

Role of food in environmental transmission of *Helicobacter pylori*

Mohammad Zamani^{1,2}
Amin Vahedi¹
Zahra Maghdouri (MSc)³
Javad Shokri-Shirvani (MD)^{*2,4}

1. Student Research Committee, Babol University of Medical Sciences, Babol, Iran.
2. Cancer Research Center, Health Research Institute, Babol University of Medical Sciences, Babol, Iran.
3. School of Medicine, Babol University of Medical Sciences, Babol, Iran.
4. Department of Internal Medicine, Ayatollah Rohani Hospital, Babol University of Medical Sciences, Babol, Iran.

*** Correspondence:**

Javad Shokri-Shirvani, Rohani Hospital, Babol University of Medical Sciences, Ganjafrooz Street, Babol, Mazandaran, Iran.

E-mail:

javadshokri121@gmail.com

Tel: 0098 1132199592

Fax: 0098 1132190181

Received: 8 Feb 2017

Revised: 17 April 2017

Accepted: 18 April 2017

Abstract

Helicobacter pylori (*H.pylori*) is a gram-negative bacterium that has infected more than half of the world's population. This pathogen colonizes the human gastric mucosa and is usually acquired during childhood. It is an important cause of peptic ulcers, chronic gastritis and stomach cancer. Among the risk factors for acquisition of *H. pylori* infection, poor socioeconomic status, poor sanitization and hygiene practices, and contaminated food and water, are the most significant ones. The main route of *H. pylori* transmission is still unknown. Studies show that *H.pylori* bacteria can spread directly from one person to the other, or indirectly from an infected person to the environment. Person to person transmission is divided into fecal-oral, gastric-oral, oral-oral, sexual routes. Presently, interpersonal pathways are more acceptable than environmental exposure routes. Literatures indicate the presence and survival of *H. pylori* in food samples, such as milk, vegetables and meat, and suggest these foods may play an important role in the environmental transmission of this pathogen. In addition, other studies report the presence of *H. pylori* in the gastric tissue of some animals (e.g. sheep and cow) and therefore, it is likely they participate in the food chain transmission as reservoirs besides human. Although there are findings which indicate the probable role of food products in the environmental transmission of *H. pylori*, there is still not enough direct evidence to confirm this and more studies are needed. However, attention to food contamination sources (unhygienic water) and controlling them may prevent transmission of pathogens associated with health.

Keywords: *Helicobacter pylori*, Transmission, Food, Water, Reservoir

Citation:

Zamani M, Vahedi A, Maghdouri Z, Shokri-Shirvani J. Role of food in environmental transmission of *Helicobacter pylori*. Caspian J Intern Med 2017; 8(3): 146-152.

H*elicobacter pylori* is a spiral-shaped gram-negative microaerophilic bacterium which is found in the human gastric mucosa. This pathogen was first isolated by Warren and Marshall (1) about 30 years ago. It was isolated from the human stomach but the principle mechanism by which it colonizes is still unclear (2, 3). Human stomach is currently the only known reservoir for this pathogen. The bacterial pathogen *H. pylori* infects about 50% of the human population around the world. Seroepidemiological studies of *H. pylori* show that the rate of infection regionally changes and it is lower in developed countries (about 30-40%) compared with developing countries (in some areas, >85%) (4-6). According to reports, its prevalence has declined in the world, which can be explained by the improvement of hygiene (7). This microorganism can be associated with the pathogenesis of some gastrointestinal diseases, such as chronic antral gastritis of type B, peptic ulcers, mucosa associated lymphoid tissue lymphoma and gastric adenocarcinoma (8-13). Furthermore, possible associations have been reported between *H. pylori* and a number of extragastric manifestations related to cardiovascular, dermatological, neurological, immunological, hematological, hepatobiliary, respiratory, and endocrine and metabolic disorders (14-17).

