

## Insufficiency of the Occurrence of Helicobacter Pylori in Oral Plaque

Wael Taha\*

Department of Oral and Craniofacial Health Sciences, University of Sharjah, UAE

### Abstract

**Objective:** Helicobacter pylori a bacteria that colonises the human stomach, is the most prevalent organism that infects people worldwide. Additionally, this bacterium has been found in various natural niches outside of the stomach, like the mouth and water. The outcomes of H. pylori identification in extra-gastric ecological niche, however, are debatable. One of the primary challenges in presenting strong evidence appears to be improving the sensitivity and specificity of the detection systems. The purpose of this study was to use a new nucleic acid detection technique called Loop-mediated Isothermal Amplification along with an analytically sensitive and specific Polymerase Chain Reaction to identify the presence of this bacterium in dental plaque samples.

**Design:** 45 participants were engaged in a descriptive cross-sectional study, and samples of dental plaque were taken with a sterile periodontal curette from at least two tooth surfaces. DNA was isolated from the samples, and PCR and LAMP procedures were used to detect the presence of H. pylori.

**Results:** Using PCR, LAMP, and positive for both tests, the prevalence of H. pylori identification in dental plaque samples was 44%, 66.67%, and 77.78%, respectively.

**Conclusion:** Participants' dental plaque samples contained high levels of H. pylori, which is consistent with the high prevalence of these germs in the general population. One of the highest reported rates ever is this one. The findings show that oral-to-oral transmission and one of the primary causes of re-infection can both be caused by dental plaque.

**Keywords:** Helicobacter pylori; Sensitivity; Loop-mediated isothermal amplification; Polymerase Chain Reaction

### Introduction

Gram-negative, spiral or curved-shaped, slow-growing, microaerophilic Helicobacter pylori bacteria. This bacterium plays a significant role in the development of chronic gastritis, gastric and duodenal ulcers, as well as gastric adenocarcinoma and MALT lymphoma. The fourth most prevalent cancer in the world, gastric cancer is a major public health concern with a high frequency in both developing and wealthy nations. Males and females contract infections at roughly the same rates until the age of 10, and there is an inverse correlation between the incidence of infection and socioeconomic position [1, 2]. In specific environmental and unfavourable conditions, H. pylori changes into a viable but non-cultivable coccoid form. The primary method of transmission of the organism is from person to person through the gastric-oral and/or faecal-oral routes.

It is assumed that the oral-oral route is one of the most plausible transmission pathways because H. pylori DNA has been found in gastric fluids, vomitus, saliva, and dental plaque. For H. pylori to colonise, it needs a certain environment, which is what the alveolar cavity, dental plaque under the gums, and periodontal pockets provide. The failure of treatment and the recurrence of the infection can both be significantly influenced by this colonisation [3]. Numerous H. pylori genes were the focus of the PCR, which has been used to identify H. pylori in clinical and environmental samples like gastric biopsies, gastric juice, saliva, dental plaque, stool, and water.

This method is considered the best for detecting the bacteria in samples from the alveolar cavity because PCR has shown to detect the greatest amount of H. pylori in dental plaque. Even while PCR is a quick and accurate method, it has several limitations, such as taking longer than LAMP, being more sensitive to inhibitors, and needing specialised equipment for thermal cycling and electrophoresis [4]. The development of the extremely sensitive and specific Loop-Mediated Isothermal Amplification technique holds the potential to overcome the limitations of PCR. This study used loop-mediated isothermal

amplification to measure the prevalence of H. pylori in tooth plaques from individuals with chronic periodontal disorders.

### Materials and Methods

45 patients who were sent to the periodontology department at Kermanshah University of Medical Sciences' school of dentistry in 2013 made up the population of this cross-sectional study. Each participant completed a release form after receiving full information about the project [5]. Patients with periodontal plaque and natural teeth in the mouth were included. Patients with diabetes, those who are expecting, and those who are HIV-positive, smokers, those who have taken antacids and medications like amoxicillin that are anti-H. Pylori.

### Results

A total of 45 dental plaques were gathered from the participants in this cross-sectional investigation. Participants' average ages ranged from 19.82 to 11.92, with a minimum and maximum age of 19.82 and 67.82, respectively [6]. 35 of the participants had a mean age of 36.25 that was female, and 10 had a mean age of 38.8 that was male. Two molecular tests, namely LAMP and PCR, were used to detect H. pylori in all of the samples after DNA extraction.

### Discussion

One of the most prevalent bacterial illnesses in the world

\*Corresponding author: Wael Taha, Department of Oral and Craniofacial Health Sciences, University of Sharjah, UAE, E-mail: waeltaha@gmail.com

**Received:** 28-Jan-2023, Manuscript No: JOHH-23-88930, **Editor assigned:** 30-Jan-2023, PreQC No: JOHH-23-88930(PQ), **Reviewed:** 13-Feb-2023, QC No: JOHH-23-88930, **Revised:** 17-Feb-2023, Manuscript No: JOHH-23-88930(R), **Published:** 24-Feb-2023, DOI: 10.4172/2332-0702.1000362

**Citation:** Taha W (2023) Insufficiency of the Occurrence of Helicobacter Pylori in Oral Plaque. J Oral Hyg Health 11: 362.

