International Journal for Multidisciplinary Research (IJFMR)

Human Campylobacteriosis: A Threat for Public Health

Sejal Malik

M.Sc. (Biotechnology), Amity Institute of Biotechnology, Amity University Noida, India

Abstract

Global public health is seriously threatened by human campylobacteriosis, which is mostly caused with the bacteria Campylobacter jejuni and Campylobacter coli. An extensive summary of the disease's epidemiology, pathophysiology, clinical presentations, diagnosis, therapy, and preventive measures is provided in this abstract. Poultry is a key reservoir for campylobacteriosis, one of the most common foodborne illnesses in the world. Via direct contact with sick animals or by consuming tainted food or water, the virus can spread. Warmer months see a rise in its incidence, which reflects varying seasons and environmental factors. When ingested, certain species of Campylobacter invade the gastrointestinal tract and produce symptoms that can range from a minor case of gastroenteritis to more serious consequences like Guillain-Barré syndrome. Young children and those with impaired immune systems are especially susceptible to its crippling consequences. Microbiological culture techniques are the mainstay of diagnosis, while molecular methods are becoming more and more popular because of their superior sensitivity and specificity. For proper therapy and to reduce the risk of complications, early identification is essential. In severe cases, treatment usually consists of supportive care and antibiotics, albeit there are hurdles due to the growth of antimicrobial resistance. Furthermore, preventive measures include appropriate food handling procedures, stringent cleanliness standards, and focused vaccination programs for livestock. Even with improvements in knowledge and treatment, human campylobacteriosis is still a serious public health issue. Because of its complicated epidemiology and the resilience of the Campylobacter species, it is imperative to use a variety of strategies to stop its spread. In summary, human campylobacteriosis poses a persistent risk to public health, requiring constant monitoring, investigation, and cross-sector cooperation. We can work to lessen its impact on society and protect the welfare of people everywhere by improving our comprehension of its mechanisms and putting in place efficient control measures.

Keywords: Campylobacteriosis, Public Health, Epidemiology

1. Introduction

The most frequent causes of campylobacteriosis are the bacteria Campylobacter jejuni, Campylobacter coli, which are among the Campylobacter species that cause the disease. These are spiral-shaped, motile, microaerophilic, gram-negative bacteria. They have flagella on ends, which helps explain why their movement resembles a corkscrew or darting. A class of bacteria known as campylobacter species primarily inhabit the digestive systems of various mammals. Because more and more species are being linked to diseases in humans and animals, campylobacter species are becoming extremely important. Campylobacter species are microaerophilic microorganisms that grow best at 42°C. They are also oxidase



E-ISSN: 2582-2160 • Website: <u>www.ijfmr.com</u> • Email: editor@ijfmr.com

and catalase positive. Infection with Campylobacter is linked to drinking raw milk, undercooked chicken, and tainted water. Usually, patients get a 5-to 7-day self-limited diarrheal sickness. Patients who are elderly and immunocompromised are most vulnerable to protracted disease and morbidity. Despite the availability of efficient treatment and eradication methods in animal reservoirs, there has been a sharp rise in the number of cases in both developed and developing countries worldwide.

There are two types of Campylobacter species: Lior serotypes and penner serotypes. Approximately 100 Lior serotypes as well as 600 penner serotypes have been identified. Only these thermotolerant Campylobacter species has been shown to have clinical importance among these Lior serotypes and penner serotypes.

1. Pathogenic campulobacter specie-

Approximately 400–500 million infections cases worldwide are caused by pathogenic Campylobacter species annually. Campylobacter jejuni, C. sputorum, C. helveticus, C. lari, C. fetal, C. mucosalis, C. coli, C. upsaliensis, and C. ureolyticus are among the pathogenic species of Campylobacter that have been linked to infections in humans.

2. C. Junini

A mobile microaerophilic, zoonotic, thermophilic bacterium called C. jejuni is thought to be the primary cause of foodborne bacterial gastroenteritis globally. It belongs to the genus Campylobacter and has helical morphology and polar flagella, which allow it to pass through viscous liquids and the mucus membrane of the digestive system. The main enteric pathogen with notable differences in pathogenicity patterns between strains is C. jejuni. More infections than any other pathogenic species of Campylobacter are caused by C. jejuni, which is also the primary species of Campylobacter that frequently causes diarrhea in humans.

