Review Article

Campylobacter Species in the Middle East

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ABSTRACT

Campylobacter is a contributing factor in many pathogen conditions. The present study aimed to review the previous findings of studies on campylobacteriosis in the Middle East region. Acute gastroenteritis is caused by Campylobacter species, known as a widespread infectious disease with worldwide disbursement. There are various reports on the incidence of *Campylobacter* in the Middle East indicating infection rates of 1-14%. *Campylobacter jejuni* (*C. jejuni*) and *C. coli* are the main responsible campylobacteriosis for intestinal diseases. The occurrence of human campylobacteriosis is sporadic, transmission from one person to another person is too rare and the incidence is usually higher in warm seasons and tropical environments. The presence of *Campylobacter* species as normal flora in stool and its contamination in poultry meat and eggs can lead to infections. Lowering the number of *Campylobacter* in animal carcasses can greatly decrease the risk of infection in consumers. Due to the high *Campylobacter* spp. antibiotic resistance, the governments are required to devise controlling programs. People in the Middle East should buy meat from stores that are under sanitary monitoring.

1. Introduction

Gastrointestinal infections are correlated with morbidity and even cause mortality at different ages[1,2]. Recently, many outbreaks of gastrointestinal and diarrheal illnesses in the Middle East have been reported due to numerous microbial pathogens[3-6]. One of the most common and central causes of infectious diarrheal illness and gastroenteritis in adults and children worldwide is campylobacteriosis[7].

The *Campylobacter jejuni* (*C. jejuni*) and *C. coli* are the main cause of campylobacteriosis resulting in a number of gastrointestinal diseases[8,9]. The occurrence of this disease is usually higher in elderlies, young children under 4 years of age, and young adults who are 20-40 years old[2,10,11]. The transmission of this organism from one person to another person rarely occurs[12]. *Campylobacter* infection rate is reported remarkably higher than those of shigellosis and salmonellosis[13]. This high rate of infection needs to be investigated with regard to the epidemiology of *Campylobacter* infection. Red blood cells and leukocytes have been found in most of the stool samples of infected patients with slightly elevated levels of peripheral White Blood Cell

(WBC) counts[14,15].

Campylobacteriosis is prevalent in third-world countries, and the main sources of human infections are food and environmental contamination[16]. In Egypt and Iraq, *Campylobacter* is the second most important cause of pediatric diarrhea[17,18]. The present review aimed to gather information on the prevalence of campylobacteriosis in the Middle East to educate researchers and policymakers to plan effective control strategies to overcome this overwhelming infection.

2. Incidence of disease

The prevalence rate of *Campylobacter* infection is associated with pathogen agents, geographical restrictions, and habitual conditions. Middle Eastern countries often do not have any national surveillance programs for controlling this infection. As can be seen in Table 1, there are different reports about the prevalence of *Campylobacter* in these countries indicating different infection rates[3,19,20].

Table 1. Prevalence of *Campylobacter* species in meats of animals and human samples in the Middle East

Country	City or state	Year	H-A*	Age	Incidence rate
Iran	Shiraz	2004	H	All ages	9.6%
	Zanjan	2013-2014	H	Adult	6.4%
	Tehran	2004-2005	H	All ages	5.4%
	Hamedan	2013-2014	H	Child	10%
	Ardabil	2016	H	Adult	6%
	Babol	2002	H	Child	4.6%
	Semnan	2007	H	Child	12.4%
	Mashhad	2005	A	-	76%
	Esfahan	2006-2008	A	-	47.1%
Egypt	Cairo	1993	H	Child	8%
	Menia, Fayoum, Cairo, Qaluobya	2015	H	Child	35%
	Qena	2014-2015	A	-	24.6%
	Beheira	1995-1998	H	Child	13%
Turkey	Istanbul	2013-2015	H	All ages	4.2%
	Ankara	1993-1994	H	Child	8.3%
	Kayseri	2002-2003	H	All ages	1.43%
Iraq	Basrah	2000-2001	H	Child	13.86 %
	Baghdad	1991	H	Child	10%
Saudi Arabia	Makkah, Jeddah	2012	H	All ages	1.7%
	Jeddah	1989-1990	H	All ages	4.5%

*H-A: Human-Animal

Investigation of *Campylobacter* prevalence in the Middle East has shown that this bacterium should be considered as one of the major pathogens in inflammatory diarrheal cases. However, during 2004-2005 in Iran, the prevalence of *Campylobacter* spp. was significantly lower than the *Shigella* spp., *Salmonella* spp., and *Escherichia coli*[21].

The prevalence of *Campylobacter* species among 1010 stool samples collected from patients in the northwest of Iran during 2016 confirmed 6% of contamination[22]. There are some reports on infection rates from Iranian researchers, such as 4.6% in Babol during 2002[23], 9.6% in Shiraz during 2004[24], 5.4% in Tehran during 2004-2005[21], 8% in Tehran during 2007[25], 12.4% in Semnan during 2007[3], 10% in Hamedan during 2013-2014[26], and 6.4% in Zanjan during 2013-2014[27].

The prevalence of *Campylobacter* species in Jeddah, Saudi Arabia, was identified as 4.5%[28]. Another researcher from Saudi Arabia during 2012 reported that the prevalence of *Campylobacter* spp. in stool samples of patients in Makkah and Jeddah hospitals was just 1.7%[29]. The prevalence of *Campylobacter* species among 2130 stool samples collected from Menia, Fayoum, Cairo, and Qaluobya of Egypt determined the fecal contamination rate of 35% in children[30].

The isolation rate of *Campylobacter* species from the stool samples of children in Baghdad, Iraq, was reported as 10% during 1991[31]. Another report from Basrah, Iraq, showed that *C. jejuni* was detected in 13.86% of all diarrhea cases and 31.03% of all bloody diarrhea cases[17]. As mentioned, *Campylobacter* was the second most frequent bacterial isolate in children with diarrhea, and the second most common cause of bloody diarrhea in Basrah, Iraq[17]. The *C. jejuni* was found to be the second most common isolate with a rate of 8.3% in Turkey during 1997[32]. Other Turkish researchers reported the prevalence of campylobacteriosis in their studies as 1.43% in Kayseri during 2002-2003[33] and 4.2% in Istanbul during 2013-2015[34].

2.1. Seasonal distribution

The incidence of campylobacteriosis is higher in warm seasons and hot weather. The prevalence of this organism in the south of Iran (9.6% in Shiraz) in warm weather was higher than in the north of this country with cold weather (6.4% in Zanjan and 6% in Ardabil)[22,24,27]. The obtained result of a survey on diarrheal cases in Cairo, Egypt, was indicative of a higher rate of prevalence in young children during the rainy seasons[35]. Regardless of these reports, a study conducted in Turkey did not support the relationship between the distribution of *Campylobacter* spp. and weather changes[36].

3. Detection of *Campylobacter* species

There have been successfully developed laboratory methods for examining the diversity of *Campylobacter* enteritis, such as Polymerase Chain Reaction (PCR), Genotyping methods (ribotyping and pulsed-field gel electrophoresis), ELISA for detecting DNA, antigens in stool samples, and also specific culturing of the organism from fecal specimens[37-42].

