Journal of Tropical Diseases

Hagos and Berhe, J Trop Dis 2019, 7:4

Review Article

Open Access

Campylobacteriosis: Emphasis on its Status as Foodborne Zoonosis in Ethiopia

Yohans Hagos[1], **Mebrahtu Berhe**[2*] **and Getachew Gugsa**[1]

[1]*College of Veterinary Medicine, Mekelle University, Mekelle, Ethiopia*

[2]*Veterinary Drug and Feed Administration and Control Authority, Mekelle, Ethiopia*

*__Corresponding authors:__ Mebrahtu Berhe, Ethiopian Veterinary Drug and Feed administration and Control Authority, Ethiopia, Tel: +251 34 241 6228; E-mail: meby99@gmail.com

Received date: March 02, 2019; **Accepted date:** May 25, 2019; **Published date:** June 04, 2019

Abstract

Campylobacter is one of the major causes of gastroenteritis in humans. The bacteria is a common component of the intestinal microbiota of numerous wild bird and mammals' species, and cause disease in human typically due to ingestion of contaminated food like that of chicken, drinking untreated water or unpasteurized milk and contact with farm animals. Most cases are sporadic with a seasonal peak during the summer. Usually, the disease manifested by fever, abdominal pain, and diarrhea, which usually diagnosed based on the patient's history and the symptoms. The case campylobacteriosis was rarely investigated and under-reported in Ethiopia. Therefore, the aims of this paper are to review the nature of *Campylobacter spp.* and overview its status as a foodborne zoonosis in Ethiopia. Based on the published report from different parts of Ethiopia, thermophilic *Campylobacter spp.* were isolated from raw meats and feces samples of different domestic animals and human. The highest prevalence reported from chicken meats, and *C. jejuni* and *C. coli* were the most prevalence *Campylobacter spp.* isolated from both the foods of animal origin and human beings. The disease has significantly reported from different parts of the country, though researches do not seem to cover wider geographic areas. Campylobacteriosis control and prevention strategies should focus on prevention of transmission to human beings by implementing strict hygienic control measures along the food chain to improve the hygienic conditions during handling, slaughtering, storage and commercialization of foods.

Keywords: *Campylobacter jejuni; Campylobacter coli*; Ethiopia; Foodborne-zoonosis

Background

Campylobacter, considered as the major cause of gastroenteritis in humans worldwide [1]. The bacterias are normal flora in animals such as poultry, pigs, cattle and wild birds. Consumption of contaminated food such as poultry regarded the major source of infection, followed by drinking of contaminated water or unpasteurized milk and contact with farm animals [2]. WHO [1] estimated that *Campylobacter* caused 37,600 deaths per year worldwide. In 2014, 237,642 campylobacteriosis cases were registered in the European Union [3].

Campylobacter spp. are a leading cause of bacterial-derived food-borne disease worldwide with an estimated 400 million cases per year [4]The *Campylobacter* genus has expanded considerably, and currently comprises 25 species and 8 sub-species [5], mostly human campylobacteriosis are caused by *Campylobacter jejuni* and *Campylobacter coli* (*C. coli*). More recently, other *Campylobacter spp.* has also recognized as gastro-intestinal pathogen in both industrialized and developed countries. *C. jejuni* is the major frequently reported *Campylobacter spp.* (80% to 90%) followed by *C. coli* (5% to 10%) [6].

In many countries, human Campylobacteriosis characterizes by watery diarrhea, abdominal pain, vomiting, fever, and nausea [7]. An acute infection can cause serious long-term consequences, including the peripheral neuropathies, Guillain-Barre Syndrome (GBS) and Miller Fisher Syndrome (MFS), and functional bowel diseases, such as Irritable Bowel Syndrome (IBS) [8].

Campylobacter infections are generally mild but can be fatal among very young children, elderly and immune suppressed individuals (including cancer, HIV/AIDS and transplant patients, and often occur more frequently per year than *Salmonella spp., Shigella spp.* or *Escherichia coli* O157:H7 infections [9]. Although the disease causes mild and self-limiting, it is frequently severe in immune-compromised patients [10] or when the infection is caused by bacteria resistant to tetracyclines, fluoroquinolones or macrolides, the principal therapeutic options against these bacteria's' causing the infection [11].

The use of antimicrobial agents in food animals has resulted in the emergence and dissemination of antimicrobial resistant bacteria including antimicrobial resistant *Campylobacter,* which has a potentially serious impact on food safety in both animal and human health. The situation seems to deteriorate more rapidly in developing countries where there is widespread and uncontrolled use of antibiotics [12]. Therefore, this article was conducted with the aim of reviewing the diseases campylobacteriosis, mainly focusing on foodborne zoonosis and its status in Ethiopia.

Etiology

The word *Campylobacter* is derived from two Greece word ' campylos ' meaning curved and ' baktron ' meaning rod [7]. *Campylobacter spp.* are considered as common human pathogens. Two species, *C. jejuni,* and *C. coli* are now among the most commonly identified causes of enteritis in many countries. Although most of these *Campylobacter spp.* implicated in human enteritis, the major ones by far are the so-called 'thermophilic' or 'thermotolerant' grow at 42ºC to

43ºC, and at 37ºC. This group includes *C. jejuni, C. coli, C. lari*, and *C. upsaliensis* [13].

