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Pathogenesis of *Campylobacter jejuni* (Food-borne Pathogen), Transmission and Laboratory Techniques for their Identification

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ABSTRACT

Campylobacter is an important microorganism which spreads the bacterial food-borne diarrheal anomaly around the world particularly in the children and old age people. *Campylobacter* is spiral, non-spore-forming, rod-shaped or curved, Gram-negative, bacteria with bipolar flagella or single polar flagellum, or absence of any flagellum, depending on the species. Most *Campylobacter* species grow under microaerophilic condition. It resides in Gastrointestinal tract of poultry birds. Wild birds may also act as carrier or vectors for transmission of *Campylobacter* particularly to poultry flocks. This review article throw light on the aspects of genus, growth and viability, transmission and detection, pathogenesis and prophylaxis measures for campylobacter jejuni.

Introduction

Campylobacter is an important organism that causes zoonotic disease particularly when the unhealthy and unhygienic conditions are present and also during slaughtering of these animals the meat and meat products get contaminated with this organism from feces. This organism generally resides in intestinal mucosa of human beings, in gastrointestinal tract of farm animals, wild animals, companion animals and birds. Campylobacteriosis is commonly reported as a zoonotic disease all over the world, transmitted directly or indirectly to humans. About Eighty percent cases of all human campylobacteriosis are due to consumption of contaminated poultry (chicken) meat worldwide (Hermans et al., 2012; Bahrndorff et al., 2013). Among *Campylobacter*, *C. Jejuni* is important species, involved in human infections cause fever, abdominal pain, and diarrhea (Messaoudi et al., 2011;Acheson et al., 2001). Guillain-Barre syndrome, irritable bowel syndrome and Reactive arthritis are the most important post infections sequelae with C. jejuni. Globally, about 1/3rd of Guillian-Barre syndrome cases are associated to Campylobacter infections.

In the developed countries, this organism is important in causing food-borne diseases in human beings. It is an obligate microaerophilic (requires minimum 5% O_2 level, Nitrogen 85%, 10% CO_2) heat labile, thermophilic, Gram-negative, and exhibits optimum growth at 42˚C (Newell et al., 2003).

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Structurally *Campylobacter* is curved rod shaped, non-spore farming, and biflagellate, having one flagella or sometimes having no flagella depending on the specie. Majority of the species are resistant to cephalothin and fluoroquinolones (Koenraad et al., 1995).

Poultry birds are the main source where the colonization of this organism takes place even without showing the clinical signs and the human beings can also be infected if they consume the meat or meat products contaminated with *Campylobacter* species (Silva 2011). This organism is regarded as an important microbial specie which is responsible to spread disease from animals to human beings (Cean et al., 2015). For the first time in 1963 this organism was discovered and it was named as *vibrio fetus* then it was renamed as to *Campylobacter fetus*. The *Campylobacter jejuni* is responsible for gastro enteritis throughout the world. Apart from this it is also the causative agent of autoimmune disease guillain barre syndrome (GBS) and Miller fisher syndrome (Mann 2011). Guillain-Barré syndrome (GBS) is an immune-mediated demyelinating polyneuropathy of peripheral nervous system (PNS), it is characterized by acute or subacute symmetrical ascending motor weakness (Asbury and Cornblath 2005). About ⅔ of the GBS patients are usually reported with the infection associated with *Campylobacter jejuni*, *Cytomegalovirus* and other important bacteria Epstein-Barr virus, and *Mycoplasma pneumoniae* (Sinha et al., 2004).

Transmission

In past, many epidemiological surveys have been conducted for *Campylobacter jejuni* to check its prevalence and the results were different but a thorough and detailed review is presented by Adkin et al., 2006 and among those transmission routes, horizontal transmission was considered most reliable (Sahin et al., 2002). The vertical transmission from parent breeder to broilers varies (Gubbels et al., 2012). The prevalence of the organism in infected commercial flocks is age related and mostly found in chickens of 2-3 weeks of age (Cox et al., 2010). After infection the chickens show higher concentrations of this organism in small intestine (Mann 2011). In developing countries, the prevalence of *C. jejuni* infection is reported to be endemic among children particularly till 5 years age (Horrocks et al., 2009). In immunocompromised people and young children, campylobacteriosis needs to be treated using antimicrobial therapy but in some cases, the gastroentertitis is self limiting disease. Contaminated water is also reported to be the source of infection and thus cause campylobacteriosis in human beings (Reiny et al., 2007; Gubbels et al., 2012).

