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Survey of Helicobacter infection in domestic and feral cats in Korea

Heh-Myung Ghil[1], Jong-Hyeon Yoo[3], Woo-Sung Jung[1], Tae-Ho Chung[1], Hwa-Young Youn[1,2], Cheol-Yong Hwang[1,2,*]

[1]Department of Veterinary Internal Medicine, and [2]KRF Priority Zoonotic Disease Research Institute, College of Veterinary Medicine, Seoul National University, Seoul 151-742, Korea
[3]BK21 Program of Integrative Network Systems for Veterinarians in Basic Science, Industrial Animals and Preventive Medicine, Konkuk University, Seoul 143-701, Korea

Discovery of *Helicobacter* (*H.*) *pylori* has led to a fundamental change in our understanding of gastric diseases in humans. Previous studies have found various *Helicobacter* spp. in dogs and cats, and pets have been questioned as a zoonotic carrier. The present study surveyed the Helicobacter infections and investigated the presence of *H. felis* and *H. pylori* infections in domestic and feral cats in Korea. Sixty-four domestic cats and 101 feral cats were selected from an animal shelter. Saliva and feces were evaluated by Helicobacter genus-specific polymerase chain reaction (PCR). Genus-specific PCR positive samples were further evaluated for *H. felis* and *H. pylori* using specific primer pairs. Thirty-six of 64 (56.3%) samples from domestic cats and 92 of 101 (91.1%) samples from feral cats were PCR positive; the positive rate of feces samples was higher than that of saliva samples in both groups. *H. felis* and *H. pylori* species-specific PCR was uniformly negative. The prevalence of *Helicobacter* spp. in feral cats was approximately two-fold higher than that of domestic cats. The fecal-oral route may be more a common transmission route not only between cats but also in humans.

Keywords: cat, Helicobacter, prevalence, zoonosis

Introduction

Since the discovery of *Helicobacter* (*H.*) *pylori* [28], gastritis has been studied from a whole new perspective. To date, spiral bacteria other than *H. pylori* found in stomach of humans, animals, dogs and cats have been considered a potential reservoir of zoonosis [7-9,13,17,19,20,31,32,36, 41,44]. Two gastric Helicobacters, *H. heilmannii* and *H. felis*, are mostly associated with human gastric disease [14]. Nevertheless, eight other enterohepatic Helicobacters (*H. canis, H. pullorum, H. cinaedi, H. fennelliae, H. canadensis, H. winghamensis, H. westmaedi, and H. rappini*) have been isolated from humans [10].

In humans, *Helicobacter* spp. infections are associated with gastrointestinal diseases, cancers, and the immunocompromised. In dogs and cats, however, clinically healthy hosts are typically found. While *H. felis* is implicated as a potential pathogen in humans, many other species are still under research [14].

The route of transmission of *Helicobacter* spp. is uncertain, but is known to spread by direct contact. Oral-oral, gastro-oral, and fecal-oral routes are all possible [2]. Iatrogenic *H. pylori* infection transmitted by the endoscope or by contact with gastric fluid also has been reported [43].

H. pylori infection is predominant in the developing world, and low socio-economic status is associated with increased prevalence of the infection [27]. Fecal contamination of common sources including water [16] and soil [15] has been implicated in spread of the infection. This is supported by the findings that *H. pylori* infection rates are higher in developing countries, where untreated water and inadequately prepared vegetables contaminated with soil are common [4,15]. In animals, the DNA of *Helicobacter* spp. has been detected from sources other than gastric tissues, which include vomitus and saliva [37], dental plaques [37], and feces [18].

In Korea, *Helicobacter* spp. has been studied in many animals, for example, dogs [1,17,18,30,33,34], cats [17,22], pigs [35], mice [21], and Mongolian gerbils [24,25]. The DNA of several species of Helicobacter has been detected. '*H. heilmannii*', formerly named *Gastrospirillum hominis* in humans [29], is the most predominant species known in cats [5,7,17,31,36,41]. *H. felis* [5,20,32,36,41] and *H. canis* [8,9] are also detected in cats. *H. pylori* was isolated from a group of cats from a commercial vendor of research animals [11,12], and the bacterial DNA has been detected in bile of cats [3]. In studies where specific pathogen free cats were experimentally inoculated with *H. pylori* or *H. felis*, the

*Corresponding author
Tel: +82-2-880-1281; Fax: +82-2-880-1281
E-mail: cyhwang@snu.ac.kr

bacteria induced mild gastritis associated with lymphoid follicles, with no gastric erosions or ulcers evident during upper gastrointestinal endoscopy or at necropsy [38,39].