Taxonomy

According to the second edition of Bergey's Manual, *Campylobacter* belongs to the kingdom of Proteobacteria, which is a large and extremely complex group that currently contains over 1,300 species in 332 genera. Even though they are all related, the group is quite diverse in morphology, physiology, and lifestyle [14]. The Current taxonomic classification of *Campylobacter* is Domain: Bacteria; Phylum: Proteobacteria; Class: *Epsilonproteobacteria*; Order: Campylobacterales; Family: *Campylobacteriaceae*; Genus: *Campylobacter* and Species: *Campylobacter jejuni* etc. All the species of Campylobacter include *C. jejuni* (subsp. *Jejuni* and subsp. *doylei*), *C. coli, C. fetus* (subsp. *fetus* and subsp. *venerealis), C. lari, C. upsaliensis, C. hyointestinalis* (subsp. *hyointestinalis* and subsp. *lawsonii*), *C. sputorum* (biovar *sputorum,* biovar *bubulus*, and biovar *fecalis*), *C. helveticus, C. mucosal, C. concisus, C. curvus, C. rectus, C. hominis, C. shower, C. lawsonii* [15].

Morphology and Biochemical Properties

Campylobacter spp. are non-spore forming and Gram-negative bacteria. They can be spiral, curved or sometimes can be seen straight rods, with size ranging from 0.2 µm to 0.8 µm wide and 0.5 µm to 5 µm long. *Campylobacter* may appear as a spiral, S, V, or comma-shaped forms and can also be found in short or occasionally long chains. First *Campylobacter* cells begin to age, and then they become coccoid in shape [16]. The cells are highly motile by a kind of single or occasionally multiple flagella at one ends [17]. Rapid movement, darting motility of comma-shaped cells can be seen by a phase contrast microscope [18]. *Campylobacter* does not utilize carbohydrates and usually obtain energy from amino acids. Typical biochemical reactions create the reduction of fumarate to succinate, acetoin, indole production and negative methyl red. Most species of the *Campylobacter* reduce nitrate and positive in oxidase but only *C. jejuni* is hippurate positive. *C. jejuni* is quite sensitive to drying and storage at room temperature, but at refrigeration temperatures and appropriate humidity [17].

Survival and Growth Characteristics

Campylobacter spp. can grow best from 37°C to 42°C and in low oxygen or microaerophilic environment, such as an atmosphere of 10% CO_2; 5% O_2 and 85% N_2 [19]. *Campylobacter* is sensitive to freezing and drying [20]. The death rate of *Campylobacter* is dependent on temperature. They die more rapidly on a dry surface at room temperature than at refrigeration conditions [21]. They can survive at refrigeration temperatures (4°C) and in meat stored frozen (at -18°C to -22°C) for several weeks [20,22] and in general survive better at cooling temperatures [23]. *C. jejuni* and *C. coli* are sensitive to NaCl concentrations of 2% and above with, 0.5% NaCI thought to be optimum for their growth. *Campylobacter spp.* will not survive below 4.9 and above 9.0 PH and grows optimally at pH 6.5-7.5 [21]. *Campylobacter spp.* shown as a viable but nonculturable state when exposed to unfavorable environments, such as elevated temperature, low nutrient availability, freezing [24]. The nature of this coccoid form is uncertain. *Campylobacter jejuni* is adapting aerobic condition due to an ability to produce biofilm (Table 1) [25].

Characteristics	Minimum	Optimum	Maximum
Temperature (°C)	30	42-43	45
PH	4.9	6.5-7.5	9
NaCl (%)	-	0.5	1.5
Water activity (aw)	>0.987	0.997	-
Atmosphere	-	5%O_2+10% CO_2	-

Table 1: Growth characteristics of thermophilic *Campylobacter spp.* [26].

Host Distribution

Campylobacter spp. is found ubiquitous in the environment and many species act as reservoirs or susceptible and wild birds are known to be natural hosts. Among the vast variety of different bird species, members of the family of gulls, cranes, raptors, corvids, waterfowl, and passerines are known to harbor *C. jejuni.* Gulls, corvids, raptors, and passerines can be found foraging in the surrounding of poultry farms, whereas members of waterfowl and cranes as migratory birds help to disperse the bacteria and may contribute to spread and the introduction to poultry flocks [27,28].

Campylobacter, are detected in every possible poultry production farm. Within the group of *Campylobacter spp., C. jejuni, C. coli* and *C. lari* are found in chicken where *C. jejuni* is the most common in both layers and broilers [29]. Once *Campylobacter* introduced into a flock, the prevalence reaches up to 100% [30]. *C. coli,* found more often in turkey than in chicken, although predominantly appearing in pigs, it can occur in turkeys with a prevalence of almost 50% [31].

Other domesticated animals are known to harbor *Campylobacter spp.* as well such as livestock including sheep, cattle, goat and pig but also cats and dogs. As already mentioned, pigs are harboring predominantly *C. coli*, whereas sheep and cattle are regarded as important reservoirs for *C. jejuni* [32]. Due to its high prevalence and persistence in the environment and natural animal hosts, control or exclusion of *Campylobacter* from poultry and poultry flocks is very difficult [33].

Transmission

Campylobacter spp. carried in the intestinal tracts of many domestic animals such as cattle, sheep and poultry, as well as wild animals and birds [34]. In the case of domesticated animals; bovine, ovine, caprine, swine and especially in case of poultry, the infection can spread due to the slaughter process to raw and finished products. A human can acquire the infection by consumption of raw or decontaminated meat, or by the direct contact of raw products or cross-contamination of raw to cooked foods, swimming in natural waters, contact with contaminated animals or animal carcasses and traveling [35,36]. The incubation period can vary from one to 10 days but is usually about two to five days after exposure to the organism. The disease is communicable when infected persons excrete the bacteria in their feces. People who never took drugs have known to shed these bacteria for as long as seven weeks [37].

