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Research Article

Campylobacter jejuni: a Comprehensive Review of a Leading Foodborne Zoonotic Pathogen

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Abstract
Campylobacter jejuni is the main bacterial cause of gastroenteritis around the world and poses a major public health challenge. This bacterium lives in the digestive tracts of many animals and is a key zoonotic agent. Poultry are the main source, but cattle, sheep, wild birds, and pets also play a role in spreading it to the environment and people. People usually get infected through the fecal-oral route, most often by eating undercooked poultry, drinking unpasteurized milk, consuming contaminated water, or having direct contact with infected animals. Symptoms of *C. jejuni* infection can be mild or lead to serious long-term effects, such as Guillain–Barré syndrome. Because it spreads between animals and humans, *C. jejuni* shows how animal health, food safety, and human health are closely linked. This review brings together current knowledge about *C. jejuni*, focusing on its impact on public health, its microbiology, how it causes disease, how it interacts with hosts, and the growing issue of antimicrobial resistance (AMR). It pays special attention to how the bacteria persist, including forming biofilms, adapting their metabolism, and changing their characteristics to survive in animals, food processing environments, and the outside world. The increase in multidrug-resistant strains, often due to antibiotic use in food animals, highlights the urgent need for control strategies that consider animal, human, and environmental health together. The review also looks at advances in genomics and new ways to prevent and treat infection, such as vaccines and alternative antimicrobials, as important tools to help reduce the global impact of campylobacteriosis.

Keywords: *Campylobacter jejuni*, zoonosis, poultry, gastroenteritis, one Health.

Introduction

Campylobacter, especially *Campylobacter jejuni* and *Campylobacter coli*, causes over 95% of human campylobacteriosis cases [1,2]. Each year, *Campylobacter* infections are thought to cause about 400 to 500 million cases of diarrhea, affecting people in both wealthy and low and middle-income countries (LMICs) [2]. The impact is greatest among children under five in LMICs, where *Campylobacter* is now seen as a major cause of pediatric diarrhea. In Bangladesh, *C. jejuni* is linked to up to 25.5% of diarrhea cases in children [3]. Ahmed et al. [4] gathered statistics from regions such as North America, South America, Asia, Oceania, and parts of Europe and the Middle East. Their report included infection rates per 100,000 people, the percentage of diarrhea or gastroenteritis cases, and hospitalization data over different time periods. In North America, Canada had high infection rates (28,521 cases per 100,000 from the year1990 to 2004), while Mexico (15.7% of infant cases in 2006-2007) and the Caribbean (1,288 children per 100,000 in 2008-2012) also saw significant numbers of acute gastroenteritis caused by *Campylobacter* [4]. In South America, the disease burden varies, with moderate rates in the general population and high rates among hospitalized patients in countries like Argentina (4.6-30%) and Bolivia (4.4-10.5%) [5]. Figure 1 shows different patterns in Bangladesh, India, and China, which

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may be due to differences in surveillance and healthcare access across Asia [5,6,7]. In Oceania, Australia and New Zealand reported occasional campylobacteriosis linked to eating poultry between 1990 and 2017 [8]. Overall, *Campylobacter* infections are found worldwide, but the rates differ by region, likely because of differences in surveillance, public health systems, and reporting.

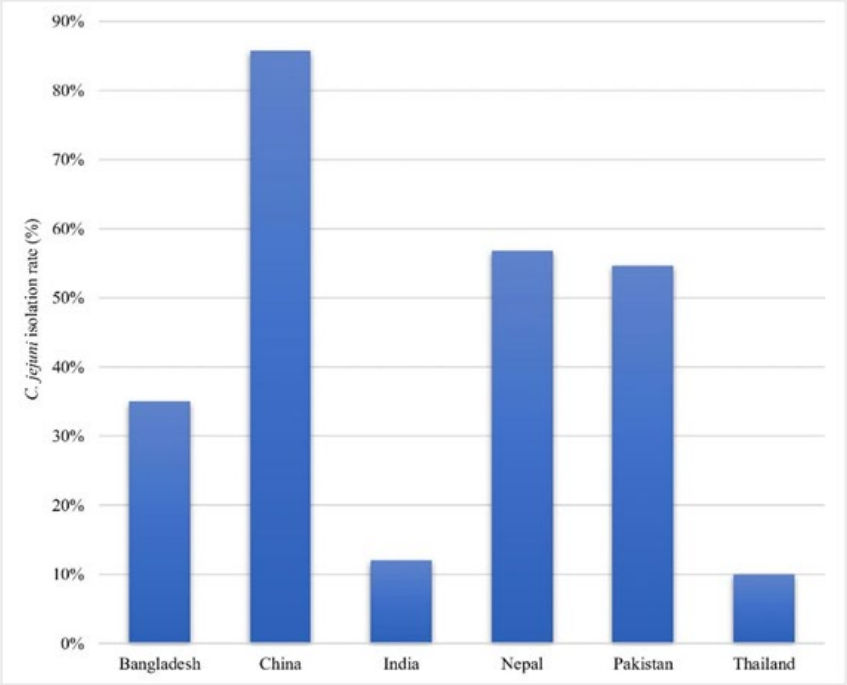

Figure 1. Infection rate of *C. jejuni* in some Asian countries [5,6,7].

Although campylobacteriosis has a low case-fatality rate of about 0.04%, it still leads to many hospitalizations and adds to healthcare costs. *C. jejuni* infection is known to cause Guillain-Barré syndrome (3.25 cases per 100,000 children), a serious immune-related neurological disorder that causes sudden muscle weakness and paralysis [3,9]. *C. jejuni* remains widespread because it can infect many hosts, adapt to different environments, spread through various routes between humans, animals, and the environment, and because antimicrobial resistance is increasing. Together, these issues make campylobacteriosis a major One Health concern.

