

# **Brief Original Article**

# Serologic evidence and risk factors for *Helicobacter pylori* infection in animals and humans

Mahmoud Elhariri<sup>1</sup>, Rehab Elhelw<sup>1</sup>, Dalia Hamza<sup>2</sup>, Heba Sayed El-Mahallawy<sup>3</sup>

- <sup>1</sup> Department of Microbiology, Faculty of Veterinary Medicine, Cairo University, Cairo, Egypt
- <sup>2</sup> Department of Zoonoses, Faculty of Veterinary Medicine, Cairo University, Cairo, Egypt
- <sup>3</sup> Department of Animal Hygiene, Zoonoses, and Animal Behaviour and Management, Faculty of Veterinary Medicine, Suez Canal University, Ismailia, Egypt

#### **Abstract**

Introduction: *Helicobacter pylori* (*H. pylori*) is one of the most common bacterial infections among humans worldwide. Although many records imply its interfamilial acquisition, the role of animals remains poorly understood. This study was undertaken to investigate the seroprevalence of *H. pylori* in animals and their human contacts in Cairo and Giza governorates, Egypt.

Methodology: Commercial enzyme-linked immunosorbent assay (ELISA) kits were used to detect IgG antibodies to *H. pylori* in dogs, cattle, and humans.

Results: Seropositive dogs (35/94; 37.2%), cattle (24/80; 30%) and humans (40/90; 44.4%) were found. Seroprevalence in animals significantly varied in different areas of sample collection, but there was no association with sex or age. Human seropositivity rates were associated with increasing age; moreover, seropositive dog owners (51.7%; 15/29), had seropositive dogs. However, infection was not associated with subject's sex, occupation, or history of animal contact.

Conclusions: Our findings indicate *H. pylori* is widely distributed in cattle and dogs and their human contacts in Cairo and Giza, Egypt. Further studies to determine infection in other occupational groups are needed. This study provides baseline information on the seroprevalence of *H. pylori*, which may be required to begin prevention control programs in our area.

Key words: dogs; cattle; human; Egypt; ELISA; Helicobacter pylori.

J Infect Dev Ctries 2017; 11(5):414-419. doi:10.3855/jidc.9339

(Received 23 August 2016 - Accepted 13 january 2017)

Copyright © 2017 Elhariri et al. This is an open-access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

#### Introduction

Helicobacter pylori (H. pylori) is one of the most common bacterial infections among humans, and has worldwide distribution. The causative agent is a helical, microaerophilic, flagellated Gram-negative bacterium that inhabits and adapts to the acidic human gastric mucosa [1]. The organism colonizes about 50% of the world population's upper gastrointestinal tracts [2], which makes the infection a public health concern worldwide [3]. Although infected individuals usually never encounter clinical symptoms except chronic gastritis, acute infections can cause acute gastritis with abdominal pain or nausea [4,5]. The ability of the organism to hydrolyze urea leads to gastric ulcers and increases the risk of developing duodenal and stomach cancer to the level that the World Health Organization (WHO) classifies as a class I carcinogen, where the infection is found in 80%–90% of patients with gastric ulcers [6,7].

The prevalence rate of *H. pylori* varies greatly by the geographic area, age, and socioeconomic status. Infection appears to be more common in developing than in developed countries [8,9]. Modes of infection are yet not clearly understood. Although spread can occur through the environment or via reservoirs or vectors, little is known about the main route of dissemination from an infected individual [10]. Transmission from person to person, through gastrooral, oral-oral, fecal-oral routes, and through exposure to contaminated food or water is highly controversial; furthermore, close and intense human contact with animals has been identified as a risk factor [11-13]. The possibility of zoonotic transmission from animals has been previously suggested [14]. Moreover, detection of anti-H. pylori antibodies in abattoir workers in France and in northern Sardinian shepherds, at higher levels than their siblings inhabiting the same house [15,16], demonstrates the zoonotic importance of the organism.

However, the exact role of animals as a reservoir of infection remains unclear.