Zoonotic Potential and Sources Of Infection

Campylobacteriosis caused by thermo-tolerant *Campylobacter spp.*, and it is the most commonly reported zoonosis in humans in Europe. Besides, it also recognized as the major causative agents of bacterial gastroenteritis in the developed country. In 2013, there were 214,779 confirmed human Campylobacteriosis cases, an average of 64.8 confirmed cases of Campylobacteriosis per 100,000 of the EU population [38]. According to the EFSA estimations, the actual number of cases believed to be around 9 million each year in Europe [39]. In developed countries, *Campylobacter* as the cause of the enteric disease is even more frequent than other bacterial infection such as *Salmonella* or entero-pathogenic *E. coli.* There is an ongoing decline of *Salmonella spp.* infections within the EU, based on strict control programs [40]. The incidence of human *Campylobacter* cases is clearly higher during summer (from June to August) [41]. Among the reported cases of human Campylobacteriosis, *C. jejuniis* the major important and most frequently detected species. In 2010, it represented 93.4% of the confirmed cases in the EU [40].

Poultry and undercooked poultry meat, considered as one of the most reservoirs for *C. jejuni* and the most common source for human gastroenteritis worldwide [42]. In contrast to poultry, carcasses of pigs and cattle are less contaminated with *C. jejuni.* This may be due to the differences in slaughterhouse processing of farm animals and poultry. The main reason for this observation might be due to fecal contamination on the slaughter process of chicken, which is not common in the slaughter process of cattle, goat, and pigs. Further, the drying of the carcasses of cattle and pigs may also play a major role in lower contamination than that of poultry [40]. Cross-contamination with new strains in the poultry processing plant has also observed. The wet environment and lack of intensive cleaning and disinfection between the slaughters of different flocks per day might lead to persistence of *Campylobacter spp.* in slaughterhouses [43].

Beside meat products, especially from poultry, other animal products are known to trigger human Campylobacteriosis such as unpasteurized milk or milk products like cheese [21,40]. Eggs do not seem to play a part in *C. jejuni* transmission to humans, however; fecal bacteria infection including Campylobacteriosis can contaminate shells [44]. Point of action for the reduction of *Campylobacters* from food products is therefore high quality in food safety and hygiene [21].

Public Health Significance

Campylobacter is a major cause of bacterial gastroenteritis in much of the developed and developing countries [45]. *Campylobacter spp.* have usual considerable attention in recent years as the main cause of bacterial enteritis in human. *Campylobacter enteritis*, considered as an important source of diarrheal illness worldwide. The pathogen is also the main causative agent of 'traveler diarrhea' accompanied by predisposing debilitating factors such as premature birth, pregnancy, chronic alcoholism, cardiovascular disease and neoplasia [46].

Campylobacteriosis can cause disease in all age groups; however, infections are recognized with increasing frequencies in immune-compromised persons, infants, children, and aged individuals. According to the Centre for Disease Control (CDC) report, Campylobacteriosis accounted for approximately one-third of laboratory-confirmed food borne disease that occurred globally in food net surveillance areas [47]. A sever consequence of diarrheal diseases in human is called Guillain-Barrè Syndrome (GBS) which is characterized by polyneuritis of the peripheral nerves that may lead to paralysis. GBS, demyelinating disorder can cause acute neuromuscular paralysis, is a serious sequel of *Campylobacter* infection [48].

Economic Significance

Campylobacteriosis, lead to severe economic loses both in the public health and food industry sector. This disease has an enormous economic impact in terms of loss of production in human welfare and treatment costs. In livestock, particularly cattle and sheep, *Campylobacter spp.* are the major cause of economic losses associated with abortion and infertility problems [49]. A study, estimating that cost-of-illness and burden, in Netherland indicated that cost-of-illness was direct health-care costs, direct non-health-care costs, and indirect non-health-care costs. costs-of-illness were estimated to total € 21 million per year, with a 90% confidence interval of € 11 to € 36 million per year. Concluding that a *Campylobacter* infection the major public health problem for the country and earn substantial costs [50]. Moreover, the total costs of Campylobacteriosis to public health systems and to lost productivity in the EU is estimated to be around € 2.4 billion a year [39].

The Status of Foodborne *Campylobacter Spp.* in Ethiopia

Many food-producing animal and poultry species carry *Campylobacter* in their intestines and foods can be contaminated during processing [42]. However, the common cases of foodborne Campylobacteriosis are linked with the handling of raw products or ingestion of undercooked, cross-contamination of raw to cooked foods [51].

A Study conducted by Dadi and Asrat [34] in Addis Ababa and Debre-Zeit cities of Ethiopia has revealed an overall prevalence of the *Campylobacter spp.* isolated from meat samples was 9.3%. Prevalence *Campylobacter* in chicken meat was recorded highest (21.7%), followed by sheep meat (10.5%), pork meat (8.5%), goat meat (7.6%) and beef (6.2%). Among these isolates, 78% of the identified agents considered as *C. jejuni* 18% were *C. coli* and 4% were *C. lari.* Moreover, Wolde-Mariam and his colleagues [52] have also conducted research in Debre-Zeit private export abattoir of the country, in which thermophilic *Campylobacter spp.* (10.1%) were isolated from goat and sheep carcasses. Among these thermophilic *Campylobacter sp,p. C. jejuni* and *C. coli* accounted for 72.5% and 27.5%, respectively, which concluded, findings were indicating that raw meat from food animals could supply as a predisposing factor for Campylobacter that could result in potential risks of infection to people through the consumption under-cooked meat.

According to Faris, [53], in general prevalence of *Campylobacter* species isolated from bovine meat in Addis Ababa was (9.4%). amongst thermophilic *Campylobacters* isolated, 78% of the identified to be *C. jejuni*, while 22% were that of *C. coli.* Raw meat could serve as a potential vehicle for transmitting *Campylobacter spp.* Hence, implementation of hygienic practice from the slaughterhouse to the retailers, proper handling and cooking of foods of meat are very important in preventing the *Campylobacter* infection.

