

# FOOD

STANDARDS

Australia New Zealand

## AGENTS OF FOODBORNE ILLNESS

A technical series  
summarising key information  
on microorganisms  
associated with  
foodborne illness

Edited by

Duncan Craig

Andrew Batholomaeus



FOOD STANDARDS  
Australia New Zealand  
Te Mana Kounga Kai – Ahitereiria me Aotearoa

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## Preface

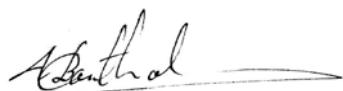
Foodborne disease is a significant cause of morbidity and mortality across the globe and the increased internationalisation of food production and distribution means pathogens associated with food know no borders. The management and reduction of foodborne disease is therefore a core objective of all food agencies. As one strategy to support this objective a number of resources have been developed and made available by food and scientific research agencies to inform food manufacturers, consumers and physicians about foodborne illness. The FDA Bad Bug Book for example, provides information to consumers and physicians primarily focused on the clinical characteristics of the foodborne illness produced by each organism, to assist in their recognition and diagnosis. Advice to manufacturers on principles of safe food production to avoid contamination with pathogenic microorganisms is also available, such as the CSIRO Food and Nutrition Sciences' "Guide to Food Safety - Make it Safe". An area not comprehensively addressed by any current, readily available resource, however, is the behaviour of foodborne pathogens under varying food and environmental conditions.

Although a limited range of organisms are responsible for the majority of foodborne disease, their potential survival, growth and toxin production, and therefore their pathogenicity, is dependent to a significant extent on the food matrix in which they are present. This interplay between the characteristics of foods and potentially pathogenic microorganisms they may contain, creates a high level of complexity and challenge in the discipline of food microbiology. Prediction, prevention and management of foodborne disease is therefore dependent on an understanding of the behaviour of microorganisms under different conditions and in different food matrices. This technical series provides monographs summarising the key biological characteristics of foodborne pathogens to support microbiological risk assessment, including hazard identification.

This series is aimed at a scientific audience with general knowledge of microbiology and provides contemporary information on the characteristics of microorganisms that may be useful to technical members of the food industry, food safety consultants and food regulators.

Each monograph contains details on growth and survival characteristics of the pathogen, symptoms of disease, virulence factors, epidemiological data (including a summary of large, well-documented outbreaks), occurrence of the pathogen in food, susceptible populations and the dose-response relationship. At the end of each monograph is a list of recommended reading and useful links for further information.

The series will be updated and expanded over future years to ensure the information remains current and relevant to its audience. FSANZ welcomes and invites comments, suggestions, corrections or additional information to enhance the material presented in these monographs.



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## Campylobacter species

Beatrice Dias-Wanigasekera

*Campylobacter* spp. are bacteria that cause the gastrointestinal disease campylobacteriosis. Most cases of *Campylobacter* disease are non-fatal, but complications of acute infection may result in symptoms that mimic appendicitis. Infection with *Campylobacter* spp. has 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 comprising 18 species, six subspecies and two biovars (Humphrey et al. 2006; Tangwatcharin et al. 2006). The two species most commonly implicated with human disease are *C. jejuni* and *C. coli*. *C. jejuni* accounts for the majority of *Campylobacter*-related human illness, with *C. Coli* accounting for 7–18.5% of human illness (Gurtler et al. 2005). *C. lari* and *C. upsaliensis* have infrequently been associated with disease in humans.

### 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). However, they are known to survive well in food production systems, such as poultry processing systems.

*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, which means that the number of *Campylobacter* spp. in foods will not increase when held at normal room temperatures (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 on survival of *Campylobacter* spp. on naturally contaminated chicken skin and minced meat at freezing temperatures (–22 °C), Sampers et al. (2010) found that numbers declined by approximately 1 log over the first 24 hour period. No further significant reduction was achieved by prolonged freezing, with *Campylobacter* spp. being detected in samples (0.1 g) 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.

*Campylobacter* spp. are highly sensitive to loss of moisture and do not survive well on dry surfaces (Fernandez et al. 1985). *Campylobacter* spp. are much less tolerant to osmotic stress caused by a change in internal water content than a number of other foodborne pathogenic bacteria. For example, *Campylobacter* spp. are not capable of multiplication in an environment where sodium chloride concentration is 2% or higher (Doyle and Roman 1982).

*Campylobacter* have varying degrees of oxygen tolerance (3–5%) between species (Forsythe 2000). Recent studies have shown that 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 strong 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 or freezing (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 when other conditions are near optimum (ICMSF 1996)

	Minimum	Optimum	Maximum
Temperature (°C)	30	37–43	45
pH	5.0	6.5–7.5	8.0
Water activity	0.987	0.997	≥ 0.997

## Symptoms of disease

Principal symptoms caused by *Campylobacter* spp. are diarrhoea (sometimes bloody), nausea, abdominal pain, fever, muscle pain, headache, and rarely, vomiting (Lastovica and Skirrow 2000). The onset of symptoms is often abrupt with cramping abdominal pains quickly followed by diarrhoea. The incubation period is 18 hours to 8 days (mean of 3 days). The unique feature of the disease is abdominal pain which may become continuous and sufficiently intense to mimic acute appendicitis. Hence this is a common reason for admission of *Campylobacter* enteritis patients to hospital (Skirrow and Blaser 2000).

Severe *C. jejuni* infection may cause reactive arthritis, pneumonia, and 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 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 is produced to a lesser degree by *C. coli* (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 wild and domestic animals, including cattle, sheep, goats, dogs, rabbits, cats, chickens, turkeys, ducks, seagulls, pigeons, blackbirds, starlings, sparrows and pigs (Smibert 1984; Nielsen et al. 1997). Rodents, beetles and houseflies have also been shown to carry *Campylobacter* spp.

*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 2008 *Campylobacter* was the most frequently notified foodborne infection in Australia, with a rate of 108 cases per 100,000 population (15,535 cases). This was a slight decrease from the 2003–2007 mean of 117 cases per 100,000 population (ranging from 111.1–121.0 cases per 100,000 population per year) (OzFoodNet 2009; NNDSS 2010).

In New Zealand the notification rate in 2008 was 156.8 cases per 100,000 population (6,693 cases). This rate was significantly lower than the reported 2007 rate of 302.2 cases per 100,000 population (ESR 2009).

While not a notifiable disease in the US, surveillance through FoodNet (representing 15% of the population) reported a rate of *Campylobacter* infection of 13.0 cases per 100,000 population in 2009. This represents a 30% decrease in surveillance data from 1996–1998 (CDC 2010b). The number of confirmed human campylobacteriosis cases reported in the EU was 40.7 per 100,000 population in 2008, ranging from 0.2 to 193.3 per 100,000 population between countries; a reduction of 5.0% from the 2007 rate (EFSA 2010b).

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). *C. jejuni* is one of the most commonly reported agents associated with foodborne illness in many developed countries, including New Zealand, the UK and the US (Mead et al. 1999; Park 2002). A main peak of *C. jejuni* during summer and a winter peak of *C. coli* has also been found in Germany (Gurtler et al. 2005).