The horizontal transmission is supposed to be important way of transmission to the poultry birds from surroundings and it happens within a flock when a bird harbors and becomes infected with this organism (Horrocks et al., 2009). The factors responsible for disease, colonization and transmission are litter, size of flock, environmental water supplies, rodents, wild animals,

insects, pets, and fecal contact (Adkin et al., 2006). Regarding the transmission through feeding route, it is not considered as an important transmission route organism however it can be considered a potential way of transmission for *Salmonella* spp. infection (White et al., 1997). The environment plays a crucial role in microorganism's transmission and also its survival. *Campylobacter* is very sensitive to the dryness so it can't be transmitted through feed (Cox et al., 2010).

Clark and Bueschkens 1985 administered *C. jejuni* in fertile eggs, and they observed that almost 11% eggs during hatching time had the pathogen in their gut. Studies conducted invivo concluded that within 1-2 days of hatching, the vertical transmission occurs and chicks' infection, in the ceacum, was reported almost 35% without any exposure of infection from farm. Lindblom et al., 1986; Chuma et al., 1994). However, Callicott et al., 2006 could not report the vertical transmission of *C. jejuni* to almost 60,000 breeders flock of *Campylobacter*-positive grandparent flocks thus further investigation and research is still in progress.

Pathogenesis

It has been detected that in case of bacterial intestinal infections, the chemotaxis of epithelial cells, the invasion and adherence of mucus are the important stages. The presence of lipopolysaccharides and lipo-oligosaccharides provides different functional roles in pathogen and host interactions as well as virulence factors. The factors responsible for virulence of *C. jejuni* are: Flagellar movement, bacterial affinity to gut mucosa and invading property (Dastia 2010). In order to get colonized in the small intestine, Campylobacter uses its flagella and then moves to colon with the help of its flagella (Snelling et al., 2005). After colonization, *C. jejuni* attaches to the intestinal mucosal cells. The pathogenicity of *C. jejuni* is associated with the production of a thermo labile enterotoxin which causes cytotoxicity, particular by *C. lari, C. fetus C. coli, C. jejuni* and *C. upsaliensis*, (Johnson and Lior 1988). The cytotoxins produced causes the cellular inflammation and thus affect the intestinal absorption (Van Deun et al., 2007). Cytolethal distending toxin (CDT) is another important toxin which is formed by CdtABC complex (Lara and Galan 2000). CdtB is an important subunit who functions like DNase and causes double-strand breaks (DSB) in the DNA thus arrest cell cycle at the M/ G2 stage. This leads to apoptosis. The binding of CdtC and CdtA to cholesterol-rich domains on the membrane of cytoplasm is essential for the transfer of CdtB to cells. (Lai et al., 2013). Production of these toxins causes death of the intestinal mucosal cells by the process of apoptosis and thus leads to diarrhea (bloody or watery). The severity is subjected to pathogenic strain as well as host's immunity level (Zilbauer et al., 2008).

Laboratory techniques for identification of Campylobacter jejuni

A number of Conventional diagnostic methods have been developed

for the identification of the campylobacter. However, detection and isolation is a lengthy task generally takes 5 to 7 days. The cause of the disease and tracking of the microorganism in real time are challenging with diagnostic procedures due to robustness and rapidity of existing methods. Once campylobacter is successfully recovered from poultry farms or matrices, the accurate detection involves phenotypic differentiation procedures such as biotyping, multilocus enzyme electrophoresis and serotyping (Eberle and kiess. 2012). A number of methods included microbiological culture techniques, immunoassays such as fluorescent antibody technique (FAT), enzyme-linked immunosorbent assay (ELISA) and molecular techniques like Polymerase chain reaction (PCR) have been established for testing food and clinical samples for Campylobacter species, including *Campylobacter jejuni* (Khan et al., 2018).

