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# *Campylobacter* colonization in poultry: sources of infection and modes of transmission

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## Abstract

Since its recognition as a human pathogen in the early 1970s, *Campylobacter jejuni* has now emerged as the leading bacterial cause of food-borne gastroenteritis in developed countries. Poultry, particularly chickens, account for the majority of human infections caused by *Campylobacter*. Reduction or elimination of this pathogen in the poultry reservoir is an essential step in minimizing the public health problem; however, farm-based intervention measures are still not available because of the lack of understanding of the ecological aspects of *C. jejuni* on poultry farms. Although *Campylobacter* is highly prevalent in poultry production systems, how poultry flocks become infected with this organism is still unknown. Many investigations indicate that horizontal transmission from environmental sources is the primary route of flock infections by *Campylobacter*. However, some recent studies also suggest the possibility of vertical transmission from breeder to progeny flocks. The transmission of the organism is not well understood, but it is likely to be through both vertical and horizontal transmission and may be affected by the immune status of the poultry host and the environmental conditions in the production system. Intervention strategies for *Campylobacter* infection in poultry should consider the complex nature of its transmission and may require the use of multiple approaches that target different segments of the poultry production system.

## Introduction

*Campylobacter jejuni*, a Gram-negative bacterium, is the most commonly reported bacterial cause of human food-borne infection in the USA and other developed countries. An estimated 2.1–2.5 million cases of human campylobacteriosis, characterized by watery and/or bloody diarrhea, occur annually in the USA (Blaser, 1997; Altekruse *et al.*, 1999; Friedman *et al.*, 2000). This pathogenic bacterium is also associated with Guillain–Barré syndrome, a demyelinating disorder which causes acute neuromuscular paralysis, respiratory muscle compromise and death (Nachamkin *et al.*, 1998; Wassenaar and Blaser, 1999). The majority of human infections result from consumption of undercooked poultry or other food products cross-contaminated with raw poultry meat during food preparation (Evans, 1992; Jacobs-Reitsma, 2000; Corry and Atabay, 2001). However, other risk factors besides poultry have been reported, including contact with house pets and the consumption of raw milk, untreated water and undercooked beef and pork (Shane, 1992; Blaser, 1997; Corry and Atabay, 2001). Reduction or elimination of poultry contamination by *C. jejuni* will thus greatly decrease the risk of campylobacteriosis for public health. To achieve

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this goal, it is essential to understand the ecology of *Campylobacter* in the poultry production system so that effective intervention strategies can be designed and implemented at the preharvest stage. Although numerous farm-based studies have been conducted in the past decades, the sources of flock infection, modes of transmission and the host and environmental factors affecting the spread of *Campylobacter* are still poorly understood. There has been a major debate on whether vertical or horizontal transmission is responsible for the introduction of *Campylobacter* into chicken flocks. In this paper, we will review the current literature on *Campylobacter* ecology in poultry. In particular, we will discuss the colonization characteristics, sources of infection and modes of transmission, and various factors that may affect the spread of *Campylobacter* on poultry farms. As *Campylobacter* contamination of broiler chickens is the major public health concern, this review will focus on the data obtained in broiler production systems.

## Prevalence and colonization

Commercial poultry are the major natural reservoirs of *C. jejuni*, and up to 100% of broilers at slaughter age may harbor the organism (Jacobs-Reitsma *et al.*, 1995, 1997). The prevalence in commercial broiler flocks varies greatly depending on the age of birds (Kazwala *et al.*, 1990; Berndtson *et al.*, 1996a, b; Evans and Sayers, 2000). *Campylobacter* is rarely detected in broiler chickens less than 2–3 weeks old under commercial production conditions, although newly hatched chickens can be experimentally infected with *C. jejuni* (Shanker *et al.*, 1988; Stern *et al.*, 1988; Young *et al.*, 1999; Sahin *et al.*, 2001a). For the majority of commercial flocks, *Campylobacter* infection is usually detected after the third week of age. Once some birds become infected, *C. jejuni* spreads rapidly to most of the birds in the flock, which remain colonized up to slaughter, leading to carcass contamination at the processing plants (Jacobs-Reitsma *et al.*, 1995; Berndtson *et al.*, 1996b; Gregory *et al.*, 1997; Evans and Sayers, 2000; Shreeve *et al.*, 2000). Shedding of *Campylobacter* by chickens varies by season, being highest in the summer (Annan-Prah and Janc, 1988; Stern, 1992; Jacobs-Reitsma *et al.*, 1994; Gregory *et al.*, 1997; Evans and Sayers, 2000; Newell and Wagenaar, 2000; Wedderkopp *et al.*, 2000, 2001). Even though *C. jejuni* is highly prevalent in broiler chickens, some flocks remain free of *Campylobacter* throughout their lifespan (Annan-Prah and Janc, 1988; van de Giessen *et al.*, 1992; Humphrey *et al.*, 1993; Berndtson *et al.*, 1996b; Wedderkopp *et al.*, 2000; Stern *et al.*, 2001). *Campylobacter* is also highly prevalent in chickens raised on organic or free-range farms (Rivoal *et al.*, 1999; Heuer *et al.*, 2001), indicating that different production systems are equally vulnerable to invasion by this organism. Besides chickens, *Campylobacter* colonization also occurs in other domestic poultry species, including ducks, turkeys, ostriches and geese, with little or no clinical consequences (Yogasundram *et al.*, 1989; Wallace *et al.*, 1998; Aydin *et al.*, 2001; Ley *et al.*, 2001).