Rapid detection of *Campylobacter* species is extremely crucial in ensuring food and water safety. The ability of PCR to amplify minute amounts of specific microbial DNA sequences has made it a powerful molecular tool[43,44]. It has been reported that multiplex PCR diagnostic tools are fast, inexpensive, and sensitive for *Campylobacter* species[45]. Multiplex PCR is one of the possible and trustworthy molecular methods for indicating the prevalence of bacterial diseases, especially *Campylobacter* infection in a single sample[44,45]. The PCR assay offers an effective alternative to traditional biochemical typing methods for the identification and differentiation of *C. jejuni, C. coli, C. lari, C. upsaliensis,* and *C. fetus* subspecies fetus[46]. There are other possible methods for the detection of *Campylobacter* infection, such as specific culture under specific

conditions[20,47].

Currently, fecal lactoferrin has become one of the important factors for investigating the fecal samples suspected of bacterial infections[27]. Acute bacterial infections of the gastrointestinal tract or high severity of bowel diseases can induce increased levels of lactoferrin[48]. It has been reported that the fecal lactoferrin values were higher in patients with *Campylobacter* (10.32 μg/g) and *Salmonella* (11.17 μg/g)[49].

4. Distribution of *Campylobacter* species

Most recent reports have determined *C. jejuni* and *C. coli* are the main causes of campylobacteriosis in intestinal diseases[9]. In addition, *C. fetus* is known as an opportunistic pathogen in humans[50]. A study conducted on food and stool samples collected from different areas of Tehran, Iran, indicated that 44% of all isolates were *C. jejuni*, 22% *C. coli*, and 33% as other *Campylobacter* strains[51]. In another study in Iraq, the prevalence rate of *C. jejuni* was estimated at 75%, compared to 25% *C. Coli*[39]. During 2006 in Ankara, Turkey, the distribution of *Campylobacter* strains was reported as 70.1% *C. jejuni*, 21.1% *C. coli*, and 8.6% *C. lari*[52]. In a study conducted in Istanbul, Turkey, in 2009, the distribution of *Campylobacter* strains was indicated as 56.5% *C. jejuni*, 33.9% *C. coli*, and 9.6% *C. lari*[53]. The highest isolation of *Campylobacter* spp. was found to be 81% *C. jejuni*, and 18% *C. coli* in the Abu Homos district of Beheira Governorate in Egypt[18].

The *C. jejuni* account for most human *Campylobacter* isolates. There are two antigenic typing schemes for *C. jejuni*; the first one is Lior scheme with 108 serotypes[54] and the second one is Penner scheme with >60 serotypes[55]. The antigen in Lior typing scheme is heat-labile that is not truly known. The antigens in the Penner typing scheme are capsule contained lipopolysaccharide (LPS) and lipooligosaccharide (LOS)[56]. However, protective antigens are not properly determined and defined.

4.1. Polymicrobial infections involving *Campylobacter*

It has been reported that *C. jejuni* antibodies have cross-reactions with *Shigella sonnei, Neisseria meningitides, Shigella flexneri*, and *Escherichia coli* plus *C. rectus* and *C. coli*[57]. The flagellar proteins of *C. jejuni* could show signs of cross-reaction with antibodies produced against *C. coli*, *Helicobacter pylori*, and *C. rectus*[58]. However, the occurrence of campylobacteriosis usually has a simultaneous infection with multiple pathogens, such as *E. coli, Listeria monocytogenes, Staphylococcus aureus, Cronobacter sakazakii, Salmonella enterica, Vibrio cholera, Shigella*, and *Yersinia* species[61-65].

5. Transmission of disease and clinical symptoms

The occurrence of human campylobacteriosis is sporadic

making it difficult to trace the reasons and routes of transmission[56]. The clinical symptoms of enteritis caused by *Campylobacter* species consist of watery, non-bloody, and non-inflammatory diarrhea which may give way to severe inflammatory diarrhea with abdominal pain and fever. However, *C. jejuni* typically leads to acute and self-limited signs[20,57,60]. The diagnosis of *Campylobacter* infection is impossible with routine clinical signs and it usually has a co-infection with multiple pathogens, such as *E. coli, Listeria monocytogenes, Staphylococcus aureus, Cronobacter sakazakii, Salmonella enterica, Vibrio cholera, Shigella,* and *Yersinia* species[61-65].

From another point of view, individuals living in developed countries with exposure to *Campylobacter* species usually suffer from severe and bloody diarrhea[66]. Another common digestive tract symptom is abdominal pain, however, vomiting is not the usual sign. Fever, headache, asthenia, and anorexia can also be observed[67-70]. The risk of developing inflammatory bowel diseases has already increased since diseases, such as ulcerative colitis and Crohn's disease, are followed by *Campylobacter* diarrhea[71].

Campylobacter infections may lead to the further development of autoimmune diseases, including reactive arthritis[72] and neurological illness (Guillain-Barre syndrome and its variant, Miller Fisher syndrome). Guillain-Barre´ Syndrome is an acute, symmetric, and ascending paralysis that is estimated to occur 30 times for every 100,000 *Campylobacter* cases and the fatality ratio is almost 10%[73,74].

5.1. Age and gender of patients

A current study on 1010 stool samples of adult patients of different ages and both genders concluded that age and gender did not have a significant effect on the prevalence of campylobacteriosis[22]. However, the occurrence of this disease was usually higher in elderly individuals above 75 years old, young children under 4 years of age, and young adults who are 20-40 years old[2,10,11].

Children experience a progressive increase in all isotypes of *Campylobacter*-specific serum antibodies in the first two years of life, followed by a continued increase in IgA titers, indicative of frequent exposure to the organism, and improved mucosal immunity[74]. The highest isolation of *Campylobacter* was reported in children of 6-12 months with diarrhea in the Abu Homos district of Beheira Governorate in Egypt[18].

During 2010-2011, Girgis et al. reported that gender is not an effective factor in the prevalence of this infection in Egypt but younger patients had more related infections to *Campylobacter* species[76]. In a study in the south of Iran, it was determined that the prevalence of *C. jejuni* in females was significantly higher than males, and also the highest contamination rate was observed in younger patients aged 11-15 years old[24]. The highest isolation rate of *Campylobacter* spp. in Turkey during 1997 was indicated in the range of 6-14 years[32]. In a study in Basrah, Iraq, the frequency of *C. jejuni* infection was

higher in children younger than 4 years of age; however, the isolation rate in males was more than the females[17]. A study in Saudi Arabia indicated that *Campylobacter* was known as an important cause of bacterial enteritis both in adults and children, however, the prevalence of this organism in males was higher than in females[28].

6. Animal sources of *Campylobacter*

Environmental contamination of *Campylobacter* species can be caused by domestic animals and poultry (as natural reservoirs of *Campylobacter* species)[78]. In another study, the prevalence of *Campylobacter* species was investigated in different animal species during 2002-2012. The findings indicated that chickens were the most common reservoir/source of *Campylobacter* infection with an average contamination of 70.9%, whereas cattle were determined as the second most common source with a rate of 19.3%, dogs and pigs were other sources with the rates of 8.6%, and 1.2%, respectively[79]. Furthermore, some reports have found this pathogen as normal flora of other animals, such as sheep carcasses at abattoir[80,81].

The isolation of thermophilic *Campylobacter* spp. was presented as 11.1%, 21.6%, and 50.4% for beef, mutton, and chicken samples, respectively, during 2009 in Istanbul, Turkey. In another study in Kayseri, Turkey, the isolation of *C. jejuni* among 6667 samples collected from humans, dogs, cattle, and chickens was reported as 1.43%, 43.50%, 31.16%, and 56%, respectively[33]. The presence of *Campylobacter* species in stool samples and eggshells of poultry as a normal flora has also been investigated[82,83].