The present study was surveyed the prevalence of Helicobacter infection and the specific presence of *H. felis* and *H. pylori* infection, as a means of clarifying the possible role of domestic and feral cats in Korea as a zoonotic source.

Materials and Methods

Animals and sampling

Saliva and feces samples of 165 domestic and feral cats were obtained. Cats were grouped by environmental criteria; domestic cats were those that were almost exclusively kept indoors and feral cats being those that had been captured roaming wild in suburban areas. In Korea, government policy dictates that overpopulating feral cats are euthanized to preserve the wild life in suburban forests. During weekly visits to an animal shelter operated by the Korea Animal Rescue and Management Association, saliva and feces feral cats (n = 101; 55 females, 46 males) were obtained. Ages and health status of the cats were not ascertained. Domestic cats (n = 64; 28 females, 36 males) were either admitted to the Veterinary Medical Teaching Hospital of Seoul National University (Korea) or were the pets of staff members. The cats had an average age of 3.1 years (range 3 months to 12 years). Twenty-three cats were healthy and 41 were clinically ill; of the latter, the clinical signs varied from simple anorexia to hepatic lipidosis, feline lower urinary tract disease, renal failure, diabetes mellitus, and lymphoma. Feces and saliva samples taken from each cat by swabbing with sterilized cotton swabs were merged in 500 µl of autoclaved phosphate buffered saline. DNA was extracted from 20 µl of each sample using DNeasy Tissue Kit (Qiagen, USA). The DNA samples eluted 100-200 µl were stored at -20°C until required.

Helicobacter genus-specific polymerase chain reaction (PCR)

Helicobacter 16S rRNA gene was amplified from each DNA sample using c97 and c98 primers (Table 1) [10]. The final reaction volume of 25 µl contained 2 µl of DNA sample, 12.5 pmole of each primer, ×1 PCR buffer (Takara Bio, Korea), 200 µM of deoxyribonucleoside triphophates mixture (Takara Bio, Korea), and 0.75 U of recombinant Taq DNA polymerase (Takara Bio, Korea). The PCR cycle was 94°C for 2.5 min followed by 40 cycles of denaturation at 94°C, annealing at 50°C, extension at 72°C for 1 min each, and a final extension at 72°C for 15 min [17]. PCR was performed using a PC808 programmed temperature control system (Astec, Japan). PCR products were electrophoresed on ethidium bromide stained 1.5% w/v agarose gels in ×0.5 TBE buffer. The separated products were visualized on ultraviolet light illuminator. PCR sensitivity and specificity of fecal samples has been previously evaluated [18].

H. pylori and *H. felis* specific PCR

H. pylori and *H. felis* specific PCR was performed with primers (Table 1) that amplify the urease B gene of *H. pylori* and *H. felis* [31]. Two microliters of each DNA sample was added to a reaction mixture containing 12.5 pmole of each primer, ×1 PCR buffer (Takara Bio, Korea), 200 µM of deoxyribonucleoside triphophates mixture (Takara Bio, Korea), and 0.75 U of recombinant *Taq* DNA polymerase (Takara Bio, Korea) to produce a total volume of 25 µl. For *H. pylori* specific PCR, samples were heated to 95°C for 5 min and 57°C for 5 min once, followed by 35 cycles of extension at 72°C for 1 min, denaturation at 94°C for 1 min, annealing at 72°C for 2 min, and a final extension at 72°C for 10 min [41]. The positive control (isolates purchased from the American Type Culture Collection; ATCC, USA) and the negative control (sterile distilled water) were carried out with every PCR. For *H. felis* specific PCR, samples were heated to 94°C for 2.5 min once, followed by 40 cycles of denaturation at 94°C, annealing at 47°C, extension at 72°C for 1 min each, with a final extension at 72°C for 15 min. The positive control (*H. felis* ATCC 49179) and the negative control (sterile distilled water) were carried out with every PCR. PCR products were electrophoresed on ethidium bromide stained 1.5% w/v agarose gels in ×0.5 TBE buffer. The separated PCR products were visualized using an ultraviolet light illuminator.