Colonization of chickens by *C. jejuni* occurs primarily in the lower intestines, where the organism is mainly found in the cecal and cloacal crypts (Beery *et al.*, 1988; Meinersmann *et al.*, 1991; Achen *et al.*, 1998). However, the organism can also be recovered to a lesser extent from the small intestines and the gizzard, and infrequently from the liver, spleen and gall bladder (Kaino *et al.*, 1988; Morishita *et al.*, 1997; Achen *et al.*, 1998; Young *et al.*, 1999). Unlike the infection in mammals (e.g. mice, swine, rabbit, monkey and humans), in which *C. jejuni* can invade intestinal epithelial cells and cause pathological changes (Caldwell *et al.*, 1983; Russell *et al.*, 1990, 1993; Babakhani *et al.*, 1993), *C. jejuni* infection in chickens has several distinct features. First, it appears that *C. jejuni* does not adhere directly to epithelial cells, but mainly locates in the mucous layer of the crypts (Beery *et al.*, 1988; Meinersman *et al.*, 1991). Secondly, no gross or microscopic lesions are induced in chickens. Thirdly, invasion of the intestinal epithelium usually does not occur. These observations indicate that *C. jejuni* is well adapted to the poultry host, and may be seen as a normal enteric flora by the host. Once a broiler chicken becomes infected, large numbers of *C. jejuni* can be detected in its intestinal tract and excreted in feces for at least 12 weeks [up to $10^8$ colony-forming units (c.f.u.)/g feces] without any apparent clinical consequences for the chicken host (Kaino *et al.*, 1988; Stern, 1992, 1995). However, cecal colonization may not always result in detectable shedding into the feces (Morishita *et al.*, 1997; Achen *et al.*, 1998; Korolik *et al.*, 1998). *Campylobacter jejuni* can also be isolated at a high rate from the crops of market-age broilers, and feed withdrawal before slaughter (a common commercial practice used to reduce fecal contamination of the carcass) significantly increases the isolation frequency from the crop (Achen *et al.*, 1998; Byrd *et al.*, 1998a, b; Willis *et al.*, 2000). However, it is not known whether the *Campylobacter* found in crops represents the organism in the ingested feces or reflects active propagation of the organism inside this organ. Thus, it would be interesting to find out if the crop serves as a natural niche for *Campylobacter* colonization in chickens.

Artificial inoculation of chickens with *C. jejuni* has revealed a number of factors that affect cecal colonization by this organism. It has been shown that the *Campylobacter* colonization rate can be influenced by the dose of inoculum (Shanker *et al.*, 1988, 1990; Stern *et al.*, 1988; Young *et al.*, 1999; Sahin *et al.*, 2001a). The minimum dose of the organism required for colonization may be as low as 35 c.f.u./bird via oral gavage (Stern *et al.*, 1988); however, the minimal infectious dose varies depending on the age of the chicken and the strain of *C.*