On other hand, increasing antimicrobial resistance of *H. pylori* has increased concerns about treatment failure and lack of control of the important mentioned gastrointestinal diseases (18, 19). Several epidemiologic risk factors for acquisition of *H. pylori* infection have been highlighted and are summarized in figure 1 (4, 20, 21). Most of risk factors are related to poor living conditions and there is no difference in this respect between the developed and developing countries (22). There are inconsistent findings regarding the association between *H. pylori* infection and some factors, such as gender and lifestyle habits (e.g. smoking and alcohol drinking) (3, 23).

The principle mechanism by which *H. pylori* infection is transmitted to humans is still not exactly defined, however, person to person and environment to person transmissions are two potential options according to studies. Interpersonal transmission may occur via several pathways, including fecal-oral, gastric-oral, oral-oral, and sexual (24-26).

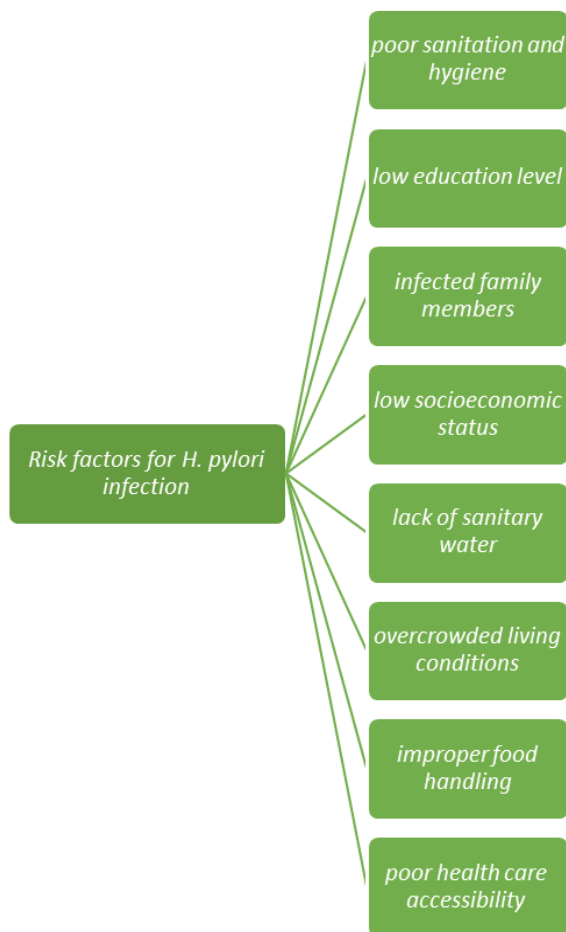


Figure 1. Main potential risk factors for *H. pylori* infection.

Evidence for the role of food in environmental transmission

A) Food as a route of transmission: The hypothesis that food is a route of transmission of *H. pylori* is supported by epidemiologic studies that have observed a higher prevalence of *H. pylori* infection and a more rapid acquisition rate in areas with poor hygienic conditions (27-29). Altogether, the food-borne transmission of *H. pylori* can be attributed to two modes of person to person and environment to person. For instance, the infection might be transmitted by ingesting foods from street vendors (22, 30). This infection may occur directly by vendors (interpersonal) or indirectly by contaminated foods (environmental). It has also been proposed that saliva could be a source of *H. pylori* (25), so food soaked in contaminated saliva can transfer the agent. On the other hand, there is some evidence supporting the role of contaminated foods in the environmental transmission of *H. pylori*, which are expressed below.

B) Detection of *H. pylori* in food samples.

Dairy products: According to the literatures, animal source foods, and specifically raw milk, have been considered as the most likely source of human infection in food chain transmission, since *H. pylori* DNA has been isolated from animal milk samples (e.g. sheep and cow) (31, 32). Of course, some other surveys reported no isolation of the organism from milk samples, which might be explained by the geographical spread of *H. pylori* (33, 34). This failure can also be due to the viable but nonculturable (VBNC) state of *H. pylori*. This state occurs to withstand environmental stressful conditions and starvation (35-37). It has been discussed that the process by which milk is contaminated may be related to lack of attention to hygiene measures during milking, cooling and storage (38). Examples of studies which have indicated that *H. pylori* DNA is present in raw milk are summarized in table 1. In addition, limited studies are available concerning the occurrence of *H. pylori* in dairy products other than milk. For example, in the study by Mousavi et al., 30% of cheese, 15% of cream, 5% of butter and 27% of ice cream samples, which were all made from unpasteurized milk, were positive for *H. pylori* (39).