6.1. Poultry as a source of *Campylobacter* spp.

Poultry and specifically broiler chickens have a metabolic temperature of 42ºC leading to an optimal environment for *Campylobacter* growth[84]. Poultry carcasses in stores that are not under sanitary monitoring and the consumption of undercooked poultry meat are important reasons for such outbreaks[85]. However, the transmission of infection from chickens to humans is greater owing to the high levels of chicken meat consumption[86,87]. In developing countries, close contact with animals, including chickens, was found to be an important risk factor for acquiring the infection[88,89]. A diarrheal disease survey in Cairo, Egypt, in young children indicated that the prevalence of *Campylobacter* spp. is highly associated with keeping fowls at home[35].

The presence of *Campylobacter* species as normal flora in stool samples and eggs of poultry is one of the major causes that might affect the prevalence of campylobacteriosis in patients[90,83]. Reducing the number of *Campylobacter* in poultry carcass can significantly decrease the risk of infection in consumers[39,91]. One of the suggested approaches for controlling the *Campylobacter*species as normal flora in poultry is using feed additives, such as probiotics and synbiotics. It is reported that a mixture of fructooligosaccharide and a galactooligosaccharide +

Bifidobacterium strain (*B. longum* subsp. *longum* PCB133) can significantly reduce the *C. jejuni* concentration in poultry feces[92]. The other common way is the use of antibiotics as growth promoters in the poultry diet; however, this pathogen is highly resistant to many antibiotics. Previously from 35 layer farms of Northern Jordan, the high resistance of this organism to ciprofloxacin (100%), tetracycline (100%), gentamicin, and amoxicillin (41%) was reported while the low resistance to nalidixic acid (21%), erythromycin (14%), and florfenicol was observed[93].

A comparative study in Esfahan, Iran, reported *Campylobacter* persistence in different poultry meat samples. The highest rate observed in quail meat (68.4%) and 56.1%, 27.4%, and 11.7% of contamination was respectively recorded for chicken, turkey, and ostrich meats. The overall prevalence of *Campylobacter* spp. in the investigated samples was 47.1% (377 from 800) which contained 76.4% *C. jejuni* and 23.6% as *C. coli*[94].

The prevalence of *Campylobacter* species among samples collected from different cities of Egypt determined that the contamination rate was 40.4% for the intestine of chicken, 37.5% for the liver of chicken[30]. A study performed in Saudi Arabia during 2012, the 81.7% prevalence of campylobacteriosis in carcass samples of chicken was presented[29]. This report was close to the report of Turkish researchers who had indicated the presence of *Campylobacter* species in 83% of chicken meat samples[50].

6.2. Other foods and water sources, and infectious dose

Campylobacteriosis can result from the consumption of suspected food sources and daily products of animals[8,95]. Recent studies indicated that the consumption of unpasteurized milk and ingestion of undercooked chicken was significantly associated with acquiring infection but there was no possible effect of poultry on the incidence of *Campylobacter* disease[85,76]. Gent et al. reported the outbreak of *Campylobacter* infection due to poor food handling in a fast food outlet located in a university that infected over 100 students[96]. Moreover, the obtained results of this outbreak led to the identification of 6 different *Campylobacter* subtypes isolated from 11 patients.

In a study focused on food samples collected from different areas of Tehran, Iran, the prevalence rates of *C. jejuni*, *C. coli*, and other *Campylobacter* strains were reported[25]. In the cities of Menia, Fayoum, Cairo, and Qaluobya, Egypt, the contamination rates of *Campylobacter* species in water and foods were estimated as 30%, 4.44%, 6.6%, and 13.33% in tap water, raw milk, Karish cheese, and yogurt, respectively[30]. During 2014-2015, samples of raw milk, Kareish cheese, and yogurt were collected from local markets in Qena City, Egypt, to evaluate the prevalence of *Campylobacter* species. The results suggested that 24.6% of samples were contaminated with *Campylobacter* spp. and the highest contamination rate belonged to raw milk while the lowest was observed in

yogurt samples[97]. Wagenaar et al. found that 23% of infected human cases with campylobacteriosis were associated with the consumption of unpasteurized milk products in Egypt[98].

This organism is occasionally isolated from streams, lakes, and ponds[99]. The survival of *Campylobacter* in surface water of the Mediterranean area has been reported[100]. A diarrheal disease survey in Cairo, Egypt, in young children indicated that the prevalence of *Campylobacter* spp. was highly associated with having an outdoor source of drinking water[35].

The current report has indicated the overall prevalence of *Campylobacter* in beef livers in Egypt was 26.66% in 2013[101] while this isolation rate was higher than the previous study conducted in Japan which reported only 5% *Campylobacter* spp. in beef livers[102]. Ghafir et al. suggested that the high level of hepatic *Campylobacter* resistance is probably due to the moisture of the liver surface, which might protect this foodborne pathogen[103]. Fecal carriage of *Campylobacter* by the slaughtered cows is another possible route of contaminating beef livers in an abattoir that may cause campylobacteriosis using undercooked beef livers[104].

Reducing the number of *Campylobacter* in poultry carcass can greatly reduce the risk of foodborne *disease*[37,91]. Few available studies reveal the exact number of cells inducing human infections. In a kindergarten, contamination of foods at the level of 10 cells of *C. jejuni* per 100 ml caused infections[105].

7. The role of immunity in *Campylobacter* infection

The presence of a specific serum immunoglobulin A (IgA), IgM, and IgG in acute campylobacteriosis has been reported[106,107]. IgA remained elevated for almost 1 month whereas IgM and IgG persisted for almost 3 months[72]. Healthy individuals with occupational exposure to *C. jejuni* produced IgM without clinical symptoms[108].

The results of a study on antibody titers ($\log_{10}$) of diarrhea patients in Cairo, Egypt, indicated that 28 patients with diarrhea had *C. jejuni*-reactive antibodies titers and 14 cases were culture-positive for this organism with significant titers of IgA and IgG. *Among* asymptomatic *culture-positive* patients (10 cases), 6 subjects had reactive IgM, and 7 subjects had IgG. However, IgA was not detected in asymptomatic patients[72]. Another study indicated that the chronic carriers of *C. jejuni* could increase the levels of IgG and IgM but not IgA[108]. The presence of antibodies in healthy populations living in areas of hyperendemicity or developing countries has also been reported[109].

8. Prevention and control

Various animal models have been tested with different vaccine formulations and each has shown varying levels of success. Most of the investigated vaccines are against *C. jejuni*[110]. The limited literature concerning virulence determinants of *C. jejuni* makes it difficult to prepare effective live attenuated vaccines[110]. Oral immunization of mice with an attenuated *Salmonella enterica* serovar Typhimurium vectoring *Campylobacter* PEB1 antigen or CjaA protein failed to protect the animals against intestinal colonization with the challenge *Campylobacter* strain even though specific serological responses were seen[111,112].

