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Campylobacteriosis: A rising threat in foodborne illnesses

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ABSTRACT
Campylobacteriosis is a foodborne illness that is contracted by eating contaminated food, particularly animal products like meat from diseased animals or corpses tainted with harmful germs. The epidemiology of campylobacteriosis varies significantly between low-, middle-, and high-income countries. Campylobacter has a complicated and poorly known survival strategy for getting past host barriers and causing sickness in humans. The adaptability of *Campylobacter* to unfavorable environments and the host's immune system seems to be one of the most crucial elements of intestinal colonization. A *Campylobacter* infection may result in fever, nausea, vomiting, and mild to severe bloody diarrhea in humans. Effective and rapid diagnosis of *Campylobacter* species infections in animal hosts is essential for both individual treatment and disease management at the farm level. According to the most recent meta-analysis research, the main risk factor for campylobacteriosis is travel, which is followed by eating undercooked chicken, being exposed to the environment, and coming into close contact with livestock. *Campylobacter jejuni*, and occasionally *Campylobacter coli*, are the primary causes of *Campylobacter* gastroenteritis, the most significant *Campylobacter* infection in humans for public health. The best antibiotic medications for eradicating and decreasing *Campylobacter* in feces are erythromycin, clarithromycin, or azithromycin. The best strategy to reduce the number of human infections caused by *Campylobacter* is to restrict the amount of contamination of the poultry flock and its products, even if the majority of infections are contracted through handling or ingestion of chicken.
Keywords: *Campylobacter*, Campylobacteriosis, Food, Poultry, Public health.

Introduction

The zoonotic disease known as campylobacteriosis is brought on by bacteria belonging to the genus *Campylobacter*, which includes the species *Campylobacter jejuni*, *Campylobacter coli*, *Campylobacter lari*, and *Campylobacter fetus* (Facciolà *et al*., 2017). This is a foodborne illness that is contracted by eating contaminated food, particularly animal products like milk and meat from diseased animals or corpses tainted with harmful germs (Chlebicz and

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Śliżewska, 2018; Agumah *et al*., 2024). The number of instances of campylobacteriosis has surpassed that of classical enteric bacteria. The information indicates that the prevalence of *Campylobacter* infections has risen recently (Behailu *et al*., 2022). The incidence of this organism's isolation from gastrointestinal infections is roughly three to four times higher than that of *Salmonella* or *Escherichia coli* (Epps *et al*., 2013; Yanestria *et al*., 2019; Ansharieta et al., 2021).

The digestive tracts of domestic animals, wild animals, and livestock serve as the organism's reservoir (Johnson *et al*., 2017). Since the *Campylobacter* species that cause human gastroenteritis can grow at temperatures above 40ºC, they are categorized as temperature tolerant (Yanestria *et al*., 2024a). The two main variables that affect *Campylobacter* infection are transmission factors and body resistance. Human campylobacteriosis infections are mostly infrequent. The most common ways that these bacteria are spread are through the consumption of raw milk, drinking water, eating undercooked chicken and cattle meat, or coming into close contact with sick dogs or cats (Goddard *et al*., 2022). The range of germs that can induce an illness in an individual is between 500 and 10,000 cells (Kreling *et al*., 2020). Children are exposed to a lower infectious dosage than adults.

Foodborne *Campylobacter* infections are a human health problem that causes approximately 8.4% of global diarrhea cases (Igwaran and Okoh, 2019). One of the four primary causes of diarrheal illness worldwide is *Campylobacter*; diarrhea is the most prevalent foodborne illness, affecting 550 million people annually (Myintzaw *et al*., 2023). Campylobacteriosis symptoms include vomiting, fever, and bloody or watery diarrhea. In general, people over 75 years old and younger children (under 4) are more likely to have *Campylobacter* infections (Guo *et al*., 2023). Furthermore, patients with hemoglobinopathy, inflammatory bowel illness, and those with compromised immune systems are among the populations most at risk of contracting a *Campylobacter* infection (Igwaran and Okoh, 2019).

Compared to low-income countries, high-income countries have a higher risk of *Campylobacter* infection. A multitude of environmental factors present a significant risk of *Campylobacter* transmission to people in low-income countries, and eating poultry meat and poultry products is the main cause of outbreaks (El-Saadony *et al*., 2023). Worldwide incidences of campylobacteriosis are caused by 60%–80% of poultry meat and its by-products. Poultry meat from laying hens, turkeys, ostriches, ducks, and broilers is frequently consumed (Suzuki and Yamamoto, 2009). To lower product contamination and thus lower the incidence of human campylobacteriosis, intervention techniques to manage *Campylobacter* in hens must be prioritized as consumer demand for poultry meat and poultry products rises (Taha-Abdelaziz *et al*., 2023). *Campylobacter*, especially in poultry, can spread quickly by the fecal-oral pathway from one animal to the entire flock in less than a week (Rawson *et al*., 2019). Once within the host body, these bacteria live in the intestines, which have the highest concentration of bacteria; the liver, deep muscles, spleen, thymus, and bursa of Fabricius have smaller concentrations of bacteria (Deng *et al*., 2020). Bacterial colonization results in serosal fluid leakage, inflammation, toxin production, invasion of host cells, and active secretion, all of which lead to epithelial diseases (O Cróinín and Backert, 2012). In humans, campylobacteriosis typically takes two to five days to incubate. Similar to the toxins produced by *E. coli* and cholera, this bacterium also produces enterotoxins (Wysok *et al*., 2022).