3. C. coli

Campylobacter coli comprises a single-flagellum, S-shaped, curved cell that is between 0.2 and 0.5 micrometers long. It resembles C. jejuni quite a bit, and both bacteria make people's intestines inflamed and diarrheal . The second most frequently reported species of Campylobacter that infects humans is C. coli.

4. C. fetus

Curved-cell, fastidious motile bacteria called C. fetus is mostly responsible for septic abortions in agricultural animals. Humans can contract an infection from C. fetus by direct contact with animals, undercooked contaminated meat eating, or ingestion of food or water tainted with animal excrement.

2. Etiology

Campylobacter species, particularly Campylobacter jejuni as well as Campylobacter coli, are the primary cause of campylobacteriosis, a bacterial infection that is common throughout the world. Investigating the microbiological traits of these infections, their origins, their modes of transmission, and the elements promoting their growth and virulence are crucial to comprehending the etiology of campylobacteriosis.

2.1 Microbiology

Gram-negative, spiral-shaped bacteria called Campylobacter species are members of the Campylobacteraceae family. Because they are microaerophilic, they can flourish in low-oxygen settings like the gastrointestinal tracts of humans and animals. The capacity of certain Campylobacter species to pierce and colonize the intestinal mucosal surface is attributed to their spiral shape and flagellar movement. As far as human infections are concerned, the most prevalent species are Campylobacter jejuni as well as



Campylobacter coli. Adhesins, cytotoxins, which and invasion-associated proteins are just a few of the virulence factors they possess. These help them connect to intestinal epithelial cells, invade host tissues, and elude the immune system.

2.2 Origins of Contamination

Numerous species of Campylobacter can be found in the environment; their reservoirs include water, soil, vegetation, and both domestic as well as wild animals as well as birds. Chickens, the turkeys, and ducks in particular are thought to be important sources of Campylobacter species because of their high intestinal tract colonization rates. Other animals that produce food, such sheep, pigs, and cattle, may also host asymptomatic strains of Campylobacter.

Raw vegetables, unpasteurized milk, and undercooked poultry are among the contaminated food items that are frequently used to spread Campylobacter illnesses to humans. Inadequate food hygiene procedures and cross-contamination during the preparation of food both contribute to the pathogen's proliferation. Apart from the consumption of contaminated food, contact with contaminated animals or their excrement, drinking tainted water from contaminated sources, including person-to-person transmission.

2.3 Routes of Transmission

Ingestion of contaminating food or water is the main way that humans contract Campylobacter species. Meat and eggs from poultry, as well as other undercooked or uncooked poultry products, are common sources of infection in cases of foodborne transmission. In the kitchen and food processing facilities, Campylobacter contamination can be introduced and spread by improper food handling, insufficient cooking temperature, and contamination during food preparation.

The ingestion of untreated water tainted with animal excrement harboring Campylobacter species can lead to the waterborne spread of campylobacteriosis. If a water source is contaminated, recreational water activities including swimming in polluted lakes or ponds may also put you at risk for infection. Direct contact with diseased animals raises the risk of Campylobacter transmission, especially when involved in veterinary or agricultural operations. People who visit zoos, animals farms, or home chicken keepers may come into contact with settings polluted with Campylobacter, which can cause diseases through animal or excrement contact.

Although less frequent, transmission from person to person of campylobacteriosis can happen in places like homes, daycare centers, and hospitals where poor cleanliness standards exist. Containing hands, the surfaces, or objects are examples of fecal-oral transmission channels that can aid in the infection's spread among close contacts.

3. Epidemiology

Infections with campylobacter are more prevalent in the summer in certain regions, but they are primarily occasional in wealthy nations. Because of the COVID-19 pandemic, the rate of campylobacteriosis has dropped in the majority of countries. The reported death rate of campylobacteriosis in the EU/EEA in 2021 was 0.03%, remaining consistent from the five years prior. Hospitalizations accounted for 23.9% of cases in 2021, while travel inside the EU was a contributing factor in 65.7% of cases involving travel. The age group of between the ages of 15 and 29 old and infants below 1 year old are the most commonly infected with campylobacter. According to one study, males are less likely than females to have Campylobacter infection, and diarrheal stools are more common than non-diarrheal stools. Additionally, the infection becomes more common as the kid grows, peaking around months 9 and 11.

