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ORIGINAL ARTICLE

PCR-based diagnosis of *Helicobacter* species in the gastric and oral samples of stray dogs

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Abstract *Helicobacter* spp. have been detected in different parts of gastrointestinal tract of dogs including the oral cavity, stomach, intestines and recently, hepatobilliary system. However, the transmission pathways of *Helicobacter* spp. have not been yet fully elucidated. Research in the last decade has proposed that oral–oral and fecal–oral transmissions, among others, may be a plausible route of this gastric infection. This study was carried out primarily to determine the existence of *pylori* and non-*pylori Helicobacter* spp. in the oral secretions and dental plaque of stray dogs of Iran as one of the possible routes of humans and animal infection and, secondly, to evaluate the accordance between oral and gastric colonization of *Helicobacter* spp. in these dogs. Forty-eight adult stray dogs were studied by PCR using 16S rRNA, *Helicobacter felis*, *Helicobacter heilmannii*, and *Helicobacter pylori* specific primers. Positive samples for 16S rRNA specific primers that did not meet the specified species of *Helicobacter* genus were randomly subjected to sequencing. *Helicobacter* spp. DNA was found in the oral and gastric specimens of 100 % of the stray dogs. There was not, however, any agreement between *Helicobacter* colonization at these two locations, at neither genus nor species level. Our study confirmed that the oral cavity of stray dogs routinely exposed to transient forms of bacteria and may temporarily harbor *Helicobacter* spp and *Wolinella* spp. Therefore, oral cavity as a source of *Helicobacter* spp. may act as a reservoir for transmission. However, it may not necessarily reflect the colonization status of the gastric mucosa.

Keywords *Helicobacter* spp · Oral infection · Stray dogs · PCR

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Introduction

During the last decade, the number of species in the genus *Helicobacter* has quickly developed, and at least 38 formally named *Helicobacters* have been recognized. *Helicobacter pylori* (*H. pylori*) is the most important species globally in human disease; however, *Helicobacter heilmannii* and *Helicobacter felis* are also associated with gastric disease in humans and are worthy of discussion (Harbour and Sutton 2008; Heilmann and Borchard 1991; Lee et al. 1988).

H. heilmannii has the largest number of known mammalian hosts among the known gastric *Helicobacter* spp. (Trebesius et al. 2001). These bacteria accompany with *H. felis* have been microscopically observed in the stomach of dogs, wild rats, cheetahs, cats, swine, various species of nonhuman primates,

and in a small percentage of humans with gastritis. But *H. felis* was occasionally observed in human gastric biopsies (Eaton et al. 1993; Lavelle et al. 1994; Queiroz et al. 1990; Stolte et al. 1994).

The prevalence of gastric *Helicobacter* spp. in dogs and cats is high, but exact mode of transmission and the role of *Helicobacter* spp. in gastrointestinal diseases are not clear. It suggested that oral–oral and fecal–oral are probable routes of transmission (Brown 2000). Detection of *Helicobacter* spp. in saliva and dental plaque supported this hypothesis that the oral cavity could be a reservoir and source of *Helicobacter* spp. infection in human and cats (Recordati et al. 2007; Shojaee Tabrizi et al. 2010). To the best of the authors' knowledge, there is no documented report investigating prevalence and association of different species of *Helicobacter* spp., including *H. pylori*, *H. felis*, and *H. heilmannii* in the oral cavity and stomach of stray dogs.

Consequently, the principal aim of the present study was to determine the prevalence of *Helicobacter* spp. in the oral cavity of stray dogs, as a possible route of transmission, and to identify the association between the oral and gastric *Helicobacters*.

Materials and methods

Animals and sampling procedure

This study has been approved by the Iranian laboratory animal ethics framework under the supervision of the Iranian Society for the Prevention of Cruelty to Animals. Forty-eight stray dogs (29 males and 19 females (mean age=5.2 years)) were captured from different suburban locations of Mashhad, Iran. In Iran, government policy dictates that overpopulating stray dogs should be euthanized to preserve the wild life in suburban areas. Health status of dogs was not ascertained in our study. Sampling performed immediately after euthanasia with acepromazine (Neurotranq 1, 0.2 mg/kg, IM) and Thiopental Sodium (Nesdonal 1, 25–30 mg/kg, IV).

Sterile cotton swabs were used to collect oral secretions, and dental plaque was removed from the first upper premolar with a sterile curette. Then, samples were immediately transferred to the 500 ml phosphate buffer saline. At necropsy, stomachs were removed and opened along the greater curvature, and samples were collected from gastric fundus, gastric body, and gastric juice. All gastric and oral specimens were frozen immediately at −20 °C until further analysis.

