Food Microbiology

Incidence and ecology of Campylobacter jejuni and coli in animals

S.M. Horrocks a, R.C. Anderson a,*, D.J. Nisbet a, S.C. Ricke b

a United States Department of Agriculture, Agricultural Research Service, Southern Plains Agricultural Research Center, Food and Feed Safety Research Unit, 2881 F & B Road, College Station, TX 77845, USA
b Center for Food Safety-IFSE, Department of Food Science, University of Arkansas, Fayetteville, AR 72704, USA

1. Introduction

Campylobacter are small, curved-to-spiral shaped, flagellated Gram-negative rods, ranging from 0.5 to 8 μm in length and from 0.2 to 0.5 μm wide [1]. Of the 17 species within the genus Campylobacter [2,3], Campylobacter jejuni and Campylobacter coli are the most important from a food safety point of view [4]. Since its emergence as a foodborne pathogen in the 1970s, Campylobacter has become one of the most common causative agents of bacterial foodborne illness [4,5]. Since its initial emergence in the 1970s, Campylobacter has become one of the most common causative agents of bacterial foodborne illness. Campylobacter species readily colonize the gastrointestinal tracts of domestic, feral and wild animals and while they rarely cause clinical disease in food animals, they can produce severe acute gastroenteritis in humans. Prevalence of Campylobacter in food animals can exceed 80% thus challenging processors to employ post-harvest pathogen reduction strategies. Reduction of pathogens before arrival to the abattoir is also of interest because the implementation of pre-harvest interventions may compliment existing post-harvest control techniques to further diminish possible retail sources of infection. Such multiple hurdle approaches that simultaneously utilize pre- and post-harvest control techniques are expected to be the most effective approach for decreasing human illness associated with foodborne pathogens.

The development of more sensitive detection methods has allowed for more accurate detection, isolation, and classification of Campylobacter spp. These advances in surveillance technology have provided improved information on the prevalence of Campylobacter spp. worldwide and now demonstrate that this pathogen can be interspecies specific rather than just limited to warm blooded hosts as was once thought.

Control of Campylobacter is presently accomplished by common cleaning and preparation practices within processing plants. The application of acid sprays, irradiation methods, chlorine and hot water rinses, and post-chilling methods have been effective in reducing the pathogen, yet contamination of product still occurs as evidenced by recovery of the organism in supermarket retail raw meats [12]. Stronger and more effective on-farm, pre-harvest control methods, combined with new and improved sanitizing techniques in food processing, may help to ensure the safety of consumer products. Eliminating or significantly reducing Campylobacter on the farm, and increasing processing hygiene practices can be effective in decreasing Campylobacter prevalence within retail meat and vegetable products, as well as reducing Campylobacter within environmental sewage and watersheds.

2. Prevalence and ecology

2.1. Overall occurrence

In 2005, Campylobacter accounted for greater than 34% of the 16,614 laboratory-confirmed infection cases report by The Foodborne Diseases Active Surveillance Network (FoodNet) [13]. Yet despite representing an overall 30% decrease in infections from that reported in 1996–1998 [13], Campylobacter are still a leading cause of human foodborne poisoning [13]. Campylobacter spp. colonize...
and their prevalence in cattle, swine and poultry can exceed 80% [15–19]. To date, *Campylobacter* have been detected nearly everywhere from farm and urban environments to slaughter plants, as well as isolated from humans, wild birds and mammals, companion animals, drinking water, farm production animals, common seals (*Phoca vitulina*) and one harbor porpoise (*Phocone phocoena*) [214–32].

