Campylobacter jejuni: Public health hazards and potential control methods in poultry: a review

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ABSTRACT: Certain strains of *Campylobacter jejuni* are implicated not only in diarrhea in humans, but also in the rare, but more serious Guillain-Barré syndrome, which may be fatal. Since poultry are the major reservoirs of *C. jejuni*, reducing contamination of poultry meat with this organism will decrease risk to the human consumer. Poultry meat which is contaminated with *Campylobacter* spp. and other human enteropathogens is safe for human consumption if handled properly while raw, and cooked completely. Recent experimental studies tend to indicate that diet formulations excluding animal proteins and fat may help towards reducing colonization of *C. jejuni* in the ceca of poultry, but attempts to combine this strategy with other methods including the use of probiotics aimed at competitive exclusion of *C. jejuni*, or prebiotics, which promote the growth of beneficial bacteria in the large intestine, and thereby reduce *C. jejuni* colonization is worth studying. Whether reduction of *C. jejuni* by these methods will cause proportionate increase of *C. coli*, which is emerging as a more drug resistant human pathogen, is not known. Colonization reduction should ideally be combined with innovative approaches at the processing plant to bring down contamination to a negligible level, although presently there is no "acceptable or safe quantitative level" for campylobacters in raw chicken meat.

Keywords: *Campylobacter* spp.; Guillain-Barré; poultry; colonization; dietary factors

Contents

1. Introduction

Campylobacter spp. have long been known as a cause of diarrhea in cattle and of septic abortion in both cattle and sheep, but they have been recognized as an important cause of human illness only from the mid 1970's. Today campylobacters are recognized globally as the major etiologic agents in human diarrheal disease (Friedman et al., 2000). Among the several species of campylobacters, *C. jejuni* ssp. *jejuni* (*C. jejuni*) is most commonly isolated from diarrheal disease in humans and animals. *C. jejuni* ssp. *doylei, C. coli*, and *C. lari* are occasionally involved in human diarrhea. According to the results of a study in France (Dachet et al., 2004), *C. jejuni* represents 68% of isolates from intestinal campylobacteriosis, and *C. coli* 18% and *C. fetus* 9%. Although *C. coli* comprises a minority of human campylobacter disease, its health burden is considerable and greater than previously thought, therefore targeted research on this organism is required for its successful control (Tam et al., 2003). One of

the most serious sequelae of infection with *C. jejuni* is Guillain Barré syndrome (GBS), a neurological disease that can be fatal (Mishu and Blaser, 1993; Hariharan et al., 1996, 1999)

Epidemiological studies have revealed a major association between *Campylobacter* infection in humans and handling and consumption of raw or undercooked poultry meat (Hopkins and Scott, 1983; Oosterom et al., 1984; Deming et al., 1987). Processing and packaging of chicken meat provides conditions that allow for the survival of campylobacters. Poultry meat tends to be moist when sold and this moisture may protect campylobacters during storage (Fricker and Park, 1989). Carrier rates from chickens sampled at slaughter and market range from 22% to 95% (Rollin, 1991; Newell and Wagenaar, 2000). In a study conducted in Prince Edward Island, the majority (52%) of broiler chickens were found to harbor *C. jejuni* in their intestinal tracts (Ahmed et al., 1992). Canada follows the pattern seen in temperate countries, with the seasonal distribution peaking in late June and lasting through August and September. Slight increases in infection due to campylobacters are recorded following holidays such as Thanksgiving and Christmas as a result of increased consumption of poultry and turkey (Lior, 1996). Control methods aimed at reducing the numbers of *C. jejuni* in the intestinal tracts of chickens can contribute to a cleaner product at market and potentially, a reduction in infection among consumers. This article takes a look at recent developments, including our own work in this regard.

