CEVA HANDBOOK
OF POULTRY DISEASES

2 CAMPYLOBACTERIOSIS

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DEFINITION

Enteritis and diarrhoea are the clinical and morphological manifestations of infections with *Campylobacter* spp., which is a minor pathogen in poultry species, but is very important for food safety and public health.

HISTORY AND SYNONYMS

A bacterium from the Vibrio genus was first isolated from sheep stillbirths in 1913, and the disease was later found in pregnant animals (Veron et al., 1973). Vibrio was first associated with enteritis in cattle dysentery (Jones et al., 1931). In 1957, the first cases of human vibriosis were reported by Dr. E. King. In 1973 *Vibrio jejuni* was classified in the genus *Campylobacter* (in Greek "curved rod") by Veron et al. (1973) who described it for the first time.

CHARACTERISTICS AND CLASSIFICATION OF THE PATHOGEN

Campylobacter possesses a wavy outer membrane, complex cytoplasmic membranes and individual flagellar basal granules (Veron et al., 1973). The microorganism is oxidase-positive, non-spore-forming, Gram-negative, thin (0.5-8 µm in length and 0.2-0.8 µm in width) with a curved, S-shaped or spiral morphology. Motility is achieved by purposeful or spiral movements. Campylobacteria belong to a specialised group of Gram-negative bacteria, known as rRNA superfamily VI (Anon, 2000). In addition to the *Campylobacter* genus, *Arcobacter* and *Helicobacter* also belong to the group. *Arcobacter* are closely related to *Campylobacter* and some of them cause intestinal diseases in humans. *Helicobacter pylori* is an acknowledged agent of gastritis and peptic ulcer disease. A common feature of the group is their ability to colonise the surface of the mucous membrane of the alimentary and reproductive tracts. The spiral shape, together with the long polar flagellum, allow for the rapid movements and thus for movement through the mucus in their preferred infection sites (Boxall, 2005).

The *Campylobacter* genus has 16 species and 6 subspecies. Relationships determined phylogenetically by DNA-rRNA hybridisation leave us with three major rRNA homology groups. The DNA of Campylobacter contains between 29% and 36% guanine and cytosine (GC) (Veron et al., 1973).

Most species are microaerophilic. Although the oxygen requirements vary, they grow best in an environment with 5-10% oxygen (Doyle, 1986).

Aerobic *Campylobacter* species were reclassified as *Acrobacter* (Vandamme et al., 1991). It is now known that the minimum temperature required by *C. jejuni* and *C. coli* to grow is about 30°C, but this does not mean that their metabolic activity stops below this level. These organisms are able to adapt to environmental stressors even when growth is not possible. Metabolic activity of *C. jejuni* has been demonstrated at 4°C (O'Dell, 1998). At the same temperature and microaerophilic conditions, chemotactic and aerotactic activities are also present, evidencing the ability of the microorganism to survive in such conditions. This flexibility allows the bacterium to metabolise in various anaerobic conditions outside of the host.

It is believed that *C. jejuni* perishes rapidly without oxygen and humidity. However, research has shown that the organism survives better *in vivo* than *in vitro* – e.g. it has been isolated from dry beach sand (Wysocki, 2002). This resistance could be linked to the ability of *Campylobacter* to form viable but non-culturable cells. The cells alter their shape from spiral into coccoid and may go undetected by common culturing techniques, but preserved the potential to regain their infectious form (Boxall, 2005).

The prevailing isolates among poultry are *C. jejuni*, followed by *C. coli* and rarely *C. lari* (Zhang, 2008).

EPIDEMIOLOGY AND PATHOGENESIS

Campylobacteria are ubiquitous. Animals and birds serve as reservoirs, contaminating the environment with infected faeces. Humans, working or living in such an environment are most susceptible to campylobacteriosis. Carriers of *Campylobacter* are significantly more numerous in the domestic bird population as opposed to wild birds (Shane, 1992; Tsai & Hsiang, 2005). This is probably due to the higher stocking density in poultry farms, which aids the spread of *Campylobacter* among birds (Zhang, 2008). Even mild diarrhoea

can moisten the litter at high population densities.