### 2.2. Human incidence

The majority of *Campylobacter* infections in humans originate from consumption of raw or undercooked meat products, however, unpasteurized milk, raw vegetables, environmental water sources, and vegetables are all potential reservoirs. One of the most common ways of acquiring a *Campylobacter* infection is during traveling. The term “Travelers’ Diarrhea” initially attributed to enteropathogens such as *Escherichia coli* has now been identified in diarrheic patients with *Campylobacter* infections. Acute symptoms of travelers’ diarrhea caused by a *Campylobacter* infection have been reported and studied in US military personnel deployed to Thailand in which positive stool samples have been observed in 33 –55% of patients with diarrheic symptoms [33–36]. Approximately 13,000 cases of travelers’ diarrhea in individuals from England and Wales have been reported as caused by *Campylobacter coli* [7]. *Campylobacter* infections have also been reported in non-traveling residents and studies on domestically acquired *Campylobacter* infections in Finnish patients have been reported [37]. Of the 3303 cases confirmed in Finland between July 1 and September 30, 1999, 533 isolates were identified resulting in an infection rate of 41.2 C. jejuni cases per 100,000 individuals. This number is much higher than CDCs report from FoodNet in 2005 [13] of 12.72 per 100,000 US individuals which has drastically decreased from 25.2 per 100,000 US individuals in 1997 [38]. It has also been suggested that those individuals native to developing countries with high *Campylobacter* prevalence may acquire natural immunity once exposed to a *Campylobacter* infection. Walz et al. [34] reported individuals with an elevated IgA titer before traveling to Thailand had a decreased risk of acquiring campylobacteriosis than those with IgA titers less than 450.

### 2.3. Poultry

Generally, *Campylobacter* colonize in high concentrations in the cecum and colon of poultry but can be found in the crop as well [39,40]. Since thermophilic *Campylobacter* grow optimally at temperatures near 42 °C [141], the higher metabolic temperatures (42 °C) found in poultry species may predispose poultry to be a prominent reservoir for thermotolerant *Campylobacter*. The increased temperature may allow thermophilic species to regulate gene expression that benefits motility and energy regulation based on specific growth requirements within a particular environmental temperature [42–44].

Several risk factors can be linked to colonization and transmission of *Campylobacter* spp. in broiler flocks such as flock size, age of birds, environmental water supplies, insects and even airborne isolates. Adkin et al. [45] identified 37 contributing factors to *Campylobacter* infection in broilers. Although seasonal and hygienic variables were shown to be possible contributors to infection, some of the most important factors for *Campylobacter* infection in birds included the presence and number of contaminated broiler houses on the same farm, and the interaction between birds and on-site workers. Transmission of *Campylobacter* from infected birds to humans is possible and risk factors often increase as contact between them increases. Nadeau et al. [46] found genotypic similarity of multiple *Campylobacter* isolates between human and poultry genotypes with the majority of the birds colonized with C. jejuni. It has been suggested that *Campylobacter* colonization may be host specific, limiting common serotypes between humans and poultry [19,31,47]. According to a study by El-Shibiny et al. [48], C. coli was found at high frequency in poultry flocks, with 38 of 42 (90%) positive samples recovered from free ranging poultry flocks and comprised as much as 50% of the total *Campylobacter* isolates within one particular flock.

Once *Campylobacter* is established within an individual bird, horizontal transmission often occurs rapidly through the flock. *Campylobacter* has been isolated from poultry as early as 8 days in free ranging chickens [48]; however, the average time for colonization of a flock takes several weeks [49–51]. The number of colony forming units (CFU) necessary to initialize colonization within birds may play a key role in horizontal transmission. One day old chicks challenged with only 40 CFU of *C. jejuni* strain 81116 recovered from a colonized cecum were colonized to populations as high as 107 CFU/g of cecum contents [52]. Although a much higher dose was needed to initially colonize the primary chicken model, the study suggests a much smaller dose may be needed for horizontal transmission within flocks. This could also explain the rapid colonization and detection of *C. jejuni* within broiler houses once *Campylobacter* is prevalent. Occasionally, however, some flocks in close proximity to infected flocks are never colonized, or at least not colonized within the surveillance times of the respective studies [50,51,53].

Other studies concerned with vertical and horizontal transmission of *Campylobacter* spp. within poultry flocks have been performed, but the evidence that favors vertical transmission is still open to debate [19,47,54–56]. Investigations on vertical transmission have shown that *C. jejuni* may potentially enter the eggshell under specific conditions [57] but the majority of supporting evidence does not suggest that vertical transmission of *Campylobacter* is a significant risk factor for the colonization of newly hatched chicks. Bull and colleagues [49] were unable to confirm vertical transmission from parents to progeny in sampled flocks as prevailing subtypes identified in colonized flocks were comparable to airborne subtypes identified either inside or outside of the broiler house. Studies on the aerosol transmission of *Campylobacter* have been reported in *Campylobacter* positive pens, while negative samples were detected in *Campylobacter*–free pens, thus raising the question as to whether or not *Campylobacter* transmission through the air is possible [51].