Most data about the rates of *H. pylori* infection in different geographical and demographic populations comes from seroprevalence studies [17]. Serological tests using enzyme-linked immunosorbent assay (ELISA) are preferred as a noninvasive alternative method to endoscopy and biopsy for rapid diagnosis of *H. pylori*. Thus, serological tests have been used extensively in screening humans in clinics and for epidemiologic studies [18]. Unfortunately, in Egypt, very little epidemiological data are available about the situation of *H. pylori* infection in humans and animals. To provide further information, the present study was undertaken to address the occurrence of *H. pylori* infection in apparently healthy humans and animals, including their owners.

# Methodology

Ethics statement

Protocols for the collection of samples were reviewed and approved by the Scientific Research Committee and Bioethics Board of Suez Canal University, Faculty of Veterinary Medicine, Ismailia, Egypt (No. 2016086).

# Sample collection

Whole blood was collected from convenience samples of apparently healthy cattle and dogs. Cattle were randomly sampled from farms in El-Badrasheen and Mazghona and Gameeit Ahmed Orabi of Giza and Cairo governorates of Egypt, respectively. Domestic dogs were those mostly kept indoor and admitted with their owners to small animal veterinary clinics in El-Haram and Heliopolis and El-Maadi for other purposes. Stray dogs were those had been captured roaming in rural and suburban areas in Giza and Cairo governorates. Human whole blood samples (n = 90) were collected from consenting apparently healthy people (n = 61) attending the Giza and Cairo hospitals for routine health examinations and from owners of

some of the sampled dogs (n = 29). Demographic data on age and sex were also obtained.

Following collection, samples were transported on ice box to Cairo University, Faculty of Veterinary Medicine, where whole blood samples were centrifuged, and aliquots of sera were separated and stored at -20°C for ELISA.

# Serological assay

The canine, bovine, and human *H. pylori* IgG (Hp-IgG) indirect ELISA kits (MyBioSource, San Diego, USA) were used according to the manufacturer's instructions to detect IgG antibodies against *H. pylori* in sera of dogs, cattle, and humans, respectively.

The sample optical densities (OD) were measured using a microplate reader (CLINDIAG, Orange, USA) at 450 nm, and the sample-to-negative ratio was determined. As recommended by the manufacturer, for human kits, samples were considered to be ELISA positive if the OD sample/OD negative  $\geq 2.1$ , while if OD sample/OD negative less than 2.1, the sample was considered as negative. For canine and bovine *H. pylroi* IgG ELISA kits, the cut-off was calculated based on the following formula: average of negative control + 0.15. Samples exceeding the calculated cut-off value were considered positive.

# Statistical analysis

PASW Statistics, SPSS version 18.0 software (SPSS Inc., Armonk, USA) was used to analyze the data. Chi-squared ( $\chi^2$ ) and Fisher's exact tests were performed to analyze *H. pylori* antibody positivity between various groups. Differences were considered statistically significant if the P value was < 0.05.

#### Results

Serologic detection of anti-H. pylori antibodies in dogs Overall, a total of 264 whole blood samples from dogs (n = 94), cattle (n = 80), and people (n = 90) were tested. Generally, there was no statistically significant difference in the seroprevalence in dogs (37.2%; 35/94), cattle (30%; 24/80), and humans (p = 0.151). A

**Table 1.** Seroprevalences of IgG antibodies to *H. pylori* by ELISA in dogs from Cairo and Giza governorates.

Source of sample collection	Dogs					Positive dogs
	Male		Female		Total positive	belonging to
	No. examined	No. of <i>H. pylori</i> + (%)	No. examined	No. of <i>H. pylori</i> + (%)	(No., %)	positive dog owners
Stray dogs	7	3 (42.9%)	11	2 (18.2%)	5 (18, 27.8%)	-
El-Maadi	14	3 (21.4%)	10	4 (40%)	7 (24, 29.2%)	5
Heliopolis	21	4 (19%)	7	1 (14.3%)	5 (28, 17.9%)	2
El-Haram	13	12 (92.3%)	11	6 (54.5%)	18 (24, 75%)	8
Total	55	22 (40%)	39	13 (33.3%)	35 (94, 37.2%)	15