Previous published data revealed the isolation of *H. pylori* from gastric tissue of several animals, taking part in the human food chain, such as sheep and cow, and this led researchers to presume them as plausible reservoirs and sources of the infection (32, 40, 41). This evidence also can support the theory of zoonotic transmission of *H. pylori*. Some studies addressed the high prevalence of *H. pylori*

among shepherds and stated that sheep may play a vital role in *H. pylori* transmission in these people and their family members and the infection may originate from these animal

species (42-44). According to the above data, and also on the possible transmission via sheep milk, *H. pylori* infection can be considered as zoonosis.

Table 1. Studies evaluating the presence of *H. pylori* DNA in milk.

| Type of milk | Samples No. | Gene | Diagnostic method | Percentage of positive samples | Reference |
|-----------------|-------------|-----------------------|-------------------|---------------------------------------|-----------|
| Sheep raw | 58 | <i>cagA</i> | PCR | 6.9 | (59) |
| Cow raw | 75 | | | 13.3 | |
| Goat raw | 42 | | | 4.7 | |
| Buffalo raw | 20 | | | 15 | |
| Camel raw | 15 | | | 6.6 | |
| Bovine bulk | 135 | <i>ureC</i> | PCR | 14.1 | (38) |
| Ovine bulk | 90 | | | 12.2 | |
| Caprine bulk | 103 | | | 8.7 | |
| Camel bulk | 55 | | | 3.6 | |
| Buffalo bulk | 64 | | | 23.4 | |
| Buffalo bulk | 210 | <i>ureC</i> | PCR | 11.4 | (60) |
| Goat raw | 160 | <i>glmM</i> | Nested PCR | 25.6 | (61) |
| Sheep raw | 130 | | | 33 | |
| Cow raw | 110 | | | 50 | |
| Cow raw | 20 | 16S rRNA | FISH | 20 | (62) |
| Sheep raw | 63 | 16 rRNA & <i>vacA</i> | PCR | 60.3 (16S rRNA) 7.9 (both of them) | (40) |
| Sheep raw | 51 | 16 rRNA & <i>vacA</i> | PCR | 60.3 (16S rRNA) 9.8 (both of them) | (63) |
| Cow raw | 25 | <i>ureC</i> | PCR | 16 | (64) |
| Cow raw | 18 | <i>ureA</i> | Semi-nested-PCR | 72 | (65) |
| Cow pasteurized | 20 | | | 55 | |
| Cow raw | 120 | <i>vacA</i> | PCR | 20.83 | (53) |
| Goat raw | 80 | | | 18.75 | |
| Sheep raw | 120 | | | 29.16 | |
| Camel raw | 50 | | | 10 | |
| Buffalo raw | 50 | | | 24 | |

Vegetables: Few reports have addressed the occurrence of *H. pylori* in vegetables. For instance, in Iran, Atapoor et al. collected 460 vegetable and salad samples from supermarkets and grocery stores and examined them by culture and PCR. *H. pylori* was detected in 9.56% of samples by the culture technique, whereas PCR results showed that 10.86% of samples were positive (45). Also, Yahaghi et al. examined 380 mixed vegetable and 50 salad samples and reported that 13.68% of vegetable samples and 14% of salad samples were contaminated with *H. pylori* (46). Besides, Goodman et al. evaluated the prevalence of *H. pylori*

infection in a rural community and reported that persons who are consumer of raw vegetables had more potential to be infected (47).

These results reveal that vegetables may be likely sources of *H. pylori* and can play a significant role in the transmission of *H. pylori* to humans. Studies indicate that raw vegetables may become contaminated by irrigation water or unpurified water source used through washing (47, 48). At any rate, careful and adequate washing of raw vegetables may decrease the incidence of such contamination events (45).