In Bahir-dar city of Ethiopia, the study conducted by Ewnetu and Mihret [54], has revealed the prevalence of thermophilic *Campylobacters*, 8% and 72.7% in man and poultry, respectively. In humans, 94.1% of the identified were *C. jejuni* and 5.9% were *C. coli, C. jejuni* was a major species of thermophilic *Campylobacters* in all categories of patients. In chicken 92.5% of thermophilic *Campylobacters* identified as *C. jejuni* and 7.5% were *C. coli*. Yeshimeet and her colleagues have also conducted another study [55], in Debre-birhan, Northen-Shoa, Ethiopia, revealed the thermophilic *Campylobacter spp.* with the prevalence of 10% and 21.4% in feces and carcass swab sample from sheep, respectively. Among the *Campylobacter* isolated from the fecal samples, 87.9% has identified to be *C. jejuni* and 12.1% were that of *C. coli*. Besides, from that of carcass swab sample, isolates *C. jejuni* and *C. coli* identified as 93.3% and 6.7% prevalence, respectively.

Pathogenesis

Not all *Campylobacter* infections produce illness. Even though all factors dependable for this phenomenon are not well known, three of the major important appears to be the number of organisms reaching the small intestine, the virulence of the infecting strain, and particular immunity of the host to the pathogen ingested [56]. *Campylobacter* establishes infection, in the small intestine first and later in the colon, and cause diarrheal illnesses with many of the appearances of naturally acquired infection. Diarrhea can be either watery or bloody, this kind of sign indicating that the extents of intestinal inflammation vary among individuals. It has been shown that this is in part correlated to differences in properties of the infecting strain. Limited evidence suggests that different *Campylobacter* strains can harbor different virulence traits [57].

Colonization of human intestine with *Campylobacter jejuni* results in the expression of several putative virulence factors, yet the mechanisms of pathogenesis are unclear [58]. Colonization may be influenced by the expression of lipopolysaccharides (LPS) on the bacterial cell surface. A mutation in the gale gene involved in the synthesis of LPS reduced the ability of *C. jejuni* to adhere and invade INT 407 cells, suggesting that LPS is a virulence factor for C. *jejuni* infection, although the *C. jejuni* was still able to colonize chickens [59].

Flagella are an important determinant in virulence since the colonization of intestinal tracts does not occur with non-motile mutants of *C. jejuni*. After immigration of the intestine, clinical disease may occur. Based on clinical sign start in patients, there are two mechanisms by which *Campylobacter* can bring disease were cause: (A) bacterial invasion and replication within the intestinal mucosa accompanied by an inflammatory response resulting in blood-containing, inflammatory diarrhea [57] and (B) adherence of *Campylobacter* to the intestine and the production of toxins, resulting in secretory diarrhea,

All strains of *C. jejuni* produce Cytolethal distending Toxin (CDT), a nuclease that results in cell cycle arrest and host DNA damage. Separately *C. jejuni* invasion of the mucosa or CDT alone triggers baso-lateral IL-8 release from the epithelium. In-vitro data suggest that *C. jejuni* can stay alive within macrophages/monocytes for several days, which may allow for some localized bacterial distribution. The pro-inflammatory cytokine IL-8 triggers an influx of PMNs from the lamina-propria, which prevents additional spread of *C. jejuni*. The resulting focal necrosis of the epithelium could result from the local inflammatory response and host cell death caused by CDT [60].

Clinical Features

In human

Most of the human *Campylobacter* enteritis is caused by *C. jejuni* and *C. coli* and these two species of *Campylobacter* are indistinguishable from each other and from acute bacterial diarrhea caused by other pathogens like *Salmonella enteritis* [61]. Campylobacteriosis may cause mild or severe diarrhea, bloody diarrhea, nausea, and stomach pain, often with fever. Abdominal pain can continue for up to 7 days and reappear of symptoms can occur [23]. An extraintestinal clinical sign of *Campylobacter* infection are quite atypical and may include endocarditis, septic arthritis, meningitis, osteomyelitis, and neonatal sepsis [62]. Bacterial infection has noted in less than 1% of patients with *C. jejuni*. Endocarditis and meningitis are rare manifestations of *C. jejuni* infection. There have been infrequent reports of *C. jejuni* infections manifested as acute cholecystitis, septic abortion, cystitis and pancreatitis [63]. Campylobacteriosis is recognized as the most commonly identified precursor event in Guillain-Barrè Syndrome (GBS) (40% to 60% of all cases), also known as post-infective polyneuropathy. The major lesions are polyradiculoneuropathy, acute inflammatory demyelinating that consequences in flaccid paralysis. Immediate arthritis occurs in approximately 1% of patients with *Campylobacter enteritis* [61].

In animals

Campylobacters are common components of the gut flora of all warm-blooded animals including livestock, domestic pets, and wild animals, and especially prevalent in avian species [64]. *Campylobacter spp.* cause abortions, enteritis, and infertility in various species of animals. The role of *C. jejuni* as a primary pathogen in farm animals is unsure [13]. *C. jejuni* and occasionally *C. coli* cause enteritis in cats, sheep, dogs, poultry, and calves some species of laboratory animals. The manifestation of the disease may be more severe in young animals. Calves typically have mucoid diarrhea, with occasional flecks of blood, either with or without fever. *C. fetus* subsp. *fetus* and *C. jejuni* can cause enzootic abortion that can result in stillbirths, abortions, and weak lambs in sheep. Morbidity in sheep can result in a reduction in milk production and in prolonged lambing. Revival with immunity to reinfection is characteristic [65].