Outbreaks due to *Campylobacter* spp. have been associated with poultry meat, raw (unpasteurised) milk and milk products, beef, pork and shellfish, with cross-contamination often being reported as risk factor (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 (Fatalities)	Food	Country	Comments	References
2007	68	Cheese	USA	Cheese from raw milk was prepared and consumed as part of community celebration activities	(KDHE 2007)
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> was implicated	(Forbes et al. 2009)
2005	79	Chicken salad	Denmark	Cross-contamination from raw chicken to the chicken salad during preparation and storage. <i>C. jejuni</i> was implicated	(Mazick et al. 2006)
2003	81	Custard prepared from UHT milk	Spain	Occurred in a school. The custard was likely to have been contaminated with <i>C. jejuni</i> from raw chicken prepared previously in the same kitchen	(Jiménez et al. 2005)
1998	79	Tuna salad	USA	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	Australia	Cucumber served at self service salad bar. Probably contaminated by raw meat	(Kirk et al. 1997)

## Occurrence in food

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 new 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 a baseline survey carried out on the incidence and concentration of *Campylobacter* spp. and *Salmonella* spp. in chicken in Australia during 2007–2008, 84.3% of post-processing carcass rinse samples (n=1104) 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).

In New Zealand, 72.7% of retail carcasses (n=500) were found to be contaminated with *C. jejuni*, as detected during 2005 to 2008. 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 revealed that 75.8% of broiler carcasses sampled (n=9213) 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 2010a).

In the UK, a survey of poultry sold at retail carried out between May 2007 and September 2008 indicated that 65.2% of samples tested (n=3274) were contaminated by *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 during March 2001 to October 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 mushroom (n=217) samples tested. 83.4% of the positive samples were contaminated with *C. jejuni* while 16.6% were contaminated with *C. coli* (Whyte et al. 2004).

## Host factors and immunity

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/natural 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 antibodies are known to appear one week after infection and IgG antibodies peak a few weeks later. IgA and IgM antibodies disappear within 2 to 3 months, while IgG antibodies remain for much longer.

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 and strain specific variations are known to occur (Havelaar et al. 2009). In some cases, acquired immunity could lead to resistance to colonization by *Campylobacter* spp. (Tribble et al. 2010).

The incidence of *Campylobacter* infection in patients with 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 illness (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 gastric acid, 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 (2009) Bad bug book: Foodborne pathogenic microorganisms and natural toxins handbook - *Campylobacter jejuni*. US Food and Drug Administration, Silver Spring.

<http://www.fda.gov/Food/FoodSafety/Foodbornellness/FoodbornellnessFoodbornePathogensNaturalToxins/BadBugBook/ucm070024.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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Parts of this document have been published in previous FSANZ microbiological risk assessments for poultry meat and dairy (including raw milk products) – these are available on the FSANZ website [www.foodstandards.gov.au](http://www.foodstandards.gov.au)

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## Hepatitis A virus

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Hepatitis A virus (HAV) infects the liver, with disease characterised by liver inflammation and the development of jaundice. HAV infection can be asymptomatic (no clinical symptoms) in mild cases, or lead to severe liver damage in chronic cases. Hepatitis A is endemic in many developing countries, while in developed countries sporadic outbreaks occur.

### Description of the organism

HAV is classified in the genus Hepatovirus, which belongs to the Picornaviridae family of viruses. The Picornaviridae family consists of small (25–28 nm) non-enveloped viruses which are generally more robust and are better able to survive in the environment compared to enveloped viruses, such as herpesvirus. HAV particles consist of a single strand of RNA contained within an icosahedral shaped protein shell (Schoub 2003; Cook and Rzezutka 2006; Levinson 2006).

HAV has one known serotype. There are seven genotypes (I–VII) of HAV, four of which (I, II, III and VII) have been associated with human illness. Genotypes I and III are further divided into A and B. The majority of human strains are genotype I (Robertson et al. 1992; Hollinger and Emerson 2001). Isolates from a particular HAV outbreak are usually of the same genotype (Normann et al. 2008).

### Growth and survival characteristics

HAV requires specific living cells (host cells) in order to replicate. This means that the level of HAV in contaminated food will not increase during processing, transport or storage (Koopmans and Duizer 2004). While not able to replicate outside the host, HAV has been shown to survive in the environment for extended periods of time (Schoub 2003; Cook and Rzezutka 2006). The survival of HAV is influenced by environmental factors such as temperature, pH, chemicals and food composition.

It has been demonstrated that under conditions simulating typical environmental exposure, HAV remains infectious after being dried and stored for one month (McCaustland et al. 1982). HAV has also been shown to survive on various non-porous surfaces such as aluminium, china and latex for 60 days, however, it does not survive as well on porous materials (Abad et al. 1994). A study by Mbithi et al. (1992) demonstrated that HAV survives and remains infectious on human hands after 4 hours and can be transferred between hands and inanimate surfaces.

Without a standard protocol determining virus survival it is difficult to compare survival rates observed between studies. HAV has been shown to survive in fresh river water, seawater, groundwater and untreated tap water (Enriquez et al. 1995; Rzezutka and Cook 2004; Cook and Rzezutka 2006). A study by Arnal et al. (1998) using artificial sterile seawater contaminated with HAV demonstrated that the genetic material of HAV was stable and remained in the water for 232 days, however, by 35 days no infectious HAV particles were detected. In general, survival of HAV in water is enhanced at low temperatures (<4 °C) (Rzezutka and Cook 2004).



Croci et al. (2002) demonstrated that when fresh produce was stored at 4 °C HAV survived and remained infective on carrots for 4 days, fennel for 7 days and on lettuce for the study duration of 9 days. The differing survival rates observed on fresh produce may be due to the difference in surface texture of the produce and the presence of anti-microbial substances. Shieh et al. (2009) showed that when spinach was stored at 5.4 °C a 1 log reduction in the level of HAV occurred over a 28.6 day period. These studies imply that HAV can persist under normal domestic storage conditions.

Chemical and physical factors can affect the heat resistance of HAV. Deboose et al. (2004) investigated the inactivation of HAV in strawberry puree and found that increasing the sucrose concentration resulted in increased heat resistance of HAV. Conversely, lowering the pH was found to decrease the heat resistance of HAV. Changing the calcium concentration had no effect. Higher fat content also increases the heat resistance of HAV, as dairy products with higher fat content require longer exposure to heat to achieve the same level of reduction in HAV (Bidawid et al. 2000b).

HAV has been found to be resistant to temperatures up to 60 °C. The temperature at which 50% of HAV particles disintegrate and release their viral RNA is 61 °C (10 minutes, pH 7). When stabilised by 1 mol/L MgCl<sub>2</sub>, 50% disintegration of HAV occurs at 81 °C (Siegl et al. 1984). In food, complete inactivation of HAV has been observed in shellfish when heated to 85 °C for 3 minutes or 95 °C for 2 minutes (Millard et al. 1987). These conditions are known to inactivate HAV in shellfish while maintaining a commercially acceptable product (Appleton 2000). For milk and cream, heating to 85 °C for 30 seconds is sufficient to cause a 5 log reduction in HAV titre (Bidawid et al. 2000b).

Low temperature has little effect on HAV survival. Butot et al. (2008) showed that frozen storage of HAV contaminated berries and herbs had little effect on HAV survival over the study period of 3 months.

HAV is highly resistant to acidic conditions and solvents. Scholz et al. (1989) demonstrated that at pH 1 (24 °C) HAV retained high infectivity after 2 hours and was still infectious after 5 hours. Under conditions that simulate the acidity of the human stomach (38 °C, pH 1) HAV remained infectious for 90 min. Also, being a non-enveloped virus, HAV is resistant to solvents such as 20% ether and chloroform (the envelop of some viruses is susceptible to ether) (Hollinger and Emerson 2001).

## Symptoms of disease

HAV infection often causes mild illness in humans, or results in no clinical disease at all. In children this is particularly common, with more than 90% of children under 5 years of age showing no symptoms (asymptomatic infection) (Issa and Mourad 2001; FDA 2009). For those individuals in which clinical disease occurs, initial symptoms include sudden onset of fever, lethargy, weakness, nausea, anorexia, arthralgias (joint pain) and myalgia (muscular pain). Flu-like symptoms may occur in children with symptomatic infection. The initial symptoms tend to abate with the onset of jaundice (yellowing of the skin and eyes and a browning of urine due to stimulation of bile pigment production), although anorexia, lethargy and weakness may persist (Koff 1998; Hollinger and Emerson 2001; FDA 2009).