This review brings together the current scientific knowledge about *C. jejuni*, a major cause of bacterial gastroenteritis and a growing foodborne threat. It focuses on the biological and environmental factors that help this microbe persist, such as its metabolic flexibility, ability to form biofilms, and survival in the viable but non-culturable (VBNC) state. The review also discusses the problem of antimicrobial resistance. It considers new control strategies, including vaccines, natural antimicrobials, and genomic diagnostics, all within the One Health approach to reduce the disease's impact on people and animals.

Material and methods

Literature search and ethical Considerations

This review exclusively analyzed publicly available, published literature and did not involve any human/animal experimentation. The approval was given under reference number SUB/MBO/Clin and Med/11, dated January 15, 2026. PRISMA 2020 guidelines were followed for the article search strategy [10]. All references were imported into Zotero reference management software for better organization.

Sampling Strategy and Rationale

The literature review used PubMed, Scopus, and Web of Science to find relevant studies. Key works published from 2018 to 2025 on epidemiology, pathogenesis, antimicrobial resistance, and global burden of *Campylobacter jejuni* were given priority.

Inclusion

The review included peer-reviewed original research on alternative approaches, as well as

studies on epidemiology, pathogenesis, antimicrobial resistance, and global burden done in vitro or in vivo**.** Clinical trials (Phases I–III) published in English from 2018 to 2025 were also included.

Exclusion

Non-English publications without validated translations, conference abstracts, patents, and non-peer-reviewed commentaries were excluded. Studies focusing exclusively on the mechanisms of epidemiology, pathogenesis, antimicrobial resistance, and global burden were also omitted. Details in this review are available within the cited references. No original datasets were generated.

A total of 800 peer-reviewed papers were found during the literature search. Among them, 72 met the eligibility criteria. A PRISMA flowchart is shown in Figure 2.

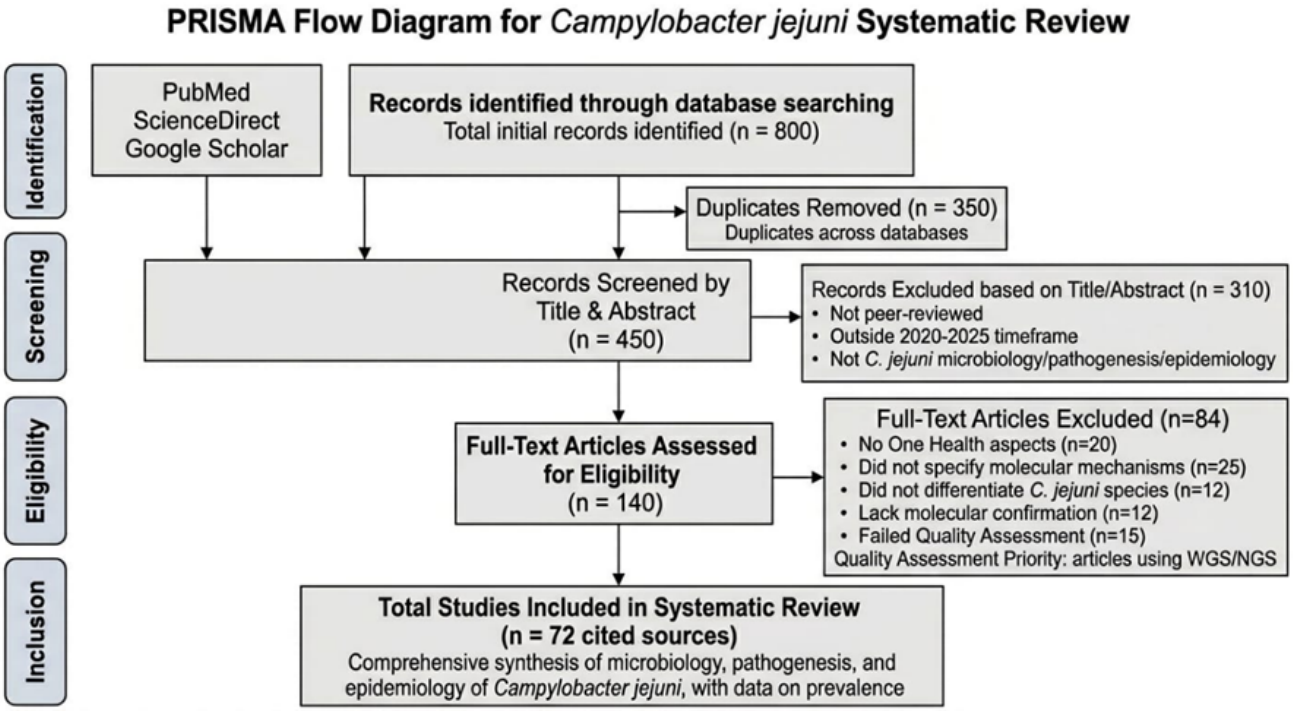

Figure 2. PRISMA flow chart describing the selection criteria of 72 added references

Results

Campylobacter jejuni is a gram-negative bacterium that needs low oxygen to grow, does not form spores, and prefers environments with extra carbon dioxide. It belongs to the Proteobacteria group. Its helical shape and flagellum help it move like a corkscrew, letting it get through the thick mucus in the gut. This movement helps it settle in hosts and cause illness. When stressed, *C. jejuni* can change shape to become round or long and thin. These forms are linked to a dormant or viable but non-culturable (VBNC) state, where the bacteria are alive but cannot be grown in standard lab tests [11]. The VBNC state helps the bacteria survive harsh conditions and makes them harder to find in studies [11,12]. *C. jejuni* grows best in low-oxygen environments with about 5% oxygen and 10% carbon dioxide, similar to the intestines of warm-blooded animals [13].

