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Prevalence and Genotyping of *Helicobacter pylori* Using *cagA* and *vacA* Among Humans and Pets (Dogs and Cats)

Article Info.

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Abstract

Helicobacter pylori is a globally distributed bacterium that colonizes the gastric mucosa and is associated with several gastrointestinal diseases in humans, including chronic gastritis, peptic ulcers, and gastric carcinoma. While transmission is primarily human-to-human, the role of animals as potential reservoirs remains under investigation. This study aimed to detect *H. pylori* in domestic cats and dogs and compare the prevalence and genotypic patterns of the *cagA* and *vacA* virulence genes with those found in human clinical samples. The study comprised 261 samples, divided into 161 animal samples (dogs and cats) and 100 human gastric biopsies of patients with gastritis. All samples were analyzed by molecular technique (PCR) to detect *H. pylori* targeting the *ureC* gene. In addition to identifying the virulence factors *cagA* and *vacA* with their genotypes (*vacA* m1, m2, s1a, s1b, s1c, and s2). The results indicate *H. pylori* infection rates of 8.2% in dogs, 4% in cats, and 25% in human samples. Notably, a high prevalence of *cagA* genes (100%), (80%), and (96%) was detected in cats, dogs, and humans, respectively. The *vacA* m2/s2 strain exhibited a high prevalence of 75% in both cats and humans, compared to 20% in dogs. These findings demonstrate a high frequency of *cagA*-positive strains in pets and humans, in addition to the predominance *vacA* m2/s2 genotype in these host species, with a moderate presence in dogs. All together indicate a potential zoonotic transmission pathway and the possibility that companion animals may serve as a reservoir and source of *H. pylori* infection.

Keywords: *Helicobacter pylori*, *ureC*, *cagA*, *vacA*, zoonotic.

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Introduction

A high percentage of the world's population is known to carry the spiral, flagellated, Gram-negative, microaerophilic bacteria called *Helicobacter pylori*, which is regarded as a serious public health concern (1). The prevalence of *H. pylori* infection ranges from approximately 30%-50% in developed countries and 70%-90% in developing countries (2). According to a few studies in Iraq, accurate statistics and information on the prevalence of this bacterium are not yet available. Scattered studies in Iraq indicated a prevalence of 11.3 to 71.3% for *H. pylori* (3). Geographically, the prevalence varies; the lowest *H. pylori* prevalences are seen in Oceania (24.4%), Western Europe (34.3%), and North America (37.1%). However, the largest infection rates are seen in Africa (79.1%), South America (69.4%), Latin America and the Caribbean (63.4%), and Asia (54.7%) (4). Domestic cats could serve as a valuable model for studying *H. pylori* disease in humans. Additionally, the isolation of *H. pylori* from domestic cats suggests the possibility that this bacterium may be zoonotic, potentially allowing transmission from cats to humans (5). In a study carried out in Taipei, Taiwan, the prevalence of *Helicobacter* species in canines was found to be 75.79% (6). Other studies have focused on the potential transmission of *H. pylori* from animals to humans. it is a possible zoonotic gastric bacterium capable of efficient interspecies transmission. Pet companion animals (particularly cats and dogs) serve as natural reservoirs for this microorganism (7). Close contact between humans and animals seems to enhance the transmission of *H. pylori*, which is suspected to be transmitted through oral-oral, fecal-oral, or gastric-oral routes, which are evidenced by isolation of *H. pylori* from saliva and feces (8). Given the high prevalence of *H. pylori* found in cats—a pathogen well-known for causing infections in humans—this possibility should be taken seriously (9). *H. pylori* is known to have a wide variety of virulence factors for thriving in the environment of the stomach. The expression of oncogenic protein cytotoxin-associated gene (*cagA*) and vacuolating cytotoxin A (*vacA*) is essential for *H. pylori* to exert pathogenesis towards the host (10). The strong link between *H. pylori* infection and gastric cancer development has led to numerous studies to clarify the role of virulence factors in the establishment of the disease.