Most patients show complete recovery from symptoms within 3–6 months of the onset of illness. Less than 0.4% of reported cases in the US are fatal, these rare deaths usually occur in the elderly. Acute liver failure due to severe HAV infection has been reported in children; however, it is more frequent in middle-aged and older people and those with underlying chronic liver disease. Acute liver failure is also a rare complication of HAV infection during pregnancy (Koff 1998; FDA 2009).

The incubation period before onset of disease is 10–50 days (mean time of 30 days). Individuals that have been infected with high levels of viral particles have a shorter incubation period (FDA 2009). HAV is shed in the faeces of infected individuals for up to 2 weeks before the onset of illness. HAV is present in the blood at the same time as viral shedding starts occurring. The virus disappears from the blood shortly after symptoms of disease start, while faecal shedding of the virus continues for another 1–2 weeks (Hollinger and Emerson 2001).

Weeks to months after apparent recovery, symptoms may recur and HAV may once again be shed in the faeces. Multiple relapses are common in children (Koff 1998).

### Virulence and infectivity

The target organ of HAV is the liver. HAV is initially ingested, infects the intestinal tract and is then transported to the liver via the bloodstream. In the liver, HAV attaches to receptors on the surface of the hepatocytes, enters these cells and replicates. Replication of HAV within the hepatocytes is not believed to result in immediate cell damage; this is thought to occur subsequent to replication and release of the virus. The host's immune response is responsible for destroying the HAV infected cells. As a consequence of this pathological damage the liver becomes inflamed (WHO 2000; Schoub 2003; Cook and Rzezutka 2006). Released viral particles enter the bile duct and pass into the gastrointestinal tract to be shed in the faeces (Cook and Rzezutka 2006). The resistance of HAV to inactivation by bile and intestinal proteolytic enzymes allows the virus to be shed in the faeces and facilitates faecal-oral transmission (Koff 1998).

### Mode of transmission

HAV is transmitted via the faecal-oral route by either person-to-person contact or consumption of contaminated food or water (Guillois-Becel et al. 2009). Poor sanitation and crowding facilitate HAV transmission (FDA 2009). Person-to-person transmission often involves young children with unrecognised HAV infection (asymptomatic infection) (Staes et al. 2000).

In contrast to person-to-person transmission, outbreaks of HAV infections usually result from faecal contamination of a single source of food or water. Foods may become contaminated in their growing and harvesting areas (usually by coming into contact with sewage polluted water) or can be contaminated by infected food-handlers (Appleton 2000; Hollinger and Emerson 2001). Infected food handlers may contaminate foods directly or contaminate surfaces on which foods are prepared. A major issue with infected food handlers is that they are often unaware they constitute a hazard, as most of the faecal shedding of HAV occurs prior to the onset of clinical symptoms (Cook and Rzezutka 2006). Food establishments with poor sanitary conditions and inadequate treatment and/or disposal of human waste (sewage), along with unsatisfactory manufacturing practices may also contribute to food contamination (Sattar et al. 2000).

Travel to areas in which HAV is endemic from low prevalence areas is known to be a risk factor for HAV infection. The likelihood of becoming infected with HAV depends on local hygienic and sanitary conditions, which vary from country to country (Koff 1998). In 2008 the majority of HAV cases reported in Australia were acquired overseas, with 45% of cases locally acquired (OzFoodNet 2009c).

HAV transmission through blood and blood products is rare. While HAV is present in the blood of infected individuals, this is only for approximately a 2 week period. However, post-transfusion HAV infection has occurred, as have outbreaks of HAV in haemophiliacs who received contaminated blood plasma-derived factor VIII concentrate (Mannucci et al. 1994; Hollinger and Emerson 2001).

## Incidence of illness and outbreak data

HAV has a worldwide distribution; however, the prevalence of infection is related to the quality of the water supply, level of sanitation and the age of the individual when infected. In most developing countries, where HAV infection is endemic, the majority of people are infected in early childhood and virtually all adults are immune. In developed countries, HAV infections are less common due to improved sanitation. As a result very few people are infected in early childhood and the majority of adults remain susceptible to infection. Hence in these countries the risk of epidemics and the occurrence of severe disease may increase as the majority of people infected during an outbreak would be adults (children are often asymptomatic) (Conaty et al. 2000; Issa and Mourad 2001; Koopmans and Duizer 2004).

Hepatitis A is a notifiable disease in all Australian states and territories. The incidence of HAV infection notified in Australia in 2008 was 1.3 cases per 100,000 population (276 cases). This is a decline from the 2003–2007 mean of 1.5 cases per 100,000 population per year (ranging from 0.8–2.2 cases per 100,000 population per year) (OzFoodNet 2009c; NNDSS 2010).

In north Queensland in 1996–1999 the average annual HAV notification rates in Indigenous and non-Indigenous people were 110 and 25 cases per 100,000 population, respectively. In 1999 a HAV vaccination program for Indigenous children in north Queensland was introduced. Consequently, in 2000–2003 the average annual HAV notification rates for Indigenous and non-Indigenous people were 4 and 2.5 cases per 100,000 population, respectively (Hanna et al. 2004). HAV is now included as part of the National Immunisation Program Schedule for Aboriginal and Torres Strait Islander children in high risk areas (DOHA 2009). HAV vaccination is also recommended for travellers to endemic areas and those at increased risk because of lifestyle or occupation (DOHA 2008).

The notification rate for HAV in New Zealand in 2008 was 2.1 cases per 100,000 population (91 cases). This was a significant increase from the 2007 rate of 1.0 cases per 100,000 population (ESR 2009). The incidence of HAV in the US has declined from 12 cases per 100,000 population in 1995 to 0.86 cases per 100,000 population in 2008. This reduction has followed the 1999 recommendation for routine vaccination of children in areas of the US with consistently elevated rates of HAV (CDC 2009; CDC 2010).

Foodborne outbreaks of HAV have been recognised for over 40 years, but are infrequently reported. This is because the 2–6 week incubation period for HAV makes it more difficult to associate the source of infection with a particular food (Appleton 2000).

Cold cut meats, sandwiches, fruits and fruit juices, milk and milk products, vegetables, salads, shellfish and iced drinks have been implicated in HAV outbreaks (FDA 2009) (refer to Table 1).

Table 1: Selected major foodborne outbreaks associated with HAV (&gt;50 cases and/or ≥1 fatality)

Year	No. cases (fatalities)	Food	Country	Comments	Reference
2009	>140	Semi-dried tomatoes	Australia	Some of the infected individuals were food handlers	(OzFoodNet 2009a; OzFoodNet 2009b)
2004	351	Orange juice	Egypt	Significant hygiene problems at the production plant; no heat treatment of the finished product	(Frank et al. 2007)
2004	269	Raw beef	Belgium	Infected food handler identified at the meat distribution plant	(Robesyn et al. 2009)
2003	601 (3)	Green onions	USA	Green onions contaminated before or during packing on the farm in Mexico	(Wheeler et al. 2005)
1997	444	Oysters	Australia	The lake from which the oysters were harvested was polluted by sewage from a local town and boats	(Conaty et al., 2000)
1997	254	Frozen strawberries	USA	Consumption of items from school cafeterias containing frozen strawberries was associated with HAV	(Hutin et al. 1999)
1996	5620	Raw seafood	Italy		(Lopalco et al. 1997)
1988	300 000+ (47)	Raw clams	China		(Cooksley 2000)

## Occurrence in food

The types of food most often implicated in HAV outbreaks are those that are either eaten raw or only slightly cooked (e.g. shellfish), or handled extensively prior to consumption (e.g. the picking and packing of raw produce in the field and the preparation of sandwiches and salads) (Koopmans and Duizer 2004; Cook and Rzezutka 2006).