DNA extraction and PCR assays

DNA was extracted from the saliva, dental plaque, and gastric samples using QIA amp DNA Mini kit (Qiagen, Hilden, Germany) according to the manufacturer's instructions. Subsequently, presence of *Helicobacter* genus was investigated using 16S rRNA gene and, then, by using specific primers. Positive samples were evaluated for different species included *H. pylori*, *H. heilmannii*, and *H. felis*. PCR amplification was performed in a final volume of 25 μl containing 3 μl of extracted DNA, 2.5 μl of 10× PCR buffer (Fermentas, Lithuania), 1.5 mM MgCl2, 0.2 mM of each deoxynucleotide triphosphate, 1.6×10^{-7} mM of each primer and 0.04 U μl^{-1} Super Taq DNA Polymerase (Gen Fanavaran Co., Iran). Primer sequences and PCR conditions are presented in Table 1. The resulting PCR products underwent gel electrophoresis [1.2 % (*w*/*v*) agarose gel with 0.3 % ethidium bromide in 10 % Tris–Borate–EDTA buffer] and visualized under UV transilluminator. PCR results were interpreted as follows: appearance of (1) the 764 bp band only, *Helicobacter* spp. positive (*H. heilmannii* and *H. felis* negative); (2) the 764 and 580 bp bands, *H. heilmannii* positive and *H. felis* negative; (3) the 764 and 241 bp bands, *H. felis* positive and *H. heilmannii* negative; (4) the 764 and 296 bp bands, *H. pylori* positive (*H. heilmannii* and *H. felis*

Table 1 Oligonucleotide primers and PCR conditions

Target genes	Primer sequence (5′→3′)	PCR fragment (bp)	PCR conditions	Reference
16S rRNA (*Helicobacter* spp.)	F: GGC TAT GAC GGG TAT CC GGC R: GCC GTG CAG CAC CTG TTTTC	764	94 °C for 4 min (94 °C for 60 s, 57 °C for 45 s, 72 °C for 60 s) 30 cycles; 72 °C for 5 min	Ulrich et al. (2002)
ureA, ureB Genes (*H. felis*)	F: GTG AAG CGA CTA AAG ATA AAC AAT R: GCA CCA AAT CTA ATT CAT AAG AGC	241	94 °C for 4 min (94 °C for 40 s, 64/1 °C for 30 s,72 °C for 30 s) 34 cycles; 72 °C for 4 min	Germani et al. (1997)
ureB Gene (*H. heilmannii*)	F: GGG CGA TAA AGT GCG CTT G R: CTG GTC AAT GAG AGC AGG	580	94 °C for 4 min (94 °C for 45 s, 58 °C for 60 s, 72 °C for 120 s) 30 cycles; 72 °C for 4 min	Neiger et al. (1998)
glmM (*H. pylori*)	F: GGA TAA GCT TTT AGG GGT GTT AGG GG R: GCT TAC TTT CTA ACA CTA ACG CGC	296	94 °C for 4 min (94 °C for 60 s, 57 °C for 45 s, 72 °C for 60 s) 30 cycles; 72 °C for 5 min	Kauser et al. (2005)

negative); and (5) all four bands: *H. felis*, *H. heilmannii*, and *H. pylori*-positive.

Sequencing

Positive samples for 16S rRNA specific primers that did not meet the specified species of *Helicobacter* genus were randomly subjected to sequencing. The sequences were analyzed by nucleotide data bank, and their similarities were determined.

Statistical analysis

Frequency of *H. felis* and *H. heilmannii* was calculated in dental plaque, saliva, gastric juice, gastric fundus, and body. Oral cavity samples include dental plaque and/or saliva samples, and gastric region samples include gastric juice or gastric fundus and/or gastric body samples. For the assessment of the association between the presence of *Helicobacter* spp. in the gastric region vs. oral cavity and dental plaque vs. saliva, chi-square test and, if required, Fisher's exact test were used. *p* Values less than 0.05 were considered as statistically significant. Kappa coefficient was also calculated to evaluate agreement between existence of *H. felis* or *H. heilmannii* in oral cavity and gastric region.