The yearly rise in instances of campylobacteriosis has raised concerns about the disease's occurrence around the world, with major socioeconomic and public health ramifications. This review article aims to provide a comprehensive explanation of campylobacteriosis, starting from etiology, history, epidemiology, pathogenesis, virulence factors, diagnosis, clinical symptoms, transmission, public health importance, treatment, antibiotic resistance, control, and vaccination. Understanding the overview of campylobacteriosis is essential to implementing effective control measures.

Etiology

Organism description

Greek terms "campylos," which means "curved," and "baktron," which means "rod," are the sources of the word *Campylobacter*. Gram-negative bacteria belonging to the Campylobacteriaceae family are called *Campylobacter* species, and they do not produce spores (Wang *et al*., 2023). There are six subspecies and seventeen species in the genus *Campylobacter*. More advancements in taxonomic standards could lead to a more precise classification of *Campylobacter* species. The two species that are most frequently linked to human illness are *C. jejuni* and *C. coli* (Pölzler *et al*., 2018). More than 80% of human *Campylobacter*-related diseases are caused by *C. jejuni*, but up to 18.6% are caused by *C. coli* (Ammar *et al*., 2020). Additionally, *C. fetus* has been connected to human foodborne diseases.

Morphology and characteristics of Campylobacter bacteria

Certain gram-negative bacteria called *Campylobacter* species do not produce spores. These organisms range in size from 0.2 to 0.8 μm in width and 0.5 to 5 μm in length. They have a spiral shape, are curved, or are occasionally observed as straight rods (Epps *et al*., 2013). *Campylobacter* can be seen in short or occasionally large chains and can have shapes such as spiral, S, V, or comma (Mushi *et al*., 2014). *Campylobacter* cells begin to age and then become coccoid in shape. Possessing one or more flagella at one end, the cells exhibit remarkable motility. A phase contrast microscope can be used to observe the comma-shaped cells' quick, darting movement (Frirdich *et al*., 2019).

Growth and survival characteristics of Campylobacter bacteria

Species of *Campylobacter* are delicate organisms. This organism is susceptible to irradiation, disinfectants, freezing, drying, heating (pasteurization/cooking), and acidic conditions (preservation) (Kim *et al*., 2021). Bacteria often do better at colder temperatures and struggle to thrive at room temperature (25°C). *Campylobacter jejuni* thrives in low-oxygen conditions, such as those with 5% O_2, 10% CO_2, and 85% N_2, and grows best at 37°C to 42°C (Kaakoush *et al*., 2007). The gut has evolved bacteria to have a single polar flagellum and a corkscrew form. These characteristics promote motility (Liu *et al*., 2018). It has been demonstrated that certain *Campylobacter* species can grow in viscous intestinal mucus under unfavorable circumstances, such as low nutrition availability, high temperature, freezing, or stationary phase (Ribardo *et al*., 2024). The cells undergo a form of transition in this condition, going from a motile spiral to a coccoid. This coccoid form's nature and function are unknown. Due to its capacity to form a biofilm, *C. jejuni* can tolerate aerobic environments (Pokhrel *et al*., 2022).

History

There is a vast and varied range of bacteria in the *Campylobacter* genus. Theodor Escherich identified the first reported case of *Campylobacter* in 1886 when he observed non-culturable, helical-shaped bacteria in colonic mucosal stains linked to diarrhea in infants who perished from an outbreak of the disease thought to be "*Cholera infantum*" (Sheppard and Maiden, 2015). In 1906, *Campylobacter* or bacteria resembling *Vibrio*, were first isolated from the uterus of a sheep that had been aborted (Tresse *et al*., 2017). A similar pathogen, *Vibrio fetus*, was identified in 1912 from aborted bovine fetuses. After fifteen years, a different *Vibrio* pathogen called *Vibrio jejuni* was found in the feces of cows that had diarrhea (Epps *et al*., 2013). *Vibrio coli* was discovered in 1944 in pigs that had diarrhea.

For more than 40 years, it has been believed that *Campylobacter* causes animal illness, especially in 1938 when *Campylobacter* spp. was reported to be involved in foodborne illness outbreaks (Sockett and Rodgers, 2001). Due to the ingestion of tainted milk, a *Vibrio jejuni*-like virus was found in the blood of 13 outbreak victims. This outbreak resulted in acute diarrheal sickness in 357 prisoners at an Illinois state institution in the United States (Poly *et al*., 2019). To distinguish this bacterium from *Vibrio* spp., it was moved to the newly formed genus *Campylobacter* in 1963 because of its unique traits, which included low DNA base composition (low G+C concentration), microaerophilic development, and nonfermentative metabolism (O'Loughlin *et al*., 2015). The Greek word "kampyo's" is the source of the genus name, which signifies bent or curled (Sheppard and Maiden, 2015).

The American bacteriologist Elisabeth King provided the first accurate description of the genus *Campylobacter*. King was determined to isolate these bacteria from feces and thought that the prevalence of *Campylobacter* was higher than the few cases that had been reported (Moore and Matsuda, 2002). Butzler was able to isolate *Campylobacter* from the feces of human patients who were experiencing diarrhea and elevated body temperatures in the early 1970s, thanks to the development of a unique filtration technique for the identification of this bacteria in veterinary medicine (Butzler, 2004). However, adding certain antimicrobial drugs, such as trimethoprim, polymyxin B, and vancomycin to the basic medium allowed for significant advancements in *Campylobacter* isolation (Bonnet *et al*., 2019).