The two most important virulence factors of *H. pylori cagA* and *vacA* have been studied extensively to support a role in the development of gastrointestinal disorders (11). The release of toxins such as *cagA* and *vacA* causes harm to the host tissue in human gastritis (12). Although *H. pylori* infection is not usually associated with any clinical symptoms, but sometimes leads to inflammation in the gastrointestinal system and results in peptic ulcer and gastric cancer (13).

The presence or absence of the *cagA* gene, which encodes the *cagA* protein, is used to classify *H. pylori* strains as *cagA*-positive or *cagA*-negative (14).

The presence of *cagA* is frequently linked to a higher incidence of inflammatory reactions and more damage to the gastric mucosa. The *vacA* antigen is one of the well-known virulence factors of this agent whose gene, *vacA*, is present in all strains. The mosaic-like structure of the *vacA* gene has both conserved and variable allelic sequences. These variable sequences are found in different regions from the N-terminal side, including signal sequence (s1 and s2) region, intermediate (i1 and i2) region, deletion (d1 and d2) region, and mid (m1 and m2) region, respectively. Whilst the cytotoxicity power of all genotypes differs from each other, in addition, two s1 and m1 regions in turn comprise several subtypes, including *vacA* s1a, s1b, s1c, m1a, m1b, and m1c (15; 16; 17). The *vacA* exerts various effects on mammalian cells by affecting functions and the integrity of the plasma membrane and membranes of other organelles (18). Researchers reported that the *vacA* gene has a strong link with the emergence of *H. pylori* infections that cause peptic ulcer disease in the population of Iraq (19). Therefore, searching for the source of the infection and the possible transfer pathway in the local geographic area is crucial.

Materials and Methods

Sample collection

The current study comprised a total 261 samples, divided into 161 animal samples were collected using sterile methods from two pet species, dogs and cats, in the shelter areas of Kerbala city, and a one hundred human patients with gastritis, who attended the Center of Gastrointestinal Tract at Al Hussain Teaching Hospital in Kerbala city between December 2022 and April 2023 for endoscopically investigation. Pet faecal samples were collected from females (75%) and males (25%), while human gastrointestinal biopsy samples were obtained from 67% and 33% of females and males, respectively.

Molecular method

Genomic DNA was extracted from all the collected samples using the Qiagen (USA) extraction kit. All the extraction steps were done according to the manufacturer's recommendations.

Identification of *H. pylori* and virulence genes

The presence of *H. pylori* bacteria in the collected samples was determined by detecting the *ureC* gene, which is an essential *H. pylori* gene, using the PCR technique, utilising a species-specific pair of primers. A similar technique was used to detect *cagA* and *vacA* genes, in addition to identifying the *vacA* sub alleles (m1, m2, s2, s1a, s1b, s1c) (Table 1).

Table 1: Primer's names, sequences, and the expected amplicon size, utilised during this study.

Target gene		Sequences 5′——▶3′	Amplicon size (bp)	Reference
ureC	**F**	GGATAAGCTTTTAGGGGTGTTAGGGG		(20)
	R	GCTTACTTTCTAACACTAACGCGC	296	
cagA	**F**	GTTGATAACGCTGTCGCTTC		(21)
	R	GGGTTGTATGATATTTTCCATAA	350	
***vacA* m1**	**F**	GGTCAAAATGCGGTCATGG		(22)
	R	CCATTGGTACCTGTAGAAAC	290	
***vacA* m2**	**F**	GGAGCCCCAGGAAACATTG		(23)
	R	CATAACTAGCGCCTTGCAC	352	
***vacA* s1a**	**F**	GTCAGCATCACACCGCAAC		(24)
	R	CTGCTTGAATGCGCCAAAC	190	
***vacA* s1b**	**F**	AGCGCCATACCGCAAGAG		
	R	CTGCTTGAATGCGCCAAAC	187	
***vacA* s1c**	**F**	CTYGCTTTAGTRGGGYTA		(25)
	R	CTGCTTGAATGCGCCAAAC	213	
***vacA* s2**	**F**	ATGGAAATACAACAAACACAC		(26)
	R	CTGCTTGAATGCGCCAAAC	286	

Polymerase chain reaction (PCR)

The genes of interest were amplified during this study by using the GoTaq® Green PCR master mix kit. Each PCR reaction was prepared for each gene in a PCR tube as outlined in Table 2. All components were subsequently subjected to vortexing and centrifugation using ExiSpinTM before placement in the PCR thermocycler.

