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Review

Dental plaque of children as probable *Helicobacter pylori* reservoir

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Helicobacter pylori **is a spiral gram negative bacterium that normally grows in the stomach, and is the main causal agent of gastritis and peptic ulcers. In 1994, The International Agency for Research on Cancer recognized *H. pylori* as a carcinogenic factor for stomach cancer. The oral cavity has been proposed as a reservoir for *H. pylori* and several authors have demonstrated the presence in adult patients of the organism in saliva and dental plaque. However, isolation of *H. pylori* in children is still questionable. Several investigators have reported the presence of *H. pylori* in the dental plaque of children, sometimes associated with gastro-esophageal reflux. These isolations has been realized by polymerase chain reaction were gold standard is the culture. On the other hand, many authors have failed to isolate *H. pylori* by culture in samples obtained from the saliva or dental plaque. We performed an extensive review of the literature to facilitate future research in this controversial topic.**

Key words: *Helicobacter pylori*, children, dental plaque, polymerase chain reaction, urease breathe test.

INTRODUCTION

Since the discovery of the presence of *Helicobacter pylori* in the stomach, there have been many studies trying to determine the source of colonization of this bacterium (Dunne et al., 2014). The oral cavity has been proposed as a reservoir for *H. pylori* by several authors that demonstrated the presence of the organism in dental plaque and saliva from adult patients (Sudhakar et al., 2008; Agarwal and Jithendra 2012; Liu et al., 2013).

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However, in children many researchers have failed to isolate the bacterium from these sites (Muñoz et al., 1999; Silva et al., 2009). This might be due to intermittent presence of the bacterium in dental plaque or due to its lesser number (Song et al., 2000). The isolation of *H. pylori* in children is still questionable, though there are studies that have associated gastro-esophageal reflux with the presence of *H. pylori* in children's oral cavity (Emiroglu et al., 2010). On the other hand, most of the studies where isolation of H*. pylori* is reported come from polymerase chain reaction laboratory test, but the standard for Helicobacter isolation is the culture (Koido et al., 2008). The main objective of this review was to verify if the hypothesis that dental plaque serves as a reservoir for *H. pylori* can be justified from the literature.

Helicobacter pylori

More than three decades ago it was thought that the human stomach was free of bacteria because of its very acid pH (Macy et al., 1978, Drasar et al., 1969). But by 1984, Dr. Robin Warren and his colleague Dr. Barry Marshall described helical-shaped bacteria that could survive and colonize the gastric chamber (Marshall and Warren, 1984). *H. pylori* is a Gram negative spiral or curve-shaped rod that normally colonizes the stomach (Owen, 1998).

Epidemiology

H. pylori infection is one of the most common infections in the world. Its prevalence varies according to geographical area. Developing and oriental countries have major rates of disease than developed and industrialized countries (Pounder and Ng, 1995). Lower socio-economic factors are related to mayor prevalence of infection (Pounder and Ng, 1995). Most of the reports on the prevalence of *H. pylori* infection come from serum-epidemiologic studies (Parsonnet, 1995). *H. pylori* is thought to be indigenous to the human population and is well adapted to the harsh conditions of the human stomach, which is colonized for the host's lifetime (Blaser, 1997). Although the principal reservoir for *H. pylori* infection appears to be humans, *H. pylori* has been isolated from non-human primates and domestic cats (Dubois et al., 1996; Handt et al., 1994). Infection is generally asymptomatic (Blaser, 1995), but may develop to a chronic gastritis (Sipponen, 1997). *H. pylori* has been recognized as a major cause of gastritis and is associated with duodenal ulcer disease, gastric ulcer disease, gastric lymphoma, and gastric cancer in humans (Konturek et al., 2006; Konturek et al., 2009; Kusters et al., 2006; Ando et al., 2006; Ito et al., 2006; Kato and Asaka, 2012). These complications of infection also display geographic preference, which may be due to variations in the strain and virulence factors of *H. pylori* (Suzuki et al., 2012). Besides, complications such as atherosclerosis with acute heart stroke and lymphomas have been associated with *Helicobacter* infections (Kinoshita, 2007; Witkowska and Smolewski, 2013).

Helicobacter infection displays no preference for the hosts gender (Kim, 2005), but its incidence increases between with increasing age of the host (2 to 20 years) and then remain stable (Kim, 2005).