Four distinct species of *Campylobacter* were identified by Veron and Chatelain in their 1973 comprehensive study on the taxonomy of microaerophilic vibrio-like organisms: *C. coli*, *C. sputorum*, *C. fetal*, and *C. jejuni* (Costa and Iraola, 2019). *C. jejuni* was not widely acknowledged as one of the most common causes of foodborne bacterial enteritis in people until the 1980s, around a century after it was initially discovered.

Public health importance

Campylobacter jejuni, and occasionally *C. coli*, are the primary causes of *Campylobacter* gastroenteritis, the most significant *Campylobacter* infection in humans for public health (Igwaran and Okoh, 2019). It is estimated that 80% of human *Campylobacter* infections are caused by chickens, which are the primary reservoir of *Campylobacter* spp. infections (Epps *et al*., 2013). Campylobacteriosis is thought to be carried asymptomatically by chickens. Most chicken flocks can become colonized by *Campylobacter* spp. (Yanestria *et al*., 2023). According to basic research conducted by the European Union, *Campylobacter* was found in 75.8% of broiler carcasses, 71.2% of a batch of broiler chickens' cecal content contained *Campylobacter*, and 61.3% of chicken skin samples from retail settings had *Campylobacter* prevalence, with 18.6% of those samples having counts of more than 1000 CFU/g (Hue *et al*., 2010).

Despite the low severity of campylobacteriosis (0.03%), there are more instances of *Campylobacter* infection in humans (Facciolà *et al*., 2017). Remarkably, among foodborne microbes, *Campylobacter* infections are the most prevalent cause of death in humans. Patients with compromised immune systems, such as those with cancer, liver disease, and acquired immunodeficiency syndrome (AIDS), are susceptible to death from *Campylobacter* infections (Louwen *et al*., 2012). In most cases, campylobacteriosis is sporadic and self-limiting. The symptoms of *Campylobacter*-induced gastroenteritis include fever, headache, vomiting, cramping or pain in the abdomen, watery or often bloody diarrhea, and weight loss (Hsu *et al*., 2023).

A *Campylobacter* infection can result in post-infectious immune disorders like Guillain Barré syndrome (GBS), a neurological disorder characterized by flaccid paralysis and advanced weakness in the extremities. It can also cause reactive arthritis (RA), or inflammation of the joints, and paralysis of the respiratory muscles (Finsterer, 2022). Not only that, but this bacterial infection also results in Miller-Fisher syndrome (MFS), which manifests as visual abnormalities (ophthalmoplegia) and coulometer weakness (areflexia and ataxia) (Ang *et al*., 2001). Campylobacteriosis is hyperendemic in underdeveloped nations, and infections with *Campylobacter* are symptomatic, virtually exclusively occurring in young children and babies (Coker *et al*., 2002). In adults or older children, the signs of this infection are uncommon since subsequent infections may be asymptomatic.

It is interesting to note that developed and developing nations have very different campylobacteriosis epidemiologies. Due to the lack of national systems for the surveillance of *Campylobacter* infections, it is challenging to assess the prevalence of *Campylobacter* infections in underdeveloped nations, since *Campylobacter* is not the most common cause of bacterial foodborne diseases (Platts-Mills and Kosek, 2014). Research articles on *Campylobacter* isolation from various specimens are the primary source of information regarding the prevalence of *Campylobacter* infections in underdeveloped nations (Chala *et al*., 2021).

Epidemiology

The epidemiology of campylobacteriosis varies significantly between low-, middle-, and high-income countries. These variations are most likely due to variations in food habits, nutritional status, environmental cleanliness, climatic conditions, immune system status (especially among elderly people and children) diagnostic methods, and the availability of natural reservoirs (Sanchez *et al*., 2020). *Campylobacter* infections are hyperendemic in children under the age of two, with up to two cases per child in low- and middle-income countries (Kiarie *et al*., 2023). Most cases in children and adults are asymptomatic. *Campylobacter* infections without symptoms are uncommon in high-income nations, but the average lifetime frequency of infections is less than one (Pascoe *et al*., 2020). Children are typically more likely to have *Campylobacter* infections, which suggests that early exposure may result in the formation of protective immunity but later asymptomatic shedding (Samie *et al*., 2022).

The exact nature of the global incidence rate of *Campylobacter* outbreaks is largely unclear, and reports of their incidence differ between nations. The underreporting of *Campylobacter* infection cases, variations in reporting systems, challenges in diagnosis, and variations in surveillance in the event of an outbreak are among the factors contributing to the inaccurate incidence of campylobacteriosis outbreaks. Outbreaks of campylobacteriosis typically result from multiple humans contracting food- or water-borne diseases, and the majority of these outbreaks are caused by animals (Llarena and Kivistö, 2020). Despite the fact that many people rely on rivers and streams as their main supply of drinking water, environmental sources like these are typically the source of campylobacteriosis outbreaks in low-income nations (Whiley *et al*., 2013). Apart from their role in the spread of human diseases in low-income nations, water sources have also been linked to epidemics of campylobacteriosis in high-income nations including Finland (Suominen *et al*., 2024), Canada (Guy *et al*., 2018), Norway (Hyllestad *et al*., 2020), Denmark (Boysen *et al*., 2014), and New Zealand (Gilpin *et al*., 2020).