Table 2: PCR reaction composition and concentration.

PCR reaction mixture	Volume
DNA sample	5 µL
Forward primer (10 pmol/ µL)	1 µL
Reveres primer (10 pmol/ µL)	1 µL
Nuclease water	3 µL
GoTaq ®Green PCR master mix (2X)	10 µL
Total volume	20 µL

PCR conditions for gene amplification

Several PCR programmes were applied to amplify the genes of interest. Regarding their sequences, amplicon size, and primers annealing temperature degrees, different PCR thermocycler conditions and parameters were used in the PCR programmes (Table 3).

Table 3: PCR thermocycler conditions protocol in the study

Target gene	Initial Denaturation	Denaturation	Annealing	Extension	Final Extension	Hold	No. of cycles
ureC	94C˚/ 4Min	94C˚/ 1Min	55C˚/1 Min.	72 C˚/ 1 Min.	72C˚/ 5 Min.	4 °C	30
cagA	94C˚/ 4Min	94C˚/ 1Min	54C˚/ 1Min.	72 C˚/ 30 Sec	72C˚/5 Min.	4 °C	30
vacA m1	95C˚/ 4 Min	95C˚/ 1Min	52C˚/ 1 Min.	72 C˚/ 30 Sec	72C˚/ 5 Min.	4 °C	35
vacA m2	95C˚/ 5Min	95C˚/ 30 S.	53C˚/ 30 Sec	72 C˚/ 1 Min.	72C˚/ 5 Min.	4 °C	35
vacA s2	95C˚/4Min	95C˚/ 40 S.	55C˚/ 1 Min.	72 C˚/ 1 Min.	72C˚/ 5 Min.	4 °C	35
vacA s1a	95C˚/ 5Min	95C˚/ 1Min	53C˚/ 1 Min.	72 C˚/ 30 Sec	72C˚/ 5 Min.	4 °C	35
vacA s1b	95C˚/ 5Min	95C˚/ 1Min	53C˚/ 1 Min.	72 C˚/ 30 Sec	72C˚/ 5 Min.	4 °C	35
vacA s1c	95C˚/ 5Min	95C˚/ 1Min	53C˚/ 1 Min.	72 C˚/ 30 Sec	72C˚/ 5 Min.	4 °C	35

DNA visualisation (agarose gel electrophoresis)

The amplified parts of the genes of interest were observed using 1.5% agarose gel electrophoresis in the presence of red safe (amb, Canada). An electrical current (90V) was applied for 45 minutes to allow the amplified DNA to transport from the cathode to the anode pole position. The transported bands were visualised on a UV transilluminator apparatus using a 500nm wavelength. Amplicon sizes were estimated by comparison with the standard size of 1500 bp DNA Ladder (Promega, USA). The resulting bands' brightest were displayed, and pictures were captured.

Statistical Analysis

The ANOVA tests have been utilised to discern statistically significant differences across multiple independent groups with a significant value less than 0.05.

Results

The current study reveals that 4% of cats, 8.2% of dogs, and 25% of human samples contained *H. pylori*, regarding the presence of the *ureC* gene, in the total of 261 samples (100 cat faecal, 61 dog faecal, and 100 human tissues endoscopically samples) (Figure 1). Table 4 shows the identification of *H. pylori* infection among pets and humans through the identification of the *ureC* gene by the PCR molecular technique.