Table 1. Primer sequences for *Helicobacter* (*H.*) spp. polymerase chain reaction

Target gene	Primer sequences	Product (bp)
H. spp. 16S rRNA	F: 5'-GCT ATG ACG GGT ATC C -3' R: 5'-GAT TTT ACC CCT ACA CCA -3'	400
H. pylori urease B	F: 5'-GGA ATT CCA GAT CTA TGA AAA AGA TTA GCA GAA AAG -3' R: 5'-GGA ATT CGT CGA CCT AGA AAA TGC TAA AGA GTT G -3'	1,707
H. felis urease B	F: 5'-ATG AAA CTA ACG CCT AAA GAA CTA G -3' R: 5'-GGA GAG ATA AAG TGA ATA TGC GT -3'	1,150

F: forward, R: reverse.

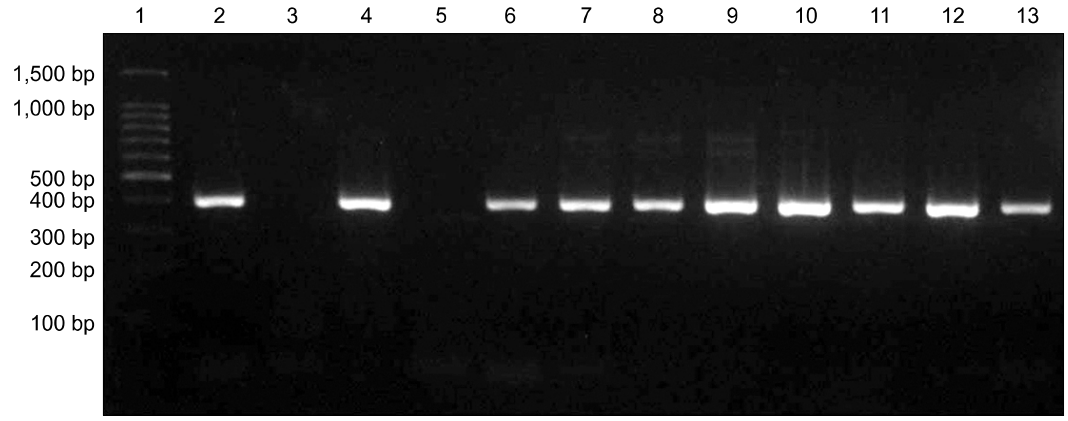

Fig. 1. PCR amplication of *Helicobacter* (*H.*) spp. genus- specific 16S rRNA gene. DNA molecular weight standard marker (Lane 1), *H. felis* positive control (ATCC 49179) of DNA product at 400 bps (Lane 2), negative control (Lane 3), feces of feral cats no.92-101 (Lanes 4-13) are shown.

Table 2. PCR prevalence of *Helicobacter* spp. infection in domestic and feral cats

	Domestic cats	(%)	Feral cats	(%)
Saliva	17	(26.6)	48	(46.5)
Feces	29	(45.3)	85	(84.2)
Saliva or Feces	36	(56.3)	92	(91.1)
Total	64	(100)	101	(100)

Purifying and nucleotide sequence analysis

A specific sized PCR product was extracted using a MEGAquick-spin gel extraction kit (Intron, Korea) to confirm the identity of the target gene PCR product. Purified PCR products were analyzed using an ABI 3100 automatic sequence analyzer (Applied Biosystems, USA).

Results

Helicobacter genus-specific PCR

On Helicobacter genus-specific PCR for 16s rRNA gene, 36 (56.3%) from 64 domestic cats were positive, and 92 (91.1%) from 101 feral cats were positive on either saliva or feces samples (Fig. 1). In domestic cats, 17 (26.6%) saliva samples and 29 (45.3%) feces samples were positive. Infection rates were higher in feral cats with 47 (46.5%) saliva samples and 85 (84.2%) feces samples being positive (Table 2). Among the 64 domestic cats for which the clinical status was known, 36 (56.3%) were positive for *Helicobacter* spp. infection. Clinically ill cats had a *Helicobacter* spp. infection rate of 63.4% (26/41), compared to 43.5% (10/23) of healthy cats, which was not statistically significant. Ill cats were not especially prone to gastrointestinal diseases, and their diagnoses mainly involved anorexia with or without hepatic lipidosis, feline urologic syndrome, diabetes mellitus, renal failure, feline infectious peritonitis, lymphoma, and otitis.

