

Campylobacter species

Campylobacter spp. are bacteria that cause the gastrointestinal disease campylobacteriosis, with symptoms that can mimic appendicitis. Most cases of campylobacteriosis are not fatal. Infection with *Campylobacter* spp. has also been associated with Guillain-Barré syndrome, which results in progressive muscle weakness or paralysis. *Campylobacter* spp. are widespread in nature and are present in the intestine of many wild and domestic animals and birds.

Description of the organism

Campylobacter spp. are Gram-negative, non-spore forming bacteria and are members of the family *Campylobacteraceae*. The genus *Campylobacter* comprises of 17 species and 6 subspecies (Nachamkin 2007; Silva et al. 2011). The two species most commonly associated with human disease are *C. jejuni* and *C. coli*. *C. jejuni* accounts for more than 80% of *Campylobacter*-related human illness, with *C. coli* accounting for up to 18.6% of human illness. *C. fetus* has also been associated with foodborne disease in humans (Gurtler et al. 2005; FDA 2012).

Growth and survival characteristics

The growth and survival of *Campylobacter* spp. depends on a variety of factors. *Campylobacter* spp. are sensitive to environmental conditions, such as temperature, availability of water and oxygen; and have limited capacity to survive environmental stress (refer to Table 1).

Campylobacter spp. grow in the 30–45°C temperature range. At 32°C, *C. jejuni* may double its number in approximately 6 hours (Forsythe 2000). *Campylobacter* spp. do not multiply at temperatures below 30°C, such that the number of *Campylobacter* spp. will not increase in foods held at room temperature (20–25°C) (Park 2002).

Although unable to grow below 30°C, *Campylobacter* spp. survive at temperatures as low as 4°C under moist conditions (Hazeleger et al. 1998; Park 2002). Survival in food is extended at refrigeration temperatures compared with room temperature, with viable cells being found after 7 months storage at 4°C (Lazaro et al. 1999). In a study of *Campylobacter* spp. that examined survival on naturally contaminated chicken skin and minced meat at freezing temperatures (–22°C), Sampers et al. (2010) found that numbers declined by approximately 1 log₁₀ over the first 24 hour period. No further significant reduction was achieved by prolonged freezing, with *Campylobacter* spp. being detected in samples by enrichment after 84 days.

Although *Campylobacter* spp. survive well at cold temperatures, they are sensitive to heat and are readily inactivated by pasteurisation treatment or domestic cooking. Heating at 55–60°C for several minutes readily destroys *Campylobacter* spp. (ICMSF 1996).

Campylobacter spp. are highly sensitive to loss of moisture and do not survive well on dry surfaces (Fernandez et al. 1985). *C. jejuni* grows best at a sodium chloride concentration of 0.5% and does not grow in the absence of sodium chloride or in the presence of 2% or higher concentrations of sodium chloride (Doyle and Roman 1982; Wallace 2003).

Campylobacter have varying degrees of oxygen tolerance (3–5%) between species (Forsythe 2000). Most strains of *Campylobacter* do not grow in the presence of air, other than a few strains that may grow under slightly oxygen rich conditions. Optimal growth occurs at 5% oxygen and 2–10% carbon dioxide (Park 2002). *C. jejuni* is able to adapt to aerobic conditions due to an ability to produce biofilms. The level of biofilm formation is higher in motile, flagellated strains than in non-flagellate, non-motile strains. This ability enhances the survival and spread in food processing environments such as poultry processing (Reuter et al. 2010).

Several studies have shown that *C. jejuni* is sensitive to acids such as formic, acetic, ascorbic and lactic acids (Murphy et al. 2006).

Campylobacter spp. have been shown to enter a viable but non-culturable state when subjected to unfavourable conditions, such as low nutrient availability, elevated temperature, freezing or stationary phase (Levin 2007). In this state, cells transform from a motile spiral form to a coccoid form (Rollins and Colwell 1986). The nature and role of this coccoid form is uncertain.