There have also been reports of *Campylobacter* infections and outbreaks in both high- and low-income nations that are caused by milk. Milk consumption is associated with outbreaks of campylobacteriosis in a number of countries, including the United States (Jaakkonen *et al*., 2020), Sweden (Artursson *et al*., 2018), Italy (Bianchini *et al*., 2014), China (Li *et al*., 2020), Japan (Ohno *et al*., 2023), India (Modi *et al*., 2015), and the Netherlands (Heuvelink *et al*., 2009). Apart from that, other countries that have recorded campylobacteriosis outbreaks due to the consumption of poultry meat are Canada (Hodges *et al*., 2019), Australia (Keerthirathne *et al*., 2022), British Columbia (Hakeem and Lu, 2021), Belgium (Gellynck *et al*., 2008), Denmark (Kuhn *et al*., 2018), England (Royden *et al*., 2021), Germany (Stingl *et al*., 2012), Poland (Szosland-Fałtyn *et al*., 2018), New Zealand (Sears *et al*., 2011), Norway (Llarena *et al*., 2022), Madagascar (Randremanana *et al*., 2014), Kenya (Mbai *et al*., 2022), Indonesia (Yanestria *et al*., 2024b), Malawi (Mason *et al*., 2013), Iceland (Stern *et al*., 2003), Estonia (Tedersoo *et al*., 2022), Guatemala (Benoit *et al*., 2014), and Peru (Harvey et al., 2003).

Humans are susceptible to campylobacteriosis due to a wide range of risk factors. According to the most recent meta-analysis research, the main risk factor for campylobacteriosis is travel, which is followed by eating undercooked chicken, being exposed to the environment, and coming into close contact with livestock (Facciolà *et al*., 2017).

Reports from the United Kingdom indicate that the occurrence of campylobacteriosis is linked to a seasonal increase in fly populations throughout the summer because of high temperatures and rainy weather that promote fly development (Nichols, 2005). This leads to the flies coming into touch with animal and human feces, which supports the theory that environmental factors cause the seasonal epidemics that are common in the summer (Penakalapati *et al*., 2017). It was also discovered that both conventional and organic environmental factors were linked to campylobacteriosis. According to a different Danish study, human campylobacteriosis incidence rose in tandem with environmental temperatures, with the biggest rise in incidence occurring between 13°C and 20°C (Patrick *et al*., 2004).

Pathogenesis

Research on the pathogenesis of *Campylobacter* is curiously typically conducted on *C. jejuni*. *Campylobacter* has a complicated and poorly known survival strategy for getting past host barriers and producing sickness in humans. It is thought that 350–10,000 cells are necessary for a *Campylobacter* infection, and the infectious dose frequently corresponds with the severity of the disease (Janssen *et al*., 2008). Immunocompromised individuals, the elderly, and children are the most common groups to contract *Campylobacter* infections (El-Tras *et al*., 2015).

Campylobacter must cross the stomach's gastric acid barrier and the upper small intestine's bile duct's extremely alkaline secretions after consuming tainted food or drink (Facciolà *et al*., 2017). Pathogenic microorganisms like *Campylobacter* can survive and proliferate when the stomach acid barrier is disrupted. Thus, individuals who take proton pump inhibitors and antacids, or who have lowered stomach acidity, may be more susceptible to campylobacteriosis (Janssen *et al*., 2008).

Once *Campylobacter* enters the lower digestive tract, it targets the distal ileum and colon epithelial cells, causing severe inflammation and cell destruction; however, in chickens, the cecum is a crucial location for *Campylobacter* colonization (Pang *et al*., 2023). *C. jejuni* is the source of invasive inflammatory illness in affluent nations. However, *Campylobacter* is the cause of non-inflammatory watery diarrhea in underdeveloped nations (Coker *et al*., 2002). Chemotaxis and motility are thought to be necessary for *Campylobacter* host colonization, adhesion, and invasion (Elmi *et al*., 2021). These bacteria require iron uptake, bile salt and gastric acid resistance, and oxidative stress tolerance for growth and survival (Kreling *et al*., 2020). Toxins produced by bacteria mediate tissue damage and inflammatory reactions (Callahan *et al*., 2021).

Numerous parameters related to survival and virulence are thought to have a major role in the pathophysiology and induction of gastroenteritis caused by *Campylobacter* spp. It is thought that the clinical and epidemiological aspects of the illness have an impact on the molecular mechanisms of *Campylobacter* infection.

Virulence factors

The expression of virulence has been linked to a number of genes. Virulence genes implicated in colonization and adhesion include *rac*R, *fla*A (flagellin A gene), *dna*J, and *cad*F (adhesin gene) (Hermans *et al*., 2011). The genes that cause invasion are virulence plasmid genes (*cia*B, *vir*B11), and (invasion-associated gene A (*iam*A) (Kovács *et al*., 2020). Virulence genes implicated in the synthesis of lipopolysaccharides are β-1,3-galactosyltransferases-encoding genes (*cgt*B and *wla*N) (Guirado *et al*., 2020). Vital pathogenic virulence genes for the expression of cytotoxin synthesis are *cdt*C, *cdt*B, and *cdt*A, or cytoletal bloating toxins C, B, and A (Talukder *et al*., 2008). The adaptability of *Campylobacter* to unfavorable environments and the host's immune system seems to be one of the most crucial elements of intestinal colonization. Under the influence of chemoattractants, organisms enter the intestinal environment through fecal-oral transfer and populate the intestinal tract (Parker *et al*., 2020). Additionally, the proximal digestive system contains a number of antimicrobial proteins, including beta-defensin gallinacin-6 (van Dijk *et al*., 2007). There has been a great deal of research done on the virulence traits of *Campylobacter*, as these traits affect how pathogenic the species is. The ability of the *Campylobacter* genus, particularly its pathogenic species like *C. jejuni*, to live and tolerate physiological stressors contributes to its pathogenicity. These bacteria have a variety of virulence factors.