Yeasts: Studies show that *H. pylori* can be found inside yeasts for example *Candida* spp. Iranian researchers in a number of studies, presented evidence for the existence of non-culturable *H. pylori* in the vacuole of *Candida* spp. from food products, such as breads, banana inner skin, yogurt, quince jam and grape juice (49, 50). On the other hand, yeasts can resist stressful conditions, for example high temperature, acidic pH and high sanitization (51). In conclusion, foodborne yeasts, such as *Candida* spp., which are often found in foods (e.g. raw milk), water and various human organs such as the oral cavity and the gastrointestinal and genitourinary tracts of humans, can act as a protector and reservoir of *H. pylori* in natural environments (49, 52).

Other food products: Saeidi et al. identified *H. pylori* in meat samples of cow (25%), sheep (37%), camel (14%), buffalo (28%) and goat (22%) (53). In their article, Hemmatinezhad et al. declared that 13.45% of ready-to-eat food samples, including cream-candy, traditional bread, salami, soup, restaurant salad, hamburger, sausage, falafel, fruit salad, chicken nugget and potato salad, were contaminated with *H. pylori* (54).

C) Survival of *H. pylori* in foods

Food samples: Some published data reveal that *H. pylori* can survive for short periods in artificially contaminated food products, such as milk, vegetables and meat, which are shown in table 2.

Table 2. Studies evaluating the survival of *H. pylori in inoculated foods.**

| Type of food | Results | Reference |
|--|--|-----------|
| Milk | Recovered up to 5 days from pasteurized skim milk at 4 °C. | (66) |
| | Survived in fresh milk without preservatives for up to 10 days at 4 °C and about 3 days at 25 °C. | (67) |
| | Survived for 9 days in pasteurized milk and 12 days in UHT milk at 4 °C. | (68) |
| | Survived until 6 days in cooled milk, 3 to 4 days in milk at room temperature or 37 °C. | (69) |
| Sucuk (Turkish fermented sausage) | Survived for 7 days at 22 °C. | (56) |
| Spinach | Survived until 6 days at room temperature. | (70) |
| Irradiated (10 kGy) & autoclaved ground beef | Survived for 7 days in irradiated & 3 days in autoclaved ground beef at 37 °C. | (71) |
| Fresh leaf lettuce, raw chicken, tofu & plain yogurt | Survived at 4 °C until 2 days in lettuce & raw chicken, 5 days in tofu & not retrieved from yogurt sample. | (67) |
| Carrots & lettuce | Survived at 8 °C for up to 72 h in sanitized carrots & lettuce, until 96 h in sterilized carrots, up to 120 h in raw carrots. | (72) |
| Kefir, curd cheese, yogurt & chicken | Survived for 24 h in kefir, 10 h in curd cheese & 3 h in in yogurt at 37 °C, survived until 72 h in these three food products at 7 °C. Survived for 48 h in chicken at room temperature. | (69) |

* The diagnostic method for *H. pylori* was culture for all above articles.

Survival conditions: Findings indicate that *H. pylori* is able to survive in water and milk, fresh fruits and vegetables, fresh meat (poultry, fish and red meat), at temperatures below 30°C (29,55). Besides, any food product presenting pH ranging from 4.9 to 6.0 and water activity higher than 0.97, can help this bacterium to have more survival potential (45). Besides, researchers found that *H. pylori*'s ability to survive in an acidic pH environment is dependently correlated with urea. Survival of *H. pylori* may be prolonged in milk due to the presence of urea in this food product (56). Based on the previously published documents, *H. pylori* does

not seem to grow in most foods, but if kept refrigerated in stores, this microorganism can survive in low-acid and high-moisture settings for a long time (55). In addition, the frequent inability of sanitation schemes in clearing pathogens on raw vegetables and fruits has been predominantly ascribed to the inability of active components in treatment solutions to reach microbial cell sites (57, 58).

