The presence of Helicobacter Pylori in dental plaque of children and their parents: is it related to their periodontal status and oral hygiene?

**ABSTRACT**

**Aim** To investigate the possible presence of H. pylori in subgingival dental plaque of children with upper gastrointestinal symptoms, as well as of their parents and to detect any association between the presence of H. pylori and oral hygiene together with the periodontal status of children and their parents.

**Materials and methods** The study comprised of 35 children with upper gastrointestinal symptoms, aged 4 to 14 years and 45 family members (mothers and/or fathers). Gastric biopsies were collected from all children for CLO-test, histology and culture. Serology was used to assess the H. pylori infection status of their parents. Before endoscopy, subgingival dental plaque from children and their parents were collected from 4 healthy and 4 diseased sites, and the clinical indices (gingival index, plaque index, bleeding on probing, pocket depth, loss of clinical attachment) after plaque collection were recorded. Statistics: The Chi-square test was performed to investigate possible differences between children and their parents and logistic regression analysis was used to evaluate the association of parental infection status with that of children.

**Results** 15 out of 35 children (42.86%) were found H. pylori-positive. In 6 out of the 15 infected children (40%) H. pylori was also identified in their subgingival plaque samples, as well as in one among the 20 non infected children. The presence of H. pylori in dental plaque was significantly associated with its presence in the gastric antrum (p=0.0274). H. pylori was identified in the dental plaque of 7 mothers corresponding to children with positive PCR in their dental plaque and of 4 fathers (one corresponding with his child found H. pylori positive in dental plaque). Children who had H. pylori identified in their dental plaque belonged to families with members also having H. pylori in dental plaque. No significant relationship between periodontal clinical parameters and detection of H. pylori in dental plaque in both children and their parents was found. However, the presence of H. pylori in the subgingival plaque samples was significantly correlated with the parental diseased sites (p=0.02).

**Conclusion** H. pylori was detected in subgingival dental plaque of children and their families, possibly acting as a “reservoir” contributing to the intra-familial spread. Efficient oral hygiene and healthy periodontal status could reduce this transmission.

**Keywords:** Helicobacter pylori; Dental plaque; Children.

**Introduction**

Helicobacter pylori (H. pylori) remains one of the most common bacterial infections in humans, with average rates of 40-50% in western countries rising to >90% in developing countries [Brown 2000; Berroteran et al., 2002].

The mode of transmission of the bacterium is vigorously debated, but most evidence suggests that it occurs from person to person and the risk factors are close contact, crowding and poor sanitation. The theory from person to person mode of transmission is supported from the elevated prevalence and high incidence of infection among institutionalised children and adults and the clustering of H. pylori infection within families [Lambert et al., 1995; Dominici et al., 1999].

The oral cavity supports many ecological niches, some of which provide the microaerophilic environment necessary for H. pylori survival and multiplication. According to this aspect, saliva and dental plaque have been implicated as a reservoir for transmission and a possible source for infection of this micro-organism and may represent risk factors for gastrointestinal re-infection and ulcer relapse after eradication with antibiotic therapy [Dowsett et al., 2003; Gurbut et al., 2003]. The hypothesis that H. pylori is a permanent or transient member of the oral microflora is still controversial. Data from adults are conflicting and in a number of attempts to culture the organism the results were negative [Bemander et al., 1993], but in the most of them the results were positive [Ferguson et al., 1993; Khandaker et al., 1993].

In Greece, H. pylori infection is common among the paediatric population and intrafamilial spread is considered high [Roma et al., 1995; Roma et al., 1999]. In another study a significantly higher prevalence of H. pylori infection in spouses of H. pylori-positive patients with duodenal ulcer was observed, when compared to spouses of H. pylori-negative patients and this observation was assessed on the base of ribotyping; similar rRNA gene patterns were demonstrated in 44.44% of cohabiting couples [Georgopoulos et al., 1996]. Additionally, Roma et al. [2009] found that in all H. pylori-positive children at
least one more family member, always included a parent, who was infected and authors concluded that family could be the main source of H. pylori infection in children. However, in Greece data regarding the detection of H. pylori in dental plaque of adults and children or within families are still lacking. Therefore, the role of dental plaque on interfamilial spread of H. pylori is still unknown.