relatively large percentage of dogs were found to have antibodies to  $H.\ pylori$  in the ELISA test (37.2%; 35/94) (Table 1). Prevalence significantly varied by the area of sample collection, with the highest found in dogs from El-Haram (75%; 18/24), El-Maadi (29.2%; 7/24), and Heliopolis (17.9%; 5/28) (p < 0.05). There was no significant difference in seropositivity in male (22/55) and female dogs (13/39) (p > 0.05) (Table 1).

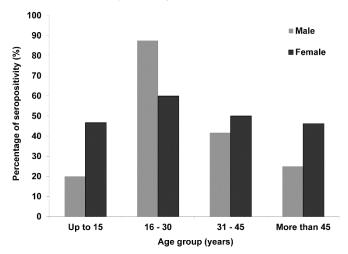
Serologic detection of anti-H. pylori antibodies in cattle

Cattle from three different farms were examined: one in Cairo (Gameet Ahmed Orabi) and two in Giza governorates (Badrashen and Mazghona), in addition to sporadic cases bred by individual farmers (Table 2). Thirty percent of the total examined cattle were seropositive (24/80). Prevalence significantly varied by the area of sample collection, with the highest found in Badrashen in Giza governorates (52%, 13/25) and Gameet Ahmed Orabi in Cairo governorate (30%, 6/20); the lowest was in Mazghona in Giza governorate (14.3%, 5/35) (p < 0.05). Seropositivity was not associated with age or type of animal production (p > 0.05) (Table 2).

Serologic detection of anti-H. pylori antibodies in humans

A total of 44.4% (40/90) of humans were found to have antibodies to *H. pylori*. There was no statistically significant difference in the seropositivity in men (17/44) and women (23/46), while there was significant difference among different age groups (p < 0.05) (Table 3, Figure 1). Although 15 seropositive dog owners had seropositive dogs, there was no statistically significant difference in seropositivity in dog owners (51.7%;

**Figure 1.** Seroprevalence of *H. pylori* in people by age and gender using ELISA. Seropositivity was associated with increasing age but not with gender. Individuals in the third decade of life were more likely to have a higher seroprevalence than those under 20 years of age.



15/29) and people with no history of animal contact (41%; 25/61) (p = 0.338) (Table 4).

#### **Discussion**

This study was undertaken as an attempt to evaluate the distribution and possible zoonotic relationship of H. pylori infection in Giza and Cairo governorates, Egypt. Overall, the differences of seroprevalence in the examined dogs, cattle, and humans was not statistically significant (p = 0.151). Seroprevalence in dogs was 37.2% (Table 1). Generally, serodiagnosis in dogs represents a big challenge, since they might be infected with several Helicobacter species [19-21].

Thirty percent of the examined cattle were seropositive (Table 2), whereas lower seroprevalence

Table 2. Seroprevalences of IgG antibodies to *H. pylori* by ELISA in cattle from Cairo and Giza governorates.

	Purpose of animal	Cairo	G		
Source of sample collection		Gameet Ahmed Orabi	Badrashin	Mazghona No. examined (+, %)	Total number examined (+, %)
		No. examined (+, %)	No. examined (+, %)		
Farms	Meat production				
	Adults	7 (4, 57.1%)		15 (5, 33.3%)	22 (9, 40.9%)
	Calves and heifers	13 (2, 15.4%)			13 (2, 15.4%)
	Total	20 (6, 30%)		15 (5, 33.3%)	35 (11, 31.4%)
	Milk production				
	Adults				
	Calves and heifers		15 (9, 60%)		15 (9, 60%)
	Total		15 (9, 60%)		15 (9, 60%)
Sporadic cases	Adults		10 (4, 40%)	15 (0, 0.0%)	25 (4, 16%)
	Calves and heifers			5 (0, 0.0%)	5 (0, 0.0%)
	Total		10 (4, 40%)	20 (0, 0.0%)	30 (4, 13.3%)
Total		20 (6, 30%)	25 (13, 52%)	35 (5, 14.3%)	80 (24, 30%)