It is proposed that vaccination may be a possible control approach, as infected people mount a strong immune response. In addition, possible immunity against *Campylobacter* in the absence of acute infection in workers of slaughterhouses was reported after initial exposure[113]. There have also been attempts to develop *Campylobacter* vaccines, whole-cell oral vaccine formulations having been tested with good results in primates[114] and a vaccine based on whole-cell formulations and purified flagellin giving some protective immunity in the mouse model[115]. A recombinant truncated flagellin protein (rFla-MBP) based on the conserved region of flagellin of *C. coli* VC167 strain used as a vaccine-induced antibody response and 60% protection against *C. jejuni* in a ferret model of diarrhea[116].

An alternative way to decrease food contamination is reducing the load of the pathogen in its main reservoir, the broiler chickens. The control programs, including increasing biosecurity and vaccination of broiler chickens, are being explored.

9. Conclusion

The prevalence of *C. jejuni* is much *more* common *than* other *Campylobacter* spp. in the Middle East leading to the incidence of *Campylobacter* infection rated between 1 and 14%. The people who live with animals should check the health of animals regularly and not consume suspected sources of food. Individuals are recommended to buy meat, particularly chicken meat, from stores that are under sanitary monitoring. Ultimately, further investigations can show all the possible pathogens responsible for polymicrobial acute diarrheal infections in addition to *Campylobacter* species and also elucidate their mechanisms of pathogenesis.

Declarations
Competing interests

Authors declare no conflicts of interest.

Authors' contribution

Authors participated equally in preparing and writing the manuscript.

Availability of data and materials

All findings and related data of the present study are prepared for publishing in the submitted journal.

References

1. Ranjbar R, Naghoni A, Afshar D, Nikkhahi F, and Mohammadi M. Rapid molecular approach for simultaneous detection of *Salmonella* spp.,

Shigella spp., and *Vibrio* cholera. Osong Public Health Res Perspect. 2016; 7: 373-377. DOI: https://www.doi.org/10.1016/j.phrp.2016.10.002

2. Ranjbar R, Rahbar M, and Naghoni A. A cholera outbreak associated with drinking contaminated well water. Arch Iran Med. 2011; 14: 339-340. Available at: https://pubmed.ncbi.nlm.nih.gov/21888459/

3. Jazayeri MA, Irajian GR, Kalantari F, Monem M, Salehian A, Rahbar H, et al. Prevalence of *Campylobacter jejuni* in diarrheic children in Semnan (Iran). Koomesh. 2008; 9: 297-300. Available at: http://eprints.semums.ac.ir/736/

4. Tajbakhsh E, Khamesipour F, Ranjbar R, and Ugwu IC. Prevalence of class 1 and 2 integrons in multi-drug resistant *Escherichia coli* isolated from aquaculture water in Chaharmahal Va Bakhtiari province, Iran. Ann Clin Microbiol Antimicrob. 2015; 14: 37. DOI: https://www.doi.org/10.1186/s12941-015-0096-y

5. El-Mohamady H, Abdel-Messih IA, Youssef FG, Said M, Farag H, Shaheen HI, et al. Enteric pathogens associated with diarrhea in children in Fayoum, Egypt. Diagn Microbiol Infect Dis. 2006; 56: 1-5. DOI: https://www.doi.org/10.1016/j.diagmicrobio.2006.02.007

6. Elamreen FHA, Abed AA, and Sharif FA. Detection and identification of bacterial enteropathogens by polymerase chain reaction and conventional techniques in childhood acute gastroenteritis in Gaza, Palestine. Int J Infect Dis. 2007; 11: 501-507. DOI: https://www.doi.org/10.1016/j.ijid.2007.01.010

7. Friedman CR, Neimann J, Caspar WH, and Tauxe RV. Epidemiology of *Campylobacter jejuni* infections in the United States and other industrialized nations. *Campylobacter* Vol. II/6 2. ed. Washington, USA : ASM International. 2000; p. 121-138. Available at: https://orbit.dtu.dk/en/publications/epidemiology-of-campylobacter-jejuni-infections-in-the-united-sta

8. Hamidian M, Sanaei M, Bolfion M, Dabiri H, Zali MR, and Rasmussen JW. Prevalence of putative virulence markers in *Campylobacter jejuni* and *Campylobacter coli* isolated from hospitalized children, raw chicken, and raw beef in Tehran, Iran. Can J Microbiol. 2011; 57: 143-148. DOI: https://www.doi.org/10.1139/w10-089

9. Skirrow M. *Campylobacter.* Lancet. 1990; 336: 921-923. DOI: https://www.doi.org/10.1016/0140-6736(90)92282-m

10. Lévesque S, Fournier E, Carrier N, Frost E, Arbeit RD, and Michaud S. Campylobacteriosis in urban versus rural areas: A case-case study integrated with molecular typing to validate risk factors and to attribute sources of infection. PloS One. 2013; 8: e83731. DOI: https://www.doi.org/10.1371/journal.pone.0083731

11. Strachan NJC, Rotariu O, MacRae M, Sheppard SK, Smith-Palmer A, Cowden J, et al. Operationalising factors that explain the emergence of infectious diseases: A case study of the human campylobacteriosis epidemic. PloS One. 2013; 8: e79331. DOI: https://www.doi.org/10.1371%2Fjournal.pone.0079331

12. Fitzgerald C. *Campylobacter.* Clin Lab Med. 2015; 35: 289-98. DOI: https://www.doi.org/10.1016/j.cll.2015.03.001

13. Altekruse SF, Stern NJ, Fields PI, and Swerdlow DL. *Campylobacter jejuni* – an emerging foodborne pathogen. Emerg Infect Dis. 1999; 5: 28-35. DOI: https://www.doi.org/10.3201/eid0501.990104

14. Engleberg NC, Correa-Villaseŉor A, North CQ, Crow T, Wells JG, and Blake PA. *Campylobacter* enteritis on Hopi and Navajo Indian reservations. Clinical and epidemiologic features. West J Med. 1984; 141: 53-56. Available at: https://pubmed.ncbi.nlm.nih.gov/6475040/

15. Blaser MJ, Berkowitz ID, LaForce FM, Cravens J, and Reller LB, Wang WL. *Campylobacter* enteritis: Clinical and epidemiologic features. Ann Intern Med. 1979; 91: 179-85. DOI: https://www.doi.org/10.7326/0003-4819-91-2-179

16. Kaakoush NO, Castaŉo-Rodrﻰguez N, Mitchell HM, and Man SM. Global epidemiology of *Campylobacter* infection. Clin Microbiol Rev. 2015; 28: 687-720. DOI: https://www.doi.org/10.1128/CMR.00006-15

17. Mohammad HF, Hassan MK, and Bakir SS. *Campylobacter jejuni* gastroenteritis in children in Basrah-Iraq. MJBU. 2004; 22: 1-2. Available at: http://un.uobasrah.edu.iq/papers/3882.pdf

18. Rao MR, Naficy AB, Savarino SJ, Abu-Elyazeed R, Wierzba TF, Peruski LF, et al. Pathogenicity and convalescent excretion of *Campylobacter* in rural Egyptian children. Am J Epidemiol. 2001; 154: 166-73. DOI: https://www.doi.org/10.1093/aje/154.2.166

19. Oberhelman RA, and Taylor DN. *Campylobacter* infections in developing countries. In: Nachamkin I, Blaser MJ, editors. *Campylobacter*. 2nd ed. Washington: American Society for Microbiology. 2000; p. 139-53. Available at: https://ci.nii.ac.jp/naid/10020280328/