We aimed to investigate the possible presence H. pylori in dental plaque of children with upper gastrointestinal symptoms, who underwent endoscopy, as well as of their parents' and to correlate the oral hygiene together with the periodontal status with the H. pylori status in the dental plaque.

Subjects and methods

Study population
The study comprised of 35 children (16 boys) with upper gastrointestinal symptoms, aged 4 to 14 years (mean age 9.25 ± 2.51 years) who were referred to the Department of Periodontics of the Dental School of Athens-Greece from the Gastroenterology Unit of the First Department of Paediatrics of University of Athens, “Aghia Sophia” Children’s Hospital in Athens. Moreover, 45 parents of the participating children (31 mothers and 14 fathers, aged 25 to 48 years, mean 35.25 ± 3.38 years) participated the study. Exclusion criteria included treatment with antibiotics less than 2 months prior to enrolment in the study, previous treatment to eradicate H. pylori and children with mental deficiency or chronic diseases.

After obtaining an informed consent from the patient’s parents, gastric biopsy specimens and gastric juice were obtained from each child in order to assess the H. pylori infection status. At the day of endoscopy and sample collection children and from at least one of the accompanying parents. Additionally, blood samples for serum anti-H. pylori IgG antibodies assessments were drawn from the participating parents. The study was approved by the local ethical committee.

Dental plaque sampling

The subgingival dental plaque samples from children and their parents were collected by one examiner (P.P.) before gastrointestinal endoscopic examination. The sampling sites for each individual participant were the buccal or interproximal sites in 4 healthy and in 4 diseased sites. Prior to plaque sampling, plaque was recorded as present or absent; then supragingival plaque was removed carefully with sterile curettes and the surfaces were dried and isolated by cotton rolls. Filter paper strips were placed in the gingival sulci/pockets for 5 seconds and the subgingival dental plaque samples were taken from each patient. The healthy sites samples were pooled together and so were the diseased sites samples. Care was taken to avoid mechanical trauma and strips contaminated with blood were discarded.

Clinical periodontal examination

Immediately after plaque collection, a standard full-mouth periodontal examination with periodontal probe WHO in order to detect the periodontal treatment needs for each participant was performed. Secondly, clinical parameters were recorded to assess periodontal health for each tooth that was used for sampling. A manual periodontal probe (UNC 15, Hu-Friedy) was used for the clinical findings which were rounded up to the nearest millimeter.

During the clinical examination we evaluated:
• Gingival index (GI, Loe and Silness 1963) around each tooth in order to evaluate the severity of inflammation at the cervical margin, recording from 0 (no inflammation) to 3 (severe inflammation, ulceration, spontaneous bleeding).
• Plaque Index (PII, Silness and Loe 1964) around each tooth in order to measure the thickness of plaque at the gingivae on the tooth surface, the scores are from 0 (none plaque visible to the eye) to 3 (gross accumulation of plaque).
• Bleeding on probing (BOP) around each tooth in order to record the presence or absence of bleeding up to 15 seconds after gentle probing.
• Probing Depth (PD) around each tooth in order to record the distance between the gingival margin and the bottom of the sulci/pocket.
• Clinical Attachment Level (CAL) around each tooth in order to record the distance between cemento-enamel junction and the bottom of the sulci/pocket.

All clinical examinations were performed by one examiner (A.T), calibrated to reduce intraexaminer error (Kappa >0.75) and to establish reliability and consistency prior to the beginning of the study.

Endoscopy

Upper gastrointestinal endoscopy was performed by three paediatric gastroenterologists using an Olympus GIF-XP20 endoscope. The endoscope and the biopsy forceps were cleaned and disinfected with glutaraldehyde after each use. Children were examined under general anaesthesia or sedation following intravenous administration of midazolam. One duodenal and at least three antral (for histological assessment, CLO test and culture) biopsies were taken from each patient. During endoscopy gastric juice was withdrawn for the detection of H. pylori by PCR. Children were considered as H. pylori-infected when they had positive culture or positive both histology and CLO test.