Conclusion Several studies indicate that this bacterium could be present in raw food products, such as milk and ready-to-eat foods like vegetables, and suggest that consumption of such foods maybe constitute a source of *H.*

pylori infection for humans. Reports also show that some animals, like cow and sheep, could act as reservoirs of this pathogen, besides humans. Confirmation of the presence of *H. pylori* in foods is mainly based on indirect results that are too few to ascribe a definite foodborne role of *H. pylori* transmission. Consequently, it is needed that more epidemiological and experimental studies be performed to corroborate this hypothesis. Finding of new food sources and reservoirs of *H. pylori* can change or improve our knowledge in the future.

Acknowledgments

We would like to thank the Department of Internal Medicine of Ayatollah Rouhani Hospital staff for all their efforts.

Conflict of interest: The authors declare no conflict of interest.

References

1. Marshall B, Warren JR. Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. *Lancet* 1984; 323: 1311-5.
2. Malfertheiner P, Link A, Selgrad M. Helicobacter pylori: perspectives and time trends. *Nat Rev Gastroenterol Hepatol* 2014; 11: 628-38.
3. Eusebi LH, Zagari RM, Bazzoli F. Epidemiology of Helicobacter pylori infection. *Helicobacter* 2014; 19: 1-5.
4. Ozaydin N, Turkyilmaz SA, Cali S. Prevalence and risk factors of helicobacter pylori in Turkey: a nationally-representative, cross-sectional, screening with the 13 C-Urea breath test. *BMC Public Health* 2013; 13: 1215.
5. Roberts S, Morrison-Rees S, Samuel D, et al. Review article: the prevalence of Helicobacter pylori and the incidence of gastric cancer across Europe. *Aliment Pharmacol Ther* 2016; 43: 334-45.
6. Khedmat H, Karbasi-Afshar R, Agah S, Taheri S. Helicobacter pylori Infection in the general population: A Middle Eastern perspective. *Caspian J Intern Med* 2013; 4: 745-53.
7. Breckan RK, Paulssen EJ, Asfeldt AM, et al. The all-age prevalence of helicobacter pylori infection and potential transmission routes. a population-based study. *Helicobacter* 2016; 21: 586-95.
8. Tokudome S, Ghadimi R, Suzuki S, et al. Helicobacter pylori infection appears the prime risk factor for stomach cancer. *Int J Cancer* 2006 ; 119: 2991.
9. Agah S, Khedmat H, Ghamar-Chehred ME, Hadi R, Aghaei A. Female gender and Helicobacter pylori infection, the most important predisposition factors in a cohort of gastric cancer: A longitudinal study. *Caspian J Intern Med* 2016; 7: 136-41.
10. Shokry Shirvani J, Siadati S, Molai M. The Frequency of helicobacter pylori infection in gastric biopsies of patients with gallbladder stones. *Govareh* 2014; 19: 208-11.
11. Tokudome S, Ando R, Ghadimi R, et al. Are there any real Helicobacter pylori infection-negative gastric cancers in Asia? *Asian Pac J Cancer Prev* 2007; 8: 462-3.
12. Zamani M, Zamani V. Helicobacter pylori antibiotic resistance: Can herbal medicine be an alternative for the treatment? *J Res Med Sci* 2016; 21: 97.
13. Kuo S, Chen L, Lin C, et al. Detection of the Helicobacter pylori CagA protein in gastric mucosa-associated lymphoid tissue lymphoma cells: clinical and biological significance. *Blood Cancer J* 2013; 3: e125.
14. Kuo CH, Chen YH, Goh KL, Chang LL. Helicobacter pylori and systemic disease. *Gastroenterol Res Pract* 2014; 2014: 358494.
15. Zamani M, Masrou Roudsari J, Zamani V. Hematologic disorder: a manifestation of helicobacter pylori infection. *Caspian J Intern Med* 2017; 8: 133-4.
16. Sotuneh N, Hosseini SR, Shokri-Shirvani J, Bijani A, Ghadimi R. Helicobacter pylori infection and metabolic parameters: Is there an association in elderly population? *Int J Prev Med* 2014; 5: 1537-42.
17. Vafaeimanesh J, Bagherzadeh M, Heidari A, Motii F, Parham M. Diabetic patients infected with helicobacter pylori have a higher Insulin Resistance Degree. *Caspian J Intern Med* 2014; 5: 137-42.
18. Zamani M, Zamani V, Shokri-Shirvani J. Helicobacter pylori Antibiotic Resistance: What is the Future of Treatment? *Iran J Pediatr* 2016; 26: e5333.
19. Thung I, Aramin H, Vavinskaya V, et al. Review article: the global emergence of Helicobacter pylori antibiotic resistance. *Aliment Pharmacol Ther* 2016; 43: 514-33.
20. Zhu Y, Zhou X, Wu J, Su J, Zhang G. Risk factors and prevalence of Helicobacter pylori infection in persistent high incidence area of gastric carcinoma in Yangzhong city. *Gastroenterol Res Pract* 2014; 2014: 481365.