Diagnosis

The urea breath test (UBT) is a very simple, innocuous and highly accurate test strongly associated to *H. pylori* infection (Bytzer et al., 2011). UBT is particularly suitable in all clinical conditions where endoscopy is not strictly necessary, and to check the success of eradication regimens (Atherton and Spiller, 1994; Logan, 1998; Savarino et al., 1999). Another frequently used test for the diagnosis of Helicobacter infection is the rapid urease test (RUT) in gastric biopsy, which is viable in commercial rapid probes (Marshall et al., 1987; Hazell et al., 1987). The antigenic determination of *H. pylori* in feces has been approved by the US Food and Drug Administration for detection and follow-up testing (Monteiro et al., 2001). Fecal antigen detection and UBT are recommended non-invasive approaches for confirmation of infection in children especially those with less than 5 years age. Recently, more sensitive and specific molecular diagnostic tests have been developed. Strategies for polymerase chain reaction (PCR)-based detection of *H. pylori* have included multiple genetic targets with varying levels of sensitivity and diagnostic accuracy. (Lu et al., 1999; Maeda et al., 1998; Gramley et al., 1999). Detection of virulence genes has been offered the best results. However culturing *H. pylori* remains the gold standard for detection of Helicobacter (Koido et al., 2008).

Serologic testing represents a primary screening approach for evaluation of *H. pylori* status in patients not immediately requiring endoscopic studies. With respect to enzyme-linked immunoassays, serum samples yield higher sensitivity and accuracy as compared to whole blood samples (European *Helicobacter pylori* Study Group, 1997; Faigel et al., 2000).

Reservoir

Since the isolation of bacteria from the stomach, researchers have searched the source of Helicobacter, including food, water and the periodontal plaque, as a reservoir from which *H. pylori* is ingested to reach the stomach and thus colonize it.

The presence of *H. pylori* in the oral cavity is still controversial as many studies claimed *H. pylori* presences based on the UBT or immunological tests without confirmation by culture or molecular assays (Al Asqah et al., 2009; Jia et

al., 2009; Koumi et al., 2011). Definitely the only way to reliably demonstrate the presence of this bacterium is its culturing, which requires special and complicated conditions, still, successful *H. pylori* cultures obtained from oral samples of 16/20 patients have been reported (D'Alessandro and Seri, 1992).

Variations in the detection of *H. pylori* by molecular techniques can be explained by differences in: 1) sampling sites (saliva, periodontal pockets, dental plaque) (Fritscher et al., 2004; Liu et al., 2008; Medina et al., (2010), Tsami et al.,2011); 2) sample handling (DNA extraction from isolation sites directly or after culturing) (Anand et al., 2014); 3) primers and geographical distribution of strains (Göttke et al., 2000), and 4) oral health status of the patient (healthy, multiple cavities, mouth ulcers, periodontal disease) (Brown, 2000). Table 1 provides a summary of results of molecular detection from oral samples from adults.

On the other hand, Table 2 summarizes the conditions related with negative results of molecular detection. The success rate in isolating *H. pylori* from the dental plaque of infected subjects varies between 0 and 88% (Pustorino et al., 1996; Pytko-Polonczyk et al., 1996; Majmudar et al., 1990; Desai et al., 1991; Nguyen et al., 1993; Bernander et al., 1993; Bickley et al., 1993; Asikainen et al., 1994; Hardo et al., 1995). A recent systemic review of literatures seems to indicate that dental plaque may be a potential reservoir for *H pylori* according to recent systematic reviews (Anand et al., 2014).

Reservoir in children

Once it was accepted that the oral cavity could serve as a reservoir for *H. Pilory*; the question arise when, during human life, the oral cavity becomes colonized. Studies in Mongolian gerbils, has demonstrated that vertical transmission occurs in the first 4 months of life (Oshio et al., 2009) while Lee and et al. (2006) could not demonstrate the vertical transmission from mother to newborns in the same murine model.

Studies in newborn and mothers from maternal child care hospital in Italy demonstrated than 34.8% of the mothers and 2.9% of the newborns have stool antigen test (SAT) positive to *H. pylori* infection (Baldassarre et al., 2009).

However same author consider that SAT is not a good test to demonstrate the vertical transmission of Helicobacter (Baldassarre et al., 2008).