Host-microbiome interaction of *C. Jejuni*

Gut microbiota plays a key role in defending host by producing metabolites. These metabolites helps to develope the mucus layer, epithelial barrier, lamina propria, and immune cells. Thus, they help to keep the barrier strong and stop pathogens from invading [14]. The balance of these metabolites and how they interact with host receptors is important for immune stability and gut health. Changes in diet, such as adding growth promoters or plant-based compounds, can shift metabolite profiles and lead to major changes in immune responses and gut health in poultry [14]. They improve the intestinal barrier by sticking to enterocytes and helping the mucosal layer grow. When microbial diversity drops, the host becomes more likely to get infections. For example, when there are fewer types of microbes, *C. jejuni* can cause more severe disease, shown by more

mucus-producing cells in birds with less gut microbiota [14].

New technologies like high-throughput sequencing have greatly improved our understanding of gut microbial communities. Unlike older culture-based methods, these tools reveal complex links, such as the connection between higher *Campylobacter* levels and more *Clostridium* and *Lachnospiraceae* in the gut [15]. Still, many studies only look at certain gut regions, even though evidence shows that interactions throughout the digestive tract affect *Campylobacter* colonization, especially in the lower intestine. Studying both upper and lower gut segments, as well as fecal samples, gives a complete picture. Animal models, like secondary abiotic IL-10$^{-/-}$ mice, are useful for studying how the host, microbiota, and pathogens interact. These studies show that the makeup of the microbiota is key to how likely an animal is to get *C. jejuni*-induced enterocolitis [16]. The microbiota protects the host in several ways, such as outcompeting harmful microbes, making short-chain fatty acids, lowering gut pH, restoring balance after disruptions, strengthening the mucosal barrier, increasing mucin production, and adjusting immune responses [18].

Gut infections from *Salmonella* or *Campylobacter* can raise the risk of developing inflammatory bowel disease. This risk can also be influenced by genetic factors in the host, like *TLR4* polymorphisms, which may make some people more likely to get infections from gram-negative bacteria [19].

Reservoirs and transmission

C. jejuni is found in the digestive systems of many birds and mammals, but it usually does not make them sick. Poultry, especially broiler chickens, are the main source, with over 60-80% of commercial flocks carrying the bacteria in their intestines [20]. During processing steps like slaughter, defeathering, and evisceration, the bacteria can spread from poultry carcasses. Handling poultry the wrong way or eating undercooked poultry meat is a leading cause of human campylobacteriosis in both cities and rural areas [20,21]. In rural and low and middle-income countries, backyard poultry and waterfowl farming, such as raising ducks and geese, are also important but often overlooked ways in which bacteria spread. Frequent contact between people and animals, along with poor hygiene and weak biosecurity, increases the risk of direct transmission and contamination at home [22,23]. The European Centre for Disease Prevention and Control (ECDC) reported 14,216 suspected cases of campylobacteriosis in 2022 (Figure 3). Most cases were linked to broiler meat (42.7%), while the smallest share (10.2%) came from other mixed contaminated food sources [24].

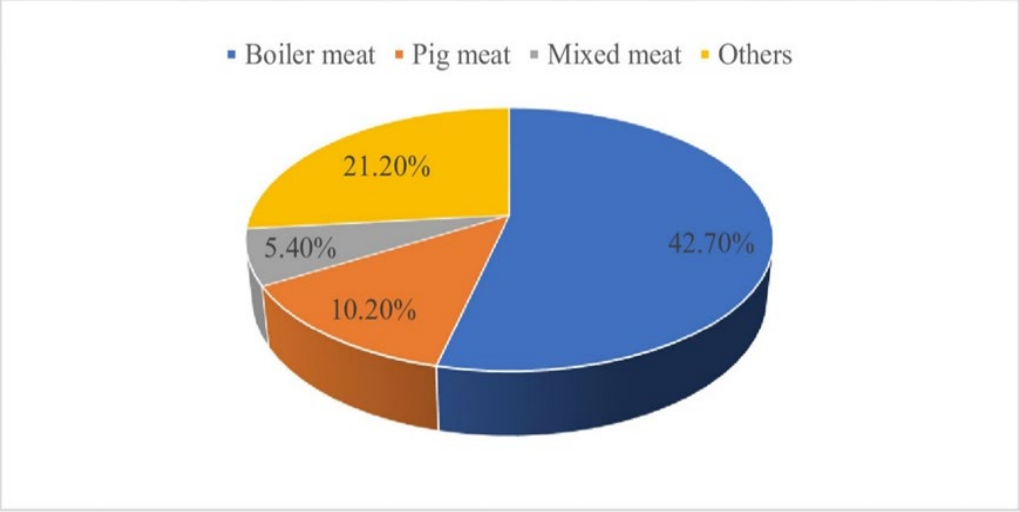

Figure 3. Suspected vehicles of infection for Campylobacteriosis [15].

Cattle and sheep are important reservoirs for *C. jejuni*, which is often found in their feces, raw milk, and on farms. Studies show that ruminants can cause up to 21% of human infections in some areas, showing their role goes beyond just poultry transmission [20]. Pets, especially young ones living closely with people, have been linked to up to 25% of human cases in some studies [24].