20. Riaz MM, Patel MJ, Khan MS, Anwar MA, Tariq M, Hilal H, et al. Clinical characteristics and predictors of positive stool culture in adult patients with acute gastroenteritis. J Pak Med Assoc. 2012; 62: 20-24. Available at: https://pubmed.ncbi.nlm.nih.gov/22352095/

21. Jafari F, Shokrzadeh L, Hamidian M, Salmanzadeh-Ahrabi S, and Zali MR. acute diarrhea due to enteropathogenic bacteria in patients at hospitals in Tehran. Jpn J Infect Dis. 2008; 61: 269-273. Available at: https://pubmed.ncbi.nlm.nih.gov/18653967/

22. Ranjbar R, and Babazadeh D. Contact with poultry and animals increases risk of Campylobacter infections in adults of Ardabil province, Iran. Universa Medicina. 2017; 36: 59-67. Available at: https://pdfs.semanticscholar.org/8d81/4a1a3dbb30ba27521d3fe42d0525b142c5ff.pdf

23. Norouzi J, Kouhi RS, and Kolaei AR. *Campylobacter jejuni* in children under 7 years old with acute enteritis. J Babol Univ Med Sci. 2002; 4: 30-32. Available at: http://jbums.org/browse.php?a_id=2841&sid=1&slc_lang=en

24. Hassanzadeh P, and Motamedifar M. Occurrence of *Campylobacter jejuni* in Shiraz, Southwest Iran. Med Princ Pract. 2007; 16: 59-62. DOI: https://www.doi.org/110.1159/000096142

25. Feizabadi MM, Dolatabadi S, and Zali MR. Isolation and drug resistant patterns of *Campylobacter* strains cultured from diarrheic children in Tehran. Jpn J Infect Dis. 2007; 60: 217-219. Available at: https://pubmed.ncbi.nlm.nih.gov/17642538/

26. Rastyani S, Alikhani MY, Sedighi I, Kazemi S, Kohan HF, and Arabestani MR. *Campylobacter jejuni* and *Campylobacter coli* in children with acute diarrhea in health centers of Hamadan, Iran. Avicenna J Clin Microbiol Infect. 2015; 2: e29791. DOI: https://www.doi.org/10.17795/ajcmi-29791

27. 47. Mobaien A, Moghaddam F, Talebi S, Karami A, Amirmoghaddami H, and Ramazani A. Studying the prevalence of *Campylobacter jejuni* in adults with gastroenteritis from northwest of Iran. Asian Pac J Trop Dis. 2016; 6: 957-960. Available at: https://www.sciencedirect.com/science/article/abs/pii/S2222180816611647

28. Zaman R. *Campylobacter* enteritis in Saudi Arabia. Epidemiol Infect 1992; 108: 51-58. DOI: https://www.doi.org/10.1017/s0950268800049499

29. Ashgar SS. Campylobacteriosis in Makkah City, Saudi Arabia. Egyptian J Med Microb. 2013; 22: 59-62. Available at: https://pesquisa.bvsalud.org/portal/resource/pt/emr-188979

30. Barakat AMA, Sobhy MM, El Fadaly HAA, Rabie NS, Khalifa NO, Ramadan ES, et al. Zoonotic Hazards of Campylobacteriosis in some areas in Egypt. Life Sci J. 2015; 12: 9-14. Available at: https://fvtm.stafpu.bu.edu.eg/Zoonotic%20Diseases/957/publications/Nashwa%20Osman%20Khalefa_Zoonotic%20Hazards%20of%20Campylobacteriosis%20in%20some%20areas%20in%20Egypt.pdf

31. Saliih DS, and Al-saad MR. Isolation and identification of thermophilic Campylobacters from diarrhoeal childrenin Baghdad. J Islamic Academy Sci. 1994; 7: 88-92. Available at: https://medicaljournal-ias.org/jvi.aspx?pdir=ias&plng=eng&un=IAS-90377

32. Uysal G, Doğru U, Aysev D, and Karabiber N. *Campylobacter jejuni* gastroenteritis in Turkish children. Infection. 1997; 25(3): 159-62. Available at: https://link.springer.com/article/10.1007/BF02113604

33. Aydin F, Gumussoy KS, Ica T, Sumerkan B, Esel D, Akan M, and Ozdemir A. The prevalence of *Campylobacterjejuni* in various sources in Kayseri, Turkey, and Molecular analysis of isolated strains by PCR-RFLP. Turk J Vet Anim Sci. 2007; 31: 13-19. Available at: https://journals.tubitak.gov.tr/veterinary/issues/vet-07-31-1/vet-31-1-3-0511-7.pdf

34. Gurol Y, Kipritci Z, and Celik G. An important agent in gastroenteritis: *Campylobacter.* J Immunol Clin Microbiol. 2016; 1: 73-74. Available at: https://dergipark.org.tr/en/pub/jicm/issue/36646/421640

35. Al Amri A, Senok AC, Ismaeel AY, Al-Mahmeed AE, and Botta GA. Multiplex PCR for direct identification of *Campylobacter* spp. in human and chicken stools. J Med Microbiol. 2007; 56: 1350-1355. DOI: https://www.doi.org/10.1099/jmm.0.47220-0

36. Bostan K, Aydin A, and Ang MK. Prevalence and antibiotic susceptibility of thermophilic *Campylobacter* species on beef, mutton, and chicken carcasses in Istanbul, Turkey. Microb Drug Resist. 2009; 15: 143-149.

DOI: https://www.doi.org/10.1089/mdr.2009.0894

37. Ranjbar R, Karami A, Farshad S, Giammanco GM, and Mammina C. Typing methods used in the molecular epidemiology of microbial pathogens: A how-to guide. New Microbiol. 2014; 37(1): 1. Available at: https://pubmed.ncbi.nlm.nih.gov/24531166/

38. On SLW, and Jordan PJ. Evaluation of 11 PCR assays for species-level identification of *Campylobacter jejuni* and *Campylobacter coli*. J Clin Microbiol. 2003; 41: 330-336. DOI: https://www.doi.org/10.1128/JCM.41.1.330-336.2003

39. Guyard-Nicodème M, Rivoal K, Houard E, Rose V, Quesne S, Mourand G, et al. Prevalence and characterization of *Campylobacter Jejuni* from chicken meat sold in French retail outlets. Int J Food Microbiol. 2015; 203: 8-14. DOI: https://www.doi.org/10.1016/j.ijfoodmicro.2015.02.013

40. Harzandi N, Jamshidi S, Dezfulian M, Bahonar A, Bakhtiari A, and Banihashemi K. Molecular detection and speciation of *Campylobacter* species in children with gastroenteritis using polymerase chain reaction in Bahonar Hospital of Karaj City. Int J Enteric Pathog. 2015; 3: e21796. DOI: https://www.doi.org/10.17795/ijep21796

41. Rokosz N, Rastawicki W, and Wołkowicz T. Microbiological diagnosis of infections caused by *Campylobacter jejuni* and *Campylobacter coli* in humans. Postepy Hig Med Dosw. 2014; 68: 48-56. DOI: https://www.doi.org/10.5604/17322693.1086079

42. Wassenaar TM, and Newell DG. Genotyping of *Campylobacter* spp. Appl Environ Microbiol. 2000; 66: 1-9. DOI: https://www.doi.org/10.1128/AEM.66.1.1-9.2000.