Although the predominant *Campylobacter* species such as *C. jejuni* and *C. coli* are often isolated and reported within most poultry flocks, *Campylobacter* diversity has also been extensively reported on poultry farms [48,49,53,58]. Initial colonization of specific *Campylobacter* subtypes has been shown to differ from the dominant subtypes prevalent at the time of slaughter [49]. The shift from one dominant species to another is poorly understood, but may reflect seasonal variations or environmental sources. At poultry processing plants, *Campylobacter* is predominantly found on the skin of infected birds mostly due to inevitable contamination from cecal and gut contents during the evisceration process. However, contamination within the muscle has also been reported in retail meat. Scherer et al. [59] found that nearly half of all retail packaged chicken legs were contaminated on the skin alone, less than 1% of samples were positive within the muscle alone while the contamination of both skin and muscle together accounted for 27%. Another study reported a *C. jejuni* incidence of greater than 71% isolated from retail chicken products in Japan [60]. Transportation coops to and from the processing plants have been shown to amplify cross-contamination between birds while detectable CFU of *Campylobacter* in chill and scald water were also observed in some plants [50]. During a 3-year surveillance study in the United Kingdom, the overall prevalence of *Salmonella* from
fresh poultry samples, retail, butchers, and frozen samples declined 41.7% while *Campylobacter* numbers declined only approximately 3% overall [61].

2.4. Cattle

In most cattle, *C. jejuni* is the prominent species recovered [62–65] although Bae et al. [62] reported that the prevalence of *C. coli* was near that of *C. jejuni* (20 versus 23.8%, respectively) in calf-rearing operations. In studies that have examined the prevalence and concentration of *Campylobacter* spp. at different gastrointestinal sites, all found that *Campylobacter* reside less frequently and at lower concentrations in the rumen than the lower gastrointestinal tract [66–68]. Grau [66] speculated that *Campylobacter* do not grow well within the rumen and that their presence was most likely the result of recent ingestion. The gallbladder, mucosal tissue [69] and bile [60] have been shown to harbor likely the result of recent ingestion. The gallbladder, mucosal tissue [69] and bile [60] have been shown to harbor likely the result of recent ingestion.

The incidence of *Campylobacter* has been reported to be higher in concentrate rather than forage-fed cattle [66,71,72] possibly due to increased stock densities, high frequency of shared access of cattle to community feed and water troughs and constant physical contact with feces from other animals during confinement. Likewise, Beach et al. [72] found higher *Campylobacter* prevalence in feedlot (64% and 68%) compared to adult pasture cattle (6.3 and 7.3%) cattle regardless of whether sampled before and after transport to the abattoir, respectively, thus further suggesting that confinement may promote increased carriage of *Campylobacter* within cattle herds. The observation that prevalence differed little between pre- and post-shipment indicates that transport may not affect *Campylobacter* shedding in cattle. *Campylobacter*-positive swabs from hides were more prevalent in feedlot cattle than adult pasture cattle. For feedlot cattle, there was a significant decrease in hide contamination pre- and post-transport to the slaughter facility but not for the pasture cattle. Besser et al. [73] reported a near 60% increase in fecal shedding of *C. jejuni* in feedlot cattle within 4 months from their initial arrival to the feedlot, which further supports the prevailing hypothesis that confinement of feedlot cattle may promote horizontal transmission and increased carriage of *Campylobacter* within a herd.

*Campylobacter* prevalence on conventional and antimicrobial free dairy farms in Wisconsin was shown to be similar at 29.1 and 26.7% respectively [74], while the prevalence of *Campylobacter* in calves was significantly higher than in cows and significantly higher in smaller farms than large farms [74]. *Campylobacter jejuni* isolates have been isolated from bulk tank milk in eastern South Dakota and western Minnesota from 9.2% of 131 samples, while *Salmonella* was prevalent in only 6.1% of samples [75]. Other studies in Ireland and China reported 1 of 62 (1.6%), and 82 of 300 (27.3%) raw milk samples to be positive for *Campylobacter*, respectively [76,77].