Figure 4 shows the environmental transmission of *C.jejuni* into human body. The fast growth of the poultry industry, now producing over 150 million tons a year, also raises the risk of human exposure from farm to table. These results show the need for One Health strategies that monitor and control animal sources, food systems, and human behavior to reduce the global impact of *C. jejuni.*

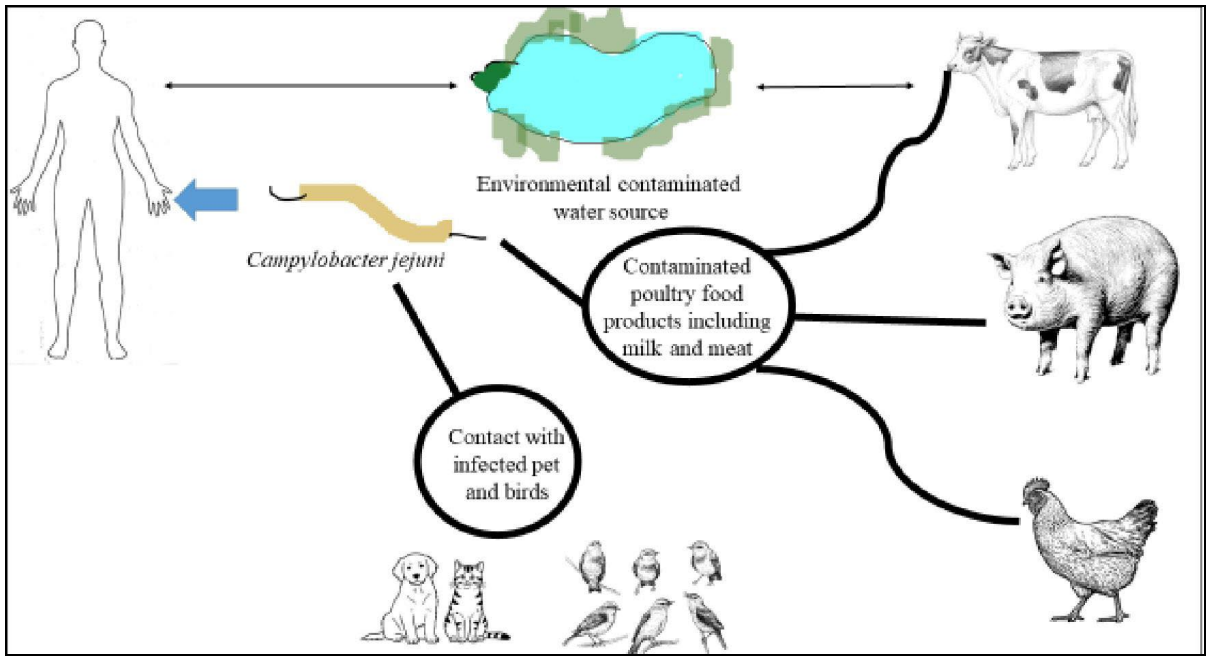

Figure 4. Environmental transmission of *C. jejuni* into the human body

Molecular epidemiology and evolution

Whole genome sequencing (WGS) and multilocus sequence typing (MLST) are detailed methods used to study the genetics and population biology of *C. Jejuni* [25]. These tools show that *C. jejuni* is highly diverse because it mutates, recombines, and adapts to many hosts and environments. Researchers have found over 97 different sequence types (STs) even in small samples, showing how varied the population is. Still, some clonal complexes like ST-21, ST-353, and ST-443 are found often and are important because they are common and linked to human disease [26,27]. *C. jejuni* cases rise in summer when higher temperatures lead to more food contamination. For example, genotypes like ST-2993 and ST-4526 were found in a large Guillain-Barré Syndrome (GBS) outbreak in Peru [28].

Molecular clock studies using genome-wide SNP data estimate that *C. jejuni* changes at a rate of about 3.4×10^{-6} substitutions per site each year [29]. This fast evolution helps researchers track how the bacteria spread, trace sources, and study their history. It also helps map how infections move from animals to people and spot new strains that may be more harmful or better adapted to certain hosts [29]. For example, ST-61 is found mainly in cattle, while ST-45 is linked to chickens or birds [30].

Pathogenesis and host-pathogen interactions

Colonization and virulence factors

C. jejuni begins colonization by moving with the help of its bipolar, sheathed flagella (Figure 5). Its helical or curved shape helps it move in a corkscrew motion, allowing it to get through the thick mucus covering the intestinal lining [31]. This movement is essential for colonizing the intestine, since bacteria that cannot move or lack flagella have trouble surviving in the host. The bacterium's movement is guided by a chemotaxis system, which uses a two-part signaling network with CheA, a membrane protein, and CheY, a protein in the cell [32]. CheV and CheW help connect these signals, allowing the bacterium to sense and react to its environment [20]. By using chemotaxis, *C. jejuni* moves toward nutrients and away from harmful conditions, which helps it survive and colonize efficiently [31-35].