21. Ding Z, Zhao S, Gong S, et al. Prevalence and risk factors of Helicobacter pylori infection in asymptomatic Chinese children: a prospective, cross-sectional, population-based study. *Aliment Pharmacol Ther* 2015; 42: 1019-26.
22. Hasosah M, Satti M, Shehzad A, et al. Prevalence and risk factors of helicobacter pylori infection in saudi children: a three-year prospective controlled study. *Helicobacter* 2015; 20: 56-63.
23. Lim SH, Kwon JW, Kim N, et al. Prevalence and risk factors of Helicobacter pylori infection in Korea: nationwide multicenter study over 13 years. *BMC Gastroenterol* 2013; 13: 104.
24. Mitchell H, Katelaris P. Epidemiology, clinical impacts and current clinical management of Helicobacter pylori infection. *Med J Aust* 2016; 204: 376-80.
25. Anand PS, Kamath KP, Anil S. Role of dental plaque, saliva and periodontal disease in Helicobacter pylori infection. *World J Gastroenterol* 2014; 20: 5639-53.
26. Dimitriadi D. Helicobacter pylori: a sexually transmitted bacterium? *Cent European J Urol* 2014; 67: 407-9.
27. Nurgalieva ZZ, Malaty HM, Graham DY, et al. Helicobacter pylori infection in Kazakhstan: effect of water source and household hygiene. *Am J Trop Med Hyg.* 2002;67(2):201-6.
28. Wynne A, Hastings EV, Colquhoun A, et al. Untreated water and Helicobacter pylori: perceptions and behaviors in a Northern Canadian community. *Int J Circumpol Health* 2013; 72: 704-5.
29. Van Duynhoven YT, Jonge Rd. Transmission of Helicobacter pylori: a role for food? *Bull World Health Organ* 2001; 79: 455-60.
30. Begue RE, Gonzales JL, Correa-Gracian H, Tang S. Dietary risk factors associated with the transmission of Helicobacter pylori in Lima, Peru. *Am J Trop Med Hyg* 1998; 59: 637-40.
31. Megraud F, Broutet N. Have we found the source of Helicobacter pylori? *Aliment Pharmacol Ther* 2000; 14: 7-12.
32. Momtaz H, Dabiri H, Souod N, Gholami M. Study of Helicobacter pylori genotype status in cows, sheep, goats and human beings. *BMC Gastroenterol* 2014; 14: 61.
33. Turutoglu H, Mudul S. Investigation of Helicobacter pylori in raw sheep milk samples. *J Vet Med B Infect Dis Vet Public Health* 2002; 49: 308-9.
34. Tabatabaei M. Application of molecular and cultural methods for identification of Helicobacter SPP. In different animal sources. *Glob Vet* 2012; 8: 292-7.
35. Percival SL, Suleman L. Biofilms and Helicobacter pylori: Dissemination and persistence within the environment and host. *World J Gastrointest Pathophysiol* 2014; 5: 122-32.
36. Ramamurthy T, Ghosh A, Pazhani GP, Shinoda S. Current perspectives on viable but non-culturable (VBNC) pathogenic bacteria. *Front Public Health* 2014; 2: 103.
37. Quaglia N, Dambrosio A, Normanno G, Celano G. Evaluation of a Nested-PCR assay based on the phosphoglucosamine mutase gene (glmM) for the detection of Helicobacter pylori from raw milk. *Food Control* 2009; 20: 119-23.
38. Rahimi E, Kheirabadi EK. Detection of Helicobacter pylori in bovine, buffalo, camel, ovine, and caprine milk in Iran. *Foodborne Pathog Dis* 2012; 9: 453-6.
39. Mousavi S, Dehkordi FS, Rahimi E. Virulence factors and antibiotic resistance of Helicobacter pylori isolated from raw milk and unpasteurized dairy products in Iran. *J Venom Anim Toxins Incl Trop Dis* 2014; 20: 51.
40. Dore MP, Sepulveda AR, El-Zimaity H, et al. Isolation of Helicobacter pylori from sheep-implications for transmission to humans. *Am J Gastroenterol* 2001; 96: 1396-401.
41. Kusters JG, van Vliet AH, Kuipers EJ. Pathogenesis of Helicobacter pylori infection. *Clin Microbiol Rev* 2006; 19: 449-90.
42. Papież D, Konturek P, Bielanski W, et al. Prevalence of Helicobacter pylori infection in Polish shepherds and their families. *Dig Liver Dis* 2003; 35: 10-5.
43. Plonka M, Bielanski W, Konturek S, et al. Helicobacter pylori infection and serum gastrin, ghrelin and leptin in children of Polish shepherds. *Dig Liver Dis* 2006; 38: 91-7.
44. Dore MP, Bilotta M, Vaira D, et al. High prevalence of Helicobacter pylori infection in shepherds. *Dig Dis Sci* 1999; 44: 1161-4.
45. Atapoor S, Dehkordi FS, Rahimi E. Detection of Helicobacter pylori in various types of vegetables and salads. *Jundishapur J Microbiol* 2014; 7: e10013.
46. Yahaghi E, Khamesipour F, Mashayekhi F, et al. Helicobacter pylori in vegetables and salads: genotyping and antimicrobial resistance properties. *Biomed Res Int* 2014; 2014:757941.