2.5. Companion animals

There has been considerable speculation about the possible transmission of *Campylobacter* from household pets to humans via constant direct physical contact. Direct transmission of *C. jejuni* from canine species to human patients has been observed [21,78] and the possibility of *Campylobacter* transmission from other household pets to humans is still a possible risk factor for campylobacteriosis.

Domesticated pets are known to harbor *Campylobacter* spp. in their digestive tracts, with incidences ranging from 11% to as much as 92% of stool samples when evaluated and characterized by either culture, polymerase chain reaction (PCR), or pulse-field gel electrophoresis (PFGE) [21,27,79,80]. Most animals are diversely colonized with numerous *Campylobacter* spp. such as observed in one study where 16 different species of *Campylobacter* were isolated in cats over a 6-year period [79]. In a 2-year study with Danish canine species, *C. jejuni* was isolated from 56 of the 278 positive samples while *C. upsaliensis* accounted for the majority (75%) of isolates [27]. *Campylobacter upsaliensis* and *C. helveticus* are the prevalent species of *Campylobacter* in dogs and cats, respectively [25,27,79–81]; however, *C. jejuni* can be found and its prevalence has been reported to be higher in dogs less than 1 year old (22%) than in dogs between 1 and 2 years of age (4.7%) has been reported [27].

2.6. Swine

Unlike poultry and cattle, *C. coli* is the more common *Campylobacter* species recovered from swine. In some studies, for instance, *C. coli* have been recovered from swine fecal samples at greater than 99% [82,83], Jensen et al. [84] studied the establishment of *C. coli* and *C. jejuni* in outdoor organically-reared pigs to monitor potential shifts from *C. coli* to *C. jejuni* in intestinal colonization. Their results demonstrated excessive fluctuations in numbers of swine colonized by *C. jejuni*, with recoveries ranging from 0, 18.8 and 78.6% among the three trials, but *C. jejuni* was never more prevalent than *C. coli* [84]. Despite being recognized as the minor *Campylobacter* species in swine, high prevalence of *C. jejuni* has been observed in cecal or rectal contents of guits, sows, and weaned piglets (76, 89, and 82%, respectively) [85]. In their report, *C. coli* (68%) was only more prevalent than *C. jejuni* (31.7%) in neonates when isolated within 24 h of birth.

Alter et al. [83] were unable to detect shedding of *C. coli* on one farm within 24 h of birth but most of the organically-reared pigs were colonized (75%) by 1 week of age. Transmission of *Campylobacter* species, primarily *C. jejuni* from other livestock species within the same farm could be a possibility; however, this has yet to be proven conclusively [86]. Isolation methods and growth fluctuations, particularly in environmental samples, may underes- timate *Campylobacter* prevalence on farms. Alter et al. [83] reported undetectable amounts of *Campylobacter* in both feedstuffs and drinking water on seven organic pig farms and only 1 of 97 water troughs were positive for *C. coli*. Within 24 weeks, the incidence of *C. coli* ranged from zero (undetected in neonates) to greater than 78% in fattened pigs post-transport to the abattoir.

The use of antibiotics may also alter a *Campylobacter* prevalence within the host. Thakur and Gebreyesus [82] reported that swine nurseries free of antibiotics exhibited a 50% increase in the number of *C. coli* isolates than recovered from traditional farms that use antibiotics. At the finishing farm, *C. coli* isolates were found in all pigs at approximately the same concentration regardless of previous antibiotic use [82]. *Campylobacter* colonization in all pigs is probably due to on the farm transmission through fecal material [83]. Piglets likely acquire *Campylobacter* from their dams as Harvey et al. [87] reported reduced rectal concentrations in neonatal pigs removed from their maternal sows and reared within separate nursery facilities compared to those piglets that remained on the sow for a period of 20 days.