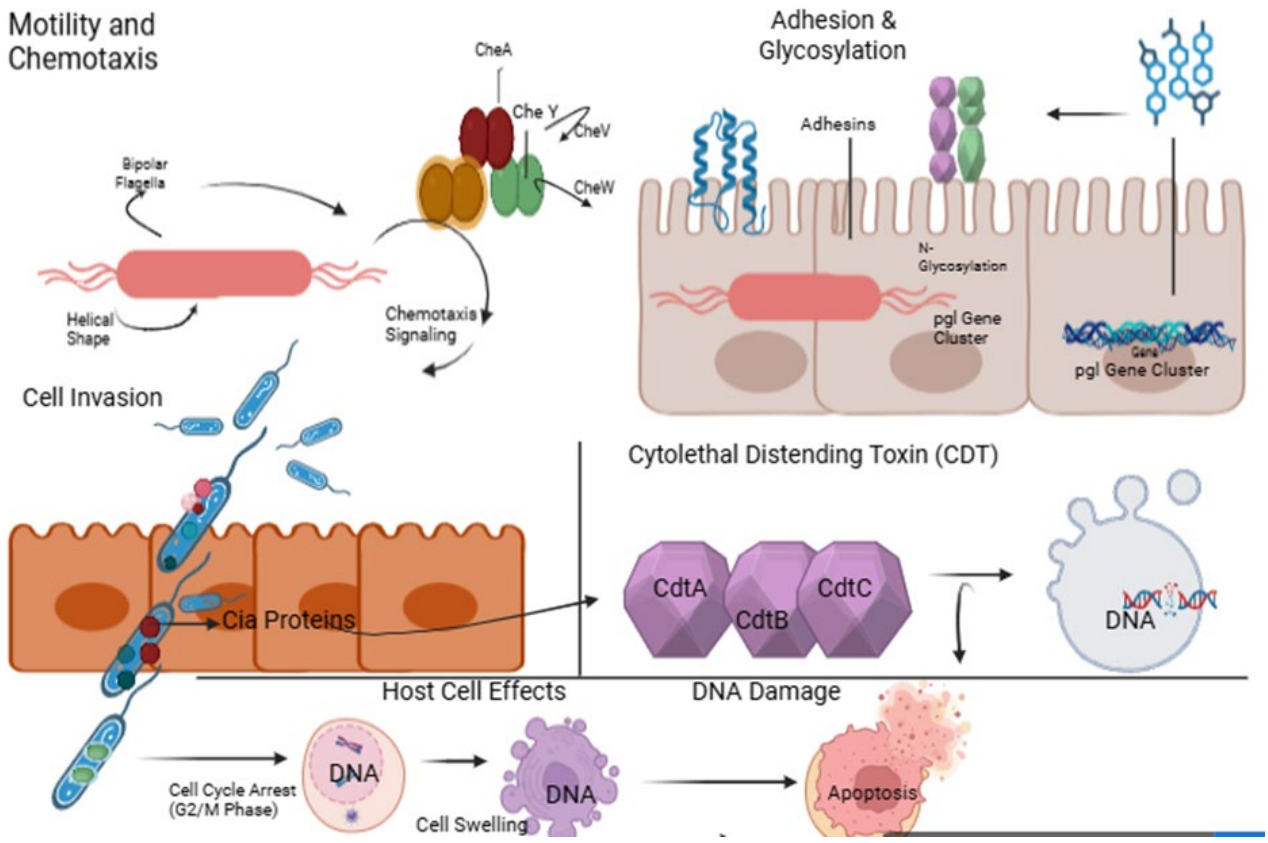

Figure 5. Virulence & pathogenesis of *Campylobacter* sp.

After *C. jejuni* first colonizes the intestine, it must stick to and invade the cells lining the gut. This process depends on the N-linked protein glycosylation system, which is controlled by the *pgl* gene cluster [36]. This system makes a heptasaccharide that attaches to asparagine on more than 75 proteins found on the cell surface and in the periplasm, including important adhesins and colonization factors. N-glycosylation is needed for protein stability, interaction with the host, and avoiding the immune system [12]. Bacteria without this system have a much harder time colonizing birds and cannot stick to or invade human intestinal cells as well [36-40].

Additional virulence factors contribute to host cell damage and disease progression. The cytolethal virulence factors also play a role in damaging host cells and causing disease [39]. The cytolethal distending toxin (CDT) is made up of three parts: CdtA, CdtB, and CdtC. This toxin causes double-strand breaks in the DNA of host cells. CdtA and CdtC help the toxin attach to the cell and allow CdtB to enter the nucleus, where it breaks down DNA [20]. Figure 5 summarizes the chemotaxis process. This leads to the cell stopping its cycle at the G2/M phase, swelling, and eventually dying.

C. jejuni also makes several *Campylobacter* invasion antigens (Cia proteins), which are released through a secretion system linked to the flagellum [38]. These proteins change host cell signaling, help the bacteria get inside cells, and support their survival inside the host, making *C. jejuni* more effective at causing infection.

Metabolism and nutrient acquisition

C. jejuni is a non-saccharolytic bacterium, so it cannot use common hexose sugars like glucose for energy. Its Embden-Meyerhof-Parnas (EMP) glycolytic pathway is incomplete because it lacks phosphofructokinase, the enzyme needed to convert fructose-6-phosphate to fructose-1,6-bisphosphate [11,41]. Because of this, *C. jejuni* mainly uses amino acids as energy sources. Serine is turned into pyruvate by L-serine dehydratase (SdaA), aspartate becomes fumarate through aspartase (AspA), and glutamate is also changed into fumarate [36]. Intermediates from the tricarboxylic acid (TCA) cycle, such as succinate, fumarate, and pyruvate, are important carbon and energy sources. *C. jejuni* shows metabolic flexibility with a branched electron transport chain, which lets it use several electron donors like formate and hydrogen that are

common in the intestine. When oxygen is limited, the bacterium can use other terminal electron acceptors, including fumarate, nitrate, and trimethylamine N-oxide [42]. This flexibility in respiration helps *C. jejuni* generate energy and survive in different host environments.

C. jejuni needs certain nutrients, which allows it to specialize in the gastrointestinal tract. There, it competes with the host and other microbes for limited resources. Some strains have a unique way to use L-fucose without phosphorylation, which may help them colonize more effectively and gain an advantage in the gut [43].

Immune response and inflammatory damage

The symptoms of campylobacteriosis mostly come from the body's immune and inflammatory responses, not from direct damage by the bacteria. When *C. jejuni* infects the gut, the innate immune system recognizes its components, which causes intestinal and immune cells to release pro-inflammatory cytokines like interleukin (IL)-1β, IL-6, IL-8, and interferon-γ (IFN-γ) [44]. IL-8 is especially important because it attracts neutrophils to the infection site. As neutrophils build up and become active, they cause injury to the intestinal lining, increase gut permeability, and lead to inflammatory diarrhea [45].