Serial surveys on newly hatched chicks originating from different breeder flocks resulted in sporadic isolation of *Campylobacter* (Doyle, 1984; Neil et al., 1984). The findings suggest that the transmission through eggs is uncommon. The weekly testing of samples from five broiler chickens flocks at the age of one week resulted in only one *Campylobacter* positive sample, but at the age of 4 weeks the spread in 3 of the test flocks was 100% (Pokamunski et al., 1986). The findings indicated a rapid horizontal transmission within the flock. The lack of detection of *Campylobacter spp.* in chickens younger than 2 weeks of age could be due to one or more factors such as increased resistance to infection at that age due to immunity, physiological status of birds or even dietary factors. It is also possible that *Campylobacter* strains appear in poultry houses only after the age of 2 weeks (Neil et al., 1984). The arrival of *Campylobacter* on farm premises may occur through a number of routes, e.g. contaminated water, feeds or via farm personnel. Many wild avian species harbour *Campylobacter* in their guts and represent a potential source of infection. Direct transmission from wild birds to broilers is rather unlikely, as most broiler fattening facilities are closed. Rotaviral or entero-like viral enterites may occur as co-infections (McFerman et al., 1983). Sources of infections may include untreated water, old litter, fleas, insects etc. Darkling beetles (*Alphitobius diaperinus*) may act as mechanical vector to spread *Campylobacter* between barns. This theory has been confirmed by isolation of identical sero- and genotypes of *Campylobacter* from broilers and insects from the same farm houses (Berndtson et al, 1996). It has been reported that, during the summer months, about 10% of fleas found around poultry houses are contaminated with *Campylobacter* (Hald et al., 2004). The seasonal pattern of campylobacteriosis in poultry flocks during the warm months is related to the increased flea populations at that time. Rodents also harbour the bacterium in their intestinal tract and act as a vector of transmission from premise to premise (Kapperud et al., 1993). The presence of other animal species on a farm (pigs, sheep, cattle etc.) may also pose a risk for infection of broiler chickens with *Campylobacter* (Nesbit et al., 2001). The main route of infection is horizontal spread. Vertical transmission of *Campylobacter* is not encountered, or only very rarely (Zhang, 2008).

Infection of flocks is accompanied by diarrhoea and wet litter in some

cases (Neil et al., 1984), but not in others (Pokamunski et al., 1986). The poor litter quality depends on the extent of infection within the flock (Neil et al., 1984). Wet litter may result in considerable economic losses to poultry farmers due to contact dermatitis manifested as sternal bursitis or pododermatitis. The most common consequences of diarrhoea are retarded growth and homogeneity of size among chickens.

The most common incidence, and highest rate of infection, for broiler flocks is detected at slaughterhouses (Oosterom et al., 1983; Pokamunski et al., 1986). An examination of frozen broiler carcasses collected from retail stores in Bulgaria found *Campylobacter spp.* in 35.2% of samples, of which 22.9% in the skin, 31.4% in breast muscle and 51.2% in thighs, although storage at 20°C to –70°C is known to considerably reduce microbe counts (Stoyanchev et al., 2007). The organisms has been detected in viscera of different avian species (Oosterom et al., 1983; Boukraa, 1991; Vashin & Stoyanchev, 2005).

Many serotypes may be isolated within the same broiler flock, reflecting the complex epizootiology of thisillness. Infection occurs after hatching, but not via the eggs. Once it has penetrated, the spread of infection through excreta is rapid until slaughtering. Both the inter- and intra-flock diversity of serotypes is high, with a variable prevalence, differing from serotypes detected at the same time in humans (Pokamunski et al., 1986). Broiler chickens are considered an important potential source of *Campylobacter* enteritis in humans, as many birds carry *C. jejuni* in their alimentary tract at slaughtering (Blaster et al., 1983).

The significance of campylobacteriosis as a foodborne infection in humans has been confirmed by numerous studies. At present, campylobacteriosis is caused mainly by *C. jejuni* and a smaller proportion of pathogenic isolates are identified as *C. coli* (Tauxe et al., 1987). *C. jejuni*–induced enteritis in humans has different manifstations. Usually, mild diarrhoea is observed (Blaser et al., 1983). The infection may sometimes remain unrecognised or undiagnosed. Detection of human campylobacteriosis depends on laboratory tests of samples and results of analytical procedures. Although in rural regions non-chlorinated water and raw milk consumption were long associated with *C. jejuni* infection outbreaks, uncooked chicken is the primary source of infection in urban communities (Skirrow, 1991; Finch & Blake, 1985). Cases of