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*jejuni* used (Kaino *et al.*, 1988; Sahin *et al.*, 2001a). The infectious dose can also be influenced by the route of challenge. Although Young *et al.* (1999) were unable to infect 1-day-old chicks with a single *C. jejuni* strain (ATCC 33291) via the cloaca, an earlier study by Shanker *et al.* (1988) demonstrated that both 2- and 14-day-old chickens required approximately 100-fold higher inocula when challenged with oral gavage rather than by the cloacal route. Experimental studies also showed that different *C. jejuni* strains have varying colonization ability in chickens (Shanker *et al.*, 1988, 1990; Stern *et al.*, 1988; Chen and Stern, 2001; Sahin, 2001a). Replacement of one *C. jejuni* strain by another has also been observed in both natural and experimental colonization studies in chickens, which indicates the possible presence of dominant *Campylobacter* isolates with the ability to displace others (Jacobs-Reitsma *et al.*, 1995; Korolik *et al.*, 1998). Although multiple *C. jejuni* isolates with different serotypes and genotypes can frequently colonize chicken flocks during the same production cycle (van de Giessen *et al.*, 1992; Jacobs-Reitsma *et al.*, 1995; Stern *et al.*, 1997; Shreeve *et al.*, 2000), infection of a single chicken with more than one strain of *Campylobacter* is a rare observation (Korolik *et al.*, 1998). To determine the colonizing factors of *C. jejuni*, Meinersmann *et al.* (1990) compared the antigenic profiles of congenic strains with different colonizing phenotypes. The study did not reveal consistent differences between the colonizing and non-colonizing *C. jejuni* strains. However, the investigators noticed the exclusive association of a 69 kDa protein with the colonizing strain. Studies using genetically defined mutants revealed that flagella, DnaJ (heat shock protein), CiaB (*Campylobacter* invasin antigen B), PldA (phospholipase A), and CadF (*Campylobacter* adhesin to fibronectin) of *C. jejuni* were involved in the colonization of chickens (Nachamkin *et al.*, 1993; Wassenaar *et al.*, 1993; Konkel *et al.*, 1998; Ziprin *et al.*, 1999, 2001). The genome sequence of *C. jejuni* NCTC 11168 revealed the presence of hypervariable homopolymeric tracts in some of the genes encoding surface structures of this pathogen (Parkhill *et al.*, 2000). The role of these hypervariable genes in host colonization remains to be determined in future studies.

There are conflicting data regarding the susceptibility to colonization of chickens of different ages. Some studies have shown that older chickens (~2–5 weeks of age) are more susceptible to *C. jejuni* colonization than younger ones (a few days old) (Kaino *et al.*, 1988; Sahin *et al.*, 2001a), while others have indicated that they are equally susceptible to *Campylobacter* colonization (Shanker *et al.*, 1988, 1990). Colonization of chickens by *C. jejuni* can also be affected by the host lineage of chickens (Stern *et al.*, 1990; King *et al.*, 1993). Stern *et al.* (1990) compared the resistance of three crossbred commercial broiler chickens to colonization by *C. jejuni* and showed significant differences in the colonization rate of various crossbred types of birds by different *C. jejuni* strains.

A general observation, and a unique characteristic of *C. jejuni* colonization in poultry, is that this organism is usually absent in chicks less than 2–3 weeks of age under commercial conditions (Annan-Prah and Janc, 1988; Jacobs-Reitsma *et al.*, 1995; Berndtson *et al.*, 1996a; Evans and Sayers, 2000; Shreeve *et al.*, 2000; Stern *et al.*, 2001), suggesting that young chickens may have age-related resistance to *Campylobacter* colonization. However, such resistance mechanisms have not been defined. Elucidation of this phenomenon is of particular interest as this may provide valuable information for strategies to reduce or even eliminate *Campylobacter* colonization in broiler chickens until slaughter. One possible contributing factor for this resistance may be the presence of *Campylobacter*-specific maternal antibodies in young chicks. In fact, we have recently demonstrated that *C. jejuni*-specific maternal antibodies are highly prevalent in egg yolks and the sera of young broiler chickens during their first week of life, and that these antibodies are active in complement-mediated killing of certain *C. jejuni* strains *in vitro* (Sahin *et al.*, 2001c). Our recent *in vivo* studies, in which 3-day-old specific pathogen-free (SPF) chicks with maternal antibody (hatched from SPF hens inoculated with *C. jejuni*) and without maternal antibody (hatched from uninoculated SPF hens) were challenged orally with different doses of various *C. jejuni* strains, indicated that *C. jejuni*-specific maternal antibodies are partially protective against colonization with both homologous and heterologous strains (Sahin *et al.*, 2002).

Besides maternal antibodies, other age-related factors, such as differences in the stage of intestinal development and the microbial flora, may influence the colonization of chickens by *C. jejuni*. An earlier study, in which inhibitory effects of cecal contents of chickens on *Campylobacter* growth *in vitro* were investigated, indicated that cecal material from younger chicks reduced the growth of the organism dramatically, while cecal contents from older chickens had no effect on the growth of the bacterium (Humphrey *et al.*, 1989). The reason for the inhibitory effect of cecal contents on *Campylobacter* is unknown. A recent study using a 16S rRNA-based method revealed the complexity of the microbiota in the cecal contents of chickens, and indicated that there are unique bacterial species associated with different age groups (Zhu *et al.*, 2002). Together, these observations suggest a possible interfering effect of microbial flora of young chicks on *C. jejuni*; however, much research is needed to define the exact role of competitive cecal microflora on *Campylobacter* colonization in chickens (Mead, 2002).