Chemotaxis

Campylobacter uses a process known as chemotaxis, which facilitates directed movement toward or away from chemical stimuli (effectors/chemo ligands, which can be either repellent or attractant) in the environment, to adapt to different environments (Kreling *et al*., 2020). Transducer-like protein (Tlps), which has a methyl acceptor domain, and core signal transduction protein make up the chemotaxis system (Clark *et al*., 2019). Ligands binding to Tlps send signals to cytoplasmic chemotaxis proteins, which in turn trigger a signal transduction cascade leading to directed movement of the flagella (Zautner *et al*., 2012). *Campylobacter* can more easily participate in substrate-specific chemotaxis when transducer-like proteins are present. This is crucial for the pathogen's capacity to adapt, grow its pathobiology, and invade the gastrointestinal tract (Chandrashekhar *et al*., 2017).

Flagellar motility

Campylobacter's motility is crucial to its survival in the digestive tract under a variety of circumstances. The mobility of flagella allows *Campylobacter* species to locate favorable environments within their hosts (Baldvinsson *et al*., 2014). The human foodborne pathogen *C. jejuni* produces two structural flagellins, FlaA and FlaG, that are heavily glycosylated by nature (Radomska *et al*., 2017).

Oxygen tension and oxidative stress defense

Campylobacter is able to withstand a number of adverse environmental conditions, such as pH fluctuations, oxygen restriction in the cecum, oxidative stress, elevated osmotic pressure, and the presence of digestive fluids, such as bile salts, from penetrating the digestive system (Kim *et al*., 2021). Peroxide resistance regulators and *Campylobacter* oxidative stress regulators control the expression of genes linked to oxidative stress resistance (Kim *et al*., 2015).

Bile resistance

Bile salt resistance is another requirement for *C. jejuni* to successfully colonize (Lin *et al*., 2003). Deoxycholate disrupts the lipid bilayer of the cell membrane and causes the unfolding and aggregation of proteins in the bacterial cytoplasm, killing detergent-like bile acids like cholate and bacteria (Talukdar *et al*., 2022).

Adhesion

Numerous adhesins produced by *Campylobacter*, both separately and in combination, have the ability to affect or mediate the attachment of bacteria to various cell structures and hosts (Kreling *et al*., 2020). The *Campylobacter* adhesion protein to fibronectin (CadF), a 37 kDa protein that binds to the fibronectin ligand present in epithelial cells and is expressed by the CadF gene, the adhesin that has been the subject of the greatest research (Konkel *et al*., 2020).

Invasion

In addition to their ability to survive intracellularly, several species of *Campylobacter* are known to secrete invasion antigens (Cia), such as CiaB, CiaC, and CiaI (Lopes *et al*., 2021). These virulence factors are essential for the bacteria's ability to infiltrate epithelial cells and populate the digestive system of their host (Callahan *et al*., 2021).

Cytolethal distending toxin

The cytolethal distention toxin is a toxin that functions similarly to DNase to generate DNA damage that stops cell division and starts apoptosis (Méndez-Olvera *et al*., 2016). Due to the toxin's parasitic nature, intestinal crypts are destroyed, which results in diarrhea.

Clinical symptoms

In humans

The clinical manifestations of human *C. jejuni* and *C. coli* enteritis are identical and cannot be distinguished from severe bacterial diarrhea brought on by other infections, such as *Salmonella* enteritis (Kemper and Hensel, 2023). A *Campylobacter* infection may result in fever, nausea, vomiting, and mild to severe bloody diarrhea (Tracz *et al*., 2005).

Stomach ache may reoccur and linger for up to seven days. In addition to more severe and persistent abdominal discomfort and occasionally blood or mucus in the stool, campylobacteriosis sickness might start with abdominal cramps, myalgia, diarrhea, chills, fever, headache, and occasionally delirium (Huayanay *et al*., 2020). Patients experience a lower percentage of extra-intestinal infections and chronic infectious problems. Less than 1% of patients with *C. jejuni* infection have been reported to have bacteremia (Gallo *et al*., 2016). Meningitis and endocarditis are uncommon signs of infection with *C. jejuni* (Tsoni *et al*., 2013). Rare cases of *C. jejuni* infections presenting as cystitis, acute cholecystitis, pancreatitis, and septic abortion have been reported (Vaughan-Shaw *et al*., 2010).

Additionally, a number of autoimmune illnesses, including GBS and RA, have been related to campylobacteriosis (Malik *et al*., 2022). It is expected that one occurrence of these two serious late-onset campylobacteriosis problems occurs for every 2000 infections (El-Saadony *et al*., 2023). As postinfectious polyneuropathy, *Campylobacter* infection is acknowledged as the most often identified antecedent event in GBS (40%–60% of all cases) (Poropatich *et al*., 2010). Acute inflammatory demyelinating polyradiculo-neuropathy is the major lesion, resulting in flaccid paralysis. About 1% of people with *Campylobacter* enteritis develop RA (Ajene *et al*., 2013).

In food and farm animals

Warm-blooded mammals and companion birds have *Campylobacter* spp. in their intestines as a component of their gut microbiota (Kreling *et al*., 2020). Many animal species are susceptible to enteritis, abortion, and infertility due to *Campylobacter* species (Dai *et al*., 2020). The role of *C. jejuni* as a major pathogen in livestock remains unclear. Enteritis is a common condition in dogs, cats, lambs, mink, calves, and certain types of laboratory animals caused by *C. jejuni* and occasionally *C. coli* (Facciolà *et al*., 2017). In young animals, the clinical indications could be more severe. Calves typically have thick, slimy diarrhea, sometimes with blood stains, and may or may not have a fever (Hansson *et al*., 2021). The enzootic abortion caused by *C. fetus* and *C. jejuni* can result in late-term abortion, stillbirth, and poor lambs (Wolf-Jäckel *et al*., 2020). In sheep, endometritis and occasionally even death can occur after infection (Sanad *et al*., 2014). In the event of an outbreak in sheep, morbidity could reach 90%, although it typically falls between 5% and 50%. Sheep morbidity can lead to delayed lambing and decreased milk yield (van den Brom *et al*., 2020). It is typical to recover with immunity against reinfection. Sheep may carry an infection for an extended period of time and continue to excrete bacteria (Ogden *et al*., 2009).

