1115). All patients agreed to participate in this study and signed the informed consent form.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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