The precise detection of *Campylobacter* species is exceptionally basic for surveillance throughout the poultry processing. The development of sophisticated and profoundly explicit measures to identify Campylobacter has potential for decreasing the food borne disease (Steven et al., 2018). For the identification and maximum growth of campylobacter, culture techniques are routinely utilized as diagnostics systems in which particular microaerophilic conditions are maintained. Furthermore few antibiotics are also used in those culture media which will suppress or stop the growth of non-campylobacter thus favoring the isolation of antibiotics resistant isolates of campylobacter. (Eberle and kiess. 2012)

Over the earlier few decades, the fast and rapid, culture-independent methods for detection of campylobacter has become increased progressively on routine basis. For the efficient identification of foodborne pathogens, nucleic acid and immune based methodologies are preferred. The immunological-based detection procedures exploit affinity of antibodies for target antigens present on the surface of the campylobacter (Zeng et al., 2016). Flow cytometry, ELISA and quantitative immunoflurescence are immune based procedures used for the detection of *C. jejuni* (Baker et al., 2016). For the identification of specific *campylobacter* epitope, polyclonal and monoclonal antibodies are used. In most antibody based technology, significant cross-reactivity with *C. jejuni* and *C. coli* is observed because there is no significant genetic divergence (Park et al., 2014). Research is still continued to evolve and focused on a major outer membrane protein epitope that specifically targeted *C. jejuni* and thus will reduce the cross-reactivity as time progressed (Qian et al., 2008). In this regard some more research is required that will include the capability to produce the monoclonal antibodies which are not affected by heat and empower the detection of Campylobacter species in thermophilic environment (Heo et al., 2009).

Molecular methods for detecting and identifying foodborne pathogens are more sensitive as comprehensive and sophisticated genomic data continue to be generated from foodborne pathogens. Nucleic acid-based methodologies

identifies unique and very specific RNA or DNA nucleotide sequences that can either amplified, visualized and sequenced for quantification, molecular typing and detection (Zeng et al., 2016). These methods are accurate, quick and commercialized kits are also available to identify foodborne pathogens including campylobacter. A principal target for differential PCR procedures includes the flaA gene, 16S rDNA gene and flaB gene (Rasmussen et al., 1996). However PCR assays are more reliable and sensitive as compared to traditional microbiological based culture assays, the specific and successful detection of Campylobacter in varied matrices still continues to be a vital challenge. This variability is dependent on numerous factors, such as the presence of polymerase inhibitors, non-culturable bacteria and fecal material and low number of cells existing in a large quantity of sample (Leskinen and Lim, 2008), Innovations to molecular based techniques (PCR) have addressed several of those limitations which includes preventing inhibitors, involving an enrichment stage before using the PCR, and coupling PCR methods with other techniques like EIA to improve the assay sensitivity and specificity.(Park et al., 2014). By coupling ELISA with PCR, researchers identified Campylobacter in contaminated environmental samples such as water that were beneath the limit of identification of conventional cultural assays (Sails et al., 2002).

Limitations

There are critical impediments related with immune based detection procedures. The previous microbiological and molecular techniques have described false positive characteristic due to cross connectivity among the *Campylobacter* species (Myers et al., 2011). The genetic specificity of *C. jejuni* and *C. coli* is not significant so problem can arise while doing immune based procedures (Park et al., 2014). Another possible way for the improvement of immunological assays would be use of proteomics to detect ideal epitopes for monoclonal antibodies to improve the resolution of discriminating between many different species and strains epithets of Campylobacter (Rodrigues et al., 2016)

In this context the commercial immune assays can draw out the false positive and vague changeability for the clinical examples (Gharst et al., 2013). When tested on frozen and chilled broiler carcass, the PCR assays proved to be more authentic and sensitive as compared to commercial ELISA (Reis et al., 2018). In a most recently conducted research trial the covalently joined the polyclonal antibodies of rabbit to gold chips and form a surface plasma resonance (SPR) sensor stage (Masdor et al., 2017). The Nano-based immunoassays are possibly viable, easy to use methodologies, which can effectively be adjusted to different poultry birds. The cotton swab immunoassay is produced to be used in processing plants through dipping the swabs into various nano-based conjugated *C. jejuni* explicit monoclonal immunizer mixed cocktails (Alamer et al., 2018). The idea was appealing for evaluating the infection level in remotely located poultry processing plants and results in efficient way for settling on in-plant decisions and also for bio-security