2. *C. jejuni* and diarrhea

Campylobacter jejuni diarrheal disease symptoms, which often include a transient watery diarrhea that progresses to a bloody diarrhea, are consistent with the idea that toxins play a role in this disease. Strains of *C. jejuni* may produce a number of toxins, mainly a cytolethal distending toxin (CDT), non-CDT cytotoxins, including Shiga toxins, and hemolysins. (Picket, 2000). Cholera-related enterotoxins have been demonstrated in several strains of *C. jejuni* originating from humans and a monkey from the United States, and strains from broiler chickens in Prince Edward Island (Hariharan and Panigrahi, 1990; Ahmed et al., 1992). Every *C. jejuni* strain that colonizes chickens may have the potential to cause human diarrhea and even to cause severe postinfectious neuropathy such as GBS (Duim et al., 2000).

3. GBS and *C. jejuni*

The clinical and epidemiological features of GBS, an acute disease of the peripheral nervous system of humans has been summarized by Hughes and Rees (1997). This disease, first described by Guillain, Barré and Srohl in 1916, is characterized by ascending paralysis, conduction block with segmental demyelination of the nerves, macrophage and lymphocytic infiltration of the nerves, and elevated protein with no cells or very few cells in the cerebrospinal fluid (Constantinescu et al., 1998; Nachamkin et al., 2000). GBS often follows nonspecific respiratory or other primary events including viral or bacterial infections (Ropper et al., 1991).

The first reports of the association between *Campylobacter jejuni* and GBS were published during 1982–1984 (Kaldor and Speed, 1984). Investigators from different parts of the world have isolated *C. jejuni* from the stools of patients with GBS at the onset of neurologic symptoms (Hariharan et al., 1996; Allos, 1997). In a study in the United Kingdom, GBS patients with *C. jejuni* infection, showed axonal degeneration, slow recovery, and severe residual disability more commonly than in GBS cases unrelated to *C. jejuni* (Rees et al., 1995). There has been speculation that the lipopolysaccharide (LPS) of at least some *C. jejuni* strains expresses an unidentified carbohydrate epitope shared with nerve (Griffin and Ho, 1993). A possible association between anti-ganglioside antibodies and *C. jejuni* associated GBS has been shown (Walsh et al., 1991; Gregson et al., 1993). Isolates of *C. jejuni* from GBS cases may belong to any of the serotypes, though Penner serotype O:19 is the most common one. Though in the United States about 75% of isolates of *C. jejuni* from diarrhea cases do not have GM1-like epitopes, all GBS associated isolates do possess GM1 or other ganglioside-like epitopes in the core region of LPS (Hariharan et al., 1999). Vriesendorp (1997) pointed out that the immune mechanisms by which infection with *C. jejuni* can create peripheral injury to axons instead of myelin should be studied. In this regard, Li et al. (1996) were able to demonstrate development of paralysis in chickens infected with a strain of *C. jejuni* isolated from a human GBS patient who developed acute motor axonal neuropathy. One-third of the experimental chickens developed paralysis 5–18 days after oral administration of the *C. jejuni* culture. Sciatic nerves from a few birds showed extensive Wallerian-like degeneration, and in some cases evidence of paranodal demyelination. These

workers also made the interesting observation that chickens can occasionally get spontaneous paralysis due to 'neuritogenic' strains of *C. jejuni*.

Recently, Murphy (2003) provided experimental evidence on the harmful effects of antibodies raised against certain *C. jejuni* strains which have been implicated in GBS, including Penner O:19 on femoral nerve of Sprague-Dawley rats. Although the antibodies were raised in rabbits, reduced conductivity in rat nerves, as well as reactivity on three cultured human neural cell lines, were evident.