The adaptive immune response is also important for fighting infection. $CD4^+$ T-helper cells, especially the Th1 and Th17 types, help clear bacteria by making cytokines that boost macrophage activity and keep neutrophil responses going [46]. Secretory immunoglobulin A (IgA) helps by stopping bacteria from attaching to the gut lining. Protection against *C. jejuni* relies on antibody-mediated, innate, and cell-mediated immune responses working together [45]. People with weakened immune systems are at higher risk for severe or long-lasting illness, which shows how important innate and T-cell immunity are in defending against *C. Jejuni* [46].

Survival mechanisms: persistence in hosts and the environment

Biofilm formation

C. jejuni forms biofilms through several steps controlled by how it senses and responds to its environment. The bacteria first attach to surfaces using flagella, outer membrane proteins, and surface polysaccharides, which helps them stick to things like stomach lining, chicken skin, water pipes, and food processing equipment. Once attached, the cells group together into microcolonies by interacting with each other and making extracellular polymeric substances (EPS). The EPS matrix, made up of polysaccharides, proteins, extracellular DNA, and lipids, gives the biofilm structure and acts as a barrier that limits the entry of antimicrobial agents and disinfectants [41]. Figure 5 shows the biofilm forming mechanism.

Environmental factors like oxygen levels, temperature, nutrients, and oxidative stress play a big role in how biofilms develop. When exposed to more oxygen, *C. jejuni* turns on stress response genes and changes its metabolism, which helps it form biofilms as a way to protect itself [47]. Inside biofilms, some cells slow down their respiration and become more varied in their metabolism, creating groups that can better handle stress. Cells in biofilms also change which genes they express, lowering those for movement and increasing those for stress defense and efflux. These changes help the bacteria survive longer in food-processing environments and can lead to contamination of the food supply [48].

Stress Responses and the VBNC state

C. jejuni enters the viable but non-culturable (VBNC) state as a way to cope with ongoing environmental stress [20]. Conditions such as exposure to oxygen, lack of nutrients, low temperatures, or changes in salt levels cause oxidative stress and use up the cell's energy, leading to major changes. In this state, the cells often change from a spiral to a coccoid shape, which reduces their surface area and lowers their metabolic needs [20]. On a molecular level, VBNC cells keep up basic metabolic activity, maintain their membranes, and produce small amounts of ATP. Stress response regulators and antioxidant systems help control reactive oxygen species, while DNA and protein repair systems stay partly active [35]. However, these cells have much lower transcription and translation, so they cannot form colonies on standard media. Importantly, VBNC cells still have virulence genes and can become active again when conditions improve, such as when they enter a suitable host. This ability to recover makes the VBNC state important for environmental survival, underestimating pathogen numbers, and causing unexpected disease.

Persister cell formation

Persister cell formation in *C. jejuni* is a survival strategy based on metabolic dormancy, not genetic resistance. Persister cells can appear randomly or in response to antibiotics and environmental stress [20]. These cells temporarily slow down their respiration, biosynthesis, and energy production. This drop in activity makes antibiotics that target active processes, like DNA replication, protein synthesis, and cell wall biosynthesis, less effective.

Persister cells form when changes in the electron transport chain and redox balance lower membrane hyperpolarization and help protect against antibiotics [49]. Toxin-antitoxin systems, stress response networks, and energy loss

pathways might also help create persisters. However, these processes are less understood in *C. jejuni* than in other bacteria. When antibiotics are gone, persister cells can quickly become active and multiply, which can cause treatment failure, longer infections, and recurring disease [50].

Antimicrobial resistance in Campylobacter: an escalating public health crisis

Antimicrobial resistance (AMR) in *Campylobacter* species develops through selective pressure, genetic changes, and strong survival tactics [51]. The widespread use of antimicrobials in medicine, livestock, and poultry has sped up the rise of resistant strains. *C. jejuni* adapts well because of its flexible genetics and ability to survive in many environments. AMR in *Campylobacter*, especially resistance to fluoroquinolones and macrolides-the main treatments for severe campylobacteriosis, is a serious public health issue. Resistance rates are high worldwide. For instance, studies show fluoroquinolone resistance in 60.6% of *C. jejuni* and 64.7% of *C. coli* from ruminants, with other antimicrobial resistance found in 38.5% and 76.5% of these isolates [51]. Antimicrobial resistance makes treatment less effective and helps bacteria survive in places with high antimicrobial use. Resistant strains can last a long time in animals and food-processing systems, increasing the risk of spreading to people. Resistance, biofilm formation, the viable but non-culturable (VBNC) state, and persister cell survival all work together to make these bacteria hard to eliminate [50]. These problems show why we urgently need coordinated surveillance and control efforts in human, animal, and environmental health.

Antimicrobial resistance in *C. jejuni* happens through changes like target modification, breaking down antibiotics, lowering drug levels inside the cell, and protective cell responses [52]. Fluoroquinolone resistance usually comes from a single change (C257T) in the *gyrA* gene, which causes a Thr86Ile change in DNA gyrase [52]. This mutation makes it harder for fluoroquinolones to bind, so DNA replication can keep going even when the drug is present.

Macrolide resistance is primarily caused by point mutations in the 23S rRNA gene, especially the A2075G mutation, which impairs antibiotic binding at the ribosomal exit tunnel. Other mutations in ribosomal proteins L4 and L22 can also change the ribosome's shape and add to resistance [53]. Getting the *ermB* gene increases resistance further by methylating the ribosomal RNA target. Tetracycline resistance comes from the *tet(O)* gene, which makes a protein that protects the ribosome by removing tetracycline from its binding site. This gene is often found on plasmids, which helps it spread between bacteria. Multidrug resistance is mostly due to the CmeABC efflux pump, a system that removes many antibiotics, bile salts, and toxins from the cell [53]. The CmeABC pump, along with target-site mutations, greatly lowers drug levels inside the cell processes. It exemplifies the One Health paradigm, highlighting that antimicrobial resistance in *C. jejuni* results from the interconnectedness of human, animal, and environmental ecosystems [54].