The disease's widespread occurrence indicates how adaptable Campylobacter is to a variety of habitats. It



is becoming more and more common for people and animals to travel internationally, making it difficult to stop the spread of Campylobacter. The CDC has worked to inform the public about disease prevention for visitors to nations where there is a high risk of coming into into contact with contaminated sources as well as illness signs and symptoms. Hand hygiene, segregating meat that is raw from other meals when cooking, fully cooking food, staying away from raw dairy, and using untreated water are some of these measures.

4. Pathophysiology

Gram-negative, microaerophilic bacteria such as C. jejuni and Campylobacter coli are the main culprits behind the gastrointestinal illness known as campylobacteriosis. Examining the complex interactions between host immune responses, mucosal integrity, and bacterial virulence factors is necessary to comprehend the pathophysiology of campylobacteriosis. The pathophysiological mechanisms of Campylobacter infection, including bacterial adhesion, invasion, toxin generation, host inflammatory reactions, and clinical symptoms, are thoroughly examined in this essay.

4.1 The Adherence and Invasion of Bacteria

Many bacterial adhesives and surface features help campylobacter species adhere to and colonize the intestinal epithelium, which is how infection begins. Bacterial adhesion to host cells is facilitated by adhesins like FlpA (fibronectin-like peptide A) and CadF (Campylobacter adhesin with fibronectin), which promote binding to parts of the extracellular matrix like fibronectin and laminin.

After adhering, Campylobacter penetrates epithelial cells by a variety of processes, which aids in the transfer of germs over the intestinal barrier. By means of endocytosis mediated by receptors or membrane ruffling, the production of invasion-associated protein molecules, like Cia proteins (This bacteria invasion antigen), facilitates the internalization of bacteria into host cells. Upon entering an epithelial cell, Campylobacter creates an intracellular niche for survival and replication while dodging the host immune system.

4.2 Toxin Generation

Numerous virulence factors, such as toxins and cytotoxins, are produced by Campylobacter species and aid in tissue damage and disease. The cdt gene cluster encodes a cytolethal distending toxins (CDT), which causes apoptosis, DNA damage, and cell cycle arrest in host cells as a result of its genotoxic actions. Intestinal epithelial integrity is compromised by CDT-mediated cytotoxicity, which encourages bacterial invasion and spread.

Other toxins, such as cytolethal extending toxins B (CdtB), which stimulates the production of proinflammatory cytokines, breaks tight junctions, and affects barrier function, may also be produced by Campylobacter strains. The clinical signs of campylobacteriosis, such as fever, diarrhea, and abdominal pain, are exacerbated by these toxins, which also cause epithelial damage and mucosal inflammation.

4.3 Host Defense Mechanisms

The etiology of campylobacteriosis is largely influenced by the host immune system, which coordinates both adaptive and innate immune responses in order to limit bacterial invasion and eradicate the infection. Innate immune cells, which include dendritic cells, macrophages, and epithelial cells, release pro-inflammatory cytokines like interleukin-8 (IL-8), the alpha form of tumor necrosis factor (TNF- α), and interleukin- beta (IL-1 β) when they recognize Campylobacter antigens.

By attracting neutrophils along with other immune-regulating cells to the infection site, these cytokines aid in tissue healing, phagocytosis, and bacterial removal. Dysregulated immune responses, on the other



hand, can result in tissue damage, excessive inflammation, and clinical consequences such reactive arthritis, inflammatory diarrhea, including Guillain-Barré syndrome (GBS).

During adaptive immunity, T and B lymphocytes that are specific to Campylobacter elicit antigen-specific responses, producing memory cells and antibodies that provide protection against reinfection. Th17 cells are essential for regulating mucosal immunity and preserving intestinal homeostasis because they secrete the interleukin-17 (IL-17) or interleukin-22 (IL-22), which support the integrity of the epithelium barrier and the release of antimicrobial peptides.