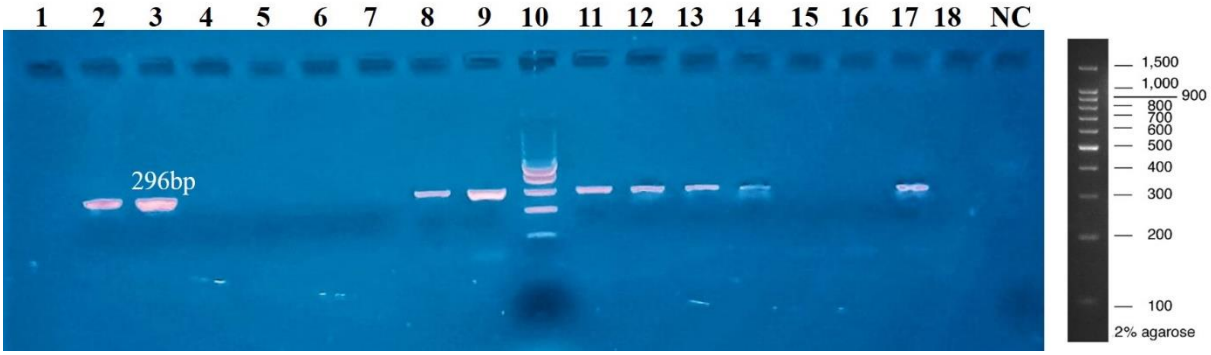

Figure 1: An agarose gel electrophoresis image shows the partially amplified *ureC* gene of *H. pylori*. Lane 10: A DNA Ladder (100 bp, Promega, USA). Lanes 2,3, 8, 9, 11 - 14, and 11 displayed a single band at approximately 296bp, indicating a positive result of the presence of the *ureC* gene. Lanes 1,4 - 7, 15, 16, and 18 did not show any band, indicating a negative *ureC* gene result. Lane NC: refers to the negative control.

Table 4: Detection of *H. pylori* in animal (Dogs and Cats) and human samples using PCR technique.

ureC	Pet cats		Pet dogs		Human	
	Number	Percentage	Number	Percentage	Number	Percentage
Positive	4	4%	5	8.20%	25	25%
Negative	96	96%	56	91.80%	75	75%
Total	100	100%	61	100%	100	100%

Identification of *Helicobacter pylori* virulence genes and subgenes in the animal and human isolates

All the *H. pylori*-positive DNA samples were subjected to virulence factor (genes) detection using gene-specific primers in the conventional PCR technique. The current finding demonstrated that the *cagA* gene was the predominant virulence factor, detected in nearly all *H. pylori*-positive samples. Specifically, *cagA* was identified in 100% of cat samples, 80% of dog samples, and 96% of human samples (Figure 2). Furthermore, the *vacA* gene was detected at a high prevalence, as *vacA* m2 and *vacA* s2 alleles were observed in 75% of cat samples. In dog samples, *vacA* m2 was the most frequently detected subtype (60%), while in human samples, *vacA* s2 showed the highest prevalence at 72% (Figures 3 and 4; Table 5).