Results

Genus-specific PCR identified *Helicobacter* spp. DNA in 45/48 (93.7 %) of dental plaque and 43/48 (89.6 %) of saliva samples. All 48 dogs were found to harbor *Helicobacter* spp. DNA in their oral cavity (dental plaque and/or saliva). Frequency of *H. felis* and *H. heilmannii* in dental plaque specimens was 3/45 (6.6 %) and 17/45 (37.7 %). Two samples of dental plaque (4.4 %) had both *H. felis* and *H. heilmannii*. PCR in saliva samples detected *Helicobacter* spp. DNA in 43/48 (89.6 %) of the subjects; 2/43 (4.6 %) were *H. felis*, and 18/43 (41.8 %), *H. heilmannii*. No saliva samples had both strains, simultaneously. A total of 5 (10.4 %) and 27 (56.2 %) dogs were found to harbor *H. felis* and *H. heilmannii* in oral cavity, respectively. Finally, no association was detected between the presence of *H. felis* or *H. heilmannii* in dental plaque and saliva ($p=1$ and $p=0.31$, respectively).

Helicobacter spp. DNA was identified in gastric juice, fundus, and body of the stomach in 46 (95.8 %), 40 (83.3 %), and 40 (83.3 %) of 48 dogs, respectively. *H. felis* and *H. heilmannii* were found in 12/46 (26 %) and 33/46 (71.7 %) of gastric juice, 12/40 (30 %) and 25/40 (62.5 %) of fundus, and 22/40 (55 %) and 16/40 (40 %) of body samples, respectively. Totally, gastric region infection with *H. felis* and *H. heilmannii* was 31 (64.6 %) and 43 (89.6 %), respectively. *H. pylori* was not found in any parts of oral cavity or stomach.

Table 2 presents the frequency of *Helicobacter* spp., *H. felis* and *H. heilmannii*, in oral cavity (saliva and dental plaque) and gastric specimens. There was no statistically difference between the frequency of *H. felis* in oral cavity and gastric specimens ($p=0.45$). Frequency of *H. heilmannii* was 27 (56.2 %) in oral cavity and 43 (89.6 %) in gastric regions, and no significant differences were identified ($p=0.86$). *H. felis* was simultaneously negative in 16 and positive in 4 dogs in oral cavity and gastric region samples, and there was a little agreement between the presence of *H. felis* in the oral cavity and gastric region (kappa=0.05). Two and 24 dogs were simultaneously negative and positive in oral cavity and gastric region for *H. heilmannii*, respectively. There was less than chance agreement between existence of *H. heilmannii* in oral cavity vs. gastric region (kappa=0.02).

Two DNA samples of dental plaques that did not meet used species of *Helicobacter* by PCR were subjected to sequencing. DNA sequencing of their 16S rRNA revealed that they were related to the *Wolinella* spp. with homology rates of 99 % (GenBank accession JN869512-JN869513).

Table 2 Frequency (in percents) of *Helicobacter* spp., *H. felis* and *H. heilmannii*, in oral cavity (saliva and dental plaque) and gastric specimens ($n=48$)

	Helicobacter spp. Number of samples (%)	*H. felis* Number of samples (%)	*H. heilmannii* Number of samples (%)	Mixed infection Number of samples (%)
Oral cavity				
Total	48 (100)	5 (10.4)	27 (56.2)	2 (4.16)
Dental plaque	45 (93.7)	3 (6.6)	17 (37.7)	2 (4.44)
Saliva	43 (89.6)	2 (4.6)	18 (41.8)	0 (0)
Gastric region				
Total	48 (100)	31 (64.6)	43 (89.6)	17 (35.41)
Gastric juice	46 (95.8)	12 (26)	33 (71.7)	9 (19.56)
Fundus	40 (83.3)	12 (30)	25 (62.5)	5 (12.5)
Body	40 (83.3)	22 (55)	16 (40)	10 (25)

Discussion

In this study, all samples of the oral cavity and gastric regions were infected with *Helicobacter* spp., except two of dental plaques which were related to *Wolinella* spp. Several studies previously showed that *Helicobacter* spp. are extremely prevalent in dog's stomach, so that 67–100 % of healthy pet dogs (Eaton et al. 1996; Happonen et al. 1998; Jalava et al. 1998) and 100 % of random-source dogs (McNulty et al. 1989), laboratory beagles, and shelter dogs have been infected (Eaton et al. 1996; Henry et al. 1987; Strauss-Ayali et al. 1999), which is compatible with our findings.

Besides *H. pylori*, the most important species of *Helicobacter* in human, two other species named *H. heilmannii* and *H. felis* are also related with gastric disorders in humans (Heilmann and Borchard 1991; Lee et al. 1988). In the current study, *H. pylori* was not detected in oral cavity and gastric regions of stray dogs. In contrast with *H. pylori*, the prevalence of *H. heilmannii* was about 56.3 and 89.6 % in dog's oral cavity and gastric regions.