**Table 3.** Seropositivity of IgG antibodies to *H. pylori* by ELISA among humans of different age groups.

	Male		Female		Total araminad
Age group (years)	No. of subjects examined	No. of <i>H. pylori</i> + (%)	No. of subjects examined	No. of <i>H. pylori</i> + (%)	Total examined (positive, %)
Up to 15	20	4 (20%)	15	7 (46.7%)	35 (11, 31.4%)
16–30	8	7 (87.5%)	10	6 (60%)	18 (13, 72.2%)
31–45	12	5 (41.7%)	8	4 (50%)	20 (9, 45%)
More than 45	4	1 (25%)	13	6 (46.2%)	17 (7, 41.2%)
Total	44	17 (38.6%)	46	23 (50%)	90 (40, 44.4%)

Table 4. Seropositivity of IgG antibodies to H. pylori by ELISA in dog owners and subjects with no history of animal contact.

Occupation of human	Ma	Fer		nale	
Occupation of human subjects	No. of subjects examined	No. of <i>H. pylori</i> + (%)	No. of subjects examined	No. of <i>H. pylori</i> + (%)	Total (positive, %)
Apparently healthy	28	11 (39.3%)	33	14 (42.4%)	61 (25, 41%)
Dog owners	16	6 (37.5%)	13	9 (69.2%)	29 (15, 51.7%)
Total	44	17 (38.6%)	46	23 (50%)	90 (40, 44.4%)

was reported in other studies [22,23]. Previous detection of *H. pylori* from bovine feces [23,24] and/or milk [23,25] and seroconversion in farm workers [26,27], however, might suggest the probable role of this animal species in transmission of infection to humans in case of inappropriate farm management practices.

In our investigation, the overall seroprevalence in humans was relatively high (44.4%) (Table 3). This was somehow higher than results shown in previous studies from other countries: United Kingdom (27.6%) [28], Australia 15.4% [29], and United State (36.3%) [30], while it was lower than reports in Uganda (87%) [31], China (62%) [32], and neighboring Arabian countries, mainly Libya (94%) [33] and Sudan (65%) [34]. The diversity of crowding, socioeconomic status, and environmental and hygiene factors may play an important role in increased rates of *H. pylori* infection in developing countries.

Results showed an association between seroprevalence and increasing age of the examined subjects (Table 3, Figure 1). Similarly, this was reported in other studies [29,35]. Individuals in the third decade of life were more likely to have a higher seroprevalence than those under 20 years of age. This may be because Egyptian youth in such age groups like to have food away from home during their outside activities in trips or camps; thus, they might be infected from consumption of contaminated food or water sources. Generally, it is estimated that colonization of H. pylori in gastric mucosa is associated with old age, male sex, and low socioeconomic status [11,12].

The controversial finding is that around fifty percent (51.7%; 15/29) of the dog owners who reacted

on ELISA had seropositive dogs (Table 4). Although there was no significant difference in the seropositivity between dog owners and the other group, contact with dogs was identified as a risk factor for acquiring *H. pylori* infection in other studies [36,37]. Similar to our findings, in other seroepidemiological studies, the exact relationship between pet ownership and human seropositivity cannot be clearly established [38,39].

It is worth mentioning that an association had been reported between animal contact and seropositivity in abattoir workers and veterinarians working in abattoirs and meat processing plants in New Zealand [40,41]. However, the cross-reactivity with antibodies to other gastrointestinal organisms which might be acquired from slaughtered animals was not excluded in those studies. Thus, these findings are questionable, and it was suggested that this higher prevalence was due to cross-reactivity to Campylobacter jejuni [14]. In another study, high seroprevalence was reported in shepherds from northern Sardinia in comparison to their siblings inhabiting the same house who had no contact with sheep. Authors claimed that contact with sheep and sheepdogs was a risk factor for infection [16]. However, other authors failed to isolate H. pylori and/or to detect its antibodies due to natural infection from stray and pet cats [42,43] and pigs in abattoirs [44]. These questionable data suggest doubtful zoonotic transmission of such agent, and whether animals are true reservoir hosts for H. pylori or not is still not obvious. It seems that infection might be contracted from a common source (e.g., drinking water, consumption of raw vegetables) or might suggest that H. pylori infection could be an anthroponosis (infections maintained mainly in humans that may be transmitted to animals) [42].

