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Persistence of *Helicobacter pylori* in the oral cavity after systemic eradication therapy

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Abstract

Aim: The present study aimed to evaluate if the oral cavity of chronic periodontitis patients can harbor *Helicobacter pylori* after systemic eradication therapy.

Materials and Methods: Samples of 30 patients (15 with gingivitis and 15 with chronic periodontitis) positive for *H. pylori* in the stomach were evaluated. Samples were collected 3 months after triple systemic antibiotic therapy from saliva, microbiota from the dorsum of the tongue, supra- and sub-gingival plaque as well as gastric biopsies. DNA of each sample was extracted by the boiling method and used as a template in polymerase chain reaction with the primers JW22/23.

Results: Eighteen patients (60%) harboured *H. pylori* in their mouths. Five patients (16.6%) were positive in saliva, two (6.6%) on the dorsum of the tongue, nine (30%) in supra-gingival plaque, 14 (46.6%) in sub-gingival plaque and three (10%) in the stomach. There was no statistically significant difference between study groups.

Conclusion: Eradication of *H. pylori* after therapy was more effective for the stomach than for the mouth ($p < 0.001$). Mouths of patients with gingivitis or with chronic periodontitis, who are positive for *H. pylori* in their stomachs, may be considered as reservoirs of these bacteria.

Key words: gastritis; gingivitis; *Helicobacter pylori*; periodontitis; plaque

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Helicobacter pylori are microaerophilic Gram-negative, spiral-shaped bacteria, originally classified as *Campylobacter pyloridis* (Marshall & Warren 1984). This microorganism has been considered an important gastro-intestinal pathogen associated with gastritis, gastric and duodenal ulcerations (Lee et al. 1993), and the higher risk of gastric carcinoma (Parsonnet et al. 1991).

H. pylori gastric infection can be successfully treated with systemic antibiotic therapy. The percentage of eradication obtained ranges between 80% and 90%, when triple therapy (combination of antimicrobials, antibiotics and drugs that increases the gastric pH) is used for 7–10 days (Gabryelewicz 1996, Harris 1998).

Nevertheless, gastric re-infection by these bacteria may be observed in some patients (Patchett et al. 1992). Therefore, studies have been carried out to evaluate sites other than the stomach that could be implicated in the eventual transmission of the bacteria and in the re-infection process. The oral cavity has been pointed out as a region with great potentiality to harbor *H. pylori* and originate gastric re-infection (Mapstone et al. 1993), and has been the focus of a number of studies which presented inconclusive results (Banatvala et al. 1993, Mapstone et al. 1993, Nguyen et al. 1993, Cammarota et al. 1996, Oshowo et al. 1998, Song et al. 1999, Miyabayashi et al. 2000, Avcu et al. 2001).

H. pylori have already been observed in saliva (Ferguson et al. 1993, Mapstone et al. 1993, Oshowo et al. 1998, Miyabayashi et al. 2000, Song et al. 2000a, b, Umeda et al. 2003, Gebara et al. 2004), in the microbiota from the dorsum of the tongue (Namavar et al. 1995, Oshowo et al. 1998, Birek et al. 1999, Özdemir et al. 2001, Gall-troselj et al. 2001), in dental plaque (ÖShames et al. 1989, Banatvala et al. 1993, Mapstone et al. 1993, Khandaker et al. 1993, Olsson et al. 1993, Khandaker et al. 1993, Cammarota et al. 1996, Oshowo et al. 1998, Song et al. 1999, Miyabayashi et al. 2000, Özdemir et al. 2001, Avcu et al. 2001), in supra-gingival plaque (Pytko-Polonczyk et al. 1996, Song et al. 2000a, b, Umeda et al. 2003, Gebara et al. 2004), in sub-gingival plaque (Pytko-Polonczyk et al. 1996, Riggio & Lennon 1999, Gebara et al. 2004), on the surface of oral ulcerations (Leimola-Virtanen et al. 1995, Mravak-Stipetic et al. 1998, Birek et al. 1999) and on the surface of oral neoplasias (Okuda et al. 2000).

Although the bacteria have been detected in different sites of the oral cavity, there are some unanswered ques-

tions such as whether *H. pylori* are resident or transient microorganisms of the oral cavity (Pavelic et al. 2000) and whether the oral cavity could be considered as a reservoir of these bacteria even after eradication therapy (Mapstone et al. 1993, Miyabayashi et al. 2000, Umeda et al. 2003).