5. Diagnosis

Global public health is greatly concerned about campylobacteriosis, which is mostly caused by the microorganisms named Campylobacter jejuni and Campylobacter coli. For the purpose of putting control measures in place, starting the right therapy, and stopping the infection from spreading, a prompt and precise diagnosis is essential. This paper offers a thorough analysis of the many diagnostic techniques and strategies—such as laboratory procedures, clinical assessment, differential diagnosis, and developing technologies-that are employed in the identification of Campylobacter infections.

5.1 Medical Assessment

From minor gastrointestinal symptoms to serious systemic consequences, campylobacteriosis can present with a wide range of clinical presentations. Frequent signs and symptoms include fever, nausea, vomiting, diarrhea (sometimes bloody), cramping in the abdomen, and lethargy. Severe cases may result in consequences such reactive arthritis, electrolyte imbalance, and dehydration, especially in susceptible groups like young children.

In order to do a clinical evaluation on a patient who may have campylobacteriosis, a thorough medical history including past travel, dietary preferences, animal exposure, and symptom start must be obtained. A physical exam may reveal fever, pain in the abdomen, and symptoms of dehydration. However, clinical diagnosis is difficult due to the generic character of signs and overlap with various gastrointestinal infections, which emphasizes the significance of laboratory confirmation.

5.2 Diagnosis in Lab

Confirming a diagnosis for campylobacteriosis and determining the causal agent primarily depend on laboratory testing. There are numerous techniques in microbiology, molecular biology, and immunology that can be used to identify different kinds of Campylobacter in clinical materials such as blood, feces, and rectal swabs.

Stool Culture: The most reliable method for identifying Campylobacter infections is still conventional culture. Stool samples are placed onto selective medium, like Campylobacter blood-free selection agar (CCDA), and then left to incubate for 48–72 hours at 42°C in a microaerophilic environment. Small, transparent, gravish and white colonies with a distinctive "darting" or spreading development pattern are the outward appearance of campylobacter colonies. Typically, biochemical assays such oxidase and catalase responses, gram staining, as hippurate hydrolysis are used to confirm the species of Campylobacter.

Molecular Methods: The rapid, sensitive, and precise detection of Campylobacter Genome in clinical specimens is made possible by PCR (polymerase chain reaction) techniques. Target genes are amplified and found using particular primers and fluorescent probes, such as the hipO gene, 23S rRNA, and 16S rRNA. Multiplexing real-time PCR (qPCR) technologies allows for the simultaneous detection of numerous diseases and allows for the quantitative measurement of bacterial load. In surveillance studies,



E-ISSN: 2582-2160 • Website: <u>www.ijfmr.com</u> • Email: editor@ijfmr.com

outbreak investigations, and situations when culture-based techniques produce unfavorable results, PCR assays are especially helpful.

Commercially available enzyme immunoassays (EIAs) identify Campylobacter antigens in stool specimens, which include Campylobacter-alike organism (CLOs) or Campylobacter-specific proteins. These tests can be carried out in medical labs without the need for specialist equipment and have quick turnaround times. They are less popular than PCR or culture techniques, and their specificity and sensitivity might change based on the target antigen and test design.

Testing using Serology: Serological assays can help with epidemiological research and retrospective diagnosis by measuring antibodies (IgM, IgG) produced in reaction to a Campylobacter infection. However, because of delayed antibody generation and a cross-reaction with other infections, serological testing is not as useful in acute cases.

6. Treatment

Diarrhea, pain in the abdomen, fever, and sometimes vomiting are the symptoms of a self-limited gastrointestinal infection called campylobacteriosis, which is mostly caused by the bacteria Campylobacter jejuni as well as Campylobacter coli. While the majority of instances end on their own without the need for special care, some patient demographics and extreme presentations may call for medical attention. The fundamentals of treating and managing campylobacteriosis are examined in this essay. These include medical treatment, supportive measures, antibiotic therapy, preventing issues, and public health initiatives.