Table 1: Limits for growth of *Campylobacter* spp. when other conditions are near optimum (ICMSF 1996; Forsythe 2000)

| | Minimum | Optimum | Maximum |
|------------------|---------|---------|---------|
| Temperature (°C) | 32 | 42–43 | 45 |
| pH | 4.9 | 6.5–7.5 | 9.5 |
| Water activity | 0.987 | 0.997 | – |

Symptoms of disease

Symptoms of campylobacteriosis include diarrhoea (sometimes bloody), nausea, abdominal pain, fever, muscle pain, headache, and vomiting. The incubation period before onset of disease is usually 2–5 days, with illness generally lasting for 2–10 days. The unique feature of the disease is the severity of abdominal pain which may become continuous and sufficiently intense to mimic acute appendicitis (Young and Mansfield 2005; FDA 2012). As a consequence of *C. jejuni* infection a small number of individuals develop a secondary condition such as reactive arthritis or Guillain-Barré syndrome, in which a harmful immune response of the body attacks part of the peripheral nervous system leading to symptoms of muscle weakness or paralysis (Havelaar et al. 2009).

Virulence and infectivity

Campylobacter spp. have four main virulence properties: motility, adherence, invasion and toxin production. The exact nature of how *Campylobacter* spp. adhere to and invade the intestinal epithelial cells is not fully understood (Levin 2007). It is thought that the combination of its spiral shape and flagella leads to rapid motility that enables the organisms to penetrate through the intestinal lining unlike conventional bacteria (Levin 2007; Bhavasar and Kapadnis 2007).

Campylobacter organisms produce two types of toxins: enterotoxin and cytotoxins. The enterotoxin of *C. jejuni* is similar to the *Vibrio cholerae* toxin and the *Escherichia coli* heat-labile toxin. This enterotoxin is produced to a lesser degree by *C. coli*. It has been suggested that enterotoxin produced by *Campylobacter* spp. results in watery diarrhoea, as opposed to

bloody diarrhoea due to cytotoxin production. However, in some studies enterotoxigenic strains have been isolated from asymptomatic carriers (Wassenaar 1997). There have been at least six types of cytotoxins identified in *Campylobacter* spp. This includes a 70 kDa cytotoxin, a Vero/HeLa cell cytotoxin, a cytolethal distending toxin (CDT), a shiga-like toxin, a haemolytic cytotoxin and a hepatotoxin. The CDT toxin has been shown to cause cell distension and cell disintegration of human tumour epithelial cells (Pickett et al. 1996). Active CDT toxin has been found in roughly 40% of over 70 *Campylobacter* strains tested (Johnson and Lior 1988). However, the role of enterotoxin and cytotoxins in *Campylobacter* pathogenesis has not been fully characterised.

Mode of transmission

Campylobacter spp. are transmitted to humans via the faecal-oral route, predominantly through the consumption of contaminated food or water or direct contact with infected animals (CDC 2010a). They are often present in the intestines of domestic and wild animals, such as cattle, sheep, poultry, dogs, wild birds and rodents, and are shed in the faeces of these animals (Hu and Kopecko 2003; Ellis-Iversen et al. 2012).

Campylobacter spp. present on raw meats may contaminate work areas and the hands of kitchen staff before being transferred to ready-to-eat foods or causing self-infection (Coats et al. 1987). External packaging material of raw meat (raw chicken, game-fowl, lamb and beef) has been reported to be a vehicle of cross-contamination of *Campylobacter* spp. in retail premises and consumer homes (Burgess et al. 2008).

Incidence of illness and outbreak data

Campylobacter infection is notifiable in all Australian states and territories except in New South Wales. In 2012 *Campylobacter* was the most frequently notified foodborne infection in Australia, with a rate of 102.3 cases per 100,000 population (15,664 cases). This was a decrease from the previous 5 year mean of 112.8 cases per 100,000 population (ranging from 107.4–119.9 cases per 100,000 population per year) (NNDSS 2013).

In New Zealand the notification rate in 2011 was 151.9 cases per 100,000 population (6,692 cases). This was a decrease from the 2010 rate of 168.2 cases per 100,000 population (Lim et al. 2012).