## Horizontal transmission

Circumstantial evidence has been accumulated in favor of horizontal transmission from the environment as the

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most probable source of poultry infection by *C. jejuni*. Potential sources include old litter, untreated drinking water, other farm animals, domestic pets, wildlife species, houseflies, insects, equipment and transport vehicles, and farm workers. However, none of these suspected sources has been identified conclusively as the formal source of infection for broiler farms. This is because, in many cases, comparison of isolates from broilers and the environment by phenotypic or genotypic typing methods was not performed, leading to questions about the significance of these putative sources of infection (Rosef and Kapperud, 1983; Kazwala *et al.*, 1990; Gregory *et al.*, 1997; Stanley *et al.*, 1998a; Studer *et al.*, 1999; Craven *et al.*, 2000). In studies in which the isolates from various sources were typed, the poultry isolates were frequently found to be different from those obtained in the immediate vicinity of the chicken farms (Rosef *et al.*, 1985; van de Giessen *et al.*, 1992, 1998; Jacobs-Reitsma *et al.*, 1995; Stern *et al.*, 1997; Nesbit *et al.*, 2001; Petersen *et al.*, 2001a). In addition, *C. jejuni* was most probably detected in suspect sources after the broilers had become infected, suggesting that broilers, instead of being infected from environmental sources, might be the source of environmental contamination (Kazwala *et al.*, 1990; Jacobs-Reitsma *et al.*, 1995; Berndtson *et al.*, 1996a; Stern *et al.*, 2001). In many situations, it was very difficult to determine which event (flock infection or environmental contamination) occurred first, because no study plan was included to monitor the direction of *Campylobacter* transmission.

Since *C. jejuni* is very sensitive to oxygen and drying, the organism is generally unable to grow in feed, litter or water under normal ambient conditions (Kazwala *et al.*, 1990; Humphrey *et al.*, 1993; Jacobs-Reitsma, 2000). The organism is usually absent in fresh litter or feed samples before broilers are infected (Humphrey *et al.*, 1993; Pearson *et al.*, 1993; Jacobs-Reitsma *et al.*, 1995; Gregory *et al.*, 1997; van de Giessen *et al.*, 1998). Used litter may become contaminated by *C. jejuni* and may play a role in maintaining *C. jejuni* in the farm environment (Montrose *et al.*, 1985). However, a recent study by Payne *et al.* (1999), in which *Campylobacter* isolates were typed using randomly amplified polymorphic DNA–PCR (polymerase chain reaction) and 23S rRNA–PCR, did not support the role of litter in the transmission of the organism to successive flocks in the same poultry house. In European countries, since broiler houses are usually cleaned and disinfected and the litter is changed between consecutive flocks, litter seems an unlikely source of infection in commercial broiler production (Evans, 1992). Also, a recent nationwide epidemiological study in the USA indicated that there were no marked differences in the prevalence and onset time of *Campylobacter* shedding among flocks on different grow-out farms having different practices of litter use (Stern *et al.*, 2001). Because of its low moisture content, feed is an unlikely source for the introduction of *C. jejuni* into the broiler houses (Evans, 1992; Jacobs-Reitsma *et al.*, 1995; van de Giessen *et al.*, 1998). Feed itself, however, can be contaminated from other sources, such as feces in the chicken house (Gregory *et al.*, 1997).

Groundwater is frequently used for drinking water on poultry farms, and unchlorinated water has been implicated as the source of *C. jejuni* in broiler chickens (Kapperud *et al.*, 1993; Pearson *et al.*, 1993). Because of its microaerophilic characteristics and inability to grow below 31°C (Hazeleger *et al.*, 1998), *C. jejuni* is unlikely to propagate in environmental water. The presence of this organism in streams, rivers, groundwater and drinking water is a sign of recent contamination with feces of livestock or wild birds (Stanley *et al.*, 1998a; Jones, 2001). Therefore, it is more likely that water is a passive source of infection rather than a niche for the growth of *C. jejuni*. Also, drinking water on poultry farms generally becomes positive with *C. jejuni* only after the chickens are colonized, suggesting that drinking water is not an original source of contamination (Kazwala *et al.*, 1990; Jacobs-Reitsma *et al.*, 1995, 1997; Berndtson *et al.*, 1996b; van de Giessen *et al.*, 1998).