6.1 Clinical Assessment and Identification

A comprehensive clinical evaluation that includes the individual's medical history, signs, and epidemiological risk factors is the first step in the diagnosis of campylobacteriosis. Clinical signs include fever, lethargy, cramping in the abdomen, runny or bloody diarrhea, and rarely nausea or vomiting. It is important to inquire about recent travel, contaminated water or food consumption, exposure at work, and interactions with sick people.

Microbiological examination of stool specimens is used to confirm campylobacteriosis in a laboratory setting. While molecular diagnostic techniques like PCR (polymerase chain reaction) and immunoassays using enzymes (EIA) provide quick and sensitive alternatives, stool culture is still the most reliable approach for identifying Campylobacter species, especially in outbreak situations or cases that need to be diagnosed right once.

6.2 Antimicrobial Treatment

Antimicrobial therapy is not recommended in the majority of instances with uncomplicated campylobacteriosis because of the infection's self-limiting nature and worries about the emergence of antibiotic resistance. Antimicrobial medicines, however, might be administered in severe or protracted instances, those with compromised immunity, or individuals at risk of consequences in order to speed up symptom resolution and stop the infection from spreading systemically. Antibiotic susceptibility patterns, patient-specific characteristics, local guidelines, and the severity of the illness all play a role in the selection of an antibiotic medication. Azithromycin, ciprofloxacin, and tetracyclines, such as doxycycline, are examples of regularly used antibiotics that are active against Campylobacter species.

Because of its effectiveness, safety record, and easy-to-follow dosage schedule, azithromycin is frequently chosen as first-line treatment, especially for children and expectant mothers. Ciprofloxacin should only be used in patients who cannot tolerate azithromycin or in situations where resistance to macrolide is



suspected. Tetracyclines are less frequently utilized because of worries about resistance and side effects, but they can be regarded as alternative agents. In patients with severe instances or extraintestinal consequences such meningitis, invasive infections, or bacteremia, antimicrobial treatment should be started very away. Treatment usually lasts three to seven days, while longer courses can be required for those with chronic symptoms or those who are immunocompromised.

7. Strategies for public health

Comprehensive public health policies that target various phases of the chain of production and consumption of food are necessary to prevent and control campylobacteriosis. In order to detect and reduce the sources of contamination, regulatory agencies are essential in carrying out surveillance operations, enforcing food safety regulations, and carrying out outbreak investigations. The goal of pre-harvest biosecurity policies, livestock vaccinations, and antimicrobial management is to lower the colonization of Campylobacter in food animals. In order to prevent transmission of foodborne illnesses in food manufacturing and preparation, it is crucial to follow appropriate hygiene procedures, thoroughly cook chicken items, pasteurize milk, avoid cross-contamination. and Public education efforts that stress the value of good food handling, hand cleanliness, and drinking safe water can increase consumer awareness and encourage behavior change. Working together, healthcare professionals, public health organizations, food manufacturers, regulators, and consumers can effectively tackle the complex issues surrounding campylobacteriosis and protect public health. In summary, a multidisciplinary strategy involving clinical care, supporting measures, antibiotic therapy, complication prevention, and public health efforts is required for the management and treatment of campylobacteriosis. Healthcare professionals, public health officials, and legislators can lessen the impact of campylobacteriosis and advance the health and well-being of afflicted individuals and communities by putting evidence-based interventions into practice at different levels.