4. Colonization of *C. jejuni* in poultry

Colonization of the gastrointestinal (GI) tracts by *C. jejuni* is the most significant contributing factor in the contamination of poultry meat (Grant et al., 1980). The organisms are transferred onto the meat during mechanized processing of the birds (Genigeorgis et al., 1986). Reducing colonization levels and prevalence in broilers during grow-out is an important part of decreasing *Campylobacter* contamination of poultry meat. However, prevalence on processed carcasses is almost always less than in the intestinal tracts of birds during production. There are fluctuations in levels throughout processing. Prevalence and levels of campylobacters on carcasses decrease after scalding but increase again followig picking, probably because of cross contamination from the mechanical picker. However, by the time carcasses exit the chill tank, *Campylobacter* spp. levels and prevalence are lower than when they entered the processing plant (Berrang and Dickens, 2000). Therefore, the ideal way to reduce the incidence of human infection would be to significantly reduce the GI colonization of these organisms in broiler chickens.

The GI colonization by *C. jejuni* in birds is very complex and involves interaction of the host and pathogen, which is influenced by many environmental factors. In a study in France, it was found that a variety of factors at farm level increased the risk of occurrence of *Campylobacter* in broiler flocks. These included high temperature and static air in poultry houses, poor water quality, absence of boot dips, and presence of litter-beetles (Refrégier-Petton et al., 2001). Vertical transmission from breeder hens is also a possibility (Cox et al., 2002).

Campylobacter is ecologically adapted to the avian GI tract and selects the ceca for colonization because the microenvironment is conducive to its survival and multiplication (Beery et al., 1988). The organism colonizes the cecal crypt mucus without attaching to the microvilli. It exhibits chemotactic attraction to l-fucose, a component of mucin, and utilizes mucin as a sole substrate for growth (Beery et al., 1988; Hugdahl et al., 1988). Therefore, changes in mucin composition are likely to influence *C. jejuni* colonization in the GI tract.

5. Control by reduction of colonization

Certain strategies, such as competitive exclusion (CE) have been utilized to take advantage of bacterial antagonism and thus reduce the colonization of pathogenic organisms in the GI tract of birds. Interestingly, certain dietary substrates cause changes in mucin composition, there by influencing the colonization of mucus-dwelling organisms. Udayamputhoor et al. (2003) compared the effects of three diet formulations containing different protein sources (animal, plant, and a combination of animal and plant) on the colonization of *Campylobacter jejuni* in the GI tract of broiler chickens. The ceca of birds receiving plant-based feed had significantly less colonization than the ceca of birds receiving the other types of feed.

A strategy that has been tried in preventing colonization of pathogens in the GI tract of birds is the manipulation of indigenous microflora, and reduction of pathogens by CE. Nurmi and Rantala (1973) introduced the concept of CE, and reduced the colonization of *Salmonella* in chicks using intestinal flora of adult chickens. The introduction of flora from an adult bird into a day-old chick speeds the maturation process of the gut microflora and increases the resistance of most chicks to colonization by *Salmonella*. Bailey (1988) noted that the CE technique showed a slight reduction to as much as a four-fold reduction in the number of salmonellae, and suggested an integrated approach using CE and other control measures at farm level for colonization control of *Salmonella* in poultry. In the subsequent years, the CE approach led to the experimental use of prebiotics, probiotics, and synbiotics for reduction of colonization of enterpathogens in poultry and farm animals. Prebiotics are oligosaccharides that are not hydrolyzed in the small intestine but modify the composition of microflora in the large intestine. The objective of prebiotics is to promote the growth of specific beneficial bacteria such as *Bifidobacterium* spp. (Collins and Gibson, 1999). Probiotics, according to Fuller (1989) consist of live microbial feed supplements which beneficially affect the host ani-