The aim of this study was to evaluate whether the oral cavity of periodontitis patients, which had been submitted to systemic eradication therapy of *H. pylori* from the stomach, can be considered as a reservoir for the bacteria even after that treatment.

Materials and Methods

Patient selection and clinical evaluation

Two hundred and thirteen patients from the Gastroenterology Division of the Universidade Federal de São Paulo (UNIFESP) were examined and 30 dentate patients from 21 to 62 years old (11 males and 19 females) were selected. The selected patients signed a volunteer consent, before undergoing research procedures, agreeing to their participation in the clinical protocol.

Only patients over 18 years of age positive to the urease test and presenting gingivitis or chronic periodontitis were included in this study.

Exclusion criteria were the use of antimicrobials, inhibitors of proton bomb, H_2 blockers and bismuth derivates within the 2 months before the clinical protocol, previous eradication therapy, upper digestive haemorrhage, hypersensitivity against amoxicillin, clarithromycin or lansoprazole, immunosuppression; pregnancy and breast feeding.

All patients answered a health questionnaire and were submitted to a clinical periodontal evaluation in order to be consecutively entered to one of two groups (gingivitis, G or chronic periodontitis, P). One investigator assessed all clinical parameters as follows: clinical attachment level, probing depth and bleeding on probing. Fifteen patients presenting probing depth $\leqslant 3$ mm and at least four sites exhibiting bleeding on probing were included in the gingivitis group. Fifteen patients exhibiting bleeding on probing and at least four teeth with a probing depth $\geqslant 5$ mm were entered to the chronic periodontitis group. Patients with healthy gingiva and aggressive periodontitis patients were not included.

Subjects were submitted to a second upper digestive endoscopy, in which four biopsies were obtained from the antral region of the stomach. Two samples were placed in flasks containing 10% urea in KH_2PO_4 10 mmol/l and 1% red phenol (pH 4.5), for evaluation of urease production, within 48 h. The two other samples were placed in flasks containing 0.5 of Tris EDTA buffer and stocked in -20°C freezer for posterior microbiological evaluation.

After the second endoscopy, all patients underwent triple therapy (lansoprazole, 30 mg; amoxicillin, 1 g; and clarithromycin, 500 mg, two times a day for 7 days) in order to eradicate *H. pylori* from the stomach.

Three months after the systemic treatment, non-stimulated saliva samples produced during 1 min. were collected. A sterile wood wedge was used to collect microbiota from the tongue by scraping the posterior third of its dorsum. Supra-gingival plaque was collected from two different teeth using a Mac Call 13/14 scaler. Sub-gingival plaque was obtained after removal of supra-gingival plaque, by inserting two sterile paper points into the sulcus/pocket for 20 s. Sub-gingival sampling sites in periodontitis patients exhibited clinical probing depth $\geqslant 5$ mm and bleeding on probing, whereas in gingivitis subjects, the sampling sites showed clinical probing depth $\leqslant 3$ mm and bleeding on probing.

After collection of the samples, the patients were submitted to a third upper digestive endoscopy that followed the same parameters of the second endoscopy as previously described.

Each sample was immediately placed into a sterile tube containing 0.5 ml of Tris EDTA buffer. All samples were identified, transported in ice and stocked at -20°C, for posterior microbiological analyses.

After the endoscopy, patients complying with the study protocol received oral hygiene orientation as well as plaque and calculus removal with an ultrasonic device.

Microbiological procedures

Microbiological procedures were performed as previously described (Gebara et al. 2004). Briefly, DNA was extracted from the clinical samples by the boiling method and the detection of *H. pylori* DNA was performed by polymerase chain reaction (PCR) using the specific primers for the species JW 22/23, homologous to 16SrRNA (Riggio & Lennon 1999). Water was used as negative control for PCR reactions and DNA was extracted from reference strain *H. pylori* ATCC (American Type Culture Collection) 43629 as positive control.

Statistical Analysis

Chi square (χ^2) and Fisher's exact tests were used in order to verify any association between gingivitis and periodontitis groups and the presence of *H. pylori* in the oral cavity, in saliva, in the microbiota of the dorsum of the tongue, in supra- and sub-gingival plaque, and in the biopsies. McNemar test was used to verify differences in the prevalence of *H. pylori* before (Gebara et al. 2004) and after the treatment.