Bivalve molluscs (including oysters, mussels, clams and cockles) live in shallow, coastal and estuarine waters which can be polluted with human sewage. They are filter feeders, so collect nutrients by filtering particulate matter from the water. If molluscs are grown in water contaminated with human faeces, the molluscs can collect and concentrate HAV from the water (Appleton 2000; Moore 2001; Cook and Rzezutka 2006). HAV has been shown to be concentrated within mussels to 100-fold higher concentrations than the surrounding water and can persist for about 7 days in the mussels (Enriquez et al. 1992). HAV has been detected in oyster samples more than 2 months after the presumed contamination event, this is thought to be due to recontamination of the oysters from sediment in the water (Conaty *et al.* 2000).

The prevalence of HAV reported in shellfish ranges between 6–27%, depending on the location and analytical technique utilised. For mussels sampled from markets of major cities in south Italy, 15.6% were found to be contaminated with infectious HAV (n=180) (Crocì et al. 2003). For shellfish (clams, mussels, scallops and oysters) collected from the North Adriatic sea located between the Italian and Balkan peninsulas, HAV was detected in 6% of samples (n=235) (Crocì et al. 2007). For shellfish collected off the coast of Spain (cultured and wild mussels, wild clams and cockles), HAV was detected in 27.4% of samples (n=164) (Romalde et al. 2002). The methods utilised in these studies detect the genetic material of HAV, however, some methods are more sensitive than others under different conditions. This suggests that the level of HAV contamination could be higher than reported.

Hernandez et al. (1997) demonstrated that 20% of pooled samples of wash water collected from lettuces in Costa Rica were contaminated with HAV (n=10 pools, 5 lettuces per pool), suggesting that lettuces from this region could be a vehicle for HAV transmission.

### Host factors and immunity

People of all ages are susceptible to HAV infection (unless they have had a previous infection or vaccination). The disease is milder in young children under 6 years, with the risk of fatality increasing with age. Thus the risks are higher for unexposed older people (ESR 2001; FDA 2009).

A single HAV infection or administration of the HAV vaccine provides lifelong immunity for the individual against the virus (Leon and Moe 2006). When an outbreak of HAV occurs, if exposure can be recognised before cases begin to occur, treatment with intramuscular immunoglobulin (passive immunisation) within 2 weeks of exposure is >85% effective at preventing HAV infection. However, passive immunisation is only effective for a short time (3–6 months) and people will be susceptible to infection after another exposure (Hollinger and Emerson 2001; Issa and Mourad 2001).

### Dose response

The number of HAV particles required to cause infection is not known, however, it is presumed to be 10–100 viral particles (FDA 2009). In fact it has been suggested that a single ingested viral particle may cause infection, however, the probability of this occurring is very low (Cliver 1985). It has been estimated that up to 13,000 infectious HAV particles may be present in 1 mg of faeces (Bidawid et al. 2000a).

### Recommended reading and useful links

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## *Listeria monocytogenes*

Adèle Yates

*Listeria monocytogenes* is a bacterium that causes listeriosis, a disease that can have severe consequences for particular groups of the population. It can cause miscarriages in pregnant women and be fatal in immunocompromised people, such as cancer patients or individuals taking immunosuppressive medication. In healthy people, listeriosis generally only causes a mild form of illness. *L. monocytogenes* can be found throughout the environment. It has been isolated from domestic and wild animals and birds, as well as from soil, vegetation, fodder and water.

### Description of the organism

*L. monocytogenes* is a Gram-positive, non-spore forming rod-shaped bacterium. It belongs to the genus *Listeria* along with *L. innocua*, *L. welshimeri*, *L. selligeri*, *L. ivanovii* and *L. grayi* (Montville and Matthews 2005). Of these species, only two are considered pathogens; *L. monocytogenes* which infects humans and animals, and *L. ivanovii* which infects ruminants (although there have been rare reports of *L. ivanovii* being isolated from infected humans) (Guillet et al. 2010). There are thirteen known serotypes of *L. monocytogenes*: 1/2a, 1/2b, 1/2c, 3a, 3b, 3c, 4a, 4ab, 4b, 4c, 4d, 4e and 7. The serotypes most often associated with human illness are 1/2a, 1/2b and 4b (FDA/USDA/CDC 2003).

### Growth and survival characteristics

The growth and survival of *L. monocytogenes* is influenced by a variety of factors. In food these include temperature, pH, water activity, salt and the presence of preservatives (refer to Table 1). For a bacterium that does not form spores, *L. monocytogenes* is considered relatively resistant to heat, freezing and drying (FDA 2009).

The temperature range for growth of *L. monocytogenes* is between -1.5 and 45 °C, with the optimal temperature being 30–37 °C. Temperatures above 50 °C are lethal to *L. monocytogenes*. Freezing can also lead to a reduction in *L. monocytogenes* numbers (Lado and Yousef 2007). As *L. monocytogenes* can grow at temperatures as low as 0 °C, it has the potential to grow, albeit slowly, in food during refrigerated storage.

*L. monocytogenes* will grow in a broad pH range of 4.0–9.6 (Lado and Yousef 2007). Although growth at pH <4.0 has not been documented, *L. monocytogenes* appears to be relatively tolerant to acidic conditions. *L. monocytogenes* becomes more sensitive to acidic conditions at higher temperatures (Lado and Yousef 2007).

Like most bacterial species, *L. monocytogenes* grows optimally at a water activity ( $a_w$ ) of 0.97. However, *L. monocytogenes* also has the ability to grow at a  $a_w$  of 0.90 (Lado and Yousef 2007). Johnson et al. (1988) demonstrated that *L. monocytogenes* can survive for extended periods of time at  $a_w$  values of 0.81. *L. monocytogenes* is reasonably tolerant to salt and has been reported to grow in 13–14% sodium chloride (Farber et al. 1992). Survival in the presence of salt is influenced by the storage temperature. Studies have indicated that in concentrated salt solutions *L. monocytogenes* has a higher survival rate at lower temperatures (Lado and Yousef 2007).

*L. monocytogenes* can grow under both aerobic and anaerobic conditions, although it grows better in an anaerobic environment (Sutherland et al. 2003; Lado and Yousef 2007).

The effect of preservatives on the growth of *L. monocytogenes* is influenced by the combined effects of temperature, pH, salt content and water activity. For example, the ability of sorbates and parabens to prevent the growth of *L. monocytogenes* is enhanced at lower storage temperatures and pH. Also, when sodium chloride is added or the temperature is lowered, lactate is more effective at preventing *L. monocytogenes* growth. Decreased temperatures (such as refrigeration storage) enhances the ability of sodium diacetate, sodium propionate and sodium benzoate to prevent growth of *L. monocytogenes* (Lado and Yousef 2007).

Table 1: Limits for growth when other conditions are near optimum (Lado and Yousef 2007)

	Minimum	Optimum	Maximum
Temperature (°C)	-1	30–37	45
pH	4.0	6.0–8.0	9.6
Water activity	0.90	0.97	–

## Symptoms of disease

There are two main forms of illness associated with *L. monocytogenes* infection. Non-invasive listeriosis is the mild form of disease, while invasive listeriosis is the severe form of disease and can be fatal (FDA/USDA/CDC 2003).

The likelihood that invasive listeriosis will develop depends upon a number of factors, including host susceptibility, the number of organisms consumed and the virulence of the particular strain (WHO/FAO 2004).