43. Karami A, Ranjbar R, Ahmadi Z, and Safiri Z. Rapid detection of different serovares of *Salmonella* entrica by multiplex PCR. Iran J Pub Health. 2007; 36: 38-42. Available at: https://ijph.tums.ac.ir/index.php/IJPH/article/view/2116

44. Ranjbar R, Afshar D, and Mehrabi Tavana A. Development of multiplex PCR for simultaneous detection of three pathogenic Shigella species. Iran J Public Health. 2014; 43: 1657-1663. Available at: https://pubmed.ncbi.nlm.nih.gov/26171358/

45. El-Adawy H, Hotzel H, Tomaso H, Neubauer H, and Hafez HM. Elucidation of colonization time and prevalence of thermophilic *Campylobacter* species during turkey rearing using multiplex polymerase chain reaction. Poult Sci. 2012; 91: 454-459. DOI: https://www.doi.org/10.3382/ps.2010-01810

46. Wang G, Clark CG, Taylor TM, Pucknell C, Barton C, Price L, et al. Colony multiplex PCR assay for identification and differentiation of *Campylobacter jejuni*, *C. coli*, *C. lari*, *C. upsaliensis* and *C. fetus* subsp. fetus. J Clini Microbiol. 2002; 40: 4744-4747. DOI: https://www.doi.org/10.1128/JCM.40.12.4744-4747.2002

47. Choi SW, Park CH, Silva TM, Zaenker EI, and Guerrant RL. To culture or not to culture: Fecal lactoferrin screening for inflammatory bacterial diarrhea. J Clin Microbiol. 1996; 34: 928-932. DOI: https://www.doi.org/10.1128/jcm.34.4.928-932.1996

48. Sipponen T. Diagnostics and prognostics of inflammatory bowel disease with fecal neutrophil-derived biomarkers calprotectin and lactoferrin. Dig Dis. 2013; 31: 336-344. DOI: https://www.doi.org/10.1159/000354689

49. Chen CC, Chang CJ, Lin TY, Lai MY, Chao HC, and Kong MS. Usefulness of fecal lactoferrin in predicting and monitoring the clinical severity of infectious diarrhea. World J Gastroenterol. 2011; 17: 4218-4224. DOI: https://www.doi.org/10.3748/wjg.v17.i37.4218

50. Sauerwein RW, Bisseling J, and Horrevorts AM. Septic abortion associated with *Campylobacter fetus* subspecies fetus infection: case report and review of the literature. Infection. 1993; 21: 331-333. DOI: https://www.doi.org/10.1007/BF01712458

51. Bakhshi B, NaserI A, and lebouyeh M. Comparison of antimicrobial susceptibility of *Campylobacter* strains isolated from food samples and patients with diarrhea. Iran Biomed J. 2016; 20: 91-96. DOI: https://www.doi.org/10.7508/ibj.2016.02.004

52. Savaci M, and Özdemir H. Prevalence of thermophilic *Campylobacter* spp. in retail chicken meat in Ankara. J food Safety. 2006; 26: 244-250. DOI: https://www.doi.org/10.1111/j.1745-4565.2006.00046.x

53. Lior H, Woodward DL, Edgar JA, Laroche LJ, and Gill P. Serotyping of *Campylobacterjejuni* by slide agglutination based on heat-labile antigenic factors. J Clin Microbiol. 1982; 15: 761-768. DOI: https://www.doi.org/10.1128/jcm.15.5.761-768.1982

54. Penner JL, and Hennessy JN. Passive hemagglutination technique for serotyping *Campylobacterfetus* subsp. *jejuni* on the basis of soluble heat-stable antigens. J Clin Microbiol. 1980; 12: 732-737. DOI:

https://www.doi.org/10.1128/jcm.12.6.732-737.1980

55. Karlyshev AV, Linton D, Gregson NA, Lastovica AJ, and Wren BW. Genetic and biochemical evidence of a *Campylobacterjejuni* capsular polysaccharide that accounts for Penner serotype specificity. Mol Microbiol. 2000; 35: 529-541. DOI: https://www.doi.org/10.1046/j.1365-2958.2000.01717.x

56. Altekruse SF, Stern NJ, Fields PI, and Swerdlow DL. *Campylobacter jejuni*—an emerging foodborne pathogen. Emerg Infect Dis. 1999; 5: 28-35. DOI: https://www.doi.org/10.3201/eid0501.990104

57. Taylor DN. Campylobacter infections in developing countries. In: Nachamkin I, Blaser M, Tompkins LS, editors. Campylobacter jejuni: current status and future trends. Washington: American Society for Microbiology. 1992; p. 20-30. Available at: https://ci.nii.ac.jp/naid/10020280340/

58. Newell DG. Human serum antibody response to surface protein antigens of *C. pyloridis*. Serodiagn Immunother. 1987; 1: 209-217. DOI: https://www.doi.org/10.1016/0888-0786(87)90027-8

59. Taylor DN, Perlman DM, Echeverria PD, Lexomboon U, and Blaser MJ. *Campylobacter* immunity and quantitative excretion rates in Thai children. J Infect Dis. 1993; 168: 754-758. DOI: https://www.doi.org/10.1093/infdis/168.3.754

60. Bae JS, Yuki N, Kuwabara S, Kim JK, Vucic S, Lin CS, et al. Guillain-Barré syndrome in Asia. J Neurol Neurosurg Psychiatry. 2014; 85: 907-913. DOI: https://www.doi.org/10.1136/jnnp-2013-306212

61. Blaser MJ, Wells JG, Feldman RA, Pollard RA, and Allen JR. *Campylobacter* enteritis in the United States. A multicenter study. Ann Intern Med. 1983; 98: 360-365. DOI: https://www.doi.org/10.7326/0003-4819-98-3-360

62. Bai J, Kim YT, Ryu S, and Lee JH. Biocontrol and rapid detection of food-borne pathogens using bacteriophages and endolysins. Front Microbiol. 2016; 7: 474. DOI: https://www.doi.org/10.3389/fmicb.2016.00474

63. Ranjbar R, Hosseini MJ, and Kaffashian AR. An outbreak of shigellosis due to Shigella flexneri serotype 3a in a prison in Iran. Arch Iran Med. 2010; 13: 413-416. Available at: https://pubmed.ncbi.nlm.nih.gov/20804308/

64. Ranjbar R, Giammanco GM, and Farshad S. Serotypes, antibiotic resistance, and class 1 integrons in *Salmonella* isolates from pediatric cases of enteritis in Tehran, Iran. Foodborne Pathog Dis. 2011; 8: 47-53. DOI: https://www.doi.org/10.1089/fpd.2010.0736

65. Ranjbar R, Masoudimanesh M, Dehkordi FS, Jonaidi-Jafari N, and Rahimi E. Shiga (Vero)-toxin producing Escherichia coli isolated from the hospital foods; virulence factors, o-serogroups and antimicrobial resistance properties. Antimicrob Resist Infect Control. 2017; 6: 4. DOI: https://www.doi.org/10.1186/s13756-016-0163-y

66. Ketley JM. Pathogenesis of enteric infection by *Campylobacter*. Microbiol. 1997; 143: 5-21. DOI: https://www.doi.org/10.1099/00221287143-1-5

67. Tauxe RV, Hargrett-Bean N, Patton CM, and Wachsmut IK. *Campylobacter* isolates in the United States, 1982-1986. MMWR CDC Surveill Summ. 1988; 37: 1-13. DOI: https://www.jstor.org/stable/44784806