measures (Rodrigues et al., 2016). The utilization of PCR is the basics of various advanced techniques (Hill, 1996). Numerous PCR-based methodologies have proved to recognize *Campylobacter* species. A focal objective for differential PCR tests incorporates 16S r DNA quality (Giesendorf et al., 1992). Multiplex PCR is a brisk and reliable technique for deciding either presence or absence of different quality focuses inside single sample. Zhao et al., 2001 built up a multiplex PCR to separate *C. jejuni* and *C. coli* and to affirm hypothetical *Campylobacter* secludes on blood agar plates. Multiplex PCR have reported to be reliable and efficient method for detection of different species of campylobacter in food samples.

Prevention and Control

The important specie of *C. jejuni* colonizes poultry approximately at densities of 10^8 colony forming units (CFU)/gram of cecal contents and once this species invades the intestine of poultry it remain inside the bird for its life time and it also spreads to other healthy birds and make them diseased (Wagenaar et al., 2013). It is a surprising fact that more than 90% of the domestic poultry birds are infected at the time of slaughtering and sale (Doyle. 1992). Presently various procedures are being framed to minimize the load of the microbe (Laniewski. et al., 2014). It is explained that if 3 $\log_{10}$ reduction of *C. jejuni* in the chicken gut or 2 $\log_{10}$ reduction in the carcass of chicken is achieved then it can minimize risk to public health by almost 90% (EFSA, 2011).

Procedures which can minimize the load of *C. jejuni* in poultry birds are: 1) inject the compounds which can hinder the activity of *C. jejuni*, 2) by administering the probiotics which struggles to compete with *C. jejuni* to colonize or to produce probiotics, 3) bacteriophage specific to *C. jejuni*, and 4) making the chicks immunized by *C. jejuni* antigens (Layton et al., 2011). Presently there are lots of vaccination protocols being framed and followed to eradicate the *C. jejuni* infection in poultry birds (Rice et al., 1997). The immunization of poultry birds through oral route was proved to exhibit the serum antibody response but it didn't work out up to the required level. In a study, the effects of utilizing live attenuated *Salmonella* to deliver the CjaA ,CjaD or Dps antigens to poultry, was documented (Laniewski et al., 2014). Attenuated *Eimeria* parasites are another way to deliver CjaA antigen (Clark et al., 2012). The successful reduction in the colonization of *Campylobacter* is seen administering the nano-particle encapsulated *C. jejuni* outer-membrane proteins (Annamalai et al., 2013). The administration of egg yolk antibodies, IgY, which are specific to *Campylobacter* specie is also used as a passive immunotherapy. In this context some more trails should be conducted to identify the specific antigen and method to inhibit the colonization of *Campylobacter jejuni* (Hermans et al., 2012).

References

Acheson, D., Allos, B.M. 2001. Campylobacter jejuni infections: update on emerging issues and trends, Clin. Infect. Dis. 32(8): 1201-1206.

Adkin, A., Hartnett, E., Jordan, L., Newell, D., Davidson, H. 2006. Use of systematic review to assist the development of Campylobacter control strategies in broilers, J. Appl. Microbiol., 100:306–315.

Alamer, S., Eissa, S., Chinnappan, R., Zourob, M. 2018. A rapid colorimetric immunoassay for the detection of pathogenic bacteria on poultry processing plants using cotton swabs and nanobeads. Microchim. Acta 185: 1–10.

Annamalai, T., Pina-Mimbela, R., Kumar, A., Binjawadagi, B., Liu, Z. 2013. Evaluation of nanoparticle-encapsulated outer membrane proteins for the control of Campylobacter jejuni colonization in chickens, Poult. Sci. 92:2201–2211.

Asbury, A. K., Cornblath, D. R. 1990. Assessment of current diagnostic criteria for Guillain-Barré syndrome, Ann Neurol. 27(1):21-24.

Bahrndorff, S., Rangstrup ,C. L., Nordentoft, S., Hald, B. 2013. Foodborne disease prevention and broiler chickens with reduced Campylobacter infection, Emerg Infect Dis. 19:425-428.