References

- 1. Skirrow MB. Campylobacter enteritis: a new "disease". *Br Med J.* 1977;2(6078):9–11. [PMC free article] [PubMed] [Google Scholar]
- 2. Figura N, Guglielmetti P. Clinical characteristics of Campylobacter jejuni and Campylobacter coli enteritis. *Lancet.* 1988;1(8591):942–943. [PubMed] [Google Scholar]
- Caprioli A, Pezzella C, Morelli R, Giammanco A, Arista S, Crotti D, Facchini M, Guglielmetti P, Piersimoni C, Luzzi I. Enteropathogens associated with childhood diarrhoea in Italy. *Pediatr Infect Dis J*. 1996;15(10):876–883. [PubMed] [Google Scholar]
- 4. European Food Safety Authority, author. The European Union summary report on trends and sources of zoonoses, zoonotic agents and food-borne outbreaks in 2013. *EFSA J.* 2015;13 doi:10.2903/j. efsa. [PMC free article] [PubMed] [Google Scholar]
- 5. Platts-Mills JA, Kosek M. Update on the burden of Campylobacter in developing countries. *Curr Opin Infect Dis.* 2014;27(5):444–450. [PMC free article] [PubMed] [Google Scholar]
- 6. Wagenaar JA, French NP, Havelaar AH. Preventing Campylobacter at the source: why is it so difficult? *Clin Infect Dis.* 2013;57:1600–1606. [PubMed] [Google Scholar]
- Islam D, Ruamsap N, Aksomboon A, Khantapura P, Srijan A, Mason CJ. Immune responses to Campylobacter (C. jejuni or C. coli) infections: a two-year study of US forces deployed to Thailand. *AP-MIS*. 2014;122(11):1102–1113. [PubMed] [Google Scholar]



E-ISSN: 2582-2160 • Website: www.ijfmr.com • Email: editor@ijfmr.com

- Shyaka A, Kusumoto A, Asakura H, Kawamoto K. Wholegenome sequences of eight Campylobacter jejuni isolates from wild birds. *Genome Announc*. 2015;3(2) 23. [PMC free article] [PubMed] [Google Scholar]
- Fitzgerald C, Nachamkin I. Campylobacter and Arcobacter. In: Versalovic J, Carroll K, Funke G, Jorgensen J, Landry ML, Warnock DW, editors. *Manual of Clinical Microbiology*. Washington DC: ASM Press; 2011. pp. 885–899. [Google Scholar]
- 10. Man SM. The clinical importance of emerging Campylobacter species. *Nat Rev Gastroenterol Hepa-tol.* 2011;8(12):669–685. [PubMed] [Google Scholar]
- Vandamme P, Dwhirst FE, Paster BJ. Campylobacteraceae. In: Garrity GM, editor. *Bergey's Manual Rof Systematic Bacteriology*. New York: Springer; 2005. pp. 1145–1168. On SLW. Family I. [Google Scholar]
- Debruyne L, Gevers D, Vandamme P. Taxonomy of the Family Campylobacteraceae. In: Nachamkin I, Szymanski C, Blaser M, editors. *Campylobacter*. Third Edition. Washington, DC: ASM Press; 2008. pp. 3–25. [Google Scholar]
- Owen RJ, Leaper S. Base composition, size and nucleotide sequence similarities of genome deoxyribonucleic acids from species of the genus Campylobacter. *FEMS Microbiol Lett.* 1981;12:395400–395400. [Google Scholar]
- Chang N, Taylor DE. Use of pulsed-field agarose gel electrophoresis to size genomes of Campylobacter species and to construct a SaA map of Campylobacter jejuni UA580. *Bacteriol.* 1990;172:5211–5217. [PMC free article] [PubMed] [Google Scholar]
- Nuijten PJM, Bartels C, Bleumink-Pluym NMC, Gaastra W, Zeijst BAM. Size and physical map of the Campylobacter jejuni chromosome. *Nucleic Acids Res.* 1990;18:6211–6214. [PMC free article] [PubMed] [Google Scholar]
- Garénaux A, Jugiau F, Rama F, Jonge R, Denis M, Federighi M, Ritz M. Survival of Campylobacter jejuni strains from different origins under oxidative stress conditions: effect of temperature. *Curr Microbiol.* 2008;56(4):293–297. [PubMed] [Google Scholar]
- Levin RE. Campylobacter jejuni: A review of its characteristics, pathogenicity, ecology, distribution, subspecies characterization and molecular methods of detection. *Food Biotechnology*. 2007;21:271–347. [Google Scholar]
- Cesare A, Sheldon BW, Smith KS, Jaykus L. Survival and persistence of Campylobacter and Salmonella species under various organic loads on food contact surfaces. *J Food Prot.* 2003;66(9):1587– 1594. [PubMed] [Google Scholar]
- Damborg P, Olsen KEP, Nielson EM, Guardabassi L. Occurrence of Campylobacter jejuni in pets living with human patients infected with C. jejuni. *J Clin Microbiol.* 2004;42:1363–1364. [PMC free article] [PubMed] [Google Scholar]
- Bae W, Kaya KN, Hancock DD, Call DR, Park YH, Besser TE. Prevalence and antimicrobial resistance of thermopilic Campylobacter spp. from cattle farms in Washington State. *Appl Environ Microbiol.* 2005;71:169–174. [PMC free article] [PubMed] [Google Scholar]
- 21. Thakur S, Gebreyes WA. Prevalence and antimicrobial resistance of Campylobacter in antimicrobialfree and conventional pig production systems. *J Food Prot.* 2005;68:2402–2410. [PubMed] [Google <u>Scholar</u>]