Insects (houseflies, darkling beetles, cockroaches, mealworms) can act as mechanical vectors and may transmit *C. jejuni* from animal reservoirs to chicken flocks (Rosef and Kapperud, 1983; Shane *et al.*, 1985; Jacobs-Reitsma *et al.*, 1995, 1997). Identical serotypes and genotypes of *Campylobacter* were isolated from both broilers and insects within broiler houses; however, the direction of spread was not determined (Rosef *et al.*, 1985; Annan-Prah and Janc, 1988; Jacobs-Reitsma *et al.*, 1995; Berndtson *et al.*, 1996a; Stern *et al.*, 1997). Insects in poultry houses were usually not positive for *C. jejuni* until the organism was isolated from broilers (Berndtson *et al.*, 1996a; Nesbit *et al.*, 2001). Therefore, the possibility that insects are an original source of infection for broiler houses is small, but insects may carry the organism from one location to another within or between flocks (Rosef and Kapperud, 1983; Shane *et al.*, 1985; Berndtson *et al.*, 1996a; Gregory *et al.*, 1997).

Several studies have shown that rodents (mice and rats) and other small wild animals, such as raccoons, can carry *C. jejuni* in their intestine, and thus these wild animals are considered likely sources of introduction of *Campylobacter* into grow-out houses (Annan-Prah and Janc, 1988; Kapperud *et al.*, 1993; Berndtson *et al.*, 1994; Nesbit *et al.*, 2001). However, *C. jejuni* was not isolated from rodents found in the vicinity of broiler houses in other studies (Jones *et al.*, 1991; Gregory *et al.*, 1997). In a recent study, the persistence of some clones of *C. jejuni* during successive broiler flock rotations was suggested to be a result of survival of the organism in such reservoirs as rodents and insects, which were able to evacuate the house during cleaning and disinfection and then return (Petersen and Wedderkopp, 2001). However, many other investigators found no evidence of transmis-

sion of *C. jejuni* from the first flock to the following flocks by persistence of the organism in broiler houses (van de Giessen *et al.*, 1992, 1998; Jacobs-Reitsma *et al.*, 1995; Gregory *et al.*, 1997). Considering the limited access of rodents into broiler houses and the effective vermin control programs in most commercial poultry production facilities, the role of rodents as a common source of infection for broiler flocks is questionable (Gregory *et al.*, 1997; Evans and Sayers, 2000).

*Campylobacter* has a wide distribution in wild birds (Luechtefeld *et al.*, 1980, 1981; Kapperud and Rosef, 1983; Kinjo *et al.*, 1983; Pacha *et al.*, 1988; Kaneuchi *et al.*, 1989; Yogasundram *et al.*, 1989; Broman *et al.*, 2000; Chuma *et al.*, 2000; Fallacara *et al.*, 2001; Jeffrey *et al.*, 2001). Owing to their great mobility, wild-living birds may spread *Campylobacter* to other animals and humans through fecal contamination of pastures, forage and surface water. Wild birds in the vicinity of poultry production facilities are often found to be infected with *C. jejuni*; however, isolates from wild birds are usually different from those of chicken origin (Rosef *et al.*, 1985; Annan-Prah and Janc, 1988; Gregory *et al.*, 1997; Nesbit *et al.*, 2001). Since wild birds have a high carriage rate of *Campylobacter* in their intestines, they should be considered a potential risk for transmission of organisms into broiler flocks (Craven *et al.*, 2000). The exact role of wild-living birds in the introduction of *Campylobacter* into broiler houses will require further studies involving comparison of isolates from broilers and wild birds by genotyping methods.

The presence of other farm animals on broiler farms, including pigs, cattle, sheep and fowls other than chickens, has been found to be associated with an increased risk of *Campylobacter* infection in broilers (Rosef *et al.*, 1985; van de Giessen *et al.*, 1992, 1998; Kapperud *et al.*, 1993; Berndtson *et al.*, 1996b; Gregory *et al.*, 1997). Gregory *et al.* (1997) indicated that cattle were the single common factor among three broiler farms positive for *C. jejuni*. In that study, cattle were found to be concurrently infected with *C. jejuni*. In a follow-up study, *C. jejuni* isolates from these cattle were shown to have the same *flaA* type as the isolates from the broilers on the same farm (Stern *et al.*, 1997). Identity of genotypes between cattle and broiler isolates from the same farm was observed in another study, and cattle were suggested to be a source of infection to the broilers on the farm (van de Giessen *et al.*, 1998). However, as pointed out by the authors (van de Giessen *et al.*, 1998), the mode of spread was not known and could have been from the broilers to the cattle. In other studies, *C. jejuni* isolated from cattle was found to be different from the isolates recovered from the broilers on the same farm (Rosef *et al.*, 1985; Jacobs-Reitsma *et al.*, 1995, 1997; Nesbit *et al.*, 2001), calling into question the role of cattle as a source of poultry infection. Nevertheless, it should be kept in mind that cattle, like sheep and other farm animals, have the potential to contaminate pastures and surface waters, which in turn may act as a source of broiler infection (Stanley *et al.*, 1998b; Jones *et al.*, 1999). Like cows, pigs are also common carriers of *Campylobacter* (Annan-Prah and Janc, 1988; Gregory *et al.*, 1997; Nesbit *et al.*, 2001). Tending pigs before entering broiler houses was indicated as a risk factor for *Campylobacter* colonization of chickens (Kapperud *et al.*, 1993). Although earlier studies found pigs and broilers to be infected with the same serotype of *C. jejuni* (Rosef *et al.*, 1985; Annan-Prah and Janc, 1988), recent studies using more discriminatory typing tools showed that pigs and broilers on the same farm were usually infected with different strains of *C. jejuni* (van de Giessen *et al.*, 1992, 1998; Jacobs-Reitsma *et al.*, 1995; Stern *et al.*, 1997). In another study, no significant association was found between colonization of broilers by *C. jejuni* and the presence of pigs on the same farm (Jacobs-Reitsma *et al.*, 1994). Also, pigs are generally infected with *C. coli* instead of *C. jejuni* (Stern, 1992; van de Giessen *et al.*, 1998). Other farm animals, such as sheep, horses, cats and dogs, can also be infected with *C. jejuni* (Stern, 1992); however, their potential role as a source of broiler infection has not been established.