Histology

The biopsy samples were fixed in 10% neutral formalin, embedded in paraffin and sections were stained with haematoxylin-eosin and modified Giemsa or Masson trichrome. Histological analysis was performed by a single histopathologist, who assessed the presence of H. pylori and the degree of gastric mucosa inflammation in a blind manner.

Culture

Antral biopsy specimens were cultured under microaerophilic conditions onto Wilkins Chalgren agar medium, supplemented with horse blood and antibiotics. Colonies of H. pylori were confirmed by morphology and the catalase and urease tests. Initial efforts to isolate H. pylori from dental plaque samples were not successful due...
to overgrowth of contaminating bacteria of the oral flora.

**PCR assay methods**

DNA was extracted from subgingival dental plaque samples by a phenol chloroform extraction method [Santamaria et al., 1999]. Two PCR methods targeting two *H. pylori* genes were used for the detection of *H. pylori* in subgingival dental plaque. Only dental samples positive by both methods were considered *H. pylori* positive.

The first PCR targeted the urease A gene of *H. pylori* and was performed as previously described [Clayton et al., 1992]. The amplified products were separated by electrophoresis on a 2% agarose gel and then stained with ethidium bromide. The expected size of PCR product was 411bp. The PCR product was further subjected to restriction endonuclease AluI digestion for final confirmation.

The second PCR method used was a nested PCR targeting the 16S ribosomal RNA genes of *H. pylori* as described elsewhere [Mapstone, 1993]. Only subgingival samples giving products of the expected 109bp size were considered positive for the presence of *H. pylori* genomic DNA.

**H. pylori antibody determination**

IgG antibodies against *H. pylori* were determined by an in-house ELISA using an ultracentrifuge supernatant of whole cell sonicates of 5 *H. pylori* isolated in Greece [Pateraki et al., 1990]. The sensitivity and specificity of the method was 91% and 85% respectively in the Greek adult population (data not shown).

**Statistical analysis**

The association of a child’s *H. pylori* infection status with the parental one was estimated by Chi-square tests; Fisher’s exact test (two-sided) was used for small numbers (expected cell numbers were <5). The same statistical tests (with the Yates correction) the presence of *H. pylori* in the studied healthy or diseased sites of children and their parents were used.

Logistic regression analysis models were fit to evaluate the odds ratios (OR for 95% C.I., coefficient interval) in order to describe the independent association of maternal and parental infection status with the child’s infection status (dependent variable).

Results were considered statistically significant at 5% significance level (p<0.05).

**Results**

Gastric biopsy specimens of 15 out of 35 children (42.86%) were *H. pylori* positive according to our definition, while in 9/15 (60%) *H. pylori* was identified in gastric juice by nested PCR. *H. pylori* was detected by PCR in the subgingival dental plaque samples of 6 out of 15 (40%) *H. pylori*-positive and of 1 out of 20 (5%) *H. pylori*-negative children. The presence of *H. pylori* in the subgingival dental plaque was significantly associated with *H. pylori* detection in gastric samples (Fisher’s test, p=0.0274) and in gastric juice (Fisher’s test, p=0.039).

On the basis of serology, 33.3% of mothers were considered positive for *H. pylori* infection. *H. pylori* was detected in subgingival dental plaque in 7 (22.5%) mothers and 5 of them were also positive by serology (Fisher’s test, p=0.0173). In the two cases of dental PCR positive, serology negative mothers, the dental PCR was also positive for the respective husbands.

On the basis of serology, 55% of fathers were found positive for *H. pylori*, which were also detected in their subgingival dental plaque in 4 out of 14 (28.57%) fathers tested. Mothers’ and fathers’ *H. pylori* status as determined by serology did not correlate significantly (Fisher’s test, p=1.00), while mothers’ and fathers’ dental carriage correlated significantly (Fisher’s test, p=0.0140).