68. Skirrow MB. *Campylobacter* enteritis: A 'new' disease. BMJ. 1977; 2: 9-11. DOI: https://www.doi.org/10.1136/bmj.2.6078.9

69. Megraud F, Gavinet AM, and Camou-Junca C. Serogroups and biotypes of human strains of *Campylobacter jejuni* and *Campylobacter coli* isolated in France. Eur J Clin Microbiol. 1987; 6: 641-645. DOI: https://www.doi.org/10.1007/BF02013060

70. Blaser MJ, and Reller LB. *Campylobacter* enteritis. N Engl J Med. 1981; 305: 1444-1452. DOI: https://www.doi.org/10.1056/NEJM198112103052404

71. Gradel KO, Nielsen HL, Schønheyder HC, Ejlertsen T, Kristensen B, and Nielsen H. Increased short- and long-term risk of inflammatory bowel disease after salmonella or *Campylobacter* gastroenteritis. Gastroenterology. 2009; 137: 495-501. DOI: https://www.doi.org/10.1053/j.gastro.2009.04.001

72. Peterson MC. Rheumatic manifestations of *Campylobacter jejuni* and *C. fetus* infections in adults. Scand J Rheumatol. 1994; 23: 167-170. DOI: https://www.doi.org/10.3109/03009749409103055

73. Wierzba TF, Abdel-Messih IA, Gharib B, Baqar S, Hendaui A, Khalil I, et al. *Campylobacter* infection as a trigger for GuillainBarré syndrome in Egypt. PLoS One. 2008; 3: e3674. DOI: https://www.doi.org/10.1371/journal.pone.0003674

74. Nachamkin I, Allos BM, and Ho T. *Campylobacter* species and Guillain-Barré syndrome. Clin Microbiol Rev. 1998; 11: 555-567. DOI: https://www.doi.org/10.1128/CMR.11.3.555

75. Ismail TF, Wasfy MO, El-berry HM, Oyofo BA, Churilla AM, Peruskilf

JR, et al. Evaluation of antibodies reactive with *Campylobacter jejuni* in Egyptian diarrhea patients. Clin Diagn Lab Immunol. 1997; 4: 536-539. DOI: https://www.doi.org/10.1128/cdli.4.5.536-539.1997

76. Girgis SA, Rashad SS, Othman HB, Bassim HH, Kassem NN, and El-Sayed FM. Multiplex PCR for identification and differentiation of Campylobacter species and their antimicrobial susceptibility pattern in Egyptian patients. Int J Curr Microbiol App Sci. 2014; 3: 861-875. Available at: https://www.ijcmas.com/vol-3-4/Samia%20A.%20Girgis,%20%20et%20al.pdf

77. Platts-Mills JA, and Kosek M. Update on the burden of *Campylobacter* in developing countries. Curr Opin Infect Dis. 2014; 27: 444-450. DOI: https://www.doi.org/10.1097/QCO.0000000000000091

78. Kittl S, Heckel G, Korczak BM, and Kuhnert P. Source attribution of human *Campylobacter* isolates by MLST and fla-typing and association of genotypes with quinolone resistance. PloS One. 2013; 8: e81796. DOI: https://www.doi.org/10.1371/journal.pone.0081796

79. Shahrokhabadi R, Rahimi E, Mommtaz H, and Poursahebi R. Prevalence of *Campylobacter Jejuni* and *Coli* in sheep carcasses by using cultural and PCR methods. Zahedan J Res Med Sci. 2013; 15: 28-33. Available at: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5094166/

80. Stanley K, and Jones K. Cattle and sheep farms as reservoirs of Campylobacter. J Appl Microbiol. 2003; 94: 104-113. DOI: https://www.doi.org/10.1046/j.1365-2672.94.s1.12.x

81. Sasaki Y, Maruyama N, Zou B, Haruna M, Kusukawa M, Murakami M, et al. *Campylobacter* cross-contamination of chicken products at an abattoir. Zoonoses Public Health. 2013; 60: 134-140. DOI: https://www.doi.org/10.1111/j.1863-2378.2012.01509.x

82. Jonaidi-Jafari N, Khamesipour F, Ranjbar R, and Kheiri R. Prevalence and antimicrobial resistance of *Campylobacter* species isolated from the avian eggs. Food Control. 2016; 70: 35-40. DOI: https://www.doi.org/10.1016/j.foodcont.2016.05.018

83. Stella E. Statistical analysis of prevalence of *Campylobacter* in poultry farms related to weather conditions in Cyprus. Theses, Hungarian Veterinary Archive. 2016; Available at: http://www.huveta.hu/handle/10832/1424/browse?value=Econom ou+Stella&type=author

84. Ozbey G, and Tasdemir B. Seasonality and antibiotic resistance of *Campylobacter* in Turkish chicken meat. Vet Ital. 2014; 50: 277-283. DOI: https://www.doi.org/10.12834/VetIt.170.2543.1

85. Lindmark H, Boqvist S, Ljungstrom M, Agren P, Bjorkholm B, and Engstrand L. Risk factors for *Campylobacter*iosis: An epidemiological surveillance study of patients and retail poultry. J Clin Microbiol. 2009; 47: 2616-2619. DOI: https://www.doi.org/10.1128/JCM.00826-09

86. Stafford RJ, Schluter P, Kirk M, Wilson A, Unicomb L, Ashbolt R et al. A multi-centre prospective case-control study of *Campylobacter* infection in persons aged 5 years and older in Australia. Epidemiol Infect. 2007; 135: 978-988. DOI: https://www.doi.org/10.1017/S0950268806007576

87. Black RE, Lopez de Romaňa G, Brown KH, Bravo N, Bazalar OG, and Kanashiro HC. Incidence and etiology of infantile diarrhea and major routes of transmission in Huascar, Peru. Am J Epidemiol. 1989; 129: 785-799. DOI: https://www.doi.org/10.1093/oxfordjournals.aje.a115193

88. Oberhelman RA, Gilman RH, Sheen P, Cordova J, Taylor DN, Zimic M, et al. *Campylobacter* transmission in a Peruvian shantytown: A longitudinal study using strain typing of *Campylobacter* isolates from chickens and humans in household clusters. J Infect Dis. 2003; 187: 260-269. DOI: https://www.doi.org/10.1086/367676

89. Pazzaglia G, Bourgeois AL, Arab I, Mikhail I, Podgore JK, Mourad A, et al. Campylobacter-associated Diarrhoea in Egyptian infants: Epidemiology and clinical manifestations of disease and high frequency of concomitant infections. J Diarrhoeal Dis Res. 1993; 11: 6-13. Available at: https://pubmed.ncbi.nlm.nih.gov/8315255/

90. Westrell T, Ciampa N, Boelaert F, Helwigh B, Korsgaard H, Chriél M, et al. Zoonotic infections in Europe in 2007: A summary of the EFSAECDC annual report. Euro surveill. 2009; 14: 785-794. Avaiable at: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19100

91. Baffoni L, Gaggìa F, Di Gioia D, Santini C, Mogna L, and Biavati B. A Bifidobacterium-based synbiotic product to reduce the transmission of C. *jejuni* along the poultry food chain. Int J Food Microbiol. 2012; 157: 156-161. DOI: https://www.doi.org/10.1016/j.ijfoodmicro.2012.04.024