Farm workers loading birds for transport to slaughter may carry *C. jejuni* from one flock to another if they move between different flocks without changing clothes and boots (Berndtson *et al.*, 1996b). The organism has been isolated from footbath water, farmer's boots and transport crates (Annan-Prah and Janc, 1988; Kazwala *et al.*, 1990; Jacobs-Reitsma, 1997; van de Giessen *et al.*, 1998; Stern *et al.*, 2001). Therefore, it is reasonable to assume that *C. jejuni* may spread between broiler flocks and farms by the movement of personnel. However, a recent study showed that two adjacent broiler houses that lacked biosecurity procedures were colonized with different genotypes (determined by *fla* typing and 23S rRNA–PCR typing) of *C. jejuni*, even though these houses shared equipment and the farmer worked in both houses using the same boots (Nesbit *et al.*, 2001).

Overall, these observations indicate that *C. jejuni* is widespread in the intestinal tract of many wild and domestic animals and birds, and ubiquitous in the poultry production environment, which makes transmission from the environment to broiler houses likely. However, there are still unresolved gaps in our understanding of the transmission of *Campylobacter* to broilers from environmental sources. Also, no single factor has been found to be the major risk for infection of broilers (Humphrey *et al.*, 1993). It is most likely that the introduction of *C. jejuni* to broiler flocks is mediated by multiple sources.

## Vertical transmission

As mentioned above, many investigators have suggested that horizontal transmission from environmental sources

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is the major source of *Campylobacter* infection for broiler flocks, and vertical transmission is unlikely. The reason underlying this prevailing theory is related to several observations. First, young broiler chickens usually lack *C. jejuni* before 2 or 3 weeks of age, even though the chicks are hatched from eggs from infected parent flocks (Annan-Prah and Janc, 1988; Shanker *et al.*, 1986; van de Giessen *et al.*, 1992, 1998; Berndtson *et al.*, 1996a). Secondly, although broilers from the same parent flocks are colonized by *C. jejuni* in some production cycles, they may be free of *Campylobacter* in other cycles (Jacobs-Reitsma, 1995; Jacobs-Reitsma *et al.*, 1995). Thirdly, broiler flocks are frequently infected with strains different from those infecting breeder flocks (Chuma *et al.*, 1997a; van de Giessen *et al.*, 1998; Petersen *et al.*, 2001b). Fourthly, chicken flocks originating from the same parent flocks do not always show similar serotypes (Berndtson *et al.*, 1996b), but broilers from different hatcheries may be infected with the same clones (Petersen and Wedderkopp, 2001). Finally, isolation of *C. jejuni* from eggs from naturally or experimentally infected chickens has been very difficult and rare (Doyle, 1984; Shanker *et al.*, 1986), and so far live *Campylobacter* cells have not been detected in hatcheries or young hatchlings (Shanker *et al.*, 1986; Annan-Prah and Janc, 1988; Kazwala *et al.*, 1990; Chuma *et al.*, 1994; Jacobs-Reitsma *et al.*, 1995; Hiett *et al.*, 2002).