Campylobacter gastroenteritis in humans having consumed chicken meat are frequently reported, some of them as outbreaks (Brower et al., 1979; Pearson et al., 1987). In all instances, the manifestations were associated with consumption of undercooked chicken meat. The close contact between chickens and men is also a potential hazard for campylobacteriosis (Marquis et al., 1990). In a survey in Peru, the authors established that the risk from *C. jejuni* infection was 12 times greater among children whose families raised backyard chickens compared to families which did not. Surveys among student communities in different universities have shown that barbecued chicken is an important risk factor for infection with *Campylobacter* (Murray, 1986; Deming et al, 1987). Such a risk exists for poultry slaughterhouse workers. In one study, testing of the personnel of a slaughterhouse revealed 27–68% seropositivity rates (Jones & Robinson, 1981).

Some criteria for diagnosis of campylobacteriosis as a foodborne infection have been outlined (Butzler & Skirrow, 1979): *C. jejuni* should be predominantly isolated as a sole pathogen from faeces of enterocolitis patients; in cases of enterocolitis, the detection of *C. jejuni* bacteriaemia is a diagnostic criterion; confirmation through seroconversion of serum samples; beneficial clinical effect from administration of erythromycin in the acute phase of infection is also indicative of campylobacteriosis.

Contamination of chicken meat with *C. jejuni*

The high prevalence of *C. jejuni* among commercial birds is reflected in the contamination level of poultry products. The presence of *C. jejuni* in chilled or frozen carcasses or cuts of chicken, turkey and duck poses a risk to consumers' health. The investigations of cloacal swab samples obtained from broilers in the slaughterhouse revealed a broad range of *C. jejuni* incidence, up to 20% (Jones et al., 1991) or even 32% for contaminated crates of transported birds (Hoop & Ehrsam, 1987). In some instances, strains have been isolated from the caeca of broilers with negative cloacal swabs in slaughterhouses. The water chilling of carcasses encourages the spread of infection to all carcasses during meat processing. High levels of *C. jejuni* were detected in the scalding water, in feathers, waste water ducts and at different areas of the conveyor system (Wempe et al., 1983; Baker et al., 1987).

A reduction of surface contamination at the slaughterhouse has been observed after scalding. It is frequently reported that levels of *C. jejuni* are lower in turkey meat products as compared to chicken products (Baker et al., 1987). There are, however, data that suggest a relatively high level of contamination of turkey meat, which can be probably attributed to the efficiency of washing, water chilling, the equipment of the slaughterhouse, carcass size and the extent of off-farm contamination (Shane, 1993).

The presence of relatively high levels of *C. jejuni* in poultry carcasses and cuts poses a potential risk of foodborne campylobacteriosis. The transportation and storage of products after slaughterhouse processing do not reduce contamination levels. The residual *C. jejuni* levels in broiler chicken legs frozen at -20°C for 26 weeks were at the limit of detection (Shane, 1992). Epidemiological data related to human enterocolitis after consumption of uncooked meat make it clear that chicken meat is an important source of infection for humans.

CLINICAL SIGNS AND PATHOLOGY

In natural conditions, campylobacteriosis is usually asymptomatic. After experimental reproduction in chickens and turkey poults, transient watery diarrhoea, weight loss and retarded growth have been observed (Neil et al 1984; Lam et al., 1992). The gross lesions following experimental infection represent signs of catarrhal enteritis. The corresponding microscopic lesions consist of moderate swelling of intestinal mucosa and submucosa (Knudsen et al., 2006).

The assumption that Campylobacter could induce vibrionic hepatitis, which had prevailed among stock laying hens in the middle of the last century and is now seen only sporadically, has not been formally confirmed (Shane & Sterm, 2003).

DIAGNOSIS

Based upon microbiological examination of samples from chickens, foodstuffs and the environment. As such samples could be contaminated with numerous microorganisms, selective nutrient media and special culturing conditions – microaerophilic atmosphere – are used. Selective nutrient media contain a variety of antibiotics, to which campylobacteria are naturally resistant. They suppress the growth of accompanying microflora in samples and allow for the isolation of slowly growing *Campylobacter spp*. Identifiable colonies may appear after 48 h incubation on solid media, but for slowly growing strains this may take twice as long. The identification of organisms is based on the morphology of colonies – spirals or curved rods. Some biochemical tests (catalase, oxidase, nitrate reduction tests etc.) may also be of use (Zhang, 2008).