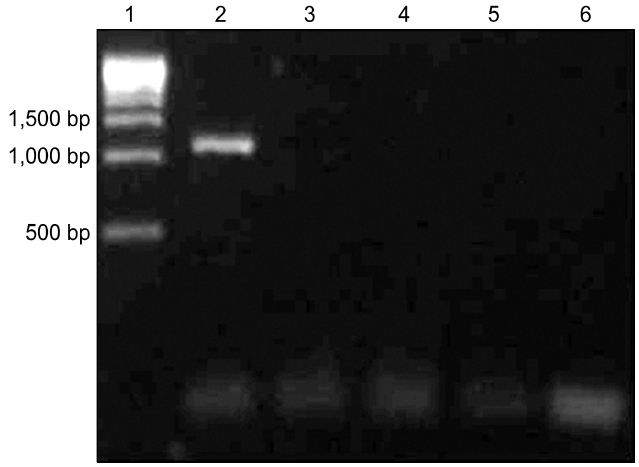

Fig. 2. PCR amplication of *Helicobacter* (*H.*) *felis* urease B gene fragment. DNA molecular weight standard marker (Lane 1), *H. felis* positive control (ATCC 49179) of DNA product at 1,150 bps (Lane 2), negative control (Lane 3), feces of domestic cats no. 12, 14, 16 (Lanes 4-6) are shown. All saliva and feces samples of positive Helicobacter genus-specific PCR were *H. felis* negative.

H. pylori and *H. felis* specific PCR

Species-specific PCR was performed on 17 saliva samples and 29 feces samples from the domestic cats and 47 saliva samples and 85 feces samples from the feral cats, which showed a positive result on genus-specific PCR. In *H. felis* specific PCR, which amplified a 1,200 bp fragment in the positive control (ATCC 49179), none of the samples were positive (Fig. 2). Also, no samples were positive on *H. pylori* specific PCR, which revealed a 1,700 bp fragment on the positive control (SS1 strain) (Fig. 3).

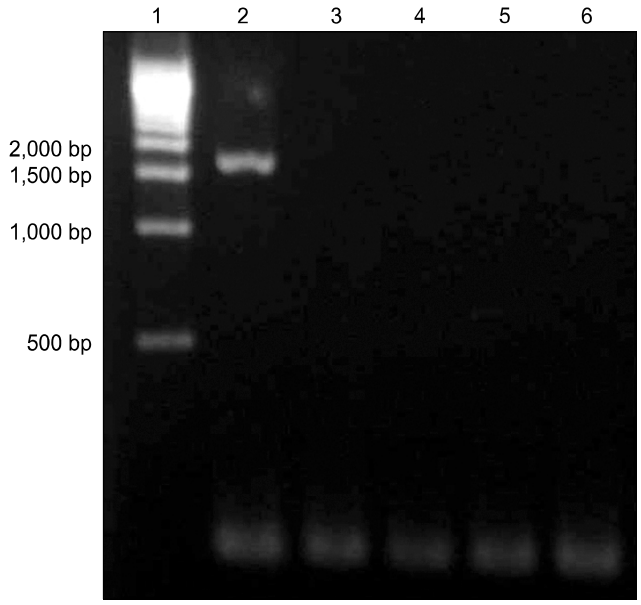

Fig. 3. PCR amplication of *Helicobacter* (*H.*) *pylori* urease B gene fragment. DNA molecular weight standard marker (Lane 1), *H. pylori* positive control of DNA product at 1,707 bps (Lane 2), negative control (Lane 3), feces of feral cats no.71-73 (Lane 4-6) are shown. All saliva and feces samples of positive Helicobacter genus-specific PCR were *H. pylori* negative.

