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Incidence and ecology of *Campylobacter jejuni* and *coli* in animals

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ABSTRACT

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.

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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* spp. are now estimated to be the most common causative agent of foodborne illnesses, followed by non-typhoidal *Salmonella* and *Shigella* spp. [5]. Of the total estimated 5.2 million annual bacterial foodborne illnesses in the United States, approximately 2.4 million have been attributed to *Campylobacter jejuni* infections [4,5]. *Campylobacter coli* was estimated to cause approximately 26,000 cases of intestinal inflammatory responses in 2000 [4–7]. The prevalence of *C. jejuni* has attracted considerable research interest because in addition to being associated with acute cases of bacterial diarrhea they also contribute to post-infection traumatic risks of acquiring immune-mediated neuropathies such as Guillian Barré Syndrome or Miller Fisher Syndrome [8–10]. More recent studies suggest that *C. jejuni* infections can lead to inflammatory bowel diseases such as Crohn's Disease [11].

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

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the gastrointestinal tracts of domestic, wild and feral animals [14,15] 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 (*Phocoena phocoena*) [2,14–32].

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 [1,41], the higher metabolic temperatures (42 °C) found in poultry species may predispose poultry to be a prominant 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 10^8 to 10^9 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