Another author has demonstrated by PCR that 46% of the mothers infected by *H. pylori* have related DNAstrains that her children. However, the vertical transmisión of these strains is not sure (Nahar et al., 2009).

Actually, the culture isolation and PCR detection has not been demonstrated in the newborn. Although, most of the studies point to maternal child transmission (Weyermann et al., 2006), it causal relation has not been demonstrated. Thus, another source of infection should be considered, and then water, food (Vale and Vítor, 2010), animals (Brown, 2000) and siblings (Schwarz et al., 2008) were associated as sources of infection of *H. pylori*.

The main concern emerge in the possibility of dental plaque as source of infection or re-infection since primary source may be mother siblings, water, food or animals; mouth is an obligatory way to transit to the gastric cavity (Brown, 2000). *H. pylori* grows better in a micro-aerophilic environmental conditions; these are the conditions of dental plaque neighborhood (Atherton, 2006). The main hypothesis is that dental plaque is colonized in early stages of the life. Then it works as reservoir, but this hypothesis cannot be completely recognized. The main obstacles to demonstrate it, merges from studies that suggest that the dental plaque colonization is caused by gastro-esophageal reflux, and not in the reverse way.

Other obstacles arise as the detection by molecular identification suffers from the same variety of outcomes as described for adults. However, the isolation of *H. pylori* in the dental plaque of children remains as a controversial topic. There are researchers who reported isolations of *H. pylori* in dental plaque (Tsami et al., 2011; Chaudhry et al., 2011; Gill et al., 1994; Liu et al., 2008; Ou et al., 2013; Valdez-Gonzalez et al., 2014), but others have been unable to detect *H. pylori* in the oral cavity (Olivier et al., 2006; Bernander et al., 1993). The study conditions that have allowed for identification of *H. pylori* for children's oral cavities or not are summarized in Tables 3 and 4 respectively (Muñoz et al., 1999; Santamaria et al., 1999; Kignel et al., 2005).

Several explanations for discordant results are: a) inadequate and poorly designed primers for microbial detection, b) inadequate sampling from patients and sample processing, c) small patient numbers (Olivier et al., 2006), and d) intermittent or and/or scarce presence of the *H. pylori* in the oral cavity (Song et al., 2000).

In developing countries, children are infected at an early age, usually before the age of two. The suggested routes of transmission are fecal-oral, oral-oral, gastric-oral (Sahay and Axon, 1996; Tursi et al., 1997); all related to poor hygienic conditions. Also vertical transmission, from mothers to baby, can happen, as well as horizontal transmission of microorganism from parents or other caregivers to children (Kitagawa et al., 2001).

The concomitant presence of gastro-esophageal reflux and *H. pylori* detection in dental plaque suggests transmission of the bacteria from the stomach to the mouth and not in the other way and arguments against the hypothesis that the dental plaque may serve as a reservoir for gastric colonization (Emiroglu et al., 2010).

Findings of *H. pylori* in children with gastritis and gastrointestinal symptoms are common (Ogunbodede et al., 2002; Medina et al., 2010). Furthermore, *H. pylori* has been detected in the dental plaque of otherwise healthy adults (Tsami et al., 2011).

Finally the virulence expression factors of *H. Pylori* in

Table 1. Authors who has reported positive isolation of *H. pylori* in adults.