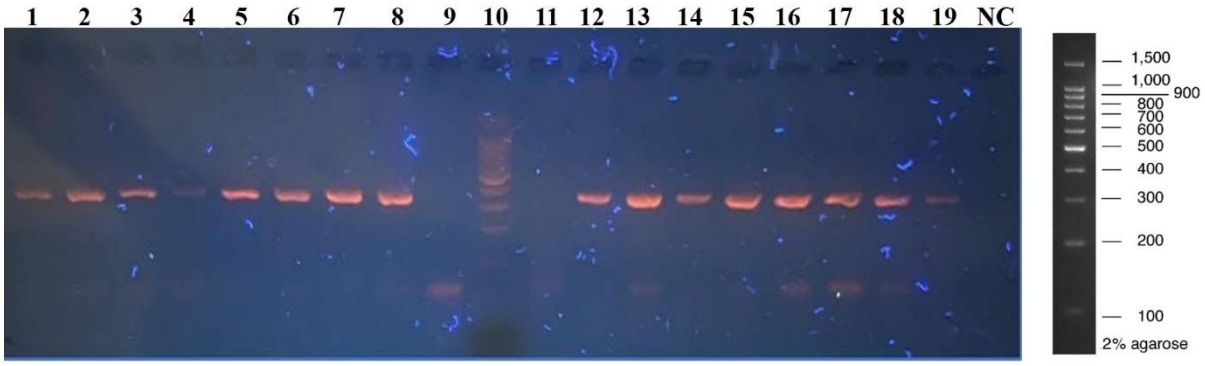

Figure 2: Agarose gel 1.5% electrophoresis image shows the partially amplified *cagA*. Lane 10: A DNA Ladder (100 bp, Promega, USA). Lanes 1-8, 12-19 show a single band at approximately 350bp, referring to the presence of the *cagA* gene. Lanes 9 and 11 display no band as a negative *cagA* gene result. Lane NC: refers to the negative control.

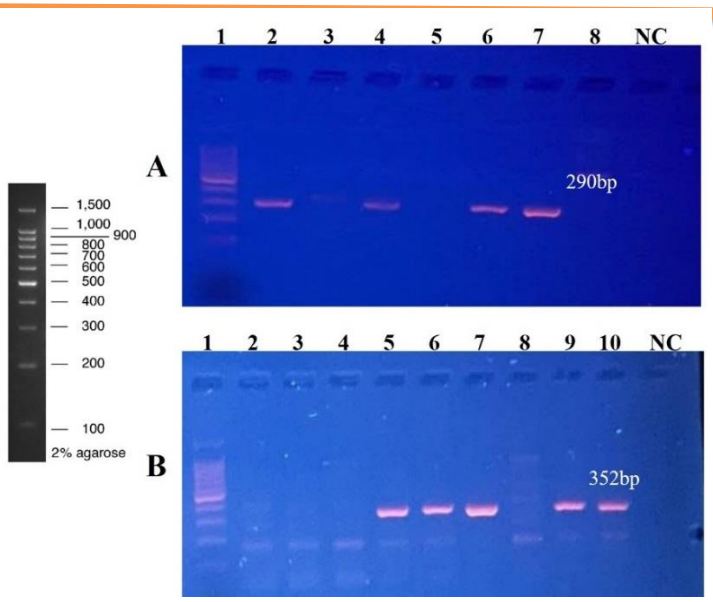

Figure 3. An agarose gel electrophoresis shows the presence of *vacA* m1 and *vacA* m2 alleles in the *Helicobacter pylori* positive samples. A) displays the *vacA* m1 presence results. Lane 1: A DNA Ladder (100 bp, Promega, USA). Lanes 2, 4, 6, and 7 demonstrate a single band at approximately 290bp as a positive result for *vacA* m1 allele. Lanes 3, 5, and 8 display no band as a negative *vacA* m1 result. Lane NC: refers to the negative control. B) displays the *vacA* m2 presence results. Lane 1: A DNA Ladder (100 bp, Promega, USA). Lanes 5-7, 9, and 10 demonstrate a single band at approximately 352bp as a positive result for *vacA* m2 allele. Lanes 2-4 and 8 display no band as a negative *vacA* m1 result. Lane NC: refers to the negative control