Results

H. pylori were detected by the urease test and PCR in all biopsies from the stomach prior to therapy. However, only three subjects (10%) remained positive for the urease test and for PCR detection in biopsies, after triple therapy. Two of these patients belonged to group P and one to group G. The same patients were positive for *H. pylori* DNA in the biopsies from the stomach after the treatment. All the other patients were negative for *H. pylori* DNA in the stomach after the therapy.

There was no statistically significant difference between the presence of *H. pylori* in the oral cavity before (Gebara et al. 2004) and after the treatment (Table 1, $p = 0.26$).

After systemic triple therapy, the bacterial DNA was detected in saliva, in the microbiota of the dorsum of the tongue, in supra- and sub-gingival plaque. The distribution of the patients, in group P or G, according to the detection of the DNA of *H. pylori* in the different sites of the oral cavity, can be seen in Table 1.

When the oral cavity was evaluated, 60% of the patients (18 patients) were positive for *H. pylori* DNA after the treatment. Eleven of these patients were from group G and seven from group P.

Ninety percent of the patients that harbored *H. pylori* in their stomachs at the beginning of the study did not harbor the bacteria anymore after triple therapy, whereas only 13.3% of the patients (four patients) that harbored the bacteria

Table 1. Patients positive to *Helicobacter pylori* DNA, before and 3 months after triple therapy

		Site					
Period	Group	Saliva, *N* (%)	Microbiota from the dorsum of the tongue, *N* (%)	Supra-gingival plaque, *N* (%)	Sub-gingival plaque, *N* (%)	Oral cavity,* *N* (%)	Stomach,* *N* (%)
Before† triple therapy	G	2 (6.6)	0 (0)	3 (10)	5 (16.6)	7 (23.3)	15 (50)
	P	1 (3.3)	0 (0)	3 (10)	4 (13.3)	6 (20)	15 (50)
After triple therapy	G	3 (10)	2 (6.6)	6 (20)	9 (30)	11 (36.6)	1 (3.3)
	P	2 (6.6)	0 (0)	3 (10)	5 (16.6)	7 (23.3)	2 (6.6)

*Eradication after triple therapy was more effective for the stomach than for the mouth ($p<0.001$ – McNemar test).
†Data from Gebara et al. (2004).
G, gingivitis group; P, chronic periodontitis group; *N,* number of patients.
There was no statistically significant difference between groups G and P (χ^2 and Fisher's exact tests).

in the oral cavity (Gebara et al. 2004) became negative for *H. pylori* DNA in their mouths after triple therapy. When the capacity of triple therapy to eradicate *H. pylori* from the mouth and stomach was evaluated, a statistically significant difference ($p<0.001$) was observed. Triple therapy was highly effective in eradicating *H. pylori* from the stomach but failed to eradicate it from the mouth of the same patients.

Discussion

The objective of this study was to evaluate if the oral cavity of chronic periodontitis patients can harbor *H. pylori* after systemic eradication therapy.

Results have shown that eradication of *H. pylori* after therapy was more effective for the stomach than for the mouth ($p<0.001$).

Before eradication therapy, all patients (100%) were positive to *H. pylori* DNA in their stomachs, whereas 13 (43.3%) of them were positive for bacterial DNA in their mouths. Three patients were positive in saliva, six patients in the supra-gingival plaque, nine in the sub-gingival plaque, and none in the microbiota of the dorsum of the tongue (Gebara et al. 2004).

Interestingly, after triple therapy, 18 patients (60%) were still positive for DNA of *H. pylori* in their mouths. Five patients were positive in saliva, two in the microbiota of the dorsum of the tongue, nine in the supra-gingival plaque, 14 in the sub-gingival plaque and only three (10%) in the biopsies of the stomach. Prevalence of *H. pylori* DNA in the oral cavity showed no statistically significant difference when groups G and P were compared or when baseline and final measurements were compared.

However, it is important to mention that the triple therapy used in this study was highly effective in eradicating the bacteria from the stomach, reaching 27 out of 30 subjects (90%, $p<0.001$), which is in agreement with the literature (Gabryelewicz 1996, Harris 1998).

Differences in the eradication efficacy achieved with triple therapy for the oral cavity and for the stomach could be due to the biofilm (Socransky et al. 1998), which is present in the oral cavity and absent in the stomach.