Baker, C.A., Rubinelli, P.M., Park, S. H., Ricke, S. C. 2016. Immunobased detection of shiga toxin-producing pathogenic Escherichia coli – a review on current approaches and potential strategies for optimization, Crit. Rev. Microbiol., 42:656–675.

Callicott. K.A., Friðriksdóttir, V., Reiersen, J., Lowman, R., Bisaillon, J.R, Gunnarsson, E., Berndtson, E., Hiett, K. L., Needleman, D. S., Stern, N. J. 2006. Lack of evidence for vertical transmission of Campylobacter spp. in chickens, Appl. Environ. Microbiol., 72(9):5794-5798.

Cean, A., Stef, L., Simiz, E., Julean, C., Dumitrescu, G., Vasile, A., Pet, E., Drinceanu, D., Corcionivoschi, N. 2015. Effect of human isolated probiotic bacteria on preventing Campylobacter jejuni colonization of poultry, Foodborne Pathog Dis. 12(2):122-130.

Chuma, T., Yamada, T., Yano, K., Okamoto, K., Yugi, H. 1994. A survey of Campylobacter jejuni in broilers from assignment to slaughter using DNA-DNA hybridization, J. Vet. Med. Sci. 56:697–700.

Clark, A.G., Bueschkens, D.H. 1985. Laboratory infection of chicken eggs with Campylobacter jejuni by using temperature or pressure differentials, Appl. Environ. Microbiol., 49: 1467– 1471.

Clark, J.D., Oakes, R.D., Redhead, K., Crouch, C.F., Francis, M.J. 2012. Eimeria species parasites as novel vaccine delivery vectors: antiCampylobacter jejuni protective immunity induced by Eimeria tenella-delivered CjaA, Vaccine. 30:2683–2688

Cox, N.A., Richardson, L.J., Buhr, R J., Fedorka-Cray, P.J. 2009. Campylobacter can remain in various organs, World Poultry. 25(12): 28-29.

Dasti, J.I., Tareen, A.M., Lugert, R., Zautner, A.E., Groß, U., 2010.

Campylobacter jejuni: a brief overview on pathogenicity-associated factors and disease-mediating mechanisms, Int. J. Med. Microbiol., Suppl. 300(4):205-211.

Doyle, M.P. 1992. Food-borne transmission and antibiotic resistance of Campylobacter jejuni. Campylobacter jejuni Current Status and Future Trends. Washington, D.C, American Society for Microbiology. 45–48.

Eberle, K.N., and Kiess, A.S. 2012. Phenotypic and genotypic methods for typing Campylobacter jejuni and Campylobacter coli in poultry. Poult. Sci, 91: 255–264.

Gharst, G., Oyarzabal, O. A., Hussain, S. K. 2013. Review of current methodologies to isolate and identify Campylobacter spp. from foods. J. Microbiol., Methods, 95: 84–92.

Giesendorf, B.A., Quint, W.G., Henkens, M.H., Stegeman, H., Huf, F.A., Hiesters, H.G. 1992. Rapid and sensitive detection of Campylobacter spp. in chicken products using the polymerase chain reaction, Appl. Environ. Microbiol., 58: 3804–3808.

Gubbels, S.M., Kuhn, K.G., Larsson, J.T., Adelhardt, M., Engberg, J., Ingildsen, P., Hollesen, L.W., Muchitsch, S., Mølbak, K. and Ethelberg, S. 2012. A waterborne outbreak with a single clone of Campylobacter jejuni in the Danish town of Køge in May 2010, Scand. J. Infect. Dis. 44(8):586-594.

Heo, S.A., Nannapaneni, R., Johnson, M.G., Park, J.S., Seo, K.H. 2009. Production and characterization of a monoclonal antibody to Campylobacter jejuni. J. Food Prot, 72: 870–875.

Hermans, D., Van, S.K., Verbrugghe, E., Verlinden, M., Martel, A., Seliwiorstow, T., Heyndrickx, M., Haesebrouck, F., De, Z.L., Deforce, D. 2014. Passive immunization to reduce Campylobacter jejuni colonization and transmission in broiler chickens, Vet. Res. 45(1): 27.

Hill, W.E. 1996. The polymerase chain reaction: applications for the detection of foodborne pathogens Crit. Rev, Food Sci. Nutr. 36:123–173.