In processing operations, individual pig carcasses may be sub- jected to multiple cleaning stations and processing equipment. A study detecting the prevalence of *Campylobacter* in swine through the different processing stations and comparing carcass, colon and rectal samples throughout a slaughter operation was
conducted by Pearce et al. [16]. When results from four different recovery methods were compared, C. coli was found in 151 of 202 isolates with recovery of C. jejuni accounting for only 1% of the samples tested. Malakauskas et al. [88] reported that C. coli was prevalent in 92 of 120 isolates obtained from fecal, carcasses and slaughter line surfaces combined, while C. jejuni isolates accounted for 28 of 120 of positive samples recovered from carcasses and slaughter line surfaces.

Campylobacter jejuni has been shown to be more resistant to selective stresses than C. coli [89]. These selective stresses may negatively influence the survivability of C. coli through processing methods such as chilling or air exposure. General processing methods such as chilling or rinsing with chlorinated water may be enough to effectively decrease less resilient C. coli, thereby decreasing total Campylobacter concentrations in retail pork products.

2.7. Sheep

The prevalence of Campylobacter in ovine species has not been as extensively studied as in other agriculture based animals. Campylobacter spp., predominantly Campylobacter fetus subsp. fetus in pregnant ewes has been known to cause acute septic abortions [90–93] and is the number one cause of abortions in sheep. Vertical transmission of Campylobacter in sheep may also play a role in initial colonization of lambs. Campylobacter isolates within gut contents (13/58) of aborted fetuses have also been detected [94].

Campylobacter spp. have been isolated from ovine liver [70,93], gallbladder [95], gut contents [95–97], and feces [96,98]. They have also been isolated from sheep carcasses at slaughter facilities at relatively low numbers (less than 1%) after chilling [99–101] while 17.5% of slaughtered sheep in Switzerland were found to harbor C. jejuni and C. coli isolates in cecal contents [96].

2.8. Wildlife and environmental sources

Wildlife serotypes have been reported to be significantly different from human or poultry serotypes when compared to the O:2 and O:4 complex types [47], while serotype similarities between humans and poultry have been shown to exist [30,47]. The species-specific relationship suggests that colonization of Campylobacter species may necessitate adaptation for survivability measures. It is also important to note that closely related strains have been identified among humans, poultry, turkey, and canine species, all of which have been domesticated and share direct contact with a common environment [21,78].

Transmission of Campylobacter among wild animals is still poorly understood. Studies have linked wild birds, flies and other small wildlife animals as possible vectors of transmission [53,102,103]. Wesley et al. [104] reported 14 out of 15 dairy herds [53] that 17.4% of wild bird fecal specimens were positive for Campylobacter at the initial time of detection of Campylobacter spp. in the poultry feces or cecal contents.

3. Serotypes and strain differentiation

Methods to determine specific thermophilic Campylobacter strains have been available for precise identification of genomic DNA. Flagellin typing (FlaA/FlaB), PFGE and amplified fragment length polymorphism (AFLP) are commonly used to identify and compare distinct genotypes among humans and animals. Zhang et al. [106] identified the specific gene (cmp gene) encoding a specific major outer membrane protein (MOMP) commonly shared by thermophilic Campylobacter species. The cmp gene encoding the MOMP may have an important role in the pathogenesis of human Campylobacter enteritis [107,108] and is considered an excellent method of classifying strains due to its ubiquitous nature within all thermophilic Campylobacter strains [106]. The cmp gene type B2 has now been identified in Campylobacter spp. originating from humans and poultry as well as type D1 in humans and turkeys [106].

Common types of Campylobacter found in both humans and poultry have been analyzed and characterized by AFLP [109] and PFGE [110]. Penner serotypes heat-stable (HS) antigens 1, 2, 4, and 21, all reported from C. jejuni isolates have been identified as shared serotypes by humans, poultry, cattle and sheep [111,112]. Serotype HS50 has been prevalent in chickens (14.4%), lamb liver (17%), ox liver (45.3%), pigs liver (5.6%) and humans (18.8%) [70]. Guévremont et al. [113] were unable to distinguish, using PFGE, genotypic similarities between swine and human isolates, with the human isolates recovered from patients experiencing diarrhea and living within close geographical proximity to the source of swine isolates. In ovine species, FlaA 16 was confirmed present in 45% of C. jejuni isolates from the intestine, while FlaA 26 was the predominant strain isolated in the feces [96]. Campylobacter coli subjected to strain differentiation were identified as subtype FlaA 8 (34%) and FlaA 1 (45%) for intestinal or gall bladder isolates respectively, while 41% from feces were restricted to FlaA 16 subtype [96]. Pulsed-field gel electrophoresis type B1 (201/293) has been determined to be the majority of C. fetus subsp. fetus isolated from sheep abortions followed by type B2 (21/293) [91].