While not a notifiable disease in the United States (US), surveillance through FoodNet (representing 15% of the population) reported a rate of *Campylobacter* infection of 13.6 cases per 100,000 population in 2010. This was similar to the 2009 rate of 13.0 cases per 100,000 population (CDC 2010b; CDC 2011). The number of confirmed human campylobacteriosis cases in the European Union (EU) was 50.3 per 100,000 population in 2011 (ranging from 0.3–178 cases per 100,000 population between countries). This was a 2.2% increase in the number of cases from 2010 (EFSA 2013).

The incidence of *Campylobacter* infections is known to be associated with seasonal changes in many countries. *Campylobacter* infection is most prevalent during spring in Australia (Unicomb et al. 2009). A main peak of *C. jejuni* during summer and a peak of *C. coli* during winter has been observed in Germany (Gurtler et al. 2005). *C. jejuni* is one of the most commonly reported agents associated with foodborne illness in many developed countries, including New Zealand, the United Kingdom (UK) and the US (Mead et al. 1999; Park 2002).

Foods associated with *Campylobacter* spp. outbreaks include poultry meat, raw (unpasteurised) milk and milk products, beef, pork and shellfish (IFT 2004) (refer to Table 2).

Outbreaks of campylobacteriosis linked to consumption of raw (unpasteurised) milk have been increasingly reported in the US (FDA 2010). *Campylobacter* infections generally occur sporadically, rather than being associated with outbreaks.

Table 2: Selected major foodborne outbreaks associated with *Campylobacter* spp. (>50 cases and/or ≥1 fatality)

| Year | No. of cases | Food | Country | Comments | References |
|------|--------------|--------------------------------|-----------------|--|-----------------------|
| 2008 | 98 | Raw peas | US | Peas were contaminated in the field with bird faeces. Same strain of <i>C. jejuni</i> isolated from peas and bird faeces | (Gardner et al. 2011) |
| 2007 | 68 | Cheese | US | Cheese from raw milk was prepared and consumed as part of community celebration activities | (KDHE 2007) |
| 2005 | 79 | Chicken salad | Denmark | Cross-contamination from raw chicken to the chicken salad during preparation/ storage. <i>C. jejuni</i> implicated | (Mazick et al. 2006) |
| 2005 | 86 | Chicken liver pate | Scotland | Pate preparation involved using undercooked chicken livers by flash frying, followed by mechanical homogenization. More than one strain of <i>C. jejuni</i> implicated | (Forbes et al. 2009) |
| 2003 | 81 | Custard prepared from UHT milk | Spain | Cross-contamination from raw chicken to custard | (Jiménez et al. 2005) |
| 1998 | 79 | Tuna salad | US | Precise route into tuna salad unknown. Rare strains of <i>C. jejuni</i> implicated. Several deficiencies identified in the camp kitchen operation | (Roels et al. 1998) |
| 1995 | 78 | Cucumber | South Australia | Cucumber served at self service salad bar. Suspected cross-contamination from raw meat | (Kirk et al. 1997) |

Occurrence in foods

Poultry meat is generally recognised as a primary source of *Campylobacter* infection in humans (Sahin et al. 2002). The reported incidence of *Campylobacter* spp. on raw meat products from other food animal species tends to be lower than those reported for poultry. Using population genetics approaches, Wilson et al. (2009) confirmed that the vast majority (97%) of sporadic *Campylobacter* infections in the UK could be attributed to animals farmed for meat and poultry. Chicken and cattle were the principal sources of *C. jejuni* pathogenic to humans, with wild animal and environmental sources responsible for the remaining 3% of human disease.

In an Australian baseline survey carried out during 2007–2008 on the incidence and concentration of *Campylobacter* and *Salmonella* in raw chicken, 84.3% of post-processing carcass rinse samples (n=1,104) were positive for *Campylobacter* spp. These results were similar to those from a retail baseline microbiological survey carried out in 2005–2006 in South Australia and New South Wales, which found that 90.0% of retail poultry samples (n=859) were contaminated with *Campylobacter* spp. (FSANZ 2010).

A retail survey conducted in New Zealand between 2005–2008 found 72.7% of poultry carcasses were contaminated with *C. jejuni* (n=500). Several internationally rare serovars as well as common human clinical serovars were isolated, both ubiquitous and supplier-associated (Mullner et al. 2010).