Diagnosis

Effective and rapid diagnosis of *Campylobacter* species infections in animal hosts is essential for both individual treatment and disease management at the farm level. Furthermore, prompt detection aids in the appropriate surveillance and monitoring of *Campylobacter* infections, which may be dangerous for human health due to zoonotic transmission.

Isolation and identification

A common procedure in *Campylobacter* species identification, which employs a range of commercially available media, is to enrich the sample in a suitable broth, like Bolton broth, and then isolate it by plating it on a particular medium, like modified cefoperazone deoxycholate charcoal medium (Bojanić *et al*., 2019). In research laboratories, a range of biochemical assays is typically conducted after bacterial separation, such as urease expression, oxidase and catalase production, nitrate and nitrite reduction, H_2S synthesis, and hydrolysis of indoxyl acetate and hippurate (Sadek *et al*., 2023).

Immunological tests

Campylobacter diagnosis can be achieved by a variety of enzyme immunoassay techniques, including flow cytometry, quantitative immunofluorescence, and enzyme-linked immunosorbent assays (ELISA). However, ELISA is the most effective method since it targets many particular antigens on the microorganism's surface (Ricke *et al*., 2019). It is possible to generate monoclonal and polyclonal antibodies to recognize particular pathogen epitopes. Moreover, antibodies can be altered, which frequently calls for the conjugation of several detection systems, like horseradish peroxidase, to boost the specificity and sensitivity of identifying various target epitopes (Hochel *et al*., 2007). It is significant to remember that immune-based detection methods can produce false-positive results even while they are somewhat sensitive to *Campylobacter* species. This has been noted when comparing conventional molecular and microbiological techniques with commercial equipment.

Molecular diagnosis

DNA or RNA sequences that are unique and extremely particular are found using technologies based on nucleic acids (Effendi *et al*., 2018; Wibisono *et al*., 2021). After that, these sequences can be separated, amplified, and shown on gel for molecular typing, identification, and quantitative analysis. DNA sequencing and polymerase chain reaction (PCR) provide quick, easy, and accurate identification of *Campylobacter* species while exposing their epidemiological traits (Yanestria *et al*., 2023). This approach also makes it possible for researchers to produce data that can be shared via web-based databases and utilized in phylogenetic analyses. This technology is known by two names, quantitative PCR (qPCR) and real-time PCR (Sails *et al*., 2003).

Differential diagnosis

The clinical manifestations of avian campylobacteriosis resemble those of other enteric infections, including rota virus, *Salmonella*, *Shigella*, *E. coli* 0157:H7, *Yersinia*, *Clostridium difficile*, and *Entamoeba histolytica* (Sadek *et al*., 2023).

Poultry

One of the primary ways that humans contract campylobacteriosis is through poultry, particularly from broiler chickens (Yanestria *et al*., 2024a). Furthermore, the chicken industry serves as a significant reservoir for additional *Campylobacter* species, including *C. jejuni*, *C. lari*, and *C. upsaliensis* (Skarp *et al*., 2016). Worldwide, the prevalence of campylobacteriosis and other *Campylobacter* infections is increased by both indigenous and imported broiler chickens (Carron *et al*., 2018). Additionally, *Campylobacter* can enter broiler chicken houses through vectors like flies, insects, or rodents, or it can travel through vehicles in the form of dust or aerosols. It can also be disseminated through excrement and be found everywhere in the environment, including surface water (Bahrndorff *et al*., 2013).

Domestic animals

In addition to a broad range of food products derived from animal groups, such as poultry, household animals (particularly cats and dogs) are the most common source of infection (Acke, 2018) and livestock are significant transmission vectors. *Campylobacter* bacteria can also be found in ruminant animals including cows, sheep, and goats (Hoque *et al*., 2021; Sanad *et al*., 2014; Cortés *et al*., 2006). Rather than in the rumen, *Campylobacter* bacteria are primarily located in the gut (duodenum, jejunum, and small and large intestine) (Chlebicz and Śliżewska, 2018). Therefore, there is a substantial danger of *Campylobacter* species transmission and spread when consuming meat from domestic animals or coming into touch with pets and companion animals.

Wildlife

Campylobacter species (particularly, *C. jejuni* and *C. coli*) are mostly commensals in birds, and they frequently invade the intestines in huge numbers (Hald *et al*., 2016). Wild animals can function as reservoirs in addition to acting as transmission vectors, such as wild birds (Olvera-Ramírez *et al*., 2023). It has been possible to isolate *C. jejuni* from wild birds, including pigeons, seagulls, geese, crows, ducks, and herons (Marotta *et al*., 2020). It is interesting to note that migrating birds have the ability to travel great distances and may provide novel genotypes of the *Campylobacter* species to various animals, including sheep, cattle, and broilers (Shad and Shad, 2019). Furthermore, wild birds congregate in metropolitan parks and recreation areas, particularly pigeons and crows. As a result, individuals with poor or neglected hygiene practices as well as children are more vulnerable to acquiring *Campylobacter* infections.