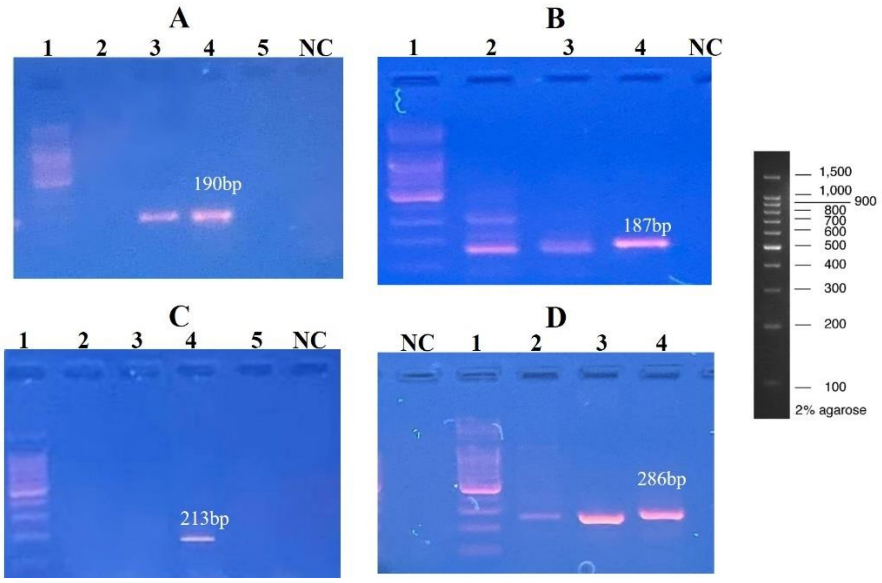

Figure 4: Agarose gel electrophoresis image shows the partially amplified *Helicobacter pylori vacA* genotypes. In all figure parts (A, B, C, and D), Lane 1: a DNA ladder (100 bp, Promega, USA). NC

indicates a negative control. A) Detection of *vacA* s1a allele. Lanes 3 and 4: display a single band at approximately 190bp as a positive result. Lanes 2 and 5 show no band, representing a negative result. B) Detection of *vacA* s1b allele. Lanes 2-4 display a single band at approximately 190bp as a positive result. C) Detection of *vacA* s1c allele. Lane 4: display a single band at approximately 213bp as a positive result. Lanes 2, 3, and 5 show no band, representing a negative result. D) Detection of *vacA* s2 allele. Lanes 2-4: display a single band at approximately 286bp as a positive result.

Table 5: The frequency of virulence factors of *H. pylori* among pets (Cats and Dogs) and human samples.

Positive samples	*cagA*	*vacA*					
		M1	M2	s2	s1a	s1b	**s1c**
Cat	4	1	3	3	1	0	0
4 (4.00%)	100.0%	25.0%	75.0%	75.0%	25.0%	0.0%	0.0%
Dog	4	2	3	2	2	0	1
5 (8.20%)	80.0%	40.0%	60.0%	40.0%	40.0%	0.0%	20.0%
Human	24	9	16	18	4	2	1
25 (25.00%)	96.0%	36.0%	64.0%	72.0%	16.0%	8.0%	4.0%

Interestingly, some *H. pylori* positive DNA samples showed more than two *vacA* subtypes. However, no more than two were detected (Table 6). The results displayed a higher prevalence of the *vacA* m2/s2 genotype in *H. pylori* from cat samples, accounting for 75% of cases. In contrast, dog samples exhibited a more diverse distribution of *vacA* sub alleles, such as m1/s2, m1/sa1, m2/s2, and m2/s1a.

Among human samples, the *vacA* m2/s2 sub alleles were the most common types, which were identified in 52% of cases. However, m2/a1c and m1/s1b were not detected during this study. Overall, the difference in *vacA* m2/s2 prevalence among the three host species was not statistically significant (p = 0.66).

Table 6: The frequency of two genotypes of *vacA* in *H. pylori* for pet and human samples

Positive samples	***vacA***								**P. value**
	M1/s2	**M1/s1a**	**M1/s1b**	**M1/s1c**	**M2/s2**	**M2/s1a**	**M2/s1b**	**M2/s1c**	
Cat	0	1	0	0	3	0	0	0	
4 (4.00%)	0.0%	25.0%	0.0%	0.0%	75.0%	0.0%	0.0%	0.0%	0.667
Dog	1	1	0	1	1	1	0	0	
5 (8.20%)	20.0%	20.0%	0.0%	20.0%	20.0%	20.0%	0.0%	0.0%	

Human	5	3	0	1	13	1	2	0
25 (25.00%)	20.0%	12.0%	0.0%	4.0%	52.0%	4.0%	8.0%	0.0%

Discussion

Helicobacter pylori infection represents a significant public health concern due to its established association with the pathogenesis of chronic gastritis, peptic ulcer disease, and gastric malignancies. Although *H. pylori* is traditionally regarded as a human pathogen, recent studies have indicated that it or closely related organisms can also be isolated from various animals, including primates, sheep, pigs, dogs, and cats. These animals may act as potential reservoirs, and close contact with them could contribute to the widespread prevalence of *H. pylori* infection in humans (27; 7).