Growing *C. jejuni* on selective media is still the main way to diagnose it, but this method has clear limits. *C. jejuni* is hard to grow and needs specific oxygen levels and temperatures. It can also enter a viable but non-culturable (VBNC) state when stressed, which can cause false negatives and make infections seem less common than they are [55]. Molecular methods, especially PCR-based tests, are faster and more sensitive, and are now common in labs. These tests can find small amounts of bacteria and are less affected by growth needs. However, tests that do not use cultures do not provide live bacteria, which limits further work like AMR profiling, serotyping, genome sequencing, and tracking outbreaks.

To address these limitations, integrated approaches such as immunomagnetic separation combined with PCR (IMS-PCR) have been created. IMS uses magnetic beads with *C. jejuni*-specific antibodies to pick out target cells from complex samples, such as stool and food [53]. This method removes PCR inhibitors and increases the number of live bacteria, making detection more sensitive. IMS-PCR allows for quick molecular identification and also helps recover live *C. jejuni* cells for culture-based tests, connecting molecular and traditional microbiology approaches [4,55,56].

Novel therapeutic and preventative strategies

Natural antimicrobial compounds, such as plant extracts and organic acids, are showing promise as alternatives to traditional antibiotics [57]. They help lower *C. jejuni* colonization by blocking important bacterial traits like movement and attachment. These compounds also support the body’s defenses by keeping the gut lining intact and reducing too much inflammation. Often, they work by affecting cell signaling pathways, including the ERK pathway [57,58]. As a result, these treatments help prevent gut inflammation and tissue damage.

Researchers are working to develop vaccines against *C. jejuni* for both people and animals. Current vaccine options focus on parts of the bacteria like capsular polysaccharides, flagellar proteins, and inactivated whole cells. Vaccinating

poultry is especially important since chickens are the main source of human infection. A good poultry vaccine could lower the amount of bacteria in chickens, reduce contamination of poultry products, and decrease the risk to people [59]. Other strategies try to block bacterial toxins, such as cytolethal distending toxin (CDT), or reduce harmful inflammation in the body instead of killing the bacteria directly. Some treatments being studied target specific cytokine signaling pathways to make the disease less severe and prevent complications like Guillain-Barré syndrome (GBS) [60]. As antibiotic resistance is rising worldwide due to heavy antibiotic use in people and animals, it is urgent to use antibiotics carefully and find new ways to treat and prevent infection.

Controlling campylobacteriosis requires a thorough approach that covers every step of food production. On farms, this includes better biosecurity, vaccinating poultry when possible, and using bacteriophages to lower *C. jejuni* levels in animals [61]. In food processing, chemical and physical cleaning methods are common. However, bacteria can still form biofilms on surfaces that touch food, which is a major problem. To solve this, researchers are testing new anti-biofilm agents, like gold nanoparticles, to break up *C. jejuni* biofilms and make cleaning in food processing more effective [61].

Research shows that handling poultry contaminated with *C. jejuni* is a major risk for human infection. Studies have found that *C. jejuni* can easily spread from raw chicken to ready-to-eat foods during normal kitchen activities. The amount of bacteria that transfers depends on the surface type and how clean it is. For instance, using the same knives and cutting boards without cleaning them led to transfer rates of 16.28%, 12.82%, and 5.32% to cucumber on bamboo, wooden, and plastic surfaces, which highlights the importance of both material and sanitation [62]. Poor cleaning and hand hygiene increase the risk of contamination, especially since these pathogens are invisible on surfaces. Safe food handling is important to prevent infection. This means cooking poultry thoroughly, avoiding unpasteurized dairy, using safe water, and keeping raw and ready-to-eat foods apart [63,64]. Washing hands, cleaning utensils, and keeping the kitchen clean also help stop bacteria from spreading [62]. However, *Campylobacter* infections still occur worldwide, which suggests that people may not always follow these guidelines or may not know about the risks.

Poultry is a main source of *C. jejuni*, which causes many human infections. Contamination often happens during slaughter and processing when poultry comes into contact with intestinal contents, letting the bacteria remain in the food chain [65]. To prevent this, farms need strong biosecurity, like controlling feed, water, soil, equipment, wildlife, and human activity [65]. However, these steps are not always followed, which reduces their effectiveness.

To reduce colonization, researchers are exploring alternatives to antibiotics, such as probiotics, prebiotics, bacteriophages, vaccines, and organic acids, especially as antibiotic use becomes more restricted [66]. Control methods are usually divided into pre-harvest steps like biosecurity, vaccination, and immune support, and post-harvest steps like processing hygiene and cleaning carcasses. However, no single method has been enough to stop Campylobacter from spreading. Because of this, a combined approach that covers all stages from farm to table is needed to lower Campylobacter contamination and related human disease [65].

Discussion

Campylobacteriosis is still a major global health issue, with *C. jejuni* being a leading zoonotic foodborne pathogen [67]. This pathogen can colonize animal hosts, especially poultry, without causing symptoms. Its ability to spread easily, survive in the environment, and adapt genetically helps it persist. Even with strong control programs, high rates in the EU/EEA and elsewhere show that current efforts are not enough to manage the complex relationships between hosts, pathogens, and the environment [68].

Accumulating evidence demonstrates that *C. jejuni* transmission and disease outcomes are strongly influenced by biological interactions beyond single-pathogen dynamics. Co-infections and microbiota-mediated effects can modify colonization efficiency, shedding intensity, and host immune responses. It alters the transmission risk. Observations from livestock systems, including protozoan-bacterial co-infections, suggest that pathogen-pathogen interactions may amplify or attenuate zoonotic spillover [67]. Another evidence from sheep co-adulterated with *Eimeria* deadbeats signifies that interactions between two pathogens can conceivably alter contamination consequences and broadcast rates [69]. These findings underscore the importance of adopting a One Health framework that integrates animal health, human health, and environmental reservoirs when designing risk models and control strategies [54]. Progress in understanding *C. jejuni* pathogenesis has been constrained by limitations in experimental models. Traditional rodent models frequently require artificial

microbiota depletion or immune manipulation, which reduces their relevance to natural infection processes. This gap has prompted increased interest in alternative models and ex vivo systems that better reflect physiological host-microbe interactions [70].