The biofilm is characterized by a complex structure that involves mixed microcolonies in a glycocalix adherent to solid surfaces (Costerton 1999). This structure provides defense from the host immunological response and from the action of antimicrobial agents (Socransky & Haffajee 2002). Thus, it would be reasonable to assume that the antibiotics effectiveness would be hampered in supra- and sub-gingival plaque.

Another finding of this study was that although triple therapy was able to eliminate *H. pylori* from oral cavity sites, bacterial DNA was observed after treatment, in initially negative sites. This could be explained by the transferring capacity of bacteria from one site to another of the oral cavity (Van Winkelhoff et al. 1988, Quirynen et al. 1996).

There were also patients who were initially negative for the bacteria in their mouths (Gebara et al. 2004) and became positive after treatment. Justification for this could be an eventual contamination of the oral cavity by the endoscope (Cammarota et al. 1996) during the second gastric examination, or by gastric-esophagi reflux, nausea and vomiting (Miyabayashi et al. 2000). In this study, contamination of the oral cavity by *H. pylori* originating from the stomach could only have happened before the eradication therapy, which was highly efficient for the stomach but not for the oral cavity.

However, it should be pointed out that detection by PCR does not implicate in cell viability or pathogenicity (Ge & Taylor 1998). Although the results of the present study do not imply in cell viability, samples were collected 3 months after triple therapy, which considerably reduces the possibilities of still having DNA remnants of bacterial cells killed by treatment because DNase activity is observed in saliva (Yaegaki et al. 1982, 1985).

This study suggests that the oral cavities of patients with gingivitis or chronic periodontitis, positive for *H. pylori* in their stomach, may be considered as reservoirs of *H. pylori* even after systemic triple therapy. Saliva, the microbiota of the dorsum of the tongue, supra- and sub-gingival plaque of these patients can harbor *H. pylori*. The triple therapy used in this study was effective in eradicating *H. pylori* from the stomach of 90% of the subjects, while it failed to eradicate *H. pylori* from the oral cavity of 60% of the subjects.

Because of the inefficiency of triple therapy to eradicate *H. pylori* from the oral cavity and the associated increased risk of gastric re-infection when bacteria is present in the mouth (Miyabayashi et al. 2000, Avcu et al. 2001), more studies should be carried out in order to elucidate if the periodontal treatment would be able to eradicate *H. pylori* from the oral cavity of patients with gingivitis or periodontitis, that are positive for this bacteria in their stomach, decreasing the risk of gastric re-infection in these patients.

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References

Avcu, N., Avcu, F., Beyan, C. et al. (2001) The relationship between gastric-oral *Helicobacter pylori* and oral hygiene in patients with vitamin B-12 deficiency anemia. *Oral Surgery Oral Medicine Oral Pathology Oral Radiology and Endodontics* **92**, 166–169.

Banatvala, N., Lopez, C. M., Owen, R., Ardi, Y., Davies, G., Hardie, J. et al. (1993) *Helicobacter pylori* in dental plaque. *Lancet* **341**, 380.

Birek, C., Grandhi, R., McNeill, K., Singer, D., Ficarra, G. & Bowden, G. (1999) Detection of *Helicobacter pylori* in oral aphthous ulcers. *Journal of Oral Pathology & Medicine* **28**, 197–203.

Cammarota, G., Tursi, A., Montalto, M., Papa, A., Veneto, G., Bernardi, S. et al. (1996) Role of dental plaque in the transmission of *Helicobacter pylori* infection. *Journal of Clinical Gastroenterology* **22**, 174–177.

Costerton, J. W. (1999) Introduction to biofilm. *International Journal of Antimicrobial Agents* **11**, 217–221.

Ferguson, J. R. D. A., Chuanfu, Li, Patel, N. R., Mayberry, W. R., Chi, D. S. & Thomas, E. (1993) Isolation of *Helicobacter pylori* from saliva. *Journal of Clinical Microbiology* **31**, 2802–2804.

Gabryelewicz, A. (1996) Critical evaluation of the treatment of peptic ulcer diseases associated with *Helicobacter pylori* infection. *Journal of Physiology and Pharmacology* **47**, 51–58.