47. Goodman KJ, Correa P, Aux HJT, et al. Helicobacter pylori infection in the Colombian Andes: a population-based study of transmission pathways. *Am J Epidemiol* 1996; 144: 290-9.
48. Mazari-Hiriart M, Ponce-de-León S, López-Vidal Y, et al. Microbiological implications of periurban agriculture and water reuse in Mexico City. *PLoS One* 2008; 3: e2305.
49. Siavoshi F, Saniee P. Vacuoles of Candida yeast as a specialized niche for Helicobacter pylori. *World J Gastroenterol* 2014; 20: 5263-73.
50. Saniee P, Siavoshi F. Endocytotic uptake of FITC-labeled anti-H. pylori egg yolk immunoglobulin Y in Candida yeast for detection of intracellular H. pylori. *Front Microbiol* 2015; 6: 113.
51. Salmanian AH, Siavoshi F, Beyrami Z, et al. Foodborne yeasts serve as reservoirs of Helicobacter pylori. *J Food Saf* 2012; 32: 152-60.
52. Dworecka-Kaszak B, Krutkiewicz A, Szopa D, Kleczkowski M, Biegańska M. High prevalence of Candida yeast in milk samples from cows suffering from mastitis in Poland. *Sci World J* 2012; 2012: 196347.
53. Saeidi E, Sheikhshahrokh A. VacA genotype status of Helicobacter pylori isolated from foods with Animal Origin. *Biomed Res Int.* 2016; 2016: 8701067.
54. Hemmatinezhad B, Momtaz H, Rahimi E. VacA, cagA, iceA and oipA genotypes status and antimicrobial resistance properties of Helicobacter pylori isolated from various types of ready to eat foods. *Ann Clin Microbiol Antimicrob* 2016; 15: 2.
55. Jiang X, Doyle MP. Effect of environmental and substrate factors on survival and growth of Helicobacter pylori. *J Food Prot* 1998; 61: 929-33.
56. Guner A, Kav K, Tekinsen KK, Dogruer Y, Telli N. Survival of Helicobacter pylori in Turkish fermented sucuk and heat-treated sucuk during production. *J Food Prot* 2011; 74: 2055-61.
57. Eraky MA, Rashed SM, Nasr ME-S, El-Hamshary AMS, Salah El-Ghannam A. Parasitic contamination of commonly consumed fresh leafy vegetables in Benha, Egypt. *J Parasitol Res* 2014; 2014: 613960.
58. Vale F, Vitor J. Transmission pathway of Helicobacter pylori: does food play a role in rural and urban areas? *Int J Food Microbiol* 2010; 138: 1-12.
59. Talaei R, Souod N, Momtaz H, Dabiri H. Milk of livestock as a possible transmission route of helicobacter pylori infection. *Gastroenterol Hepatol Bed Bench* 2015; 8: S30-6.