It was reported that some *H. pylori* serotypes are zoonotic gastric microorganisms capable of efficient interspecies transmission. Domesticated companion animals, particularly dogs and cats, serve as natural reservoirs for *H. pylori* (7). The current study found that 8.2% of pet dogs and 4% of pet cats tested positive for *H. pylori* in their stool samples, whereas 25% of human gastritis samples are positive, as shown in Table 4. This difference may be attributed to recent societal shifts towards the use of commercial pet foods, which have become integral to modern lifestyles. This notion is supported by the study of Monno *et al*. (28), which suggests that the dietary intake of certain foods and beverages can influence the acquisition of *H. pylori*. Current results are consistent with the findings of (29), who detected *H. pylori* among *Helicobacter* species in dogs and cats living with human companions, with 6% of the tested samples.

Furthermore, the current results are in line with (30), who reported a high percentage of *H. pylori* in human patient samples from Duhok governorate (northern Iraq), at a rate of 40.2%. This might be related to the similarity of people treating their pets in the same country. Other results showed that the domestic cat may be a potential model for *H. pylori* disease in humans (31).

The *cagA*-positive strain appears to be the most prevalent form of *H. pylori,* as well as sharing this gene across all three hosts, suggesting that the *cagA* gene is the predominant virulence factor in the *H. pylori* strains. Which observed in this study with prevalence rates of 96%, 100%, and 80% in humans, cats, and dogs, respectively (Table 5). A range distribution of the *cagA* gene was reported between 17% to 100% in different geographic regions (32; 33). This phenomenon may indicate the association of the *cagA* gene with the severity of disease with *H. pylori* infection and the role of the surrounding environment (34; 35).

The current study results are consistent with a study conducted in Baghdad, which reveals that among 51 *H. pylori*-positive samples, the *vacA* m2 allele was detected in 64% of patients, indicating a high prevalence of this allele (36). A similar finding was also reported by Jouimyi *et al*. (37), who analysed *H. pylori* strains in the Moroccan population at 77%. These studies provide evidence of the prevalence of *vacA* m2 and s2 alleles among human *H. pylori* strains. However, data on the prevalence of these alleles in pet animals such as cats and dogs, particularly in Iraq, are limited. Thus, there is a gap in understanding the genetic diversity and potential cross-species transmission of the bacterium.

The current finding shows that animals (dogs and cats) could be infected with *H. pylori*. So, transmission between animals and from them to humans is a possible approach. Another key finding of this study is the presence of the same *vacA* allele in all the tested samples, including human and animal. For instance, *vacA* m2 was detected in humans (64%), dogs (60%), and cats (75%). Furthermore, the results of the current study indicate that the predominant *vacA* alleles across the examined host species are m2 and s2, suggesting these variants are more commonly circulating within the local population. The presence of *vacA* in dogs and cats suggests that they could be a potential reservoir host of *H. pylori*, though at a much lower rate.

The high prevalence of the *vacA* s2 allele among human *H. pylori* isolates was reported at 72%. Whereas, in dog samples, the predominant *vacA* allele was m2 (60%), in comparted to the cat samples, both *vacA* m2 and s2 alleles were equally common, each accounting for 75%. That might raise the idea that cats play a major role in the *H. pylori* transfer between dogs and humans, as they are widely distributed and live closer with humans than dogs in our country these days.

Remarkably, the results indicate that the m2/s2 genotype is the common *vacA* combination genotype among all the studied samples, which was detected at 75%, 52%, and 20% in dogs, humans, and cats. These findings agree with Jouimyi *et al*. (37), who reported a similar common combination of *vacA* gene (in addition to i2/d2) in 52% of human chronic gastritis samples. The current study also agreed with a study conducted by El Khadir *et al.* (38), which presents a high prevalence of m2/s2 genotypes, with more than 58% in gastritis patients. In addition to the results of the study conducted in Morocco show the preponderance of *vacA* m2/s2 among gastritis patients (39). This might be related to an association between this genotype and gastritis and gastric cancer (40).