Symptoms of non-invasive listeriosis are similar to those associated with gastrointestinal illness and may include chills, diarrhoea, headache, abdominal pain and cramps, nausea, vomiting, fatigue and myalgia (muscular pain). The onset of illness is usually within 24 hours (FDA/USDA/CDC 2003; Swaminathan and Gerner-Smidt 2007). Non-invasive listeriosis is also known as listerial gastroenteritis or febrile listeriosis.

Invasive listeriosis is characterised by the presence of *L. monocytogenes* in the blood, in the fluid of the central nervous system (leading to meningoenzephalitis), or infection of the uterus or cervix of pregnant women. The latter may result in spontaneous abortion in the second or third trimester of pregnancy, or stillbirth. Influenza-like symptoms including ongoing fever and gastrointestinal symptoms such as nausea, vomiting and diarrhoea may precede the more severe form of listeriosis. The incubation period before onset of disease is typically 2–3 weeks, however, the time of onset may extend to 3 months (FDA/USDA/CDC 2003; FDA 2009).

## Virulence and infectivity

When *L. monocytogenes* is ingested, it may survive the stomach environment and enter the intestine where it penetrates the intestinal epithelial cells. The organism is then taken up by macrophages and non-phagocytic cells. The *L. monocytogenes* surface protein internalin is required for this uptake by non-phagocytic cells, as it binds to the receptors on the host cells to instigate adhesion and internalization. The bacterium is initially located in a vacuole when it is taken up by a macrophage or non-phagocytic cell. *L. monocytogenes* secrete listeriolysin O protein, which breaks down the vacuole wall and enables the bacteria to escape into the cytoplasm. Any bacteria remaining in the vacuole are destroyed by the host cell. Once located in the cytoplasm, *L. monocytogenes* is able to replicate. *L. monocytogenes* is transported around the body by the blood, with most *L. monocytogenes* being inactivated when they reach the spleen or liver. *L. monocytogenes* is able to utilise the actin molecules of the host to propel the bacteria into neighbouring host cells. In the case of invasive listeriosis, this ability to spread between host cells enables *L. monocytogenes* to cross the blood-brain and placental barriers (Montville and Matthews 2005; Kuhn and Goebel 2007; Bonazzi et al. 2009).

## Mode of transmission

The most common transmission route of *L. monocytogenes* to humans is via the consumption of contaminated food. However, *L. monocytogenes* can be transmitted directly from mother to child (vertical transmission), from contact with animals and through hospital acquired infections (Bell and Kyriakides 2005).

Healthy individuals can be asymptomatic carriers of *L. monocytogenes*, with 2–6% of healthy people being found to shed *L. monocytogenes* in their faeces. However, outbreak investigations have shown that listeriosis patients do not always shed the organism in their faeces. Therefore the role of healthy carriers in the transmission of *L. monocytogenes* is unclear (Rocourt and Cossart 1997; FDA/USDA/CDC 2003).

## Incidence of illness and outbreak data

Listeriosis is a notifiable disease in all Australian states and territories. The incidence of listeriosis notified in Australia in 2008 was 0.3 cases per 100,000 population (65 cases). This is the same as the 2003–2007 mean of 0.3 cases per 100,000 population per year (ranging from 0.2–0.3 cases per 100,000 population per year). In Australia the fatality rate in 2008 was 18% (OzFoodNet 2009b; NNDSS 2010). The notification rate for listeriosis in New Zealand in 2008 was 0.6 cases per 100,000 population (27 cases). This was the same notification rate as 2007. The fatality rate in New Zealand in 2008 was 19% (ESR 2009).

In the US the notification rate for listeriosis in 2008 was 0.29 cases per 100,000 population. This was similar to the 2007 rate of 0.27 cases per 100,000 population (CDC 2010). In the EU there were 0.3 confirmed cases of listeriosis per 100,000 population in 2008 (ranging from 0–0.9 cases per 100,000 between countries). This was a 11.1% decrease in the number of cases from 2007 (EFSA 2010).

Invasive *L. monocytogenes* infections can be life threatening, with average fatality rates being 20–30% among hospitalized patients (WHO/FAO 2004; Swaminathan and Gerner-Smidt 2007)

Most cases of listeriosis are sporadic. Despite this, foodborne outbreaks due to *L. monocytogenes* have been associated with cheese, raw (unpasteurised) milk, deli meats, salad, fish and smoked fish, ice cream and hotdogs (Montville and Matthews 2005; Swaminathan and Gerner-Smidt 2007) (refer to Table 2).

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

Year	Total no. cases (fatalities)	No. perinatal cases (fatalities)	Food	Country	Comments	Reference
2009	40(3)	8(3)	Chicken wrap	Australia	<i>Listeria</i> isolated from chicken meat supplier, deficiencies in the food safety program for chicken meat production	(OzFoodNet 2009a)
2008	57(22)	0	Deli meats	Canada	<i>Listeria</i> identified on plant equipment, company tried to correct problem with sanitation program; low sodium product	(Govt of Canada 2009)
1998–1999	108 (18)	13(4)	Frankfurters	USA	Contamination due to demolition of ceiling refrigeration unit in frankfurter hopper room	(Mead et al. 2006)
1997	1566*	0	Corn and tuna salad	Italy	Possible cross-contamination from other untreated foods	(Aureli et al. 2000)
1992	279(92)	92(29)	Jellied pork tongue	France	<i>Listeria</i> identified at manufacturing facility	(Norton and Braden 2007)
1985	142 (48)	93 (30)	Mexican-style soft cheese	USA	Cheese was made from contaminated milk that was unpasteurised or inadequately pasteurised	(Linnan et al. 1988)
1983–1987	122(34)	65(16)	Vacherin Mont d'Or cheese	Switzerland	Contamination thought to be from cellars used for ripening the cheese	(Norton and Braden 2007)
1981	41(18)	34(16)	Coleslaw	Canada	Cabbage fertilised with manure from sheep with listeriosis	(Schlech et al. 1983)

\* Non-invasive listeriosis

## Occurrence in food

*L. monocytogenes* has been isolated from various ready-to-eat products. In a study by Meldrum et al. (2010) the prevalence of *L. monocytogenes* was 4.1% in crustaceans (n=147), 6.7% in smoked fish (n=178), 2% in sushi (n=50) and 0.9% in green salad (n=335) samples in Wales. Wong et al. (2005) isolated *L. monocytogenes* from 1% of ham (n=104) and 1.7% of pate (n=60) samples in New Zealand. *L. monocytogenes* has also been isolated from dairy products. For example, *L. monocytogenes* was detected in 1.3% of fresh cheese samples in Spain (n=78), 0.2% of hard cheese samples in the UK (n=1242) and 0.3% of ice creams in Italy (n=1734) (Busani et al. 2005; Cabedo et al. 2008; Little et al. 2009). The prevalence of *L. monocytogenes* in bulk milk tank internationally is 1–60% (FSANZ 2009).

The presence of *L. monocytogenes* in ready-to-eat products is probably due to contamination occurring after the product has been processed. This contamination may occur during additional handling steps such as peeling, slicing and repackaging. Also, in the retail and food service environment, contamination may be transferred between ready-to-eat products (Lianou and Sofos 2007). The type of handling that ready-to-eat meat receives may also influence the level of *L. monocytogenes* contamination. In a survey of retail packaged meats there was a significantly higher prevalence of *L. monocytogenes* reported in products cut into cubes (61.5%) (n=13), compared with sliced products (4.6%) (n=196) (Angelidis and Koutsoumanis 2006).