Horrocks, S.M., Anderson, R.C., Nielsbet, D.J., Ricke, S.C. 2009. Incidence and ecology of Campylobacter jejuni and coli in animals. Anaerobe, 15:18–25

Johnson, W.M., Lior, H. 1988. A new heat-labile cytolethal distending toxin (CLDT) produced by *Campylobacter*, Microb. Pathog. 4(2):115-126.

Khan, J.A., Rathore, R.S, Abulreesh, H.H., Qais, F.A. Ahmad, I. 2018. Cultural and immunological methods for the detection of Campylobacter jejuni: A Review, Ind. J. Biotechnol. Pharma. Res. 6(3):4-10.

Koenraad, P. M., Jacobs-Reitsma, W. F., van der Laan, T., Beumer, R. R., Rombouts, F.M. 1995. Antibiotic susceptibility of Campylobacter isolates from sewage and poultry

abattoir drain water. Epidemiol. Infect., 115: 475–483.

Lai, C.H., Lai, C.K., Lin, Y.J., Hung, C.L., Chu, C.H., Feng, C.L., Chang C.H, Su, H.L. 2013. Characterization of putative cholesterol recognition/interaction amino acid consensus-like motif of Campylobacter jejuni cytolethal distending toxin C, PloS one, 8(6).

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

Lara-Tejero, M., Galan, J. E. 2000. CdtA, CdtB and CdtC form a tripartite complex that is required for cytolethal distending toxin activity, Infect. Immun. 69:4358–4365.

Layton, S.L., Morgan, M.J., Cole, K., Kwon, Y.M., Donoghue, D.J., Hargis, B.M., Pumford, N.R. 2011. Evaluation of Salmonellavectored Campylobacter peptide epitopes for reduction of Campylobacter jejuni in broiler chickens, Clin. Vaccine Immunol. 18(3):449-454.

Leskinen, S.D., Lim, D.V. 2008. Rapid ultrafiltration concentration and biosensor detection of enterococci from large volumes of Florida recreational water, Appl. Environ. Microbiol., 74(15):4792– 4798.

Lindblom, G.B., Sjogren, E., Kaijser, B. 1986. Natural Campylobacter colo nization in chickens raised under different environmental conditions, J. Hyg. 96:385–391.

Man, S.M.2011. The clinical importance of emerging Campylobacter species, Nat Rev Gastro Hepat. 8(12):669-685.

Masdor, N.A., Altintas, Z., Tothill, I.E. 2017. Surface plasmon resonance immunosensor for the detection of Campylobacter jejuni, Chem. Aust. 5:1–15.

Messaoudi, S., Kergourlay, G., Rossero, A., Ferchichi, M., Prevost, H., Drider, D., Manai, M., Dousset, X. 2011. Identification of lactobacilli residing in chicken ceca with antagonism against Campylobacter, Int Microbiol., 14(2):103-110.

Myers, A.L., Jackson, M.A., Selvarangan, R. 2011. False positive results of Campylobacter rapid antigen testing, Pediatr. Infect. Dis. J. 30:542.

Newell, D.G., Fearnley, C. 2003. Sources of Campylobacter colonization in broiler chickens, Appl. Environ. Microbiol., 69(8):4343-4351.

Park, S.H., Aydin, M., Khatiwara, A., Dolan, M.C., Gilmore, D.F., Bouldin, J.L. 2014. Current and emerging technologies for rapid detection and characterization of Salmonella in poultry and poultry products, Food Microbiol., 38:250–262.

Qian, H., Pang, E., Du, Q., Chang, J., Dong, J., Toh, S.L., Ng, F.K., Tan, A.L., Kwang, J. 2008. Production of a monoclonal antibody specific

for the major outer membrane protein of Campylobacter jejuni and characterization of the epitope, Appl. Environ. Microbiol., 74(3):833-839.

Rainey, P.B., Hansen, S.K., Haagensen, J.A.J., Molin, S. 2007. Evolution of species interactions in a biofilm community, Nature. 445:533–536.

Rasmussen, H.N., Olsen, J.E., Jørgensen, K. and Rasmussen, O.F. 1996. Detection of Campylobacter jejuni and Camp. coli in chicken faecal samples by PCR, Lett Appl Microbiol., 23(5):363-366.