mal by improving its intestinal microbial balance. The major components of probiotics commonly used in farm animals are *Lactobacillus*, *Bifidobacterium*, *Streptococcus*, *Pediococcus*, *Enterococcus*, *Bacillus*, yeasts (*Saccharomyces*), and filamentous fungi such as *Aspergillus* and *Torulopsis* (Berg, 1998). Synbiotics are probiotics and prebiotics used in combination. Examples are *Bifidobacterium* with fructooligosaccharide and *Lactobacillus* with lactitol. These combinations may improve survival of the probiotic organism because its specific substrate is readily available for fermentation (Collins and Gibson, 1999). However, the results with respect to the effects of prebiotics and probiotics on broiler performance and nutrient utilization is quite variable and unpredictable. All these strategies, invariably manipulate the gastrointestinal miroflora so that growth of some beneficial organisms is favored to suppress the colonization by pathogens. But some strategies reported to be helpful in reducing the colonization of *Salmonella* spp. have not been found useful in the case of *Campylobacter* spp. *Salmonella* colonizes the epithelium of the lower intestinal tract, mainly the cecum, whereas *Campylobacter* spp. are found colonizing crypt mucus without attaching to crypt microvilli. *Campylobacter jejuni* does not adhere to or penetrate epithelial cells (Beery et al., 1988, Meinersmann et al., 1991). Hence, strategies that target organisms found in the epithelium, such as receptor antagonism may not be the best in reducing colonization of *Campylobacter*. Mucous and crypt dwelling microorganisms have been used alone or in combination with other intestinal bacteria from chickens to competitively exclude *Campylobacter* colonization in poultry. These include mucus-adapted, curved bacteria resembling campylobacters called K-bcteria (Aho et al., 1992), and members of *Enterobacteriaceae*, capable of using mucin as sole substrate for growth, and producing anti-*C. jejuni* metabolites (Schoeni and Doyle, 1992). Intervention strategies that are successful with *Salmonella* spp. have also been found to be somewhat successful in *C. jejuni* colonization reduction, because of the concentration of campylobacters in cecal crypts. These included avian specific probiotics containing *Lactobacillus acidophilus*, and *Streptococcus faecium* (Morishita et al., 1997). Compared to conventional CE, use of mucosal CE microflora has recently been found to reduce *Campylobacter* colonization significantly more (Stern et al., 2001). Recently, Heres et al. (2004) noted that chickens fed acidified feed were somewhat less susceptible to an infection with *Campylobacter* than were chickens fed conventional feed. A combined use of CE strategy with prebiotics on a diet designed exclusively of plant origin may contribute a great deal in reducing the colonization of *C. jejuni* in the GI tract of birds.

According to the French antimicrobial surveillance data (Avrain et al., 2003; Desmonts et al., 2003)), between 1999 and 2002 there was a change in the *C. jejuni*/*C. coli* ratio in the ceca of standard broiler chicken, with a decrease of *C. jejuni*, and proportionate increase of *C. coli*. It will be important to monitor the *Campylobacter* species ratio in the future to determine whether this situation will remain stable. Production factors such as a ban on animal proteins and fat, and most of the growth promoters are hypothetical explanations for this observed phenomenon, but other yet unsuspected factors may explain the species ratio evolution. Furthermore, it is of utmost importance to monitor in the future the variation of *C. coli* in human campylobacteriosis as suggested by Tam et al. (2003), and its antimicrobial resistance, as *C. coli*, compared to *C. jejuni* is more resistant to a variety of animicrobials (macrolides in particular) that are useful for human therapy. It is also important to determine if diet changes and antimicrobial drugs will affect the serotypes of *C. jejuni* colonizing the chicken gut.

Other control measures including improved biosecurity protocols, good water supply in the poultry farm, and prevention of cross contamination during processing procedures in the meat plant should be implemented to decrease carcass contamination. Control measures at farm level and meat processing plant should be combined into an integrated approach for pathogen reduction. Additionally, it is important to educate people about the risks associated with handling raw poultry meat, and consuming undercooked or contaminated products.

6. Conclusions

Poultry are reservoirs of *C. jejuni*, causing diarrhea as well as rare cases of GBS, and pose a risk, but if poultry meat is properly handled and cooked, the consumer is at no risk. Reduction of intestinal colonization of chickens by dietary manipulation may be successful, and worth the effort, as indicated by the limited experimental studies or field data. Further research on combining dietary change with the use of prebiotics or probiotics on the colonization of *C. jejuni* and the less common *C. coli* may be rewarding.

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