**Copyright:** © 2023 Taha W. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

that colonised the human stomach is *H. pylori*. It is commonly acknowledged that the virus has a significant role in the development of gastritis, peptic ulcer disease, and stomach cancer. Some ecological niches outside of the stomach, including as dental and sub gingival plaque, oral lesions or ulcers, saliva, periodontal and gingival pockets in the oral cavity, as well as tap, ground, sea, and well waters, have been found to harbour *H. pylori* [7, 8]. A multifactorial condition called early childhood caries affects children under the age of six and is defined by a process called mineral transfer from the tooth to the environment. Dental plaque, which coats tooth surfaces as a firmly adhering layer made up of bacterial, inorganic, and organic components-the so-called biofilm-if left undisturbed-is closely associated to the dynamic process of dental caries.

The part performed by glucans in the organic components or matrix needs to be emphasised. Glucans are extracellular polysaccharides made from sucrose and are synthesised by glucosyltransferases, an enzyme found in bacteria like *Streptococcus mutans* [9, 10]. The water solubility of the glucan or extracellular polysaccharide depends on whether the alpha 1-3 or alpha 1-6 linkage type is more prevalent. *S. mutans* clings to surfaces with persistent adhesion and accumulation thanks to the matrix, which is primarily made of insoluble glucans. An increased number of infected tooth sites may result from *S. mutans*'s propensity to cling to and collect on dental surfaces through the synthesis of glucan, which is a very important virulence factor.

Numerous researches involving these bacteria have shown that *Mutans Streptococci* are the most prevalent pathogens responsible for ECC. *Scardovia wiggsiae*, a candidate for a newly identified caries pathogen, and *Slackia exigua*, among others, are recent evidences that the ECC has a large diversity of species [11, 12]. *Mutans streptococci* typically colonise young infants with ECC, and these children frequently engage in inappropriate eating behaviours such frequent consumption of carbohydrates and sweetened liquids. Inappropriate feeding practises that frequently involve eating sugar supply sucrose, a particular substrate for the formation of glucans. IP and the occurrence of ECC in early children have been linked. However, it is necessary to show that IP has the ability to forecast ECC. Additionally, researching the progression of caries after discovering an IP concentration in dental plaque allows us to take into account how the child will react to this component as the condition progresses [13-15]. By doing this, we avoid supposing that the development of caries was caused by a specific factor. This study was out to evaluate the associations with dental plaque IP, sugar exposure, and cariogenic bacteria and ECC. To our knowledge, this study of preschoolers from low socioeconomic backgrounds and high caries prevalence indicated for the first time that dental plaque IP can predict the development of ECC. Preschoolers with caries also had plaque with much greater IP, dental plaque IP concentration was related with a higher risk of developing caries.

## Conclusion

The participants' dental plaque samples showed a high frequency of *H. pylori*, which is consistent with the high prevalence of these bacteria in the general population. One of the highest reported rates in the entire world is this one. The findings show that dental plaques might be a major source of both oral-to-oral transfer and re-infection.

## References

- Gilbert P, Maira-Litran T, McBain AJ, Rickard AH, Whyte FW (2002) The physiology and collective recalcitrance of microbial biofilm communities. *Adv Microb Physiol* 46: 203–255.
- Kolenbrander PE, Andersen RN, Kazmerak KM, Palmer RJ (2000) Coaggregation and coadhesion in oral biofilms. In: Allison DG, Gilbert P, Lappin-Scott HM, Wilson M, editor. *Community structure and co-operation in biofilms*. Cambridge, Cambridge University Press, Society for General Microbiology Symposium 2000: 65–85.
- Zhang Y, Lei Y, Nobbs A, Khammanivong A, Herzberg MC (2005) Inactivation of *Streptococcus gordonii* SspAB alters expression of multiple adhesin genes. *Infect Immun* 73: 3351–3357.
- Marsh PD, Featherstone A, McKee AS, Hallsworth AS, Robinson C, et al. (1989) A microbiological study of early caries of approximal surfaces in schoolchildren. *J Dent Res* 68: 1151–1154.
- Suntharalingam P, Cvitkovitch DG (2005) Quorum sensing in streptococcal biofilm formation. *Trends Microbiol* 13: 3–6.
- Devine DA (2003) Antimicrobial peptides in defence of the oral and respiratory tracts. *Mol Immunol* 40: 431–443.
- Loesche WJ (1986) Role of *Streptococcus mutans* in human dental decay. *Microbiol Rev* 50: 353–380.
- Marsh PD (1999) Microbiologic aspects of dental plaque and dental caries. *Dent Clin North Amer* 43: 599–614.
- Sansone C, van Houte J, Josphura K, Kent R, Margolis HC (1993) The association of mutans streptococci and non-mutans streptococci capable of acidogenesis at a low pH with dental caries on enamel and root surfaces. *J Dent Res* 72: 508–516.
- Brailsford SR, Shah B, Simins D, Gilbert S, Clark D, et al. (2001) The predominant aciduric microflora of root-caries lesions. *J Dent Res* 80: 1828–1833.
- Tanner AC, Milgrom PM, Kent R, Mokeem SA, Page RC, et al. (2002) Similarity of the oral microbiota of pre-school children with that of their caregivers in a population-based study. *Oral Microbiol Immunol* 17: 379–387.
- Welin J, Wilkins JC, Beighton D, Wrzesinski K, Fey SJ, et al. (2003) Effect of acid shock on protein expression by biofilm cells of *Streptococcus mutans*. *FEMS Microbiol Lett* 227: 287–293.
- McNeill K, Hamilton IR (2004) Effect of acid stress on the physiology of biofilm cells of *Streptococcus mutans*. *Microbiology* 150: 735–742.
- Bradshaw DJ, Marsh PD (1998) Analysis of pH-driven disruption of oral microbial communities in vitro. *Caries Res* 32: 456–462.
- Theilade E (1986) The non-specific theory in microbial etiology of inflammatory periodontal diseases. *J Clin Periodontol* 13: 905–911.