## Host factors and immunity

People at risk of invasive listeriosis include pregnant women and their foetuses, newborn babies, the elderly and immunocompromised individuals (such as cancer, transplant and HIV/AIDS patients). Less frequently reported, but also at a greater risk, are patients with diabetes, asthma, cirrhosis (liver disease) and ulcerative colitis (inflammatory bowel disease (FDA 2009).

## Dose response

Investigations of foodborne outbreaks of non-invasive listeriosis have concluded that consumption of food with high levels of *L. monocytogenes* ( $1.9 \times 10^5/\text{g}$  to  $1.2 \times 10^9/\text{g}$ ) is required to cause illness in the general healthy population (Sim et al. 2002).

The number of *L. monocytogenes* required to cause invasive listeriosis depends on a number of factors. These include the virulence of the particular serotype of *L. monocytogenes*, the general health and immune status of the host, and attributes of the food (for example fatty foods can protect bacteria from stomach acid). Some *L. monocytogenes* serovars are more virulent than others; this may be attributed to differences in the expression of virulence factors which could influence the interactions with the host cells and cellular invasion (Severino et al. 2007). The FDA and WHO have developed separate models for both healthy and susceptible populations to predict the probability that an individual will develop listeriosis (FDA/USDA/CDC 2003; WHO/FAO 2004). The probability that a healthy person of intermediate age will become ill from the consumption of a single *L. monocytogenes* cell was estimated to be  $2.37 \times 10^{-14}$ . For more susceptible populations the probability that illness will occur was estimated to be  $1.06 \times 10^{-12}$ . A more recent assessment on invasive listeriosis in susceptible populations was performed which took into account the different serotypes of *L. monocytogenes* (Chen et al. 2006). This study showed that the probability of a susceptible individual developing invasive listeriosis ranged from  $1.31 \times 10^{-8}$  to  $5.01 \times 10^{-11}$ , suggesting that there are large differences in virulence between *L. monocytogenes* serotypes.

## Recommended reading and useful links

FDA (2009) Bad bug book: Foodborne pathogenic microorganisms and natural toxins handbook - *Listeria monocytogenes*. US Food and Drug Administration, Silver Spring.

<http://www.fda.gov/Food/FoodSafety/Foodbornellness/FoodbornellnessFoodbornePathogensNaturalToxins/BadBugBook/ucm070064.htm>

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Parts of this document have been published in previous FSANZ microbiological risk assessments for poultry meat and dairy (including raw milk products) – these are available on the FSANZ website [www.foodstandards.gov.au](http://www.foodstandards.gov.au)

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## Salmonella (non-typhoidal)

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*Salmonella* spp. are bacteria that cause salmonellosis, a common form of foodborne illness in humans. Some strains of *Salmonella* generally produce mild symptoms, while other strains cause severe disease and can be fatal. *Salmonella* spp. are carried by a range of domestic and wild animals and birds and have been widely isolated from the environment.

### Description of the organism

*Salmonella* spp. are Gram-negative, non-spore forming rod-shaped bacteria and are members of the family Enterobacteriaceae (Jay et al. 2003). The genus *Salmonella* is divided into two species: *S. enterica* (comprising six subspecies) and *S. bongori*. Over 99% of human *Salmonella* spp. infections are caused by *S. enterica* subsp. *enterica* (Bell and Kyriakides 2002; Crum-Cianflone 2008). Strains of *Salmonella* can be characterised serologically based on the presence and/or absence of O (somatic) and H (flagella) antigens. Phage typing is used to subtype *Salmonella* serotypes. The phage type is determined by the sensitivity of the bacterial cells to the lytic activity of selected bacteriophages (Bell and Kyriakides 2002; Jay et al. 2003).

The formal names used to describe types of *Salmonella* are rather cumbersome, for example *S. enterica* subsp. *enterica* serotype Typhimurium. For practical reasons, the shortened versions of these names are commonly used, such as *S. Typhimurium* (Bell and Kyriakides 2002).

Some *Salmonella* serotypes are host-adapted to individual animal species and may differ vastly in the severity of the disease they cause; others such as *S. Typhimurium* have a broad host range, with an ability to infect a wide range of animals, including humans (Jay et al. 2003; Wallis 2006).

*S. Typhi* and *S. Paratyphi* are specifically associated with infections in humans, leading to severe disease called enteric fever. *S. Typhi* and *S. Paratyphi* produce clinical syndromes referred to as typhoid and paratyphoid fever, respectively. Enteric fever is rare in developed countries, with the majority of cases associated with overseas travel (Darby and Sheorey 2008). For example, in Australia in 2008, 92.5% of notified cases of typhoid fever reported recent overseas travel (OzFoodNet 2009).

### Growth and survival characteristics

Salmonellae have relatively simple nutritional requirements and can survive for long periods of time in foods and other substrates. The growth and survival of *Salmonella* spp. is influenced by a number of factors such as temperature, pH, water activity and the presence of preservatives (refer to Table 1).

The temperature range for growth of *Salmonella* spp. is 5.2–46.2 °C, with the optimal temperature being 35–43 °C (ICMSF 1996). Although freezing can be detrimental to *Salmonella* spp. survival, it does not guarantee destruction of the organism. There is an initial rapid decrease in the number of viable organisms at temperatures close to the freezing point as a result of the freezing damage. However, at lower temperatures *Salmonella* spp. have the ability to survive long term frozen storage (Jay et al. 2003). Strawn and Dayluk (2010) showed that *Salmonella* was able to survive on frozen mangoes and papayas stored at -20 °C for at least 180 days.

Heat resistance of *Salmonella* spp. in food is dependent on the composition, pH and water activity of the food. The heat resistance of *Salmonella* spp. increases as the water activity of the food decreases. Foods which are high in fat and low in moisture, such as chocolate and peanut butter, may have a protective effect against heat. In low pH conditions the heat resistance is reduced (Jay et al. 2003; Shachar and Yaron 2006; Podolak et al. 2010).

*Salmonella* spp. will grow in a broad pH range of 3.8–9.5, with an optimum pH range for growth of 7–7.5 (ICMSF 1996). The minimum pH at which *Salmonella* spp. can grow is dependent on temperature, presence of salt and nitrite and the type of acid present. Volatile fatty acids are more bactericidal than organic acids such as lactic, citric and acetic acid. Outside the pH range for growth, cells may become inactivated, although this is not immediate and cells have been shown to survive for long periods in acidic products (Bell and Kyriakides 2002; Jay et al. 2003).

Water activity ( $a_w$ ) has a significant effect on the growth of *Salmonella* spp., with the optimum  $a_w$  being 0.99 and the lower limit for growth being 0.93. *Salmonella* spp. can survive for months or even years in foods with a low water activity (such as black pepper, chocolate, peanut butter and gelatine) (ICMSF 1996; Podolak et al. 2010).

*Salmonella* spp. are similar to other Gram negative bacteria in regard to susceptibility to preservatives commonly used in foods. Growth of *Salmonella* spp. can be inhibited by benzoic acid, sorbic acid or propionic acid. The inhibition of *Salmonella* spp. is enhanced by the use of several preservative factors in combination, such as a preservative in combination with reduced pH and temperature (ICMSF 1996; Banerjee and Sarkar 2004; Ha et al. 2004).

*Salmonella* spp. are classed as facultative anaerobic organisms as they do not require oxygen for growth (Jay et al. 2003).

