

Oral *H. Pylori* Infection

JKC Yee*

Research Lab of Oral *H. pylori*, Everett, WA, USA

***Corresponding author:** JKC Yee, Research Lab of Oral *H. pylori*, Everett, WA, USA, Email:

Published Date: December 22, 2016

INTRODUCTION

During the past 20 years, there has been disagreement regarding the existence of *H. pylori* in the oral infection. It was proposed that no living *H. pylori* exists in the oral cavity and that the positive results detected by a Polymerase Chain Reaction (**PCR**) in the oral cavity may be a fragment of *H. pylori*, instead of living bacteria, as part of a reflux from the stomach: *H. pylori* could not be cultivated from PCR-positive samples. The *H. pylori* come from the stomach reflux only to survive in the oral cavity for a few hours because of the high oxygen concentration in the oral cavity. If the above proposed idea is correct, then the fragment or dead *H. pylori* should not have any negative effect on the drug eradication of *H. pylori* infections of the stomach [1,2].

However, the proposed idea contradicts with PCR studies, the fact of oral hypoxia environment, eradication can't eliminate Oral *H. pylori* infection; *H. pylori* can be cultured in the oral cavity, the same original source of Oral and Stomach *H. pylori*. Lower rate of eradication on stomach *H. pylori* when oral *H. Pylori* positive and Meta analysis [3].

There are several reports indicated non-gut organs have been harbored of *H. pylori*, such as vagina [4], nasopharyngeal sinus cavities [5], coronary plaque [6], otitis media [7], breast [8]. However, further confirmation study should be follow up.

After review of studies in past 5 years, there are four important facts here we want emphasized in *H. pylori* colonized in oral cavity;

UBT C13 is a gold standard for diagnosis of stomach *H. pylori*, but is not so for detection in the mouth. We found that UBT C13 has color blind that see *H. pylori* in the stomach, but can't detecting *H. pylori* in oral cavity. In medical practice, patients with negative results in UBT C13 suggest that their stomach infection of *H. pylori* is cured. In fact, patients can present negative UBT results and yet exhibit *H. pylori* infection due to oral infection. The clinical study provides evidence that *H. pylori* oral infection is nonetheless present. In Asia, more than 90% of the population suffered from oral *H. pylori* infection but had negative UBT results [9]. This study also showed that oral antigen screening test could identify individuals who have no risk for *H. pylori* gastric infection. It further identified persons with no symptoms but with antigenic evidence of possible oral *H. pylori* infection who are thus at risk for developing gastric disease. This information was not provided by UBT methods [10].

Drug treatment on stomach *H. pylori* infection has no effective in *H. pylori* infection of oral cavity. *H. pylori* exist in between the teeth and gums called "bio- film membrane" (Bifilm), also known as plaque barrier. It is resistance when the drug into this area. This is why conventional treatment for *H. pylori* eradication *H. pylori* infection, but is not efficacy of oral *H. pylori* in dental plaque. Miyabayashi etc. [11] found the eradication success rate was significantly lower in the oral *H. pylori*-positive cases (12/23, 52.1%) than in the negative cases (22/24, 91.6%) at 4 weeks after the therapy ($p = 0028$). Two years later, only 16 of the 23 (69.5%) oral *H. pylori*-positive cases were disease-free, as compared to 23 of the 24 (95.8%) oral *H. pylori*-negative cases ($p = 018$). They concluded *H. pylori* in the oral cavity affected the outcome of eradication therapy and was associated with a recurrence of gastric infection and recommend that oral *H. pylori* should be examined by nested PCR and, if positive, should be considered a causal factor in refractory or recurrent cases. Our study show the efficacy rate of treatment on stomach *H. pylori* infection at 82.26% for patients received treatment of mouthwash combined with drug eradication; but only at 61.33% efficacy when patients received drug eradication on stomach. So treatment of oral cavity *H. pylori* raise about 20% efficacy when combined treatments of both mouth and stomach [10].

There is non-antibiotic treatment for oral *H. pylori* infection available. Our studies indicated e-polylysine (L) and the Glycerol Monolaurate (GM) used in mouth washing solution. The L is typically produced as a homo-polypeptide of approximately 25-30 L-lysine residues. The epsilon (e) refers to the linkage of the lysine molecules. In contrast to a normal peptide bond that is linked by an alpha carbon group, the lysine amino acids are molecularly linked by the epsilon amino group and the carboxyl group. L belongs to the group of cationic polymers. In water, L contains a positively charged hydrophilic amino group. It is adsorbed electrostatically to the cell surface of the bacteria, followed by a stripping of the outer membrane. This eventually leads to the abnormal