92. Al-Natour MQ, Alaboudi AR, Osaili TM, and Obaidat MM. Resistance of *Campylobacter jejuni* isolated from layer farms in Northern Jordan using microbroth dilution and disc diffusion techniques. J food Sci. 2016; 81: M1749-M1753. DOI: https://www.doi.org/10.1111/1750-3841.13363

93. Rahimi E, and Tajbakhsh E. Prevalence of *Campylobacter* species in poultry meat in the Esfahan city, Iran. Bulg J Vet Med. 2008; 11: 257-262. Available at: http://www.uni-sz.bg/bjvm/vol11-no4-05.pdf

94. Kittl S, Heckel G, Korczak BM, and Kuhnert P. Source attribution of human Campylobacter isolates by MLST and fla-typing and association of genotypes with quinolone resistance. PloS One. 2013; 8: e81796. DOI: https://www.doi.org/10.1371/journal.pone.0081796

95. Gent RN, Telford DR, and Syed Q. An outbreak of *Campylobacter* food poisoning at a university campus. Commun Dis Publ Health. 1999; 2: 39-42. Available at: https://pubmed.ncbi.nlm.nih.gov/10462894/

96. El-Zamkan MA, and Abdel Hameed KG. Prevalence of *Campylobacter jejuni* and *Campylobacter coli* in raw milk and some dairy products. Vet World. 2016; 9: 1147-1151. DOI: https://www.doi.org/10.14202/vetworld.2016.1147-1151

97. Wagenaar JA, French NP, and Havelaar AH. Preventing *Campylobacter* at the source: Why is it so difficult?. Clin Infect Dis. 2013; 57: 16006. DOI: https://www.doi.org/10.1093/cid/cit555

98. Marler B. The spread of *Campylobacter* can be prevented through proper sanitation and cooking procedures. Food poisoning resources. About *Campylobacter* - Copyright. 2005; 2015.

99. Rodيguez S, and Araujo R. Effect of environmental parameters on the inactivation of the water borne pathogen Campylobacter in a Mediterranean river. J. Water Health. 2012; 10: 100-107. DOI: https://www.doi.org/10.2166/wh.2011.044

100. Khalifa NO, Afify JSA, and Rabie NS. Zoonotic and molecular characterizations of *Campylobacter jejuni* and *Campylobacter coli* isolated from beef cattle and children. Global Veterinaria. 2013; 11: 585-591. DOI: https://www.doi.org/10.5829/idosi.gv.2013.11.5.818

101. Enokimoto M, Kubo M, Bozono Y, Mieno Y, and Misawa N. Enumeration and identification of Campylobacter species in the liver and bile of slaughtered cattle. Int J Food Microbiol. 2007; 118: 259-263. DOI: https://www.doi.org/10.1016/j.ijfoodmicro.2007.07.057

102. Ghafir Y, China B, Dierick K, De Zutter L, and Daube G. A seven-year survey of Campylobacter contamination in meat at different production stages in Belgium. IntJ Food Microbiol. 2007; 116: 111-120. DOI: https://www.doi.org/10.1016/j.ijfoodmicro.2006.12.012

103. Noormohamed A, and Fakhr MK. A Higher prevalence rate of *Campylobacter* in retail beef livers compared to other beef and pork meat cuts. Int J Environ Res Public Health. 2013; 10: 2058-2068. DOI: https://www.doi.org/10.3390/ijerph10052058

104. Riordan T, Humphrey TJ, and Fowles A. A point source outbreak of Campylobacter infection related to bird-pecked milk. Epidemiol Infect. 1993; 110: 261-265. DOI: https://www.doi.org/10.1017/s0950268800068187

105. Blaser MJ, and Duncan DJ. Human serum antibody response to *Campylobacter jejuni* infection as measured in an enzyme-linked immunosorbent assay. Infect Immun. 1984; 44: 292-298. DOI: https://www.doi.org/10.1128/iai.44.2.292-298.1984

106. van Spreeuwel JP, Duursma GC, Meijer CJ, Bax R, Rosekrans PC, and Lindeman J. *Campylobacter* colitis: Histological, immunohis-tochemical and ultrastructural findings. Gut. 1985; 26: 945-951. DOI: https://www.doi.org/10.1136/gut.26.9.945

107. Svedhem AH, Gunnarsson H, and Kaijser B. Diffusion-in-gel en-zyme linked immunosorbent assay for routine detection of IgG and IgM antibodies to *Campylobacter jejuni.* J Infect Dis. 1983; 148: 82-92. DOI: https://www.doi.org/10.1093/infdis/148.1.82

108. Blaser MJ, Berkowitz ID, LaForce FM, Cravens J, Reller LB, and Wang WL. *Campylobacter* enteritis: Clinical and epidemiological features. Ann Intern Med. 1979; 91: 179-185. DOI: https://www.doi.org/10.7326/0003-4819-91-2-179

109. Albert MJ. Vaccines against *Campylobacter jejuni.* Austin J Clin Immunol. 2014; 1: 1013. Available at: https://austinpublishinggroup.com/clinical-immunology/fulltext/ajci-v1-id1013.php

110. Sizemore DR, Warner B, Lawrence J, Jones A, and Killeen KP. Live, attenuated *Salmonella Typhimurium* vectoring *Campylobacter* antigens. Vaccine. 2006; 24: 3793-3803. DOI: https://www.doi.org/10.1016/j.vaccine.2005.07.026

111. Laniewski P, Kuczkowski M, Chrzastek K, Wozniak A, Wyszynska A, Wieliczko A, et al. Evaluation of the immunogenicity of *Campylobacter jejuni* CjaA protein delivered by *Salmonella* enterica sv. Typhimurium strain with regulated delayed attenuation in chickens. World J Microbiol Biotechnol. 2014; 30: 281-292. DOI:

https://www.doi.org/10.1007/s11274-013-1447-5

112. Cawthraw SA, Lind L, Kaijser B, and Newell DG. Antibodies, directed towards Campylobacter jejuni antigens, sera from poultry abattoir workers. Clin Exp Immunol. 2000; 122: 55-60. DOI: https://www.doi.org/10.1046/j.1365-2249.2000.01349.x

113. Baqar S, Applebee LA, and Bourgeois AL. Immunogenicity and protective efficacy of a prototype Campylobacter killed whole-cell vaccine in mice. Infect Immun 1995; 63: 3731-3735. DOI: https://www.doi.org/10.1128/iai.63.9.3731-3735.1995

114. Lee LH, Burg E 3rd , Baqar S, Bourgeois AL, Burr DH, and Ewing CP. Evaluation of a truncated recombinant flagellin subunit vaccine against *Campylobacter jejuni*. Infect Immun. 1999; 67: 5799-5805. DOI: https://www.doi.org/10.1128/IAI.67.11.5799-5805.1999

115. Tribble DR, Baqar S, and Thompson SA. Development of a human vaccine. In: *Campylobacter* 3rd edition, Nachamkin I, Szymanski C, Blaser MJ, ed,. Washington, DC: American Society for Microbiology. 2008; p. 429-444. DOI: https://www.doi.org/10.1128/9781555815554.ch24

116. Wagenaar JA, Mevius DJ, and Havelaar AH. Campylobacter in primary animal production and control strategies to reduce the burden of human Campylobacteriosis. Rev Sci Tech. 2006; 25: 581-594. Available at: https://pubmed.ncbi.nlm.nih.gov/17094699/