Gall-Troselj, K., Mravak-Stipetic, M., Jurak, I., Ragland, W. L. & Pavelic, J. (2001) *Helicobacter pylori* colonization of tongue mucosa-increased incidence in atrophic glossitis and burning mouth syndrome (BMS). *Journal of Oral Pathology & Medicine* **30**, 560–563.

Ge, Z. & Taylor, D. E. (1998) *Helicobacter pylori* – molecular genetics and diagnostic typing. *British Medical Bulletin* **54**, 31–38.

Gebara, E. C. E., Pannuti, C., Faria, C. M., Chehter, L., Mayer, M. P. A. & Lima, L. A. P. A. (2004) Prevalence of *Helicobacter pylori* detected by PCR in the oral cavities of periodontitis patients. *Oral Microbiology and Immunology* **19**, 1–4.

Harris, A. (1998) Current regimens for treatment of *Helicobacter pylori* infection. *British Medical Bulletin* **54**, 195–205.

Khandaker, K., Palmer, K. R., Eastwood, M. A., Scott, A. C., Desai, M. & Owen, R. J. (1993) DNA fingerprints of *Helicobacter pylori* from mouth and antrum of patients with chronic ulcer dyspepsia. *Lancet* **342**, 751.

Lee, A., Fox, J. & Hazell, S. (1993) Pathogenicity of *Helicobacter pylori*: a perspective. *Infection and Immunity* **61**, 1601–1610.

Leimola-Virtanen, R. E., Happonen, R.-P. & Syrjanen, S. M. (1995) Cytomegalovirus (CMV) and *Helicobacter pylori* (HP) found in oral mucosal ulcers. *Journal of Oral Pathology & Medicine* **24**, 14–17.

Mapstone, N. P., Lynch, D. A. F., Lewis, F. A., Axon, A. T. R., Tompkins, D. S., Dixon, M. F. et al. (1993) Identification of *Helicobacter pylori* DNA in the mouths and stomachs of patients with gastritis using PCR. *Journal of Clinical Pathology* **46**, 540–543.

Marshall, B. J. & Warren, J. R. (1984) Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. *Lancet* **1** (8390), 1311–1315.

Miyabayashi, H., Furihata, K., Shimizu, T., Ueno, I. & Akamatsu, T. (2000) Influence of oral *Helicobacter pylori* on the success of erradication therapy against gastric *Helicobacter pylori*. *Helicobacter* **5**, 30–37.

Mravak-Stipetic, M., Gall-Troselj, K., Lukac, J., Kusic, Z., Pavelic, K. & Pavelic, J. (1998) Detection of *Helicobacter pylori* in various oral lesions by nested polymerase chain reaction (PCR). *Journal of Oral Pathology & Medicine* **27**, 1–3.

Namavar, F., Roosendaal, R., Kuipers, E. J., De Groot, P., Van Der Bijl, M. W., Peña, A. S. et al. (1995) Presence of *Helicobacter pylori* in the oral cavity, oesophagus, stomach and faeces of patients with gastritis. *European Journal of Clinical Microbiology & Infectious Diseases* **14**, 234–237.

Nguyen, A.-M. H., Engstrand, L., Genta, R. M., Graham, D. Y. & El-Zaatari, F. A. K. (1993) Detection of *Helicobacter pylori* in dental plaque by reverse transcription polymerase chain reaction. *Journal of Clinical Microbiology* **31**, 783–787.

Okuda, K., Ishihara, K., Miura, T., Katakura, A., Noma, H. & Ebihara, Y. (2000) *Helicobacter pylori* may have only a transient presence in the oral cavity and on the surface of oral cancer. *Microbiology and Immunology* **44**, 385–388.

Olsson, K., Wadström, T. & Tyszkiewicz, T. (1993) *H. pylori* in dental plaques. *Lancet* **341**, 956–957.

Oshowo, A., Gillam, D., Botha, A., Turnio, M., Holton, J., Boulos, P. & Hobsley, M. (1998) *Helicobacter pylori*: the mouth, stomach and gut axis. *Annals of Periodontology* **3**, 276–280.

Özdemir, A., Mas, M. R. & Sahin, S. (2001) Detection of *Helicobacter pylori* colonization in dental plaques and tongue scrapings of patients with chronic gastritis. *Quintessence International* **32**, 131–134.

Parsonnet, J., Friedman, G. D., Vandersteen, D. P., Chang, Y., Vogelman, J. H., Orentreich, N. & Sibley, R. K. (1991) *Helicobacter pylori* infection and the risk of gastric carcinoma. *New England Journal of Medicine* **325**, 1127–1131.