Author	Patients	Isolation (%)	Method
Majmudar et al. (1990)	40	100	CLOtest, culture and smear
Desai et al. (1991)	43	98	CLOtest
D'Alessandro et al. (1992)	20	80	Culture
		100	Urease
Malaty et al. (1992)	239	24	IgG antibodies
Nguyen et al. (1993)	25	38.8	RT-PCR immunofluorescence
Mapstone et al. (1993)	30	5-17	PCR
Song et al. (1994)	40		Rapid urease test, anti-Hp fluorescein-labelled antibody staining, bacterial culture and electronic microscopy.
Cellini et al. (1995)	31	64	Urease test
		3.2	Culture
Zhou and Yang (1995)	3519	61.2	PCR
Yang (1993)	29	72.4	PCR
Herdo et al. (1995)	62	54	PCR
		8	Culture
Luzza et al. (1995)	152	86	IgG antibodies
Wallfors et al. (1995)	110	48	PCR
Cammarota et al. (1996)	31	3.2	PCR
Pustorino et al. (1996)	63	6	Culture
Peach et al. (1997)	217	30.6	IgG antibodies
Contractor et al. (1998)	100	81	Rapid urease test
		13	Rapid urease test
Oshowo et al. (1998)	208	7	PCR
		1	Culture
Amendula et al. (1998)	20	5	Culture, PCR
Mattama et al. (1998)	62	1.6	Culture
Kamat et al. (1998)	248	4	PCR
Riggio et al. (1999)	73	33	PCR
Song et al. (1999)	40	27-100	PCR
Dore et al. (1999)	24	40.9	PCR
Huw et al. (1999)	13	84.6	PCR
Butt et al. (1999)	173	100	CLOtest
Miyabayashi et al. (2000)	47	25.5	PCR
Song et al. (2000)	42	97	PCR
Kim et al. (2001)	46	6.9-28.6	PCR
Younj et al. (2001)	5	100	Electronic microscopy
Avcu et al. (2001)	108	28.5-100	Camphylobacter-like organism test gels
Honda et al. (2001)	60	42-70	IgG ELISA
Kitagawa et al. (2001)	1588	29.2	IgG antibodies and PCR
Goosem et al. (2002)	58	3	PCR
Al-Refai et al. (2002)	135	89	Urease
Mazuda et al. (2002)	116	42.3	IgG antibodies
	116	40	
Ozdemir et al. (2001)	81	79	Urease
Butt et al. (2002)	78	100	CLOtest
		88	Citology
Huw et al. (2002)	32	84	PCR
Berroteran et al. (2002)	32	37.5	PCR
Suk et al. (2002)	65	43	PCR

Table 1. Contd.

Umeda (2003)	57	35.1	PCR
Gürbüz et al. (2003)	75	90	CLOtest
Ogunbodede et al. (2002)	66	69.7	Culture and biopsy
Siddig et al. (2004)	52	92.3	Urease test
Al-Hawajri et al. (2004)	24	50	PCR
Cześnikiewicz et al. (2004)	100	48.3	Culture
Kignel et al. (2005)	49	2	PCR
Anand et al. (2006)	134	71-89	Rapid urease test and serology
Gebara et al. (2006)	30	46	PCR
Chitsazi et al. (2006)	88	34	Rapid urease test
De Souza et al. (2006)	97	99.3	Rapid urease test
Loster et al. (2006)	40	48	Culture and PCR
Chumpitaz et al. (2006)	115	3.5	Culture
Teoman et al. (2007)	67	28.3	PCR
Liu et al. (2008)	126	55	PCR
Bürgers et al. (2008)	94	17	PCR
Sudhakar et al. (2008)	15	11.9	Culture and RUT
Liu et al. (2009)	443	59.4	PCR
Al Asqah et al. (2009)	101	65	Rapid urease test
Silva et al. (2009)	62	36.6	PCR
Jia et al. (2009)	148	19.1	Rapid urease test
Medina et al. (2010)	98	18	PCR
Eskandari et al. (2010)	67	5.97	PCR
Leszczyńska et al. (2009)	164	81.2	Immunoassay
Trevizani et al. (2010)	78	47.4	PCR
Chaudhry et al. (2011)	150	37.5	PCR
Assumpção et al. (2010)	71	89	PCR
Fernández et al. (2011)	200	17	PCR
Silva et al. (2010)[a]	30	20	PCR
Silva et al. (2010)[b]	115	25	PCR
Momtaz et al. (2010)	250	14.4	PCR
Koumi et al. (2011)	56	41.5	Rapid urease test
Diouf et al. (2011)	109	14.7	PCR
Momtaz et al. (2012)	300	77.6	PCR
Agarwal et al. (2012)	30	60	PCR and culture
Liu et al. (2013)	574	68.2	

dental plaque suggest that the bacteria express active virulence factors while growing in the dental plaque. This finding plus major expression of factors and major bacterial counts in the periodontal disease suggest that dental plaque works not only as reservoir by contamination but it really infect and cause damage in the periodontal tissue (Tsami et al., 2011).

There are studies that have shown that strains that are present in the dental plaque and in the stomach are different. In this way, the controversy continue because enough information does not exist that demonstrate transmission from the mouth to the chamber gastric or chamber gastric-dental plaque (Cai et al., 2014).