It was very difficult in this investigation to implicate or rule out infection of *H. pylori* to animal contact alone since combinations of other factors, including hygienic conditions, environmental factors, and socioeconomic status, contribute to the spread of the disease.

#### **Conclusions**

H. pylori is common in humans and animals in Cairo and Giza governorates, Egypt. Epidemiology of H. pylori is complex, and the zoonotic risk has not been clearly identified in this study. Further investigations with greater numbers of samples are essential to study the mechanism of disease transmission and potential risks for acquisition of infection. The present results provide a background and baseline data that may be required for commencing Helicobacter control programs in the studied area.

### Acknowledgements

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

#### References

- Ruggiero P (2010) Helicobacter pylori and inflammation. Curr Pharm Des 16: 4225-4236.
- Atherton J (2006) The pathogenesis of H. pylori-induced gastro-duodenal diseases. Annu Rev Pathol 1: 63-96.
- Magalhaes Queiroz DM, Luzza F (2006) Epidemiology of Helicobacter pylori infection. Helicobacter 11 Suppl 1: 1-5.
- Butcher GP (2003) Gastroenterology: An Illustrated Colour Text, 1st edition. Edinburgh; New York: Churchill Livingstone. 128 p.
- Bytzer P, Dahlerup JF, Eriksen JR, Jarbøl DE, Rosenstock S, Wildt S (2011) Diagnosis and treatment of *Helicobacter pylori* infection. Dan Med Bull 58: C4271.
- Lynch NA (2002) Helicobacter pylori and Ulcers: A Paradigm Revised. Available: http://www.faseb.org/Portals/2/PDFs/opa/pylori.pdf. Accessed 14 March 2016.
- Banerjee HN, Gramby M, Hawkins Z (2011) Molecular diagnosis of *Helicobacter pylori* strain by 16S rDNA PCR amplification and direct sequencing. J Bioprocess Biotech 1: 1-2
- Mitchell HM, Li YY, Hu PJ, Liu Q, Chen M, Du GG, Wang ZJ, Lee A, Hazell SL (1992) Epidemiology of *Helicobacter* pylori in southern China: identification of early childhood as the critical period for acquisition. J Infect Dis 166: 149-153.
- Pounder RE, Ng D (1995) The prevalence of *Helicobacter* pylori infection in different countries. Aliment Pharmacol Ther 9 Suppl 2: 33-39.
- Vale F, Vítor J (2010) Transmission pathway of *Helicobacter* pylori: does food play a role in rural and urban areas? Int J Food Microb 138: 1-12.