Water

According to a number of earlier studies, drinking water poses the greatest risk to the spread of campylobacteriosis (Ferrari *et al*., 2019; Whiley *et al*., 2013). The ability of *Campylobacter* to form a biofilm in the water pipes of broiler chicken houses contributes to the species' increased growth and survival and is a key risk factor for colonization in chicken flocks (Pokhrel *et al*., 2022). Recent research conducted in Ireland found that on seven out of twenty farms, there were serious issues with getting rid of *Campylobacter* species colonies in water pipes following disinfection (Battersby *et al*., 2017). More people than any other documented enteric disease are at risk of contracting campylobacteriosis when they drink water from private wells as opposed to municipal surface water systems (Galanis *et al*., 2014). Another factor in the spread of *Campylobacter* is contaminated pet waste and wild bird droppings that pollute outdoor water sources (Pitkänen, 2013).

Other sources

Person-to-person transmission, whether by fomites or faecal-oral routes, is uncommon but has great potential and a propensity to spread. There is a documented 3% person-to-person transmission of campylobacteriosis in the United Kingdom (Wensley *et al*., 2020). The ingestion of raw milk derived from dairy cows has also been linked to numerous cases of campylobacteriosis outbreaks (An *et al*., 2018). Several other *Campylobacter* species, such as *C. hyointestinalis*, *C. fetus*, and *C. ureolyticus*, can also be found in unpasteurized milk (Koziel *et al*., 2012; Koziel *et al*., 2014; Grouteau *et al*., 2023). A genomic study of the milk revealed a considerable level of fecal contamination. *Campylobacter* infections can also be dangerously transmitted by insects such as flies, roaming animals, and chicken products (Royden *et al*., 2016; Frosth *et al*., 2020).

Treatment

The best antibiotic medications for eradicating and decreasing *Campylobacter* in feces are erythromycin, clarithromycin, or azithromycin (Wieczorek and Osek, 2013). If therapy was initiated prior to the third day after the onset of symptoms, there was a noticeable improvement. Research on children with *C. jejuni*-caused diarrhea revealed that early erythromycin treatment led to a significant improvement in symptoms (Munoz *et al*., 2022). Clinical experience and controlled research demonstrate that patients with high fever, bloody stools, diarrhea, and more than eight evacuations per day respond better to antibiotic treatment (Eiland and Jenkins, 2008).

Despite the lack of animal clinical efficacy trials, it has been reported that dogs and cats can recover from this illness when given erythromycin, the recommended medication for humans, three times a day for seven days at a dose of 11.1 to 15.5 mg/kg (Gibreel and Taylor, 2006). There is currently discussion about *Campylobacter* resistance to widely used antimicrobials in both veterinary and human situations.

In the past 20 years, there has been a noticeable rise in the amount of antibiotic resistance in bacteria isolated from humans, particularly to fluoroquinolones (Luangtongkum *et al*., 2009). This high level of resistance is still present today in many parts of the world. The first-choice medications for treating adult campylobacteriosis are those in the fluoroquinolone class (Portes *et al*., 2023). Global reports relate the approval of the use of these antimicrobials in poultry production to *Campylobacter* sp. infections that are resistant to fluoroquinolones (Zhao *et al*., 2010).

Rastall (2004) looked into alternate sources to antibiotics for the treatment of campylobacteriosis and discovered that probiotics (*Lactobacillus acidophilus*) combined with prebiotics (fructooligosaccharides and lactosucrose) had a positive impact on the balance of intestinal microflora, improved immune status, and aided in the recovery from campylobacteriosis infection. Following the usage of probiotics, pathogens are competitively excluded, resulting in this microflora balance.

Antibiotic resistance

In the US, 1% of *C. jejuni* strain isolates from infections in humans in 2010 had erythromycin resistance, compared to 43% for tetracycline and 22% for ciprofloxacin (Rodrigues *et al*., 2021). Almost the same data were reported in the same year about *C. jejuni* strain isolates from chicken flesh. In 2010, strains of *C. jejuni* recovered from chicken flesh in the European Union showed resistance to erythromycin in 2% of cases, tetracyclines in 21%, and fluoroquinolones in 52% of cases (Marotta *et al*., 2023). Antibiotic resistance in *C. coli* is higher than in *C. jejuni* in the US and Europe. The frequent use of veterinary antibiotics (enrofloxacin and danofloxacin) in the pharmacological treatment of chickens appears to be the root cause of the high level of resistance to fluoroquinolones (Cox and Popken, 2006). Consequently, the use of fluoroquinolones in chickens was outlawed in the US in 2005 (Price *et al*., 2007). Resistance to these antibiotics is uncommon in Australia, where the use of fluoroquinolones has not been allowed (Cheng *et al*., 2012). It is well recognized that travel, in both industrialized and developing nations, is often linked to fluoroquinolone-resistant illnesses. According to a recent study by Ghunaim *et al*. (2015), *C. jejuni*'s erythromycin resistance was comparatively low, accounting for just 8.6% of isolates, while ciprofloxacin resistance accounted for 63.2% of isolates. There have also been reports of high ciprofloxacin resistance in the United Arab Emirates, where 85.4% of isolates exhibited resistance (Sonnevend *et al*., 2006). Published ciprofloxacin resistance rates were 2% in Australia (Bell *et al*., 2022) and 40% lower in Poland (Jeżak and Kozajda, 2022).