Sequence analysis

Direct sequencing of the PCR product of the specific 400 bp band confirmed the amplified DNA fragments to be from the genus Helicobacter. Direct sequencing of the genus specific PCR product was performed on two randomly selected feces samples. One of the sequencing results was 100% identical with *H. canis* 16S rRNA gene. It displayed 99% similarity to the sequence of *Helicobacter* spp. 'feline isolate' 16S rRNA, *H. canis* strain Lausanne 16S rRNA, and *H. canis* NCTC 12220 16S rRNA. The other sequencing result resulted in 97% homology to *H. canis* strain MIT 51402, *H. canis* strain ATCC 51401, *H. cholecystus* strain ATCC 700242, and *H. bilis* strain FL56.

Discussion

The prevalence of *Helicobacter* spp. between 64 domestic and 101 feral cats in Korea was compared in this study. Prevalence of *Helicobacter* spp. in either saliva or feces samples was 91.1% in feral cats and 56.3% in domestic cats suggesting that feral cats under shelter environments are at greater risk of *Helicobacter* spp. infection. The precise route of transmission of *Helicobacter* spp. is yet unknown, but other than fecal-oral or oral-oral routes, gastric juice after vomiting as the natural route of transmission is considered [2]. This gastro-oral hypothesis seems to be convincing considering the fact that the infection is typically acquired in early childhood in humans, specifically with epidemiologic vomiting and childhood overcrowding [2,23]. Moreover, since infection is predominant in developing countries and among intimate familial members [26], it seems likely that the cats in shelter environments are prone to *Helicobacter* spp. infection. Positive infection rates were higher on feces samples in both domestic and feral cats (46.5% and 84.2%, respectively) than on saliva samples (26.6% and 45.3%, respectively). This may suggest that under natural circumstances fecal-oral transmission is more likely than oral-oral transmission among cats. In a previous study, only fecal contact remained as a significant risk factor in an indirect study by questioning, and the seroprevalence for *H. pylori* increased significantly with age [6]. Nevertheless, healthy adult cats vomit naturally on occasion to spit out hairballs, which makes the gastro-oral route conceivable. The incidence of positive outcome between saliva and feces samples were random, meaning positive feces samples did not always have positive saliva samples, nor did positive saliva samples not always have positive feces samples.

Clinical signs were unknown in feral cats, and domestic cats did not have apparent gastrointestinal signs. Forty-one domestic cats admitted to the hospital for sickness mostly had systemic diseases, and gastrointestinal signs were of simple anorexia coupled with stress or secondary mucosal bleeding due to azotemia. In clinically healthy domestic cats the rate of *Helicobacter* spp. infection was 43.5% (10/23) and in clinically ill domestic cats the rate was 63.4% (26/41). Correlation between the infection rate and the clinical illness including gastrointestinal signs was not confirmative in this study due to the small number of cats involved. Further study of a larger cat population would be needed.

In species-specific studies, neither *H. felis* nor *H. pylori* were found. This is consistent with the results of some previous studies. A Swiss study of 58 cats reported that no amplification of *H. felis* or *H. pylori* were detected in PCR [31]. However, other studies that utilized PCR for examination of gastric biopsy samples reported *H. felis* was in four of 17 [41], two of 21 [17], and one of 15 [5] cats, and one of 10 cheetahs [42]. Although the numbers of cats in the present study was higher than in previous studies, not a single *H. felis* positive sample was evident. Presently, direct sequencing of two 16S rRNA gene-specific PCR products was conducted from purified isolates of genus-specific PCR. One of the two products displayed 100% similarity to a *H. canis* 16S rRNA sequence of 336 base pairs. The 16S rRNA sequences of *H. felis* and those of *H. bizzozeronii* and *H. salomonis* display 98.2-100% similarity [19], and *H. canis* differs by 8.1-10.1% from these species [40]. This implies that there is a significant genetic difference within the 16S rRNA gene of these Helicobacter species. *H. canis* also has been reported in

cats in the United States [8,9].

In Korea, *Helicobacter* spp. studies in cats [17,22] have been fewer and not perceived as urgent a public health issue as similar studies conducted in dogs [1,17,30,33,34]. However, given the burgeoning population of domestic cats in Korea, and the likelihood that many of these cats are kept indoors in close contact with adults and children, careful study of the zoonotic potential of cats is warranted. While cats have not been regarded as a potential zoonotic threat for *Helicobacter* infections, the results of this study show prompt a re-examination of that view. It is suggested that care be taken especially when handling feces of domestic cats.

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