- 11. De Bock M, Van den Bulck K, Hellemans A, Daminet S, Coche JC, Debongnie JC, Decostere A, Haesebrouck F, Ducatelle R (2007) Peptic ulcer disease associated with *Helicobacter felis* in a dog owner. Europ J Gastroenter Hepat 19: 79-82.
- Haesebrouck F, Pasmans F, Flahou B, Chiers K, Baele M, Meyns T, Decostere A, Ducatelle R (2009) Gastric Helicobacters in domestic animals and nonhuman primates and their significance for human health. Clin Microb Rev 22: 202-223
- Lavelle JP, Landas S, Mitros FA, Conklin JL (2015) Acute gastritis associated with spiral organisms from cats. Dig Dis Sci 39: 744-750.
- 14. Fox JG (1995) Non-human reservoirs of *Helicobacter pylori*. Aliment Pharmacol Ther 9 Suppl 2: 93-103.
- Husson MO, Vincent P, Grabiaud MH, Furon D, Leclerc H (1991) Anti-Helicobacter pylori IgG levels in abattoir workers. Gastroenterol Clin Biol 15: 723-726.
- Dore MP, Bilotta M, Vaira D, Manca A, Massarelli G, Leandro G, Atzei A, Pisanu G, Graham DY, Realdi G (1999) High prevalence of *Helicobacter pylori* infection in shepherds. Dig Dis Sci 44: 1161-1164.
- 17. Parsonnet J (1995) The incidence of *Helicobacter pylori* infection. Aliment Pharm Therap 9 Suppl 2: 45-51.
- 18. Neiger R, Simpson KW (2000) *Helicobacter* Infection in dogs and cats: Facts and fiction. J Vet Int Med 14: 125-133.
- Eaton KA, Dewhirst FE, Paster BJ, Tzellas N, Coleman BE, Paola J, Sherding R (1996) Prevalence and varieties of Helicobacter species in dogs from random sources and pet dogs: Animal and public health implications. J Clin Microb 34: 3165-3170.
- Jalava K, On SLW, Vandamme P, Happonen I, Sukura A, Hänninen ML (1998) Isolation and identification of Helicobacter spp. from canine and feline gastric mucosa. App Environ Microb 64: 3998-4006.
- Neiger R, Tschudi M, Burnens AP, Göke B, Schmassman A (1999) Diagnosis and identification of gastric *Helicobacter* species by PCR in dogs. Microb Ecol Health Dis 11: 234-240.
- Vaira D, Ferron P, Negrini R, Cavazzini L, Holton J, Ainley C, Londei M, Vergura M, Dei R, Colecchia A (1992) Detection of Helicobacter pylori-like organisms in the stomach of some food-source animals using a monoclonal antibody. Ital J Gastroenter 24: 181-184.
- Safaei HG, Rahimi E, Zandi A, Rashidipour A (2011)
   Helicobacter pylori as a zoonotic infection: the detection of H.
   pylori antigens in the milk and faeces of cows. J Res Med Sci
   16: 184-187.
- Fujimura S, Kawamura T, Kato S, Tateno H, Watanabe A (2002) Detection of *Helicobacter pylori* in cow's milk. Lett Appl Microbiol 35: 504-507.
- Rahimi E, Kheirabadi EK (2012) Detection of *Helicobacter* pylori in bovine, buffalo, camel, ovine, and caprine milk in Iran. Foodborne Path Dis 9: 453-456.
- Bener A, Adeyemi EO, Almehdi AM, Ameen A, Beshwari M, Benedict S, Derballa MF (2006) *Helicobacter pylori* profile in asymptomatic farmers and non-farmers. Int J Environ Health Res 16: 449-454.
- Celiński K, Kurzeja-Mirosław A, Słomka M, Cichoz-Lach H, Madro A, Kasztelan-Szczerbińska B (2006) The effects of environmental factors on the prevalence of *Helicobacter pylori* infection in inhabitants of Lublin Province. Ann Agric Environ Med 13: 185-191.