A baseline survey carried out in the EU in 2008 revealed that 75.8% of broiler carcasses sampled (n=9,213) were contaminated with *Campylobacter* spp. The prevalence of *C. jejuni* and *C. coli* were 51.0% and 35.5%, respectively. *Campylobacter* spp. were also commonly detected in live poultry, pigs and cattle (EFSA 2010).

In the UK, a survey of poultry sold at retail carried out during 2007–2008 indicated that 65.2% of samples tested (n=3,274) were contaminated with *Campylobacter* spp. *C. jejuni* was present in 52.9 % of the samples while 47.1% contained *C. coli* (FSA 2009).

In a survey of retail food stuffs in Ireland between 2001–2002, *Campylobacter* spp. were found in 49.9% of raw chicken (n=890), 37.5% of raw turkey (n=88), 45.8% of raw duck (n=24), 3.2% of raw beef (n=221), 5.1% of pork (n=197), 11.8% of lamb (n=262), 0.8% of pork pate (n=120), 2.3% of raw oysters (n=129), and 0.9% of fresh mushrooms (n=217) tested. Of the positive samples, 83.4% were contaminated with *C. jejuni* and 16.6% were contaminated with *C. coli* (Whyte et al. 2004).

Host factors that influence disease

It is now known that individuals and populations express acquired immunity against *Campylobacter* infections. This immunity may be achieved via non-specific host-defence mechanisms (innate immunity) as well as via a pathogen specific immune response (adaptive immunity). The bacterial factors that induce the innate response in humans are known to be variable among strains of *Campylobacter* spp. and therefore influence the extent of the innate immune response (Havelaar et al. 2009). Following infection by *C. jejuni*, immunoglobulin (Ig) A and IgM antibodies appear one week after infection and IgG antibodies peak a few weeks later. IgA and IgM antibodies disappear within two to three months, while IgG antibodies remain for much longer (Havelaar et al. 2009).

IgA antibodies directed against *Campylobacter* spp. are present in breast milk (Ruiz-Palacios et al. 1990; Nachamkin et al. 1994). Therefore, susceptibility in early infancy may

be reduced by passive immunity acquired from milk and/or placentally transferred immunity from immune mothers (Havelaar et al. 2009). Available data suggests that young children under the age of four (with the exception of early infants) and young adults in the age range of 20 to 30 years old are most susceptible to *Campylobacter* spp. infection (WHO/FAO 2009).

The bacterium-specific immune response limits the disease and leads to the development of protective immunity. Phagocytes and *Campylobacter*-specific secreted IgA antibodies play a part in this immune response. Repeated exposure is known to increase levels of protective immunity, however, this immunity is often strain specific (Havelaar et al. 2009). In some cases, acquired immunity could lead to resistance to colonisation by *Campylobacter* spp. (Tribble et al. 2010).

The incidence of *Campylobacter* infection in patients with acquired immune deficiency syndrome (AIDS) has been calculated to be 40-fold higher than that in the general population (Sorvillo et al. 1991). People with AIDS, immunosuppressive therapy, and liver disease are predisposed towards *Campylobacter* infections (Pigrau et al. 1997).

Dose Response

Volunteer studies have shown that 800 cells are able to cause campylobacteriosis in healthy adults (Black et al. 1988). The dose-response relationship and the illness-to-infection ratio appeared to differ between different *C. jejuni* isolates (Medema et al. 1996). Due to the sensitivity of *C. jejuni* to acids, it has been suggested that ingesting *Campylobacter* spp. with buffers such as milk or water which aid rapid wash through the gastric acid of the stomach, may reduce the oral infective dose (Blaser et al. 1980). Recent data confirm that doses of less than 100 cells have been associated with human illness (Teunis et al. 2005; Tribble et al. 2010).

Recommended reading and useful links

FDA (2012) Bad bug book: Foodborne pathogenic microorganisms and natural toxins handbook, 2nd ed, US Food and Drug Administration, Silver Spring, p. 17–20.
<http://www.fda.gov/Food/FoodbornenessContaminants/CausesOfIllnessBadBugBook/ucm2006773.htm>

WHO/FAO (2009) Risk assesment of *Campylobacter* spp. in broiler chickens. World Health Organization and Food Agriculture Organization of the United Nations, Geneva.
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