Patchett, S. E., Beattie, S., Shankaran, K., Mehta, P. R. & Prabhu, S. R. (1992) *Helicobacter pylori* and duodenal ulcer recurrence. *American Journal of Gastroenterology* **87**, 24–27.

Pavelic, J., Gall-Troselj, K., Jurak, I. & Mravak-Stipetic, M. (2000) *Helicobacter pylori* in oral aphthous ulcers. [Letter to the Editor]. *Oral Pathology & Medicine* **29**, 523–525.

Pytko-Polonczyk, J., Konturek, S. J., Karczewewska, E., Bielanski, W. & Kaczmarczyk-Stachowska, A. (1996) Oral cavity as permanent reservoir of *Helicobacter pylori* and potential source of reinfection. *Journal of Physiology and Pharmacology* **47**, 121–129.

Quirynen, M., Papaionnou, W. & Van Steenbrghe, D. (1996) Intraoral transmission and the colonization of hard surfaces. *Journl of Periodontology* **67**, 986–993.

Riggio, M. P. & Lennon, A. (1999) Identification by PCR of *Helicobacter pylori* in subgingival plaque of adult periodontitis patients. *Journal of Medical Microbiology* **48**, 317–322.

Shames, B., Krajden, S., Fuksa, M., Babida, C. & Penner, J. L. (1989) Evidence of the same strain of *Campylobacter pylori* in the stomach and dental plaque. *Journal of Clinical Microbiology* **27**, 2849–2850.

Socransky, S. S. & Haffajee, A. D. (2002) Dental biofilms: difficult therapeutic targets. *Periodontology 2000* **28**, 12–55.

Socransky, S. S., Haffajee, A. D., Cugini, M. A., Smith, C. & Kent, R. L. Jr (1998) Microbial complexes in subgingival plaque. *Journal of Clinical Periodontology* **25**, 134–144.

Song, Q., Haller, B., Schmid, R. M., Adler, G. & Bode, G. (1999) *Helicobacter pylori* in dental plaque. A comparion of different PCR primer sets. *Digestive Diseases and Sciences* **44**, 479–484.

Song, Q., Haller, B., Ulrich, D., Wichelhaus, A., Adler, G. & Bode, G. (2000a) Quantification of *Helicobacter pylori* in dental plaque samples by competitive polymerase chain reaction. *Journal of Clinical Pathology* **53**, 218–222.

Song, Q., Lange, T., Spahr, A., Adler, G. & Bode, G. (2000b) Characteristic distribution pattern of *Helicobacter pylori* in dental plaque and saliva detected with nested PCR. *Journal of Medical Microbiology* **49**, 349–353.

Umeda, M., Kobayashi, H., Takeuchi, Y., Hayashi, J., Morotome-Hayashi, Y., Yano, K. et al. (2003) High prevalence of *Helicobacter pylori* detected by PCR in the oral cavities of Periodontitis patients. *Journal of Periodontology* **74**, 129–134.

van Winkelhoff, A. J., Van Der Velden, U., Clement, M. & De Graaff, J. (1988) Intra-oral distribution of black-pigmented *Bacteroides* species in periodontitis patients. *Oral Microbiology and Immunology* **3**, 83–85.

Yaegaki, K., Ogura, R., Kameyama, T. & Sujaku, C. (1985) Biochemical diagnosis of reduced salivary gland function. *International Journal of Oral Surgery* **14**, 47–49.

Yaegaki, K., Sakata, T., Ogura, R., Kameyama, T. & Sujaku, C. (1982) Influence of aging on DNase activity in human parotid saliva. *Journal of Dental Research* **61**, 1222–1224.

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Clinical Relevance

Scientific rationale for the study: H. pylori are important gastrointestinal pathogens associated with gastritis, peptic ulcers and an increased risk of gastric carcinoma. The oral cavity has been pointed out as a possible source of *H. pylori* in the gastric re-infection process after treatment.

Principal findings: Triple therapy is able to eliminate *H. pylori* from the stomach; however it fails to eradicate bacteria from the mouth.

Practical implications: The oral cavity of urease positive patients with gingivitis or with chronic periodontitis might be considered as reservoirs for *H. pylori,* enhancing the risk for re-infection of the stomach.