E-ISSN: 2582-2160 • Website: <u>www.ijfmr.com</u> • Email: editor@ijfmr.com

- 22. Ternhag A, Törner A, Svensson A, Giesecke J, Ekdahl K. Mortality following Campylobacter infection: a registry-based linkage study. *BMC Infect Dis.* 2005;5:1–5. 12, 13. [PMC free article] [Pub-Med] [Google Scholar]
- 23. Jones K. Campylobacters in water, sewage and the environment. *Appl Microbiol*. 2001;90:68–79. [PubMed] [Google Scholar]
- 24. Newell DG, Shreeve JE, Toszeghy M, Domingue G, Bill S, Humphrey T, Mead G. Changes in the carriage of Campylobacter strains by poultry carcasses during processing in abattoirs. *Appl Environ Microbiol.* 2001;67:2636–2640. [PMC free article] [PubMed] [Google Scholar]
- 25. Newell DG, Fearnley C. Sources of Campylobacter colonization in broiler chickens. *Appl Environ Microbiol.* 2003;69:4343–4351. [PMC free article] [PubMed] [Google Scholar]
- 26. Inglis GD, Kalischuk LD, Busz HW. Chronic shedding of Campylobacter species in beef cattle. *J Appl Microbiol*. 2004;97:410–420. [PubMed] [Google Scholar]
- 27. Stafford RJ, Schluter PJ, Wilson AJ, Kirk MD, Hall G, Unicomb L. Population-attributable risk estimates for risk factors associated with campylobacter infection, Australia. *Emerg Infect Dis.* 2008;14:895–901. [PMC free article] [PubMed] [Google Scholar]
- Meldrum RJ, Griffiths JK, Smith RMM, Evans MR. The seasonality of human Campylobacter infection and Campylobacter isolates from fresh, retail chicken in Wales. *Epidemiol Infect*. 2005;133:49–52. [PMC free article] [PubMed] [Google Scholar]
- Mullner P, Spencer SEF, Wilson DJ, Jones G, Noble AD, Midwinter AC, Collins-Emerson JM, Carter P, Hathaway S, French NP. Assigning the source of human Campylobacteriosis in New Zealand: a comparative genetic and epidemiological approach. *Infect Genet Evol.* 2009;9:1311–1319. [Pub-Med] [Google Scholar]
- 30. Nachamkin I, Szymanski MC, Blaser JM. *Campylobacter*. 3rd edition. Washington DC, USA: ASM Press; 2008. [Google Scholar]
- Verhoeff-Bakkenes L, Jansen HAPM, Veld PH, Beumer RR, Zwietering MH, Leusden FM. Consumption of raw vegetables and fruits: A risk factor for Campylobacter infections. *Int J Food Microbiol.* 2011;144:406–412. [PubMed] [Google Scholar]
- Rapp D, Ross CM, Pleydell EJ, Muirhead RW. Differences in the fecal concentrations and genetic diversities of Campylobacter jejuni populations among individual cows in two dairyherds. *Appl Environ Microbiol.* 2012;78:7564–7571. [PMC free article] [PubMed] [Google Scholar]
- 33. Moore JE, Corcoran D, Dooley JSG, Fanning S, Lucey B, Matsuda M, McDowell DA, Megraud F, Millar BC, Mahoney RO, et al. Campylobacter. *Vet Res.* 2005;36:351–382. [PubMed] [Google Scholar]