4. Seasonal patterns

Various outbreaks and seasonal peaks of campylobacteria have been reported in the warmer months; however, other studies have failed to identify specific climatic fluctuations in Campylobacter prevalence. Stern et al. [50] measured seasonal fluctuations in Campylobacter prevalence over the course of 1 year. The majority of positive Campylobacter samples from cecal contents occurred in the spring while the majority of positive fecal samples were identified in the summer months (approximately 38% and 46%, respectively). No significant differences could be determined, however, due to the short sampling time (only one full year). In a New Zealand study during the months of August and February 64 of 113 raw chicken samples were positive for Campylobacter [112]. From the identified isolates, the study identified variable outbreaks of different C. jejuni serotypes found either in the winter or summer. Campylobacter isolation from the skin of retail chicken legs in Berlin, Germany was most prevalent in February (100%) while muscle samples showed 70% contamination in September [59] and both muscle and skin showed decreasing Campylobacter concentrations from August through December. Acute seasonal outbreaks of Campylobacter on retail raw chicken over a 3-year period in Wales were observed between March and June of 2002, April and June of 2003, and June to August of 2004, with the highest annual peaks falling within the months of June 2003, December 2003 and August 2004 [61]. Other
studies have compared the seasonal prevalence of *Campylobacter* in humans and broilers using temperature, sunlight, humidity, and precipitation [24]; however, explanations for peak outbreaks in either humans or animals have been inconclusive. More research is still needed to determine the significant risk factors for seasonal fluctuations within *Campylobacter* isolates.

5. Control and management strategies

To reduce the risk of campylobacteriosis, careful management practices focus on innovative methods to avoid cross-contamination from raw meat products. Predominantly, the reduction of contamination of raw meats is handled at the processing plants through a post-harvest cleaning process. Pre-chilled carcasses that harbor *Campylobacter* [114,115] may lead to contamination of retail consumer products. Product contamination by conventional evisceration processes fluctuates greatly depending on the processing plant [115–117]. Reduction of pathogens before arrival to the abattoir is of concern because pre-harvest interventions may diminish possible retail sources of infection, thereby decreasing human illness associated with foodborne pathogens [118].

5.1. Pre-harvest control and intervention strategies

Considering that *Campylobacter* colonization of food animals is perhaps most often due to direct physical contact among infected and uninfected animals, it becomes intuitive that interventions that target primary sources of infection should be able to reduce the infectious cycle. The application of specific management, hygiene and biosecurity practices is commonly used to potentially prevent initial infection of production animals by *Campylobacter*, but whether these can remain effective over the productive life of the animal remains doubtful for many systems [119,120]. Consequently, pre-harvest interventions that can be easily applied and adapted to various production systems to reduce concentrations of *Campylobacter* in animals prior to processing, particularly when applied in conjunction with post-harvest interventions, may yield the most effective way to reduce the risk of human infection. Antimicrobial treatments exist for reducing gastrointestinal concentrations of bacterial pathogens such as *Campylobacter*; however, because of potential residues and issues relating to antimicrobial resistance, use of antibiotics for pre-harvest control of *Campylobacter* is undesirable. Therefore, considerable research has been directed toward the development of alternative pre-harvest interventions to reduce the carriage of *Campylobacter* in food animals on the farm, but at present, none are widely available or accepted.