Despite the observations counter to the role of vertical transmission, increasing evidence suggests that vertical transmission of *C. jejuni* may occur from breeder flocks to broiler farms through the egg. Earlier studies showed that, even if at a low level, *C. jejuni* could be isolated from both the outer (Doyle, 1984) and the inner (Shanker *et al.*, 1986) surface of eggshells laid by naturally infected commercial layers or broiler breeders. We also detected *C. jejuni* in a small number of freshly laid eggs obtained from layer chickens which were experimentally infected with *C. jejuni*, when a pool of whole eggs were mixed in a blender and subjected to selective enrichment for isolation (Sahin *et al.*, 2001b). Shane *et al.* (1986) isolated the organism from both the interior surface of the eggshell and the egg contents after swabbing feces containing *C. jejuni* onto the surface of the eggs. Following experimental infections of eggs with *C. jejuni* by either the temperature differential method (Clark and Bueschkens, 1985) or inoculation of egg albumen via direct injection (Shanker *et al.*, 1986), the organism was recovered from both the contents of unhatched eggs and from the newly hatched chicks. Our preliminary studies indicated that *C. jejuni* was able to survive up to 2 weeks in eggs with or without anti-*Campylobacter* antibody kept at 18°C after artificial injection into the egg yolk (Sahin *et al.*, 2001b), which is in contrast to the short survival rate of the organism at low temperatures *in vitro* (Solomon and Hoover, 1999; Jacobs-Reitsma, 2000). Thus, these observations (Clark and Bueschkens, 1985; Shanker *et al.*, 1986; Sahin *et al.*, 2001b) plus a recent finding that 'viable but not culturable' forms of *C. jejuni* could be resuscitated by injection into the yolk sac of embryonated eggs (Cappelier *et al.*, 1999) indicate that, once *C. jejuni* enters inside the egg, it can survive there long enough to potentially infect the hatchlings. The ability of *C. jejuni* to survive in egg yolk, even in the presence of high levels of *Campylobacter*-specific antibody, for a long time is probably related to lack of complement in the yolk.

Detection of *Campylobacter* DNA in eggs and young hatchlings has been shown in several studies. Chuma *et al.* (994) found that as many as 35% of newly hatched chicks contained *C. jejuni* DNA, as determined by a DNA–DNA hybridization method. However, the investigators were unable to detect any live *Campylobacter* cells by the enrichment culture method, suggesting that there were no live *Campylobacter* cells in the chickens, or that the organisms were in a 'viable but not culturable' state. Similarly, *C. jejuni* DNA was detected in the cecal contents of newly hatched chickens and 18-day-old embryos by PCR and/or Southern blot hybridization but not by conventional culture with selective enrichment (Chuma *et al.*, 1997b). Recently, Hiett *et al.* (2002) reported PCR detection of *Campylobacter* DNA in fluff and eggshell samples from hatcheries, although the same samples yielded no live organisms when conventional culture methods were used.

Following experimental infection of Japanese laying quails with *C. jejuni*, the organism was recovered from the eggshell surfaces and egg contents (Maruyama and Katsube, 1990). Since no *Campylobacter* was isolated from the shell surface of several eggs which had the organism in their contents, and since *C. jejuni* was isolated from the liver, matured yellow follicles and lower oviduct of these laying quails, it was thought that contamination of the egg with *C. jejuni* was caused by systemic infection of the quail's reproductive tract. *C. jejuni* has also been isolated from the ovaries and oviducts of healthy laying chicken hens (Jacobs-Reitsma, 1997; Camarda *et al.*, 2000). Camarda *et al.* (2000) compared several *C. jejuni* isolates recovered from the intestinal and reproductive tracts of laying hens using genotyping methods (*fla* typing and pulsed-field gel electrophoresis), and showed that identical *Campylobacter* strains could colonize both sites. In addition, the results suggested that colonization of the oviduct with *Campylobacter* was via an ascending infection from the cloaca, and that certain strains of *Campylobacter* could colonize the oviduct better than others. However, the exact role of infected reproductive organs in the contamination of eggs requires further research. *C. jejuni* was able to invade and survive in egg contents for at least 3 days after immersion of Japanese quail eggs into bacterial suspensions for 30 s (Maruyama *et al.*, 1995). The same study also showed that the organism could survive up to 86 days at 4°C after injec-

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tion into egg yolk, suggesting that *C. jejuni* could infect egg contents and survive there for a long time.

Strict adherence to biosecurity on poultry farms has had limited success in preventing the infection of broilers with *C. jejuni* (Berndtson *et al.*, 1996a, b; Shreeve *et al.*, 2000). In addition, chickens housed in a protective laboratory environment still became colonized by *Campylobacter* (Lindblom *et al.*, 1986). Application of strict control measures in two different broiler houses, such as cleaning and disinfection of the houses between successive cycles, did not prevent the broiler flocks from becoming colonized by *Campylobacter*, even though some reduction in the percentage of *Campylobacter*-positive flocks was achieved (van de Giessen *et al.*, 1998). Similarly, strict observance of high standards of hygiene and biosecurity practised before placement of day-old broilers and during the entire grow-out period by all personnel reduced the prevalence of *Campylobacter*, but it did not prevent some flocks from being colonized by *C. jejuni* (Gibbens *et al.*, 2001). Thus, even when the likely sources of horizontal transmission are controlled, broiler chickens still become infected with *C. jejuni*, raising the possibility that vertical transmission of *C. jejuni* may occur.