Enzyme immunoassays developed for direct detection of *Campylobacter spp*. on the basis of antigen-antibody interaction in faeces or foods are much more rapid than routine culturing methods (Hindien et al., 2000).

MANAGEMENT, PREVENTION AND CONTROL

There are two major approaches to the challenge of eliminating *Campylobacter spp*. from the food chain. On one hand, efforts may be directed towards reduction of the contamination of live animals with *Campylobacter spp*. at the farm, while the other method would be to focus on decontamination of the final product.

Biosecurity

A strict compliance to biosecurity standards is required to prevent the penetration of *C. jejuni* in a flock. A complete decontamination of poultry houses is required, beginning with full removal of the litter in order to prevent the spread of infection from the previous batch

of birds. Before each repopulation of the facility, a new litter is recommended. The reuse of the old litter during the next cycle, as is the practice in some farms, requires at least an intermediate 7-day interval for removal of existing moist litter aggregates, where *C. jejuni* could survive. Biosecurity measures should be taken to prevent the introduction of infection through contaminated shoes, rodents, wild birds or fleas.

The decontamination of drinking Water, together with decontamination of water tanks and troughs, may reduce the infection level at the farm. Appropriate measures can significantly reduce isolation rates in individual birds from 80% to 2%, but the discontinuation of decontamination measures may result in high recolonisation rates (Shane, 1992).

More general biosecurity measures include: compliance to the "all-in all-out" principle, utilisation of poultry houses fit for cleansing and disinfection, maintenance of houses and surrounding vegetation (grass, trees) in a good condition, appropriate drainage of premises and land; availability of sites for disinfection of shoes, hands etc. in poultry house anterooms; compliance of drinking water to sanitary and hygiene standards, restriction of the number of visitors to poultry houses to a strict minimum and equipment of visitors with protective clothing.

Swabs should be regularly collected from houses with a history of previous colonisation with *Campylobacter spp.* After washing and disinfection, poultry houses should be completely dry before the birds are brought in.

Vaccination

So far, there are no commercially available vaccines for control of *Campylobacter* in industrial poultry farms. Positive results obtained after vaccination of some mammalian species suggest that successful vaccination of birds may yet be possible. It appears that birds are protected against *Campylobacter* infection through maternal immunity. The rapid development of genetically-engineered vaccinology and targeted modulation of immune response are promising steps towards the development of efficient vaccines(de Zoete et al.,2007).

Slaughterhouse control

The risk for contamination of meat with *Campylobacter spp.* originating from the skin or alimentary tract of carrier animals cannot be prevented by current practices at slaughterhouses. The introduction of good manufacturing practices (GMP) and the identification of hazards and potential points for control may help minimise the level of contamination. One of the major concerns is to prevent the contamination of carcasses with faeces. Clinically infected slaughterhouse workers and food chains should be also taken into consideration as possible sources of contamination.

Pre-slaughter management of poultry will determine the extent of contamination of the skin, feathers and colonisation density of poultry intestines. These factors are important for the contamination of raw chicken meat.

Faecal contamination rates at the slaughterhouse can be reduced by withdrawing food at least 12 hours before slaughter. Furthermore, transportation vehicles, crates and modules should be de-contaminated after each batch in order to remove faecal matter.

A modified scalding technology reducing the microbial contamination has been proposed (Veerkamp, 1991). The method includes three phases of scalding with hot water. Increasing the water temperature in tanks and maintaining a pH of 9.0 can substantially reduce *C. jejuni* counts from 82 to 1 microbial cell per 100 ml, but has no effect on carcass contamination (Humphrey & Lanning, 1987). This technique can also result in altered organoleptic properties, when the immersion time was more than 3 min.

The improvement of carcass washing, the control on chillers and avoiding manual manipulations through introduction of automated equipment and improved hygiene of conveyor belts contribute to reduction of *C. jejuni* levels and help reduce the internal contamination of the slaughterhouse.

Chlorination, organic acids at various concentrations and gamma radiation have all been tested in attempts to eliminate *Campylobacter spp.* (Shane, 1992). So far, the chemical treatment of carcasses is prohibited except for the use of chlorinated water (within the permitted concentrations for commercial use), organic acids and ionised radiation (Steele, 2001; Whyte et al., 2001).

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