H. heilmannii is an important *Helicobacter* that can cause gastritis, peptic ulcer, and even gastric low-grade lymphoma in humans (Morgner et al. 2000; Regimbeau et al. 1998). Unlike many species of *Helicobacter* spp., such as *H. pylori* and *H. felis*, it has been isolated from different types of mammals (Trebesius et al. 2001). Several studies showed that the prevalence of *H. heilmannii* was less than 0.5 % among human patients who underwent upper gastrointestinal endoscopy for dyspeptic symptoms (Flejou et al. 1990; Heilmann and Borchard 1991; McNulty et al. 1989; Morris et al. 1990); however, it has been as high as 6 % in Thailand and China (Yali et al. 1998; Yang et al. 1998). This controversy could be related to different parameters in various geographic regions, but, as our study revealed a high prevalence of *H. heilmannii* in stray dogs, human contact with infected animals may be an important risk factor for acquiring this infection.

This study showed low prevalence of *H. felis* in oral cavity (10.4 %) and high prevalence of *H. felis* in gastric regions (64.6 %). In other studies, 8.4 and 62.7 % of dogs were affected with *H. felis* in gastric regions (Jalava et al. 1998; Van den Bulck et al. 2005).

Even though the exact route of transmission is unclear, direct contact, fecal–oral, oral–oral, and gastro–oral are likely ways (Axon 1995). With respect to some reports of non-*pylori Helicobacter* gastritis in humans and high prevalence of *H. felis* and *H. heilmannii* in dogs, zoonotic features of *Helicobacter* infections should be considered (Meining et al. 1998; Stolte et al. 1994).

There are a few reports about the prevalence of *Helicobacter* spp. in oral cavity and its association with occurrence of *Helicobacter* spp. in gastric regions (Solnick and Schauer 2001). In a study by Recordati et al. (2007), nested PCR showed *Helicobacter* spp. DNA in 36 (94.7 %) gastric biopsies, 17 (44.7 %) dental plaque, and 19 (50 %) saliva samples. In this study, no statistical relationship (p=0.053) between the degree of gastric colonization in histology and the presence of *Helicobacter* spp. DNA in the oral cavity was shown (Recordati et al. 2007).

In accordance with previous studies, in the current study, the association between *H. felis* and *H. heilmannii* in oral cavity and gastric region was not shown. Shojaee Tabrizi et al. (2010) also found no correlations between *Helicobacter* colonization at oral cavity and gastric regions of stray cats (Shojaee Tabrizi et al. 2010). In contrast, a number of authors reported statistically significant correlation between the existence of *H. pylori* in the gastric region and the oral cavity (Morales-Espinosa et al. 2009; Rasmussen et al. 2010). Although we did not find any relationship between presence of *H. felis* or *H. heilmannii* in oral cavity and gastric region, high prevalence of *H. heilmannii* in oral cavity favors the oral–oral spread and proposes that the oral cavity may be a reservoir for *Helicobacter* spp., though may not inevitably indicate the colonization status of the gastric mucosa.

Our study showed relatively low prevalence of *H. felis* (6.6 and 4.6 %) and high prevalence of *H. heilmannii* (37.7 and 41.8 %) in dental plaque and saliva samples. There was no association between presence of *H. felis* or *H. heilmannii* in dental plaque and saliva. In another study on humans, *H. pylori* DNA was detected in 42.3 and 47.4 % of saliva and dental plaque samples, respectively (Rasmussen et al. 2010), although no statistically significant difference was observed between strains in the saliva and dental plaque (Rasmussen et al. 2010).

Direct sequencing of two 16S rRNA gene-specific PCR products of dental plaque was related to the *Wolinella* spp. with homology rates of 99 %. *Wolinella* spp. is related to Helicobacteraceae family with ribosomal DNA similar with *Helicobacter* spp. members. Frequency of this bacterium in the oral cavity of dogs is believed to be high (Craven et al. 2011). Results of this study, in accordance with Craven et al. (2011), revealed that *Wolinella* spp. may be found in the oral cavity of dogs. However, further investigation is required to detect the real prevalence of *Wolinella* in the oral cavity of dogs and its role in pathogenesis of gastrointestinal disorders.

In conclusion, this study showed low prevalence of *H. felis* and high prevalence of *H. heilmannii* in oral cavity of stray dogs. Meanwhile, it revealed that the contamination of dog's oral cavity may not neccessarily equal to the gastric *Helicobacter* infection, but it may possibly operate as a reservoir for gastric infectivity. Furthermore, the oral–oral route should be considered as an important route of transmission for non-*pylori Helicobacter* spp. in stray dogs.

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