Other important issue emerges from the fact that *H. pylori* isolation in the most studies is realized by PCR were culture is the gold standard for the diagnosis.

PERSPECTIVE

Recent literature seems to favor the view that the dental plaque may serve as a reservoir for *H. pylori*, both in healthy adults and patients suffering from oral disease, especially periodontal disease (Anand, 2014).

Table 2. Authors who has been reported negative isolation of H. pylori in adults.

Author	Patients	Method
Dalen et al. (1993)	94	Culture
Bickley et al. (1993)	15	Culture
Von Recklinghausen et al. (1994)	49	Culture
Asikainen et al. (1994)	336	PCR
Luman et al. (1996)	1020	Culture
Cheng et al. (1996)	244	Culture
Savoldi et al. (1998)	80	Immunoperoxidase test
Muñoz et al. (1999)	53	PCR and culture
Birek et al. (1999)		PCR
Sahin et al. (2001)	23	PCR-RFLP
Olivier et al. (2006)	79	Histology and PCR
Silva et al. (2009)	62	PCR

Table 3. Authors who has been reported positive isolation of *H. pylori* in children.

Author	Patients	Isolation (%)	Method
Gill et al. (1994)	22	82	Rapid urease test
Muñoz et al. (1999)	53	84.3	PCR and culture
Qureshi et al. (1999)	100	28.3-40	IgG antibodies
Song et al. (2000)	6	100	PCR
Patty et al. (2002)	140	7.8	PCR
Allaker et al. (2002)	15	68	PCR
	22	0	Culture
Ogunbodede et al. (2002)	12	69	Culture and histology
Fritscher et al. (2004)	105	5.7	PCR
Liu et al. (2008)	240	51	PCR
Medina et al. (2010)	98	18	PCR
Wichelhaus et al. (2011)	11	82	PCR
Tsami et al. (2011)	35	42.8	PCR
Hirsch et al. (2012)	3	66	PCR
Ou et al. (2013)	138	21.7	PCR

The identification of *H. pylori* by culturing or PCR was quite consistent. The design of a variety primers and probes enables to detect several strains types according to their geographical distribution and even though they are present in small quantities (Diouf et al., 2011; Song et al., 2000). The successful identification of *H. pylori* in the oral cavity is mostly achieved from dental plaque samples, and seems to be more difficult from saliva samples (Song et al., 2000).

The microaerophilic conditions of the periodontal pockets appear to favor the growth of bacteria, while saliva may serve as a transient vehicle. In adult patients, the flaws in the isolation may be related to the sample site; as we have already mentioned saliva samples may be inadequate (Madinier et al., 1997).

Appropriated culture conditions are essential for *in vitro* growth of the bacteria. Failure to culture bacteria may be due to lack of experience in handling these bacteria, such as small variations in the culture medium, technique or incubation conditions (Ndip et al., 2003). Similarly, the failure to detect *H. pylori* by PCR may be due to methodological flaws from the site of the fetched sample, the sample size, the primers used, the reagents used and failures in amplification protocols (Al Sayed et al., 2014). The isolation of *H. pylori* from children is not exempted from the problems associated with isolation in adults.

The concomitant presence of gastro-esophageal reflux and *H. pylori* identification in the oral cavity is an

Table 4. Authors who has been reported negative isolation of *H. pylori* in children.

Author	Patients	Method
Santamaría et al. (1999)	53	PCR
Oliver et al. (2005)	79	PCR and culture
Song et al. (2000)	6	PCR
Allaker et al. (2002)	22	Culture
Hirsch et al. (2012)	3	Culture

argument against the hypothesis that the dental plaque serves a reservoir. Indeed, the presence of *H. pylori* in the periodontal pockets may be a contamination of the normal oral microflora by gastric reflux (Kurtaran et al., 2008).

It is important to determine the infection moment as well as to establish the route of transmission. Another problem is to demonstrate the presence of *H. pylori* in Edendule adult patients (Cheng et al., 1996), but it does not happen in kids (Alarcón et al., 2013; Mourad-Baars et al., 2010).

In conclusion, although *H. pylori* has been identified in the oral cavity of children, additional studies are needed to support the hypothesis that the dental plaque serves as a reservoir of *H. pylori* in children.

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