- 28. Moayyed P, Axon AT, Feltbower R, Duffett S, Crocombe W, Braunholtz D, Richards ID, Dowell AC, Forman D, Leeds HELP Study Group (2002) Relation of adult lifestyle and socioeconomic factors to the prevalence of *Helicobacter pylori* infection. Int J Epidemiol 31: 624-631.
- Moujaber T, MacIntyre CR, Backhouse J, Gidding H, Quinn H, Gilbert GL (2008) The seroepidemiology of *Helicobacter* pylori infection in Australia. Int J Infect Dis 12: 500-504.
- 30. Smith JG, Li W, Rosson RS (2009) Prevalence, clinical and endoscopic predictors of *Helicobacter pylori* infection in an urban population. Connect Med 73: 133-137.
- 31. Newton R, Ziegle JL, Casabonne D, Carpenter L, Gold BD, Owens M, Beral V, Mbidde E, Parkin DM, Wabinga H (2006) *Helicobacter pylori* and cancer among adults in Uganda. Infect Agent Cancer 1: 5.
- 32. Shi R, Xu S, Zhang H, Ding Y, Sun G, Huang X, Chen X, Li X, Yan Z, Zhang G (2008) Prevalence and risk factors for *Helicobacter pylori* infection in Chinese populations. Helicobacter 13: 157-165.
- Bakka AS, El-Gariani AB, AbouGhrara FM, Salih BA (2002)
   Frequency of *Helicobacter pylori* infection in dyspeptic patients in Libya. Saudi Med J 23: 1261-1265.
- 34. Abdallah TM, Mohammed HB, Mohammed MH, Ali AA (2014) Sero-prevalence and factors associated with *Helicobacter pylori* infection in Eastern Sudan. Asian Pac J Trop Dis 4: 115-119.
- 35. Graham DY, Malaty HM, Evans DG, Evans DJJr, Klein PD, Adam E (1991) Epidemiology of *Helicobacter pylori* in an asymptomatic population in the United States. Effect of age, race, and socioeconomic status. Gastroenterology 100: 1495-1501.
- Lindo JF, Lyn-Sue AE, Palmer CJ, Lee MG, Vogel P, Robinson RD (1999) Seroepidemiology of *Helicobacter pylori* infection in a Jamaican community. Trop Med Int Health 4: 862-866.
- Dore MP, Malaty HM, Graham DY, Fanciulli G, Delitala G, Realdi G (2002) Risk Factors Associated with *Helicobacter* pylori Infection among Children in a Defined Geographic Area. Clin Infect Dis 35: 240-245.

- Ansorg R, Vonheinegg EH, Vonrecklinghausen G (1995) Cat owner's risk of acquiring a *Helicobacter pylori* infection. Zentralbl Bakteriol Int J Med Microbiol Vir Parasitol Infect Dis 283: 122-126.
- 39. Bode G, Rothenbacher D, Brenner H, Adler G (1998) Pets are not a risk factor for *Helicobacter pylori* infection in young children-results of a population-based study in southern Germany. Pediatr Infect Dis J 17: 909-912.
- Morris, A, Nicholson, G, Lloyd, G, Haines D, Rogers A, Taylor D (1986) Seroepidemiology of *Campylobacter* pyloridis. NZ Med J 99: 657-659.
- 41. Vaira D, D'Anastasio C, Holton J, Dowsett JF, Londei M, Bertoni F, Beltrandi E, Grauenfels P, Salmon PR, Gandolfi L (1988) *Campylobacter pylori* in abattoir workers: Is it a zoonosis? Lancet 2: 725-726.
- 42. El-Zaatari FAK, Woo JS, Badr A, Osato MS, Serna H, Lichtenberger LM, Genta RM, Graham DY (1997) Failure to isolate *Helicobacter pylori* from stray cats indicates that *H. pylori* in cats may be an anthroponosis-An animal infection with a human pathogen. J Med Microbiol 46: 372-376.
- Neiger R, Dieterich C, Burnens AP, Waldvogel A, Corthésy-Theulaz I, Halter F, Lauterburg B, Schmassmann A (1998) Detection and prevalence of *Helicobacter* infection in pet cats. J Clin Microbiol 36: 634-637.
- 44. Grasso GM, Ripabelli G, Sammarco ML, Ruberto A, Iannitto G (1996) Prevalence of *Helicobacter*-like organisms in porcine gastric mucosa-a study of swine slaughtered in Italy. Comp Immunol Microbiol Infect Dis 19: 213-217.

# **Corresponding author**

Heba S El-Mahallawy Department of Animal Hygiene, Zoonoses, and Animal Behaviour and Management Faculty of Veterinary Medicine, Suez Canal University Ring Rd., Kilo 4.5, 41522, Ismailia, Egypt

Phone: +20 100 294 2291

Email: dr\_ba1012\_2@hotmail.com, Heba\_elmahalawi@vet.suez.edu.eg

Conflict of interests: No conflict of interests is declared.