Diagnosis

Campylobacteriosis, diagnosis can be recognized by the demonstration of the infection by direct examination of feces, or by isolation of the organisms. Bacteria of the genus *Campylobacter* have a characteristic morphology and a darting type of motility that permits their identification by direct examination of broth suspensions of feces. However, *C. jejuni* indistinguishable from *C. coli* by this procedure and the test is considerably less sensitive than isolation by culture [59]. A confirmed diagnosis requires a culture of the organism from feces or blood. Fecal samples should arrive in the laboratory within a few hours after collection or, if a delay is likely, should inoculate into a transport medium [66].

The beginning of selective agars proved successful for isolation from human feces and so recognizes campylobacters as a major cause of enteritis in human. Several different blood-based and non-blood-based media (Blaser, Butzler, Skirrow) containing different antimicrobial supplements and growth factors have been developed for the identification of *Campylobacters* from fecal specimens [67]. Since *campylobacters* are micro-aerophilic, the most convenient method to achieve a favorable atmosphere is with commercially available gas-generating envelopes that are activated in the anaerobic jars. Plates should be incubated for 48 to 72 hours and examined daily for growth Suspect colonies should be screened with three presumptive tests: oxidase test, wet mount preparation under dark-field or phase-contrast microscope, and Gram stain. If a dark-field or phase-contrast microscope is not available, colonies may be rapidly screened for typical cell morphology by staining with Gram's crystal-violet solution [68].

Further identification to the species level requires other tests including antibiotic sensitivity to cephalothin and nalidixic acid, growth temperature and biochemical tests, mainly the hippurate test [69]. Polymerase chain reaction (PCR) can be applied in the clinical diagnosis of campylobacteriosis, and application of multiplex PCR for the finding and speciation of this pathogen; however, this guideline have been optimized for isolates obtained from pure-cultures and artificially spiked stool specimens [70].

Treatment and Antimicrobial Resistance

In immune-competent individuals, *Campylobacter* enterocolitis is generally self-limited, with mild to moderate symptoms, and antibiotic therapy is not required for most patients. While supportive care with oral rehydration is the preferred treatment [56]. Antibiotic therapy may be careful with febrile patients, immune suppressed, bloody diarrhea and those whose clinical sign worsen or continue for more than one week from the time of diagnosis. Macrolides (e.g. Erythromycin) are the first choice of drug, and Fluoroquinolones (e.g. Ciprofloxacin) are often the second choice of drug recommended for the treatment of human Campylobacteriosis[71].

Recent data indicates a rising trend of *Campylobacter* resistance to antibiotics with unstable patterns being seen in different countries and regions [72]. Antibiotic resistance has emerged between campylobacter mainly because of the wide use of the antimicrobials, especially fluoroquinolones, macrolides, and tetracyclines in food animal, and in human medicine too [73, 74]. In general, the majority of thermo-tolerant *Campylobacter spp.* are resistant to a large number of beta-lactam antibiotics, particularly for Ampicillin, Amoxicillin, and Cefotaxime [75].

Prevention and Control Strategies

At farm level

Campylobacteriosis is common in domestic animals and wild, and therefore in the environment, it is important to reduce contamination of chicken rearing houses from such sources. Installing hygienic barriers between the internal and external environments, such as strict hygienic routines before entering, controlling the entry of farm personnel, have shown to be effective. Poultry production in a free-range system has a much greater risk of infection compared to conventional production and increased the difficulties in control [42]. Flock vaccination starts to show green light in minimizing excretion rate. Nevertheless, the elimination of intestinal *Campylobacter* carriage from a food-producing animal may not be an easy task and thus the risk of infection from these sources will remain [76].

At the food animals processing level

Bacterial load counts on meat increase during slaughter and processing steps. In studies of turkeys and chickens at slaughter, bacterial counts increased by approximately 10-100 folds during defeathering, and increase up to the highest level after evisceration. However, bacterial counts on meat could decline through hygienic processing and slaughter steps such as forced-air blast chilling of carcasses, scalding or mild heat treatment, use of lactic acid spraying of swine carcasses, chlorinated sprays, maintenance of clean working surfaces, and terminal radiation [61].

At home level

The consumer is the last bond in the food chain and has to contact with residual pathogens in food. A major procedure required in the kitchen to diminish the risk of infection with *Campylobacter spp.* Consists of the application of the basic doctrine of safe food preparation. Furthermore, awareness of basic measures such as separation of ready to eat, raw food and hand washing, some of the traditional food preparation practices should be depressed, for example, increases the risk of contamination and the practice of washing dressed poultry carcasses in the kitchen sink is unnecessary [77]. appropriate hygienic preparation of food, travel to underdeveloped countries (hyper-endemic *Campylobacter* transmission area), avoidance of eating raw meat, avoidance of unpasteurized dairy products, and exposure to animals such as pet animal with diarrhea (particularly puppies and kittens) should be avoided [61].

Conclusion

Human campylobacteriosis caused by thermo-tolerant *Campylobacter spp.* continued to be one of the major commonly reported zoonotic disease, which results in a serious consequence of diarrheal in human and severe economic losses worldwide. According to the research findings reported from Ethiopia, thermophilic *Campylobacter spp.* have been identified both row types of meat and fecal samples of different domestic animals (chicken, cattle, sheep, goat, and pig) and human; however, highest prevalence (72.7%) has reported from that of chicken meat. *C. jejuni* and *C. coli* were the predominant species, recorded with a prevalence rang of (93.3% to 72.5%) and (27.5% to 6.7%) respectively. Although the scientific researches regarding *Campylobacter spp.* published from the country are few in number, findings have indicated that undercooked meat from food animals mainly that of chicken could provide as the main source of campylobacteriosis infection in Ethiopia, through the consumption of undercooked meat. Hence, the concern should focus on breaking the transmission from these sources to human beings. In countries like Ethiopia, controlling *Campylobacter* infection at farm level (i.e. poultry) is difficult, in which the livestock production was highly laid on backyard system. Therefore, implementation of hygienic practice from slaughterhouse up to the consumers, using chlorinated water in birds' feed and to perform slaughtering, skinning, and evisceration under aseptic conditions, proper handling and cooking meats of animal origin are very important points, besides, further researches should focus on study samples other than meat and economic significance of the diseases.