distribution of the cytoplasm, causing damage to the *H. pylori* cell. GM is the mono-ester formed from glycerol and lauric acid. *H. pylori* is extremely sensitive to GM, however there are no reports of L or GM killing *H. pylori* in vivo. Since both have had a safe record in the food industry, we use L-GM successfully eliminate *H. pylori* of oral cavity within 2 to 3 months. In China alone, more than 280 million people carry oral *H. pylori*, which results in 28 million recurrences of stomach *H. pylori* infection and the abuse of antibiotics by overuse [12]. The massive antibiotic pollution that appears in food, water, and children's urine has become a serious concern worldwide. Antibiotic abuse kills 80,000 Chinese people every year and leads to extra medical spending of 11.7 billion dollars across the country, which could become a global problem. This is why we recommended use non-antibiotic formula to take care on *H. pylori* in oral cavity.

There are three important technologies developing to make a strong foundation of a colonized site of the oral cavity. PCR is a high sensitivity test for oral *H. pylori*, but it is not convenient in clinical settings. So, first a high sensitivity and specificity test in saliva such as HPS test should be established. Then we will have a much easier time of running clinical trial on a large number of patients to obtain a greater number of data, to find the positive correlation between oral and stomach *H. pylori* infection [9]. Second most important technology is developing a cell culture suitable for low concentration *H. pylori* in the oral cavity. Because the concentration of *H. pylori* in stomach is five magnitudes higher than that of the oral cavity (107 CFU/mL versus 102 CFU/mL [13,14]), it would be insufficient to use conventional stomach culturing techniques for detecting oral *H. pylori*. After the method of a new cell culture is established, we would confirm if HPS technology can be confirmed by cell culture data [10]. As a final step, we need to develop a technology to eliminate *H. pylori* from the oral cavity instead of an antibiotic drug.

References

1. Marshall B. Speech at Helicobacter pylori infection of the Seventh National Forum of China. Beijing. 2012; 26-27.
2. Al-Ahmad A, Kürschner A, Weckesser S, Wittmer A, Rauberger H, et al. Helicobacter pylori resident or transient in the human oral cavity? J Med Microbiol. 2012; 61: 1146-1152.
3. Yee J.K.C. Oral Cavity is Second Colonized Site beside Stomach- a milestone discovery. World J Gastroenterol. 2016; 22: 641-648.
4. Minakami H, Hayashi M, Sato I. Does Hp colonize the vagina of pregnant women? J infect. 2000; 41: 112-113.
5. Morinaka S, Ichimiya M, Nakamura H. Detection of Hp in nasal and maxillary sinus specimens from patients with chronic sinusitis. Laryngoscope. 2003; 113: 1557-1563.
6. Kowalski M. Hp infection in coronary artery disease: influence of Hp eradication on coronary artery lumen after percutaneous transluminal coronary angioplasty. The detection of Hp specific DNA in human coronary atherosclerotic plaque. J Physiol Pharmacol. 2001; 52: 3-31.
7. Yilmaz T, Ceylan M, Akyon Y, Ozcakay O, Gursel B. Hp: A possible association with otitis media with effusion. Otolaryngol Head Neck Surg. 2006; 134: 772-777.
8. Kast RE. Some fibrocystic breast change may be caused by sexually transmitted *H. pylori* during oral nipple contact: Supporting literature and case report of resolution after gut *H. pylori* eradication treatment. Medical Hypotheses. 2007; 68: 1041-1046.
9. Yee KC, Wei MH, Yee HC, Everett KD, Yee HP, and Hazeki- Taylor N. A screening trial of Helicobacter pylori-specific antigen tests in saliva to identify an oral infection. Digestion. 2013; 87: 163-169.
10. Wang XM, Yee KC, Hazeki-Taylor N, Li J, Fu HY, Huang ML, et al. Oral Helicobacter pylori, its relationship to successful eradication of gastric *H. pylori* and saliva culture confirmation. J Physiol Pharmacol. 2014; 65: 559-566.

11. Miyabayashi H, Furhata K, Shimizu T, Ueno I, Akamatsu T. Influence of oral *Helicobacter pylori* on the success of eradication therapy against gastric *Helicobacter pylori*. *Helicobacter*. 2000; 5: 30-37.
12. Huang, R, Ding, P, Huang. D. and Yang, F. Antibiotic pollution threatens public health in China. *The Lancet*. 2015; 385: 773-734.
13. Megraud F, Lehours P. *Helicobacter pylori* Detection and Antimicrobial Susceptibility Testing. *Clin Microbiol Rev*. 2007; 20: 280-322.
14. Song Q, Zirnstein GW, Swaminathan B, Gold BD. Pretreatment with Urea-Hydrochloric Acid Enhances the Isolation of *Helicobacter pylori* from Contaminated Specimens. *J Clin Microbiol*. 2001; 39: 1967-1968.