Recent advances in technology have greatly improved both epidemiological and mechanistic studies. Standardized methods like automated DNA extraction, fluorometric quantification, and Illumina sequencing now allow for detailed analysis of gut microbial communities and their response to *C. jejuni* colonization [71]. These tools help identify which microbiota patterns are linked to resistance or susceptibility, guiding targeted interventions. However, antimicrobial resistance (AMR) is still a growing problem. Studies show that *C. jejuni* and *C. coli* from food animals often have high resistance to fluoroquinolones, especially ciprofloxacin [72]. The rise of multidrug-resistant strains makes first-line treatments for severe campylobacteriosis less effective. To prevent *C. jejuni* transmission, farms need better biosecurity, such as controlled access, strict hygiene, clean water, and good vector control to limit flock colonization. Poultry vaccines are still being developed but could help reduce intestinal carriage and food contamination in the future. Strict rules on antimicrobial use in livestock are also needed to stop the spread of resistant strains. During processing and distribution, using hazard analysis and critical control point (HACCP) protocols, better chilling and decontamination, and high sanitation standards is important. Since *C. jejuni* biofilms can stay on food-contact surfaces, new anti-biofilm methods like enzymatic treatments, bacteriophages, or nanoparticle-based agents could greatly improve control [61].

From a public health view, it is important to teach people about safe food handling, avoiding cross-contamination, and cooking poultry thoroughly. Using whole-genome sequencing in surveillance can improve outbreak detection, source tracking, and risk assessment. Sustainable solutions should focus on alternatives to regular antibiotics. Anti-virulence therapies, bacteriophage use, and microbiota-based methods like probiotics and competitive exclusion have shown promise in lowering *C. jejuni* rates without causing resistance [59]. Checking water sources and wildlife is also key to reducing indirect transmission. In the end, controlling *C. jejuni* as a zoonotic pathogen requires teamwork across different fields, following One Health principles. Ongoing progress in genomics, microbiome studies, and sustainable strategies will be crucial to reducing the long-term public health impact of campylobacteriosis.

Several important research gaps and limitations make it hard to fully understand and control *C. jejuni*. One main limitation is that we do not fully understand how gut microbes interact. Many studies only look at certain parts of the intestine, like the ceca or feces, and ignore how the whole digestive system works together. This makes it hard to understand how microbes affect *C. jejuni* colonization and survival. While we know the microbiota can protect against infection, we still do not know enough about how the host, microbiome, and pathogen interact, especially in natural infections. Another important gap is that many experimental models do not accurately reflect real host conditions. For example, animal models like microbiota-depleted or immunocompromised rodents do not match the biology of humans or poultry, so the results may not apply well to real infections.

The review also points out problems with current diagnostic methods, especially the heavy use of culture-based techniques. Since *C. jejuni* can become viable but non-culturable (VBNC), it often goes undetected, leading to underestimates and false negatives that hurt surveillance accuracy. Molecular methods are more sensitive, but they do not provide live samples needed for testing resistance or tracking outbreaks. These issues show the need for better, combined diagnostic tools. Current control methods are also not effective enough. Even with strict biosecurity and farm measures, contamination rates stay high, which suggests problems with how these steps are carried out and understood. No single method before or after harvest has worked well on its own. Therefore, combined and multi-level approaches are needed. The increase in antimicrobial resistance (AMR) is another big research gap. While some resistance mechanisms are known, we still do not understand well how they develop, what environmental factors cause them, or how they interact with other microbes. This makes it difficult to find good ways to control resistance.

Finally, not having a standard global surveillance system is a major problem. Right now, there is no single system that brings together genomic, environmental, and epidemiological data for full surveillance. Differences in how data is collected and reported make it hard to assess risks and track outbreaks. All these issues show the need for integrated, multidisciplinary research using the One Health approach.

Conclusions

Campylobacter jejuni is a bacterial pathogen distinguished by metabolic flexibility, adaptability and genetic diversity. It is the primary bacterial cause of gastroenteritis worldwide and is

commonly linked to poultry, food supply chains, and robust survival strategies. Its ability to form biofilms and persist under adverse conditions enhances its transmission among animals, the environment, and humans. Rising antimicrobial resistance further complicates the management of campylobacteriosis. Widespread antibiotic use in both human and veterinary medicine has contributed to resistance, reducing the effectiveness of standard therapies and exposing limitations in current treatment options. This challenge highlights the urgent need for coordinated One Health strategies. Rapid, sensitive, and culture-independent diagnostic tools capable of detecting viable but non-culturable (VBNC) cells are essential for accurate surveillance and effective patient management. Accelerated vaccine development for both animals and humans is necessary to reduce colonization, transmission, and disease incidence. Further investigation of immune responses and disease pathogenesis is critical for identifying novel therapeutic targets, particularly to prevent severe complications such as Guillain–Barré syndrome. Additionally, the implementation of integrated surveillance systems using whole-genome sequencing is vital for real-time monitoring of strains, resistance patterns, and outbreaks at both local and global levels. Future research should focus on elucidating resistance mechanisms and host-microbiome interactions to inform new prevention and treatment strategies. Reducing the public health impact of *C. jejuni* will require sustained collaboration among experts in microbiology, immunology, veterinary medicine, food safety, and public health. Effective control of campylobacteriosis depends on such coordinated, evidence-based efforts.

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