Acknowledgment

We would like to thank Mekelle University College of Veterinary Medicine for evaluating this research review work, and allowing us with free access to internet and library services.

Funding

This review work has not received any fund.

Authors' Contribution

MB and YH wrote this manuscript. Both authors have read and approved the final manuscript.

Competing Interest

The authors declare that they have no competing interests.

References

1. World Health Organization (2018) Campylobacter. Facts Sheets. Geneva, Switzerland.
2. Silva J, Leite D, Fernandes M, Mena C, Gibbs PA, et al. (2011) Campylobacter spp. as a foodborne pathogen: A review. Front Microbiol 2: 200.
3. European Food Safety Authority and European Centre for Disease Prevention and Control (EFSA and ECDC) (2015) The European Union summary report on trends and sources of zoonoses, zoonotic agents and food-borne outbreaks in 2014. EFSA Journal 13: 43294.
4. Mpalang RK, Boreux R, Pierrette M, Ni Bitiang K, Daube G, et al. (2014) Prevalence of Campylobacter among goats in Congo. Infect Dev Ctries 8: 168-175.
5. Kaur T, Singh J, Huffman M, Petrzelková K, Taylor N, et al. (2011) Campylobacter troglodytis sp. nov., isolated from feces of human-habituated wild chimpanzees (Pan troglodytes schweinfurthii) in Tanzania. Appl Environ Microbiol 77: 2366-2373.
6. Fitzgerald C, Whichard J, Nachamkin I (2008) Campylobacter, In Nachamkin, I, Szymanski, C, Blaser M (Eds.). Diagnosis and antimicrobial susceptibility of Campylobacter species,Washington, DC: ASM Press pp: 227-243.
7. Blaser MJ (2000) C. jejuni and related species. In Gerald L.M., Douglas R.G. and Bennett. S.E. Principle and practice of infectious Disease, 5th Ed. Hurchil Livingston, pp: 2276-2285.
8. Loshaj-Shala A, Regazzoni L, Daci A, Orioli M, Brezovska K, et al. (2015) GuillainBarr´e syndrome (GBS): New insights in the molecular mimicry between C. jejuni and human peripheral nerve (HPN) proteins. J Neuroimmunol 289: 168-176.
9. Allos BM (2001) C. jejuni infections: update on emerging issues and trends. Clin Infect Dis 32: 1201-1206.
10. MagazMartínez M, GarridoBotella A, Pons Renedo F, Oliva Del Río B, Agudo Castillo B, et al. (2016) Fatal Campylobacter jejuni ileocolitis. Rev Esp Enferm Dig 108: 662-663.
11. Ghosh R, Uppal B, Aggarwal P, Chakravarti A, Jha AK (2013) Increasing antimicrobial resistance of Campylobacter jejuni isolated from paediatric diarrhea cases in a tertiary care hospital of New Delhi, India. J Clin Diagn Res 7: 247-249.
12. Ebrahim R (2010) Occurrence and resistance to antibiotics of Campylobacter spp. in retail raw sheep and goat meat in Shahre Kord, Iran Glob Vet 4: 504-509.
13. Padungton P, Kannen JB (2003) Campylobacter spp. in human, chickens, pigs, and their antimicrobial resistance. J Vet Med Sci 65: 161-70.
14. Prescott LM, Harley JP, Klein DA (1999) Microbiology, 4th ed. McGraw-Hill co, Boston Burr Ridge WCB, pp: 393-420.
15. Garrity GM, Bell IA, Lilburn TG (2004) Taxonomic outline of the prokaryotes, Bergey's manual of systematic bacteriology, 2nd ed. Springer, Beringey's manual trust, pp: 137-138.
16. Moran AP, Upton ME (1987) Factors affecting production of coccoid forms by C. jejunion solid media during incubation. J App Bacteriol 62: 527-537.
17. Ursing JB, Lior H, Owen RJ (1994) Proposal of minimal standards for describing new species of the family Campylobacteraceae. Int J Syst Bacteriol 44: 842-845.
18. On SLW, Bloch B, Holmes B, Hoste B, Vandamme P (1995) Campylobacter hyointestinalis subsp. lawsonii subsp. nov., isolated from the porcine stomach, and an emended description of Campylobacter hyointestinalis. Int J Systemat Bacteriol 45: 767-774.
19. Gharst G, Oyarzabal OA, Hussain SK (2013) Review of current methodologies to isolate and identify Campylobacter spp. from foods. J Microbiol Methods 95: 84-92.
20. Sampers I, Habib I, De Zutter L, Dumoulin A, Uyttendaele M (2010) Survival of Campylobacter spp. in poultry meat preparations subjected to freezing, refrigeration, minor salt concentration, and heat treatment. Int J of Food Microbiol 137: 147-153.
21. Teh AHT, Lee SM, Dykes GA (2019) Association of some Campylobacter jejuni with Pseudomonas aeruginosa biofilms increases attachment under conditions mimicking those in the environment. PLoS One 14: e0215275.
22. Oyarzabal OA, Oscar TP, Speegle L, Nyati H (2010) Survival of C. jejuni and C.coli on retail broiler meat stored at -20, 4, or 12 degrees C and development of Weibull models for survival. J Food Protec 73: 1438-1446.
23. Andrew P, Emily V, Mai P, Ian Y, Nicole B (2013) Using risk factor weighting to target and create effective public health policy for Campylobacteriosis prevention in Ontario, Canada. Amer J Public Health Res 1: 32-37.
24. Levin RE (2007) C. jejuni: A review of its characteristic, pathogen city, ecology, distribution, subspecies characterization and molecular methods of detection. Food Biotechnol 21: 271-347.
25. Reuter M, Mallett A, Pearson BM, Vliet AHM (2010) Biofilm formation by C. jejuni is increased under aerobic condition. Appl Env Microbiol 76: 2122-2128.
26. International Commission on Microbiological Specifications for Foods (ICMSF) (1996) Microorganisms in Foods, in microbiological specifications of food pathogens. International Commission on Microbiological Specifications for Foods. London: Blackie 5, pp: 45-65.
27. Craven SE, Stern NJ, Line E, Bailey JS, Cox NA, et al. (2000) Determination of the incidence of Salmonella spp., C. jejuni, and Clostridium perfringens in wild birds near broiler chicken houses by sampling intestinal droppings. Avian Dis 44: 715-720.
28. Keller JI, Shriver WG, Waldenstrom J, Griekspoor P, Olsen B (2011) Prevalence of Campylobacter in wild birds of the mid-Atlantic region, USA. J Wild Dis 47: 750-754.
29. Eyers M, Chapelle S, Van camp G, Goossens H, Wachter RDE (1993) Discrimination among thermophilic Campylobacter spp. by Polymerase Chain Reaction amplification of 23S rRNA gene fragments. J Clin Microbiol 31: 3340-3343.
30. Alter T, Weber RM, Hamedy A, Glunder G (2011) Carry-over of thermophilic Campylobacter spp. between sequential and adjacent poultry flocks. Vet Microbiol 147: 90-95.
31. Weber R, Alter T, Glünder G (2011) Campylobacter in different poultry species: Prevalence and spread between flocks. In: Referatesammlung, 80. Technical discussion about poultry diseases, DVG-Fachgruppe poultry diseases, Hannover, pp: 23-28.
32. Weber A, Lembke C, Schafer R (1985) Season-related incidence of C. juni and C. coli in feces samples of healthy slaughter cattle. Zbl Vet Med B 32: 197-201.
33. Petersen L, Wedderkopp A (2001) Evidence that certain clones of C. jejuni persist during successive broiler flock rotations. Appl Environ Microbiol 67: 2739-2745.