Table 1: Limits for growth when other conditions are near optimum (ICMSF 1996; Podolak et al. 2010)

	Minimum	Optimum	Maximum
Temperature (°C)	5.2	35–43	46.2
pH	3.8	7–7.5	9.5
Water activity	0.93	0.99	>0.99

## Symptoms of disease

Outcomes of exposure to non-typhoidal *Salmonella* spp. can range from having no effect, to colonisation of the gastrointestinal tract without symptoms of illness (asymptomatic infection), or colonisation with the typical symptoms of acute gastroenteritis. Gastroenteritis symptoms are generally mild and may include abdominal cramps, nausea, diarrhoea, mild fever, vomiting, dehydration, headache and/or prostration. The incubation period is 8–72 hours (usually 24–48 hours) and symptoms last for 2–7 days (WHO/FAO 2002; Darby and Sheorey, 2008). Severe disease, such as septicaemia sometimes occurs, predominantly in immunocompromised individuals. This occurs when *Salmonella* spp. enters the bloodstream, leading to symptoms such as high fever, lethargy, abdomen and chest pain, chills and anorexia, and can be fatal (in less than 1% of cases). A small number of individuals develop a secondary condition such as arthritis, meningitis or pneumonia as a consequence of infection (Hohmann 2001; WHO/FAO 2002; FDA 2009).

*Salmonella* spp. are shed in large numbers in the faeces of infected individuals at the onset of illness. In the case of non-typhoid disease, bacterial shedding continues for about 4 weeks after illness in adults and 7 weeks in children. In 0.5% of non-typhoid cases individuals become long-term carriers and continue shedding the bacteria on an ongoing basis (Jay et al. 2003; Crum-Cianflone 2008).

## Virulence and infectivity

Once ingested, *Salmonella* spp. must survive the low pH of the stomach, adhere to the small intestine epithelial cells and overcome host defence mechanisms to enable infection (Jay et al. 2003).

*Salmonella* possesses a number of structural and physiological virulence factors enabling it to cause acute and chronic disease in humans. The virulence of *Salmonella* varies with the length and structure of the O side chains of lipopolysaccharide (LPS) molecules at the surface of the cell. Resistance of *Salmonella* to the lytic action of complement (part of the immune response) is directly related to the length of the O side chain (Jay et al. 2003). Other important virulence factors include the presence and type of fimbriae, which is related to the ability of *Salmonella* to attach to epithelium cells, as well as the expression of genes responsible for invasion into cells (Jones 2005). Some of these virulence genes are encoded on *Salmonella* pathogenicity islands (SPI). SPI-1 is required for invasion of the microorganism into intestinal epithelial cells, while systemic infections and intracellular accumulation of *Salmonella* are dependent on the function of SPI-2 (Valle and Guiney 2005).

*Salmonella* spp. produce a heat labile enterotoxin, resulting in the loss of intestinal fluids (causing diarrhoea). This enterotoxin is closely related functionally, immunologically and genetically to the toxin of *Vibrio cholerae* and the heat labile toxin of pathogenic *E. coli* (Jay et al. 2003). Most *Salmonella* strains also produce heat labile cytotoxin which may cause damage to the intestinal mucosal surface and results in general enteric symptoms and inflammation. Infection with non-typhoidal *Salmonella* is generally limited to a localised intestinal event. However, the presence of virulence plasmids has been associated with non-typhoidal *Salmonella* spp. surviving in phagocytes and spreading from the small intestine to the spleen and liver (Jay et al. 2003; Hanes 2003).

Multiple antibiotic resistant strains of *Salmonella* have emerged, an example being *S. Typhimurium* definitive phage type 104 (DT104). Multi-resistant *S. Typhimurium* DT104 infects both humans and animals, such as cattle and sheep. To date, this organism is not endemic in Australia, although it is a significant health problem in European countries, North America, the Middle East, South Africa and South-East Asia (Jay et al. 2003).

## Mode of transmission

*Salmonella* spp. are transmitted by the faecal-oral route by either person-to-person contact, consumption of contaminated food or water, or from direct contact with infected animals (Jay et al. 2003).

## Incidence of illness and outbreak data

Salmonellosis is one of the most commonly reported enteric illnesses worldwide, being the second most frequently reported cause of enteric illness in Australia (behind campylobacteriosis). It is a notifiable disease in all Australian states and territories, with a notification rate in 2008 of 38.9 cases per 100,000 population (8,310 cases). This was similar to the 2003–2007 mean of 40.1 cases per 100,000 population per year (ranging from 35.2–45.2 cases per 100,000 population per year) (OzFoodNet 2009; NNDSS 2010).

The salmonellosis notification rate varied between jurisdictions from 31 cases per 100,000 population in Victoria to 226 cases per 100,000 population in the Northern Territory. Children aged between 0–4 years had the highest notification rate, with 300 cases per 100,000 population reported for 2008 (OzFoodNet 2009). The higher rate of notified cases in this age group may reflect an increased susceptibility upon first exposure, but may also be a result of other factors such as an increased likelihood of exposure and increased likelihood to seek medical care.

The distribution of *Salmonella* serovars in Australia varies geographically, however *S. Typhimurium* was the most commonly reported serovar in 2008, representing 42% of all notified infections. Internationally, *S. Enteritidis* is frequently reported as cause of human illness, however it is not endemic in Australia, with >80% of notified cases reporting recent overseas travel (Greig and Ravel 2009; OzFoodNet 2009)

The notification rate for salmonellosis in New Zealand in 2008 was 31.5 cases per 100,000 population (1,346 cases). This was slightly higher than the 2007 rate of 30.1 cases per 100,000 populations (ESR 2009). In the US 16.92 cases of salmonellosis were notified per 100,000 population in 2008. This was a slight increase from the 2007 rate of 16.03 cases per 100,000 population (CDC 2010a). In the EU the notification rate for salmonellosis was 26.4 cases per 100,000 population in 2008 (ranging from 0–126.8 cases per 100,000 between countries). This was a 13.5% decrease in the number of cases from 2007 (EFSA 2010).

Outbreaks attributed to *Salmonella* spp. have been associated with eggs, poultry, raw meat, milk and dairy products, fresh produce, salad dressing, fruit juice, peanut butter and chocolate (Jay et al. 2003; Montville and Matthews 2005) (refer to Table 2).

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

Year	Serovar	Total no. cases (fatalities)	Food	Country	Comments	Reference
2009-2010	<i>S. Montevideo</i>	272	Salami containing red or black pepper	USA	Pepper was added to the salami after the kill step, pepper samples were positive for <i>S. Montevideo</i>	(CDC 2010b)
2008	<i>S. Montevideo</i>	61	Chicken	USA	Cross contamination of other food items with raw chicken, undercooking of chicken. <i>S. Montevideo</i> isolated from raw chicken	(Patel et al. 2010)
2006-2007	<i>S. Tennessee</i>	628	Peanut butter	USA	Environmental samples from the plant were positive for <i>S. Tennessee</i>	(CDC 2007)
2005-2006	<i>S. Oranienburg</i>	126	Alfalfa	Australia	Alfalfa at the production facility were positive for <i>S. Oranienburg</i>	(OzFoodNet 2006)
2005	<i>S. Typhimurium</i> PT135	63	Eggs used in bakery products	Australia	<i>S. Typhimurium</i> PT135 isolated from cream piping bag and bench of bakery. Issues with handling raw eggs, inadequate hygiene practices and cross-contamination. Eggs were dirty (externally) and from the same farm	(Stephens et al. 2007)
2001-2002	<i>S. Oranienburg</i>	>439	Chocolate	Germany	The high fat content of chocolate increases the heat resistance of <i>Salmonella</i> spp.	(Werber et al. 2005)
1999	<i>S. Typhimurium</i> PT135a	507	Unpasteurised fruit juice	Australia	<i>S. Typhimurium</i> PT135a was found on the oranges. It was also found in the fungicide tank and wax tank (through which the oranges passed) of the packing shed	(Federal Court of Australia 2003)
1985	<i>S. Typhimurium</i>	16,284 (7)	Pasteurised milk	USA	Potential cross-contamination between the unpasteurised milk and pasteurised milk tank	(Ryan et al. 1987; Montville and Matthews 2005)

## Occurrence in food

The primary reservoir of *Salmonella* is the intestinal tract of warm and cold-blooded vertebrates, with many animals showing no sign of illness. Unlike diseased animals which can be removed from production and/or treated, these asymptomatic (carrier) animals can shed large numbers of *Salmonella* spp. in their faeces and are therefore an important source of contamination. Faecal shedding of *Salmonella* spp. leads to contamination of the surrounding environment including soil, crops, plants, rivers and lakes. A wide range of foods have been implicated in foodborne salmonellosis, particularly those of animal origin and those foods that have been subject to faecal contamination (ICMSF 1996; Jay et al. 2003).