60. Kazemeini HR, Rahimi E, Kianpour F. Prevalence of helicobacter pylori in buffalo milk in Iran. *Iranian J Publ Health* 2014; 43: 174. Available at: file:///C:/Users/hashemi/Downloads/4845-4972-1-SM%20(2).pdf
61. Quaglia N, Dambrosio A, Normanno G, et al. High occurrence of Helicobacter pylori in raw goat, sheep and cow milk inferred by glmM gene: a risk of food-borne infection? *Int J Food Microbiol* 2008; 124: 43-7.
62. Angelidis AS, Tirodimos I, Bobos M, et al. Detection of Helicobacter pylori in raw bovine milk by fluorescence in situ hybridization (FISH). *Int J Food Microbiol* 2011; 151: 252-6.
63. Dore MP, Sepulveda AR, Osato MS, Realdi G, Graham DY. Helicobacter pylori in sheep milk. *The Lancet* 1999; 354: 132.
64. Safaei HG, Rahimi E, Zandi A, Rashidipour A. Helicobacter pylori as a zoonotic infection: the detection of H. pylori antigens in the milk and faeces of cows. *J Res Med Sci* 2011; 16: 184-7.
65. Fujimura S, Kawamura T, Kato S, Tateno H, Watanabe A. Detection of Helicobacter pylori in cow's milk. *Lett Appl Microbiol* 2002; 35: 504-7.
66. Poms RE, Tatini SR. Survival of Helicobacter pylori in ready-to-eat foods at 4 degree C. *Int J Food Microbiol* 2001; 63: 281-6.
67. Fan XG, Chua A, Li TG, Zeng QS. Survival of Helicobacter pylori in milk and tap water. *J Gastroenterol Hepatol* 1998; 13: 1096-8.
68. Quaglia N, Dambrosio A, Normanno G, et al. Survival of Helicobacter pylori in artificially contaminated ultrahigh temperature and pasteurized milk. *Food Microbiol* 2007; 24: 296-300.
69. Böhmeler G, Gerwert J, Scupin E, Sinell H. The epidemiology of helicobacteriosis in humans; studies of the survival capacity of the microbe in food. *Dtsch Tierarztl Wochenschr* 1996; 103: 438-43.
70. Buck A, Oliver JD. Survival of spinach-associated Helicobacter pylori in the viable but nonculturable state. *Food Control* 2010; 21: 1150-4.
71. Jiang X, Doyle MP. Optimizing enrichment culture conditions for detecting Helicobacter pylori in foods. *J Food Prot* 2002; 65: 1949-54.
72. Gomes BC, De Martinis EC. Fate of Helicobacter pylori artificially inoculated in lettuce and carrot samples. *Braz J Microbiol* 2004; 35: 145-50.