Moreover, according to the findings of (41), who reported the *vacA* alleles s1a/m2 and *vacA* m1a/m2, positive strains were predominant in gastric biopsy samples of dogs, even though they often exhibit mild or no clinical symptoms. These findings together would suggest that dogs may serve as reservoirs for *H. pylori* (29). Consistent with our study findings, it can be indicated that companion animals, including dogs and cats, represent a significant source of zoonotic transmission and pose public health risks. Furthermore, additional investigations are warranted to explore the relationship between the presence of the *cagA* gene and the pathogenicity of *H. pylori*.

Conclusion

The high prevalence of the *H. pylori* virulence genes *cagA* and *vacA* m2/s2 in both cats and humans, along with a moderate presence in dogs, suggests a potential zoonotic transmission pathway. And suggesting the possibility that companion animals may serve as a reservoir and source of *H. pylori* infection in humans.

Conflicts of interest

The authors declare that there is no conflict of interest.

Ethical Clearance

This work is approved by The Research Ethical Committee.

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الكشف عن انتشار وتحديد النمط الجيني لبكتيريا الملوية البوابية باستخدام جينات *cagA* و*vacA* بين الانسان والحيوانات الأليفة (الكلاب والقطط)

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الخلاصة

تعد الملوية البوابية (*H. pylori*) بكتيريا منتشرة بشكل واسع عالميا، تستوطن الغشاء المخاطي للمعدة، وترتبط بالعديد من أمراض الجهاز الهضمي (لدى الانسان)، بما في ذلك التهاب المعدة المزمن، وقرحة وسرطان المعدة. يعرف انتقال هذه البكتريا بشكل رئيسي من إنسان إلى آخر، بينما لا يزال دور الحيوانات كمستودعات محتملة للعدوى قيد البحث. هدفت هذه الدراسة إلى الكشف عن الملوية البوابية في القطط والكلاب المنزلية، ومقارنة انتشار وأنماط جينات الضراوة *cagA* و*vacA* مع تلك الموجودة في العينات السريرية البشرية. شملت الدراسة 261 عينة، مقسمة إلى 161 عينة حيوانية (كلاب وقطط) و100 خزعة معدية بشرية لمرضى التهاب المعدة. حُللت جميع العينات بتقنية الـ PCR للكشف عن الملوية البوابية باستهداف جين *ureC*. بالإضافة إلى الكشف عن عوامل الضراوة (*cagA* و*vacA*) مع تحديد أنماط *vacA* الجينية (*vacA* m1، m2، s1a، s1b، s1c و s2). تشير النتائج إلى معدلات إصابة بنسبة 8.2% لدى الكلاب، و4% لدى القطط، و25% لدى البشر. والجدير بالذكر، أنه تم رصد انتشار مرتفع لجينات *cagA* (100%) و(80%) و(96%) لدى القطط والكلاب والبشر على التوالي. كما أظهرت النتائج انتشارًا مرتفعًا في سلالة *vacA* m2/s2 بنسبة 75% لدى كل من القطط والبشر، مقارنةً بنسبة 20% لدى الكلاب. ان اكتشاف ارتفاعًا في السلالات الإيجابية لـ *cagA* لدى الحيوانات الأليفة والبشر، بالإضافة إلى تواتر في النمط الجيني *vacA* m2/s2 لدى هذه المضائف، مع ضهور معتدل لدى الكلاب، يشير إلى مسار انتقال حيواني محتمل، مع احتمالية أن تكون الحيوانات الأليفة بمثابة مستودع ومصدر لعدوى البكتيريا الحلزونية البوابية.

الكلمات المفتاحية: بكترية الملوية البوابية ، *ureC*، *cagA*، *vacA*، الامراض حيواني المنشأ.