At the time of slaughter, *Salmonella* infected animals may have high numbers of organisms in their intestines as well as on the outside of the animal (faecal contamination of hides, fleece, skin or feathers) (Bryan and Doyle 1995; Jay et al. 2003). In Australia, *Salmonella* spp. have been isolated from 3% of chilled cattle carcass samples (n=100) (Fegan et al. 2005). The distribution of *Salmonella* spp. on contaminated meat carcasses is not uniform. For example, a US study by Stopforth et al. (2006) found that the prevalence of *Salmonella* spp. on fresh beef ranged from 0.8% (rib eye roll, n=133) to 9.6% (strip loins, n=52) depending on the cut of meat. Cross contamination during processing may also lead to increased prevalence of *Salmonella* in finished products (Bryan and Doyle 1995).

*Salmonella* spp. are found in a range of foods. The prevalence of *Salmonella* spp. in bulk tank milk internationally is 0–11.8% (FSANZ 2009a). In shellfish (mussels, clams, oysters and cockles) collected off the coast of Spain, *Salmonella* spp. were detected in 1.8% samples (n=2980) (Martinez-Urtaza et al. 2003). Boughton et al. (2004) isolated *Salmonella* spp. from 2.9% of retail pork sausages samples in Ireland (n=921), and in Spain, *Salmonella* spp. were detected in 2% of cooked ham samples (n=53) and 11.1% of cured dried pork sausage samples (n=81) (Cabedo et al. 2008).

An Australian survey found 43.3% of chicken meat at retail (n=859) was positive for *Salmonella* spp. The most prevalent serovar was *S. Sofia*, with 30.5% of chicken meat samples positive for this serovar (Pointon et al. 2008). Although *S. Sofia* accounts for a large proportion of salmonellae isolated from poultry in Australia it is rarely associated with human or animal illness as it appears to be a non-virulent serovar (Gan et al. 2011). The predominance of *S. Sofia* in poultry is a uniquely Australian observation as *S. Sofia* is essentially geographically isolated to Australia (Mellor et al. 2010).

*S. Enteritidis* (in particular phage type 4) is a globally important *Salmonella* serotype that can infect the reproductive tract of poultry and contaminate the internal contents of eggs, however, it is not endemic in Australian egg layer flocks (FSANZ 2009b).

## Host factors and immunity

People of all ages are susceptible to *Salmonella* spp. infection. However, the elderly, infants and immunocompromised individuals are at a greater risk of infection and generally have more severe symptoms (Jay et al. 2003; FDA 2009).

## Dose response

Human feeding trials were undertaken during the 1950s to determine the relationship between the dose of *Salmonella* spp. ingested and the level of illness incurred. These studies showed that ingestion of between  $10^5$ – $10^{10}$  organisms caused infection (McCullough and Eisele 1951a; McCullough and Eisele 1951b; McCullough and Eisele 1951c; McCullough and Eisele 1951d). However, there are a number of limitations on the use of this feeding trial data. Firstly, the volunteers selected were all healthy adult males, so the results may underestimate the risk to the overall population. Secondly, low doses which are more likely to exist in real food contamination events were not considered (Kothary and Babu 2001; Bollaerts et al. 2008). Investigation of salmonellosis outbreaks has estimated dose ranges of  $<10$ – $10^9$  organisms (depending on the food) and as such, doses resulting in illnesses may be much lower than those reported in the feeding trials (Todd et al. 2008).

The WHO/FAO (2002) developed a dose-response model based on outbreak data. Using this model the probability of illness for ingestion of 100 organisms was  $1.3 \times 10^{-1}$ . However, it should be noted that the data used in this model have a certain degree of uncertainty, which required assumptions to be made. This is because it was difficult to determine the actual dose ingested (based on the level of the organism in the food at the time of consumption and the amount of food consumed), as well as determining the actual number of people exposed or ill during the outbreak.

## Recommended reading and useful links

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Parts of this document have been published in previous FSANZ microbiological risk assessments for poultry meat, eggs and dairy (including raw milk products) – these are available on the FSANZ website [www.foodstandards.gov.au](http://www.foodstandards.gov.au)

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## Shiga toxin-producing *Escherichia coli* (STEC)

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*Escherichia coli* are bacteria that form part of the normal gut flora of humans and other warm-blooded animals. Although most *E. coli* are considered harmless, certain strains can cause severe illness, particularly Shiga toxin-producing *E. coli* (STEC). Infection with STEC is the main cause of haemolytic uraemic syndrome (HUS), a condition which can be fatal.

### Description of the organism

*E. coli* are Gram-negative, rod-shaped bacteria and are members of the family Enterobacteriaceae. Other species of the genus *Escherichia* include *E. adecarboxylata*, *E. blattae*, *E. fergusonii*, *E. hermanii* and *E. vulneris*.

Pathogenic *E. coli* are classified into specific groups based on the mechanisms by which they cause disease and clinical symptoms. These categories include enterohaemorrhagic *E. coli* (EHEC), enteroaggregative *E. coli* (EAEC), enteroinvasive *E. coli* (EIEC), enteropathogenic *E. coli* (EPEC), enterotoxigenic *E. coli* (ETEC) and diffusely adhering *E. coli* (DAEC) (Montville and Matthews 2005). STEC are Shiga toxin producing *E. coli*, they are also known as verocytotoxin-producing *E. coli* (VTEC). The STEC strains that cause haemorrhagic colitis (bloody diarrhoea) belong to the EHEC group (Yoon and Hovde 2008). EHEC is the most serious of the pathogenic *E. coli* in developed countries, however, in developing countries EPEC is a major issue in children (Meng and Schroeder 2007; Ochoa et al. 2008).

Strains of *E. coli* can be characterised serologically based on the detection of specific O (somatic), H (flagella) and K (capsule) antigens. For most *E. coli* strains the O and H antigens are sufficient to identify the strain. For example, *E. coli* O157:H7 is the leading cause of STEC infections internationally (Meng and Schroeder, 2007; Gyles 2007).

### Growth and survival characteristics

The growth and survival of *E. coli* depends on a number of environmental factors such as temperature, pH, water activity and the composition of the food (refer to Table 1).

The temperature range for growth of *E. coli* is 7–8 to 46 °C, with an optimum temperature of 35–40 °C (ICMSF 1996). Heat resistance of *E. coli* in food is dependent on the composition, pH and water activity of the food. The heat resistance of *E. coli* increases as the water activity of the food decreases. Also, *E. coli* is more resistant to heat when it is in stationary phase compared to log phase growth (Desmarchelier and Fegan 2003). Low temperature has little effect on *E. coli* survival. Strawn and Danyluk (2010) showed that *E. coli* O157:H7 was able to survive on mangoes and papayas stored at -20 °C for at least 180 days.

*E. coli* grow in a broad pH range of 4.4–10.0, with an optimum pH of 6–7 (Desmarchelier and Fegan 2003). A study by Molina (2003) demonstrated STEC are tolerant