Because of the microaerophilic and thermophilic nature of *Campylobacter*, focusing on reducing gastrointestinal concentrations of the organism may be a superior target to suppress environmental microbial activity. Studies have demonstrated that gut concentrations of *C. jejuni* in broilers and *C. coli* in turkeys were significantly reduced when administered bacteriocins (121–123). Likewise, administration of bacteriophage significantly reduced *C. jejuni* concentrations in broilers (124). Chemical feed additives are also of interest to reduce *Campylobacter* colonization in supplementation and some, namely, short chain nitrocompound supplementation, are now being examined. Certain nitro-compounds, such as 2-nitro-1-propanol, decreased the *in vitro* survivability of *C. jejuni* and *C. coli* [125] as well as other foodborne pathogens such as *Salmonella Typhimurium*, *E. coli* O157:H7, *Enterococcus faecalis*, and *Listeria monocytogenes* [126,127]. Results from initial in vivo studies further demonstrated the potential of the nitrocompounds to reduce gastrointestinal concentrations of *Campylobacter* and *Salmonella* in poultry and swine [128,129]. The nitrocompounds markedly inhibit the oxidation of formate and hydrogen by mixed populations of gastrointestinal microbes [130], both of which are important reducing substrates used by *Campylobacter* for energy conservation during respiration on fumarate, nitrate, sulfites or oxygen [131–133].

Unlike most other gut bacteria, *Campylobacter* lack a key enzyme, 6-phosphofructokinase, involved in energy metabolism [134] and thus do not ferment sugars and carbohydrates, but rather utilize amino acids as major energy substrates. Velayudhan and colleagues [135] demonstrated that a *C. jejuni* mutant lacking L-serine dehydratase activity, and thus deficient in L-serine catabolism, was unable to colonize the avian gut and suggested that inhibiting amino acid catabolism may be a potential target to reduce the competitiveness of this bacterium in the gut. More recently, a pre-harvest intervention that targets a specific metabolic processes of *Campylobacter* has been investigated, revealing that the deaminase inhibitors diphenyliodonium chloride and thymol significantly reduced recovery of *Campylobacter jejuni* during mixed culture of porcine fecal bacteria in Bolton broth at 39 °C [136].

Another possible treatment to reduce gastrointestinal pathogens is through the administration of select intestinal microflora for competitive exclusion measures [137,138]. Competitive exclusion is achieved by administering mixed bacterial cultures orally to increase the resistance to infection by pathogens before initial colonization. However, most competitive exclusion techniques reduce *Campylobacter* populations but fail to eliminate intestinal andecal concentrations. While competitive exclusion has been used mainly in neonates to prevent colonization of undesirable microflora, it may be less effective in displacing established species. The possibility of supplementing feeds with selected organic acids exists and has reduced fecal concentrations of *Campylobacter*, but has pronounced effects on growth suppression in broiler chickens [139]. Avian species have fewer taste receptors in the oral cavity compared to those of bovine, porcine and ovine species. The addition of lactic and acetic acids to feeds intended for animal species other than poultry may decrease palatability and potentially reduce feed intake. In addition, hindgut fermentation results in the production of substantial quantities of volatile fatty acids which exhibit direct inhibitory activity against some bacteria as well as affecting indirect inhibition via alteration of colonic pH. Supplementation of acid rich feeds may intensify the amount of water needed to normalize the colonic pH leading to acute cases of diarrhea.

5.2. Post-harvest control and intervention strategies

A predominant issue in the control of foodborne pathogens is cross-contamination of carcasses within the processing facilities. Numerous treatments on carcasses have been implemented to reduce pathogens before final distribution to retail outlets. *Campylobacter jejuni* can be inactivated (99%) with only 1 ppm of free chlorine (pH 6 ––8 and 25 °C) after 15 min [140]; however, *C. jejuni* and other bacterial pathogens have been reported to increase resistance (greater than 50 fold) and survivability to chlorinated water by infecting and utilizing protozoa species as a temporary reservoir [141,142]. A pre-harvest method where chlorine was added to the drinking water was reported effective in controlling *Campylobacter* colonization in poultry [143] but was ineffective in controlling *Campylobacter* prevalence in feedlot cattle water troughs [73]. Chlorine additives are considered effective in post-harvest poultry applications, however, 40.5% of samples were positive for *Campylobacter* after chilling in water treated with as much as 50 ppm chlorine [50]. Gaseous ozone treatments to chicken meats challenged with *Salmonella infantis* or *Pseudomonas aerugi- nosa* have been unsuccessful in reducing pathogen concentrations [144] and may not be effective for controlling *Campylobacter*. Electron irradiation [145] and even hot water immersion techniques
References


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