34. Dadi L, Asrat D (2008) Prevalence and antimicrobial susceptibility of thermotolerant Campylobacter strains in retail raw meat products in Ethiopia. Ethiop J Health Dev 22: 195-196.
35. Heuvelink AE, Van heerwaarden C, Zwartkruis-Nahuis A, Tilburg JJ, Bos MH, et al. (2009) Two outbreaks of Campylobacteriosis associated with the consumption of raw cows' milk. Int J Food Microbio 134: 70-74.
36. Ricotta EE, Palmer A, Wymore K, Clogher P, Oosmanally N, et al. (2014) Epidemiology and antimicrobial resistance of international travel-associated Campylobacter infections in the United States, 2005-2011. Ame J Public Health 104: 108-114.
37. Young KT, Davis LM, Dirita VJ (2007) C. jejuni: Molecular biology and pathogenesis. Nat Rev Microbiol 5: 665-679.
38. European Food Safety Authority (EFSA) (2015) The European Union Summary Report on Trends and Sources of Zoonoses, Zoonotic Agents and Food-borne Outbreaks in 2013. EFSA J 13: 3991.
39. European Food Safety Authority (EFSA) (2011) Scientific opinion on Campylobacter in broiler meat production: Control option and performance objective and/or targets at different stages of the food chain. EFSA J 9: 2105.
40. European Food Safety Authority (EFSA) (2012) The European Union summary report on trends and sources of zoonoses, zoonotic agents and food-borne outbreaks in 2010. Euro Survei 10: 2597.
41. Louis VR, Gillespie IA, O'brien SJ, Russek-Cohen ED, Pearson A, et al. (2005) Temperature-driven Campylobacter seasonality in England and Wales. Appl Environ Microbiol 71: 85-92.
42. Humphery T, O'brien S, Madsen M (2007) Campylobacters as zoonotic pathogens: A food production perspective. Int J Food Microbiol 117: 237-257.
43. Melero B, Juntunen P, Hanninen ML, Jaime I, Rovira I (2012) Tracing C. jejuni strains along the poultry meat production chain from farm to retail by pulsed-field gel electrophoresis, and the antimicrobial resistance of isolates. Food Microbiol 32: 124-128.
44. Cox NA, Richardson LJ, Maurer JJ, Berrang ME, Fedorka-CRAY PJ, et al. (2012) Evidence for horizontal and vertical transmission in Campylobacter passage from hen to her progeny. J Food Prot 75: 1896-1902.
45. Centre for Disease Control (CDC) (2009) Preliminary food net data on the incidence of infection with pathogens transmitted commonly through food-10 states, Centers for disease control and prevention. Morb Mortal Wkly Rep 59: 418-422.
46. Mandrell E, Miller G, Motarjemi Y, Adams C (2006) Campylobacter in: Emerging food borne pathogens. Wood head publishing Ltd, pp: 476-521.
47. Centre for Disease Control (2008) Preliminary food net data on the incidence of infection with pathogens transmitted commonly through food in 10 states, Centers for Disease Control and Prevention. Morb Mortal Wkly Rep 57: 366-370.
48. Shane M (2000) Campylobacter infection of commercial poultry. Rev Sci Tech 19: 376-395.
49. Beatriz O, Ana H (2011) Emerging thermotolerant Campylobacter spp in healthy ruminants and swine. Department of Animal Health, Spain. Foodborne Patho Dis 8: 807-813
50. Mangen MJ, Havelaar AH, Wit GA (2004) Campylobacteriosis and sequelae in the Netherlands; Estimating the disease burden and the cost-of illness. Wageningen University and Research Centre, Agricultural Economics Research Institute RIVM report 1-20.
51. European Food Safety Authority (EFSA) (2014) The European union summary report on trends and sources of zoonoses, zoonotic agents and food-borne outbreaks in 2012. EFSA J 12: 3547.
52. Woldemariam T, Asrat D, Girma Z (2009) Prevalence of thermophilic Campylobacter spp. in carcasses from sheep and goat in Ethiopia. Ethiop J Health De 23: 229-233.
53. Faris G (2015) Identification of Campylobacter spp. and their Antibiotic resistance pattern from raw bovine meat in Addis Ababa, Ethiopia. Int J Microbial Immu Res 4: 001-005.
54. Ewnetu D, Mihret A (2010) Prevalence and antimicrobial resistance of Campylobacter isolates from humans and chickens in Bahir Dar, Ethiopia. Foodborne Pathog Dis 7: 667-670.