Finally, another line of evidence for vertical transmission of *C. jejuni* from breeder flocks to the progeny comes from a few observations that the isolates from both the breeders and the broilers had the same serotypes or genotypes. Pearson *et al.* (1993, 1996) performed multiyear studies on a highly populated broiler chicken farm and provided evidence for the involvement of both vertical and horizontal transmission. Once the conditions for horizontal transmission were under control, a pattern of intermittent shed positivity within the same broiler flock and the lack of diversity of types isolated during the entire study period became apparent, which indicated a common source of *C. jejuni* introduced by vertical transmission (Pearson *et al.*, 1996). Furthermore, the isolation rate (42.9%) of *C. jejuni* in market-age broilers supplied by hatchery B was found to be significantly higher than that (17.6%) in broilers supplied by hatchery A in the same study. In two instances, when both hatcheries were used to hatch chicks to stock the same farm flock, *C. jejuni* was found only in those sheds with chicks supplied by hatchery B. Together, the result suggested that there was a common source of infection to the broiler farm (Pearson *et al.*, 1996). Recently, Cox *et al.* (1999) compared *C. jejuni* isolates from breeders and their progeny, and showed that the isolates from both places were of the same clonal origin, as determined by sequencing the short variable region of *flaA*. The investigators interpreted the result as cultural evidence for vertical transmission of *C. jejuni*. Despite the observations supporting the possibility of a low rate of vertical transmission, live *Campylobacter* organisms have not yet been detected in the contents of commercial eggs, young hatchlings, or hatcheries (Doyle, 1984; Shane *et al.*, 1986; Shanker *et al.*, 1986; Baker *et al.*, 1987; Chuma *et al.*, 1994; Sahin *et al.*, 2001b; Hiett *et al.*, 2002). Thus, the exact role of vertical transmission in introducing *Campylobacter* to broiler flocks remains to be answered in future studies.

## Conclusions

Despite extensive studies, the ecology of *C. jejuni* in the poultry reservoir is still poorly understood, particularly with respect to the sources of infection and routes of transmission. In cases in which *C. jejuni* isolates have been typed using molecular methods, it is apparent that great genetic diversity exists among *Campylobacter* strains from within a flock and among adjacent flocks on the same farm. The existence of both *C. jejuni*-free and *Campylobacter*-colonized flocks on the same farm further complicates the understanding of the ecological features of this important human pathogen. These findings illustrate the complexity of the dynamics of *C. jejuni* transmission on poultry farms. Current knowledge indicates that multiple routes, including both vertical and horizontal transmission, are involved in the original introduction of *C. jejuni* into broiler flocks. It is likely that there is not a single dominating source for *Campylobacter* transmission on broiler farms. Rather, diverse sources of infection may exist on different farms. Once a flock is infected, the extent of *Campylobacter* colonization in the broiler flock is likely to be influenced by host-related factors (e.g. immune status of the birds) and environmental conditions in the production system (e.g. management practices, biosecurity measures and the presence of other farm animals). Therefore, it may be necessary to target different segments of the broiler production system using several methods in order to effectively reduce or eliminate *C. jejuni* infection in broiler flocks.

In the future, well-conceived epidemiological studies using powerful genotyping methods will be required in order to provide a complete understanding of the ecology of *Campylobacter* on poultry farms. There are multiple molecular typing tools currently available for *Campylobacter*, which have been reviewed recently by Wassenaar and Newell (2000). When appropriately designed and used, the molecular typing tools will provide key information on the transmission of *Campylobacter* in broiler production systems. Among the unsolved mysteries regarding *Campylobacter* ecology, one of great interest is the lack of colonization of young broilers by *C. jejuni* in commercial production systems. Regardless of the sources of infection and modes of transmission, young broiler chickens less than 2–3 weeks of age are usually *Campylobacter*-free. If the reasons for the lack of infection in young chickens are elucidated, they may be exploited to raise broiler flocks free from *C. jejuni* until slaughter, eliminating a source

of carcass contamination in processing plants. Possible factors related to this phenomenon may be the presence of maternal antibodies, age-related differences in the intestinal environment, such as competitive microflora and specific receptors for the organism, and differences in management practices. Future studies on these aspects of *Campylobacter* ecology may help in the development of effective intervention strategies to control this food-borne pathogen at the preharvest stage.

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# Digestive Physiology of Pigs

Edited by **J E Lindberg** and **B Ogle**, *Department of Animal Nutrition* and *Management, Swedish University of Agricultural Sciences, Uppsala, Sweden*

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