Reis, L.P., Menezes, L.D.M., Lima, G.K., Santos, E.L.D.S., Dorneles, E.M.S., Assis, D.C.S.D., Lage, A.P., Cançado, S.D.V., Figueiredo, T.C.D. 2018. Detection of Campylobacter spp. in chilled and frozen broiler carcasses comparing immunoassay, PCR and real time PCR methods, Ciênc. Rural. 48(2).

Rice, B.E., Rollins, D.M., Mallinson, E.T., Carr, L., Joseph, S.W. 1997. Campylobacter jejuni in broiler chickens: colonization and humoral immunity following oral vaccination and experimental infection, Vaccine. 15(17-18):1922-1932.

Rodrigues, R.C., Haddad, N., Chevret, D., Cappelier, J.M., Tresse, O. 2016. Comparsion of proteomics profiles of Campylobacter jejuni strain Bf under microaerobic and aerobic conditions, Front. Microbiol., 7:1–12.

Sahin, O., Morishita, T.Y., Zhang, Q. 2002. Campylobacter colonization in poultry: sources of infection and modes of transmission, Anim. Health Res. Rev. 3:95–105.

Sails, A.D., Bolton, F.J., Fox, A.J., Wareing, D.R., Greenway, D.L. 2002. Detection of Campylobacter jejuni and Campylobacter coli in environmental waters by PCR enzyme-linked immunosorbent assay, Appl. Environ. Microbiol., 68:1319–1324.

Silva, J., Leite, D., Fernandes, M., Mena, C., Gibbs, P.A., Teixeira, P. 2011. Campylobacter spp. as a foodborne pathogen: a review, Front. Microbiol., 2:200.

Sinha, S., Prasad, K.N., Pradhan, S., Jain, D., Jha, S. 2004. Detection of preceding Campylobacter jejuni infection by polymerase chain reaction in patients with Guillain-Barré syndrome, Trans. R. Soc. Trop. Med. Hyg. 98(6):342-346.

Snelling, W., Matsuda, M., Moore, J., Dooley, J. 2005. Under the microscope - Campylobacter jejuni, Lett Appl. Microbiol., 41:297–302.

Steven, C.R., Kristina, M.F., Chaney, W.E., Shi, Z., Pavlidis, H., Yang, Y. 2018. Developments in rapid detection methods for the detection of foodborne Campylobacter in the United States, Front. Microbiol., 9:3280-3389.

The European Food Safety Authority (EFSA). 2011. Scientific Opinion on Campylobacter in broiler meat production: control options and performance objectives and/or targets at different stages of the food chain, EFSA Journal 9:2105.

Van Deun, K., Haesebrouck, F., Heyndrickx, M., Favoreel, H., Dewulf, J., Ceelen, L., Dumez, L., Messens, W., Leleu, S., Van Immerseel, F., Ducatelle, R. 2007. Virulence properties of Campylobacter jejuni isolates of poultry and human origin, J. Med. Microbiol., 56(10):1284-1289.

Wagenaar, J.A., French, N.P., Havelaar, A.H. 2013. Preventing Campylobacter at the source: why is it so difficult, Clin Infect Dis. 57:1600–1606.

White P.L., Baker A.R., James W.O. 1997. Strategies to control Salmonella and Campylobacter in raw poultry products, Rev. Sci. Tech. 16:525–541.

Zeng, D., Chen, Z., Jiang, Y., Xue, F., Li, B. 2016. Advances and challenges in viability detection of foodborne pathogens, Front. Microbiol., 7:1–12.

Zhao, C., Ge, B., Villena, J.D., Sudler, R., Yeh, E., Zhao, S. 2001. Prevalence of Campylobacter spp., Escherichia coli, and Salmonella serovars in retail chicken, turkey, pork, and beef from the greater Washington, D.C. area, Appl. Envrion. Microbiol., 67:5431–5436.

Zilbauer, M., Dorrell, N., Wren, B.W., Bajaj-Elliott, M. 2008. Campylobacter jejuni mediated disease pathogenesis: an update, Trans. R. Soc. Trop. Med. Hyg. 120:123–129.