Table 1 summarises the association of children’ *H. pylori* infection status with the *H. pylori* dental carriage of the corresponding parent. Out of *H. pylori*-infected children, 42.8% had mothers and 33.3% fathers with dental PCR positive for *H. pylori*, while for non-infected children it was 5.9% and 25% respectively. *H. pylori* was detected by PCR in both parents of one biopsy-negative child that also had *H. pylori* in his subgingival dental plaque samples. In general, children who had *H. pylori* identified in their subgingival dental plaque belonged to families having also *H. pylori* in subgingival dental plaque. However, it seems that the presence of *H. pylori* in mother’s subgingival dental plaque and not the father’s was associated with *H. pylori* infection in the respective child. By the logistic regression analysis, it was determined that mothers with positive dental PCR have 2.5 times more possibility to infect their children with *H. pylori* (OR=2.571 95%CI=1.354 to 4.885); however this possibility is only 1.2 times for the fathers with positive dental PCR (OR=1.250 95%CI=0.362 to 0.319).

Table 2 and Table 3 summarise respectively the relationship of findings between the periodontal clinical data of children or their parents and the detection of *H. pylori* in subgingival plaque samples, collected from healthy and diseased sites. In children, we did not find any statistically significant relationship between periodontal

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**Table 1 - Parental H. pylori dental carriage in relation to H. pylori status of children.**

<table>
<thead>
<tr>
<th></th>
<th>No.</th>
<th>H. pylori carriage</th>
<th>Fisher (p)</th>
<th>LR</th>
<th>Odds r. (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mother dental PCR (n=31)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>7</td>
<td>6/14 (42.8%)</td>
<td>1/17 (5.9%)</td>
<td></td>
<td>2.081 (1.354 to 4.885)</td>
</tr>
<tr>
<td>Negative</td>
<td>24</td>
<td>8/14 (57.2%)</td>
<td>16/17 (94.1%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Father dental PCR (n=14)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive</td>
<td>4</td>
<td>2/6 (33.3%)</td>
<td>2/8 (25%)</td>
<td></td>
<td>1.000 (0.362 to 0.319)</td>
</tr>
<tr>
<td>Negative</td>
<td>10</td>
<td>4/6 (66.7%)</td>
<td>6/8 (75%)</td>
<td></td>
<td>1.250 (0.362 to 0.319)</td>
</tr>
</tbody>
</table>

**lr** = Logistic regression - Odds ratio
Clinical and the detection of H. pylori either in healthy or diseased sites (Table 2). Similar results were found for their parents (Table 3), with the only exception of the dental plaque index, which had a statistically significant relationship with the presence of H. pylori in the subgingival plaque samples of diseased sites ($\chi^2=7.48$, p=0.02).

**Discussion**

In the present study H. pylori was detected in subgingival dental plaque samples of 40% biopsy-proven H. pylori-positive children but only in 5.3% of H. pylori-negative children. The H. pylori infection status of the mother and not of the father as determined by serology, correlated significantly with the H. pylori infection status of the child. H. pylori was also detected—at least in one member—in subgingival dental plaque samples in 40% of families of H. pylori-positive children.

It is interesting that children who had H. pylori identified in their subgingival dental plaque belonged to families who also had H. pylori in subgingival dental plaque, both of them collected from periodontal diseased sites. It was detected in both parents of the biopsy negative child who had also H. pylori in his dental plaque. In general, children living in families in which one, or both parents were infected, had a significantly higher rate of infection than children with no parents infected.

To our knowledge this is the first study in Greece investigating the incidence of H. pylori in subgingival dental plaque of children undergoing endoscopy and their families and suggesting that the oral presence of H. pylori may play a critical role in gastric infection and not only an intermittent and transient role. Our results are in agreement with the findings of other studies suggesting that H. pylori in dental plaque may represent a risk factor for gastrointestinal re-infection and ulcer relapse after antibiotic therapy [Gurbuz et al., 2003; Yang et al., 2003] or that oral cavity is a reservoir for gastric infections [Dowsett et al., 2003].

Studies concerning the isolation of H. pylori from the dental plaque are controversial. Our study is in agreement with previous ones which have been successful in detecting H. pylori by PCR from dental plaque of infected subjects, but the positivity varied from 1% to 88% [Brown, 2000]. However, other investigators have not been able to isolate H. pylori from dental plaque or have only occasionally reported the presence of this organism in the mouth [Hardo et al., 1995; Oshowo et al., 1998]. Our findings are in contrast with those of a previous study concerning patients with duodenal ulcer and hospital employees infected by H. pylori as determined by urea breath test, which did not find any correlation between H. pylori status in saliva, dental plaque and periodontal status, as determined by PCR [Dore-Davin et al., 1999].