55. Yeshimebet C, Daniel A, Patamapom A, Winya L (2013) Prevalence and antimicrobial susceptibility of thermophilic campylobacter isolated from sheep at Debre-Birhan, North-Shoa, Ethiopia. Kasetsart J Nat Sci 47: 551-560.
56. Masci JR, Wormser GP (2005) Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases, 6th Edition Edited by Gerald L. Mandell, John E. Bennett, Raphael Dolin Philadelphia: Elsevier Churchill Livingstone, Clinical Infectious Diseases 41: 277.
57. Janssen R, Krogfelt KA, Cawthraw SA (2008) Host-pathogen interactions in Campylobacter infections: the host perspective. Clin Microbiol Rev 21: 505-518.
58. Van Vliet AHM, Ketley JM (2001) Pathogenesis of enteric Campylobacter infection. J Appl Microbiol 90: 45S-56S.
59. Gillespie SH and Hawkey PM (2006) Principles and practice of clinical bacteriology 2nd ed. John Wiley and Sons Ltd, The Atrium, Southern Gate, Chichester, England, pp: 587.
60. Kopecko DJ, Hu L, Zaal KJM (2001) Campylobacter jejuni microtubule-dependent invasion.Trends Microbiol 9: 389-396.
61. Skirrow MB (2002) Microbiology and epidemiology of Campylobacter infection: Clinical features and treatment of Campylobacter infection in children. 10: 2-4.
62. Pires SM (2014) Burden of disease of food borne pathogens in Denmark. 1st edt. FoodBurden DK-Technical Report.
63. Nachamkin I (1999) Campylobacter and Arcobacter. In: Murray P.R. and Baron E.G.O., Manual of linical Microbiology 7th Ed. ASM press; Washington DC. America. Society of Microbiology, pp: 716-726.
64. European Food Safety Authority (EFSA) (2010) Analysis of the baseline survey on the prevalence of Campylobacter in broiler batches and of Campylobacter and Salmonella on broiler carcasses in the EU, 2008, Part A: Campylobacter and Salmonella prevalence estimates. EFSA J 8: 1522.
65. Aiello SE, Mays A (1998) The Merck veterinary manual, 8th Edited. Whitehouse Station, NJ: Merck and Co, pp: 988-997.
66. Sack DA, Lyke C, McLaughlin C, Suwanvanichkij V (2001) Antimicrobial Resistance in Shigellosis, Cholera and Campylobacteriosis: WHO/CDS/CSR/DRS/2001.8.
67. Cheesbrough M (2006) District laboratory practice in tropical countries Part 2 second edition. Part 2.
68. Vandepitte J, Verhaegen J, Engbaek K (2003) Basic laboratory procedures in clinical bacteriology, WHO, Geneva.
69. Senok AC, Botta GA (2009) Campylobacter enteritis in the Arabian Gulf University of Sharijah, United Arab Emirates. J Infect Dev Countries 3: 74-82.
70. Person S, Olsen KE (2005) Multiplex PCR for identification of C. coli and C. jejuni from pure cultures and directly on stool samples. J Med Microbiol 54: 1043-1047.
71. Engberg J, Aarestrup FM, Taylor DE, Gerner-Smidt P, Nachamkin I (2001) Quinolone and macrolide resistance in C. jejuni and C .coli: resistance and trends in human isolates. Emerg infecti Dis 7: 24-34.
72. Moore JE, Barton MD, Blair IS, Corcoran D, Dooley JS, et al. (2006) The epidemiology of antibiotic resistance in Campylobacter. Microbes Infect 8: 1955-1966.
73. Fraqueza MJ, Martins A, Borges AC, Fernandes MH, Fernandes MJ, et al. (2014) Antimicrobial resistance among Campylobacter spp. strains isolated from different poultry production systems at slaughterhouse level. Poul Sci 93: 1578-1586.
74. Kovalenko K, Roasto M, Šantare S, Berzins A, Horman A (2014) Campylobacter spp. and their antimicrobial resistance in Latvian broiler chicken production. Food Control 46: 86-90.
75. Tajada P, Gomez-Garces JL, Alos JI, Balas D, Cogollos R (1996) Antimicrobial susceptibilities of C. jejuni and C. coli to 12 beta-lactam agents and combinations with betalactamase inhibitors. Antimicrob Agents Chemother 40: 1924-1925.

76. Lis A, Wyszynska A, Raczko A (2003) Bivalent anti-Campylobacter/ Salmonella chicken vaccine- effect of oral immunization with cocktail vaccine prototype. Int J Med Microbiol 293: 15-23.

77. Food Safety Authority of Ireland (FSAI) (2002) Control of Campylobacter spp. in the food chain.Food Safety Authority of Ireland, Dublin, Ireland, pp: 2-3.