Azithromycin, a macrolide, is typically the next line of treatment when resistance to these medications is established, yet resistance to this class of antibiotics has also been documented (Dai *et al*., 2020). The use of antibiotics indiscriminately at different points in the chicken production chain can contribute to the establishment of resistant forms of campylobacteriosis since the disease is spread through food consumption (Epps *et al*., 2013). Therapeutic options are limited due to the emergence of strains that are resistant to various medicines, including the antibiotics of choice for humans.

Control

The best strategy to reduce the number of human infections caused by *Campylobacter* is to restrict the amount of contamination of the poultry flock and its products, even if the majority of infections are contracted through the handling or consumption of chicken (Newell *et al*., 2011). A common way to stop *Campylobacter* infection outbreaks is to steer clear of unpasteurized milk (Facciolà *et al*., 2017; Tyasningsih *et al*., 2022). It is important to stress that those who are pregnant, have young children, are elderly, have weakened immune systems, are visiting low- or middle-income countries, or are campers should not drink unclean water (Asuming-Bediako *et al*., 2019).

Farm

Both domestic and wild animals, as well as the environment, frequently contract campylobacteriosis. As a result, it is critical to lessen the amount of contamination from these sources in chicken coops. It has been shown that using hygiene barriers at poultry house's entrances, limiting the access of livestock workers, offering hand washing stations, putting in place boot dips, and using specific house boots and overshoes, are all beneficial practices (Wagenaar *et al*., 2006). One efficient way to stop the spread of contamination in slaughterhouses is to segregate *Campylobacter*-positive animals from negative animals and to kill *Campylobacter*-positive animals (Taha-Abdelaziz *et al*., 2023). It has been demonstrated that more frequent emptying and cleaning of water troughs lowers the chance of *Campylobacter* colonization in animals (Ellis-Iversen *et al*., 2009).

Food animals processing

Large concentrations of *Campylobacter* are found in the digestive tracts of chickens, and these bacteria have the ability to contaminate food products, the environment, and slaughterhouses (Kreling *et al*., 2020). At this stage, the amount of *Campylobacter* organisms is decreased by treatment with organic acids, UV radiation, and chemical dipping tanks for corpses (Szott *et al*., 2022). Reducing the amount of bacteria in the kitchen through heating, freezing, or radiation eliminates pathogens and lowers the degree of cross-contamination (Taha-Abdelaziz *et al*., 2023).

Home

The prevention of *Campylobacter* species infection depends on proper food preparation and consumption practices. It is possible to guarantee that temperatures high enough to destroy *Campylobacter* species organisms are reached by using a meat thermometer and properly cooking the meat (de Jong *et al*., 2012). After handling raw poultry or other meats, cutting boards and cutlery should be cleaned in hot, soapy water and used again to prepare other raw meals (Luber *et al*., 2006). Hand washing and the separation of raw and ready-to-eat food must be put into practice. Individuals experiencing acute diarrheal illness ought to refrain from handling and preparing food until the condition has resolved (Hatchette and Farina, 2011). Eating raw meat, unpasteurized dairy products and exposure to animals such as pets suffering from diarrhea should be avoided (Facciolà *et al*., 2017). Prior to eating, everyone should wash their hands, especially if they are handling pets or other animals (Taha-Abdelaziz *et al*., 2023).

Vaccination

The most effective control strategy is generally thought to be vaccination against *Campylobacter*. Numerous field and experimental investigations on sheep and experimental research on guinea pigs have shown the efficacy of vaccination (Burrough *et al*., 2010). However, there is limited cross-protection between strains or species, numerous strains of a species may be involved in the disease, and abortion can be induced by two or more separate species (*C. fetus*, *C. jejuni*, and *C. coli*) (Costa and Iraola, 2019). These factors make immunization complex. Therefore, even after using polyvalent vaccinations, vaccination may not offer full protection.

Conclusion

The primary cause of the infectious disease, campylobacteriosis, is the *Campylobacter* species. *Campylobacter* is an emerging global foodborne bacterial pathogen attributed to the cause of foodborne diseases and death, resulting in enormous social and economic losses. *Campylobacteriosis* is particularly prevalent in the paediatric and elderly population. This illness is zoonotic, which means that eating certain foods can spread it from animals to people.

Campylobacter has been isolated from farm animals and foods of animal origin. Worldwide, poultry and their by-products remain the major cause of foodborne illnesses, especially campylobacteriosis. The prevention of *Campylobacter* species infection depends on proper food preparation and consumption practices. The consumption of undercooked poultry products and the contamination of carcasses and other food products continue to be an important public health concern. More challenging is the increasing incidence, emergence, and spread of antibiotic-resistant *Campylobacter* species which has further complicated and limited treatment options. In order to curtail and avert this challenging public health problem, it is imperative to establish strong measures that are directed towards its eradication, especially good hygienic practices during food handling. Efforts to curtail *Campylobacter* contamination in the food industry also need to become a more determined priority. To comprehensively understand the disease burden of campylobacteriosis, more research studies with multiple intervention within the "one health" approach are needed to evaluate the under-reporting of diarrhoea incidents at national and intercontinental levels by enhancing and encouraging local capacities for continuous disease surveillance and monitoring. Also, more research that will devise alternative strategies or new therapeutics against campylobacteriosis will also greatly help in turning the tide against this emerging foodborne and zoonotic disease.

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Authors' contributions

ARK, IBM, and MKJK drafted the manuscript. SRA, MHE, FNAEPD, and SMY revised and edited the manuscript. OSMS, IF, AHF, and KHPR took part in preparing and critically checking this manuscript. AH, KAF, RR, and SA edited the references. All authors read and approved the final manuscript.

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Conflict of interest

The authors declare that there is no conflict of interest.

Data availability

All references are open access, so data can be obtained from the online web.

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