Helicobacter pylori infection is mainly acquired in the early childhood, and the risk of infection declines rapidly after five years of age, while frequently the acquisition age is lower in developed countries [Rothenbacher et al., 1999; Rowland et al., 2006]. The main transmission routine is family members, and especially infected mothers, may play a key role in the transmission of H. pylori within the family. Mothers have a longer and closer contact with their children. By contrast, the role of the father is thought to be minor in terms of the risk of H. pylori infection [Zhou et al., 2000].

Probably the initial site of infection is the oral cavity, in which H. pylori persists in low numbers for a long time and does not colonize the stomach. The most likely routes of oral infection include saliva, dental plaque and refluxed gastric contents or vomit. An intriguing hypothesis is if the dental plaque, as a biofilm on the tooth surface which has been defined as a diverse community of microorganisms, is a significant factor that indicates whether oral H. pylori may or may not cause gastric infection [Dowsett et al., 1999; Choudhury et al., 2003]. Especially, if the accumulation of supragingival dental plaque develops

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**TABLE 2** - Relationship between the periodontal clinical parameters of the children and detection of H. pylori in subgingival plaque samples by nested-PCR from periodontal healthy and diseased sites.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Healthy sites</th>
<th>Diseased sites</th>
<th>$\chi^2$</th>
<th>L.S. (p)</th>
<th>$\chi^2$</th>
<th>L.S. (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pocket depth</td>
<td>4.85</td>
<td>0.08</td>
<td>5.23</td>
<td>0.07</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loss of clinical attachment</td>
<td>4.85</td>
<td>0.08</td>
<td>5.23</td>
<td>0.07</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gingival index</td>
<td>1.23</td>
<td>0.53</td>
<td>0.72</td>
<td>0.39</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plaque Index</td>
<td>0.78</td>
<td>0.67</td>
<td>1.37</td>
<td>0.50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bleeding on probing</td>
<td>0.17</td>
<td>0.67</td>
<td>0.19</td>
<td>0.64</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

L.S. = Level of significance
$\chi^2$ = Chi-square test

**TABLE 3** - Relationship between the periodontal clinical parameters of parents and detection of H. pylori in subgingival plaque samples by nested-PCR from periodontal healthy and diseased sites.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Healthy sites</th>
<th>Diseased sites</th>
<th>$\chi^2$</th>
<th>L.S. (p)</th>
<th>$\chi^2$</th>
<th>L.S. (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pocket depth</td>
<td>5.48</td>
<td>0.13</td>
<td>5.25</td>
<td>0.15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loss of clinical attachment</td>
<td>5.48</td>
<td>0.13</td>
<td>5.25</td>
<td>0.15</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gingival index</td>
<td>2.70</td>
<td>0.43</td>
<td>3.88</td>
<td>0.14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plaque Index</td>
<td>3.85</td>
<td>0.14</td>
<td>7.48</td>
<td>0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bleeding on probing</td>
<td>1.48</td>
<td>0.22</td>
<td>0.20</td>
<td>0.64</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

L.S. = Level of significance
$\chi^2$ = Chi-square test
gingival inflammation, some of the coronal epithelium is lost and consequently the plaque biofilm begins to extend subgingivally, junctional epithelium is no longer attached closely to the tooth and has transformed into pocket epithelium forming initially gingival “false” pocket and secondly, after the apical migration of this, the true periodontal pocket. So, since periodontal disease in the mouth plays a critical role in survival and multiplication for H. pylori then the periodontal pocketing provides the microaerophilic environment of its colonisation. Previous studies indicate that in periodontitis and periodontal pockets or in other clinical conditions such as oral mucosal ulcerations, the number of H. pylori increases and reaches levels sufficient to cause gastric infection [Slomiany et al., 2002; 2003].

Accordingly, the aim of our study was to investigate the presence of H. pylori in subgingival dental plaque of children as well as of their parent’s and its relationship with the oral hygiene and the periodontal status of the children and their parents. Our efforts to isolate H. pylori in culture were not successful due to overgrowth of contaminating bacteria in the growth medium not allowing proper detection. The inability to culture H. pylori from oral samples could be attributed to the insufficient number of cells for detection, the presence of inhibitors, the presence of uncultivable but viable coccoid forms in polymicrobial oral specimens and possibly a more transient colonization of the oral cavity by H. pylori.

Therefore, we used polymerase chain reaction (PCR), a sensitive assay technique and especially the nested PCR assay technique able to enhance sensitivity and specificity for the detection of H. pylori in clinical specimens [Brow 2000; Berroteran et al., 2002; Chaudhry et al. 2010]. We employed a single and a nested PCR method to detect the presence of H. pylori in each dental plaque sample in a blind matter. The samples were collected before endoscopy to prevent oral contamination caused by gastroesophageal reflux at the time of endoscopy. To avoid contamination, stringent procedures were employed when performing PCR. Only samples positive by both methods were considered positive. Dental plaque may harbor more than 350 different bacterial species, and H. pylori-like organisms in the oral microflora may give false-positive PCR results on analysis of plaque material. The possibility of a bacterium of the oral flora other than H. pylori to carry both, the 16S RNA and the urease A genes is extremely low. However the possibility of false-positive results due to presence of these genes in different bacteria cannot be ruled out. In our hands the PCR methods used gave similar results, with the nested PCR slightly more sensitive, giving three more positive results than the urease A gene PCR.

In the present study a statistically significant relationship between dental plaque index and detection H. pylori in subgingival dental plaque samples, which were collected from diseased sites in parents, was found. The above finding showed that the presence of subgingival dental plaque and consequently the presence of periodontal inflammation were correlated with the presence of H. pylori in the subgingival plaque samples. Our results are in agreement with the findings of Peach et al. [1997], who in a study on Australian adults found that positive H. pylori status was associated with a high plaque score and with visiting the dentist less than once a year. However, our findings are in contrast with the results of Namiot et al. [2010], who in their study in patients aged 19-74 years found that the presence of H. pylori in dental plaque of natural teeth was not associated with oral health or dental plaque removal practice. Similarly, in contrast to the findings of the present study, Hardo and al. [1995] did not find any association between H. pylori infection and time between visits in the dentist, numbers of times per week that patients brushed their teeth, oral hygiene index and periodontal status of dyspeptic patients, while Berroteran et al. [2002] have demonstrated that there was no correlation between H. pylori infection and dental hygiene, dental caries, periodontal disease or use of dentures. The absence of association between periodontal pocket depth and detection of H. pylori in subgingival dental plaque by nested PCR is in agreement with the findings of Dowsett et al. [1999]. However these investigators found a significantly positive relationship between fingernail and tongue and concluded that oral carriage play a significant role in the transmission of the infection. These conflicting results regarding the frequency of H. pylori detection in dental plaque could be due to differences in the methods of subgingival or supragingival dental plaque samples collection, the PCR assay detection technique, the ethnic differences in the study populations groups. Also, this could be due to the oral hygiene status and the status of the periodontal tissues, health or disease, the eating habits in relation or not to socioeconomic class, dental individual care and oral contamination caused by gastro-oesophageal reflux or to the presence of few other types of bacteria in the mouth or in the dental plaque that inhibits the growth of H. pylori.

In the present study we examined the hypothesis that oral to oral transmission of H. pylori between children and their parents may accrue by subgingival dental plaque, especially if the periodontal health of them has been lost. According to the results of this study, we could support the hypothesis that daily toothbrushing especially of parents, could prevent periodontal disease, which could be one of parameters contributing to the prevention of the interfamilial spread of H. pylori.

Conclusion

The present study gives further evidence that H. pylori is detected in subgingival dental plaque of children and their families and that may play an important role, acting as a “reservoir” for the intra-familial spread and transmission. The key contributing to avoidance of interfamilial transmission of H. pylori could be meticulous oral hygiene and healthy periodontal status, since the presence of subgingival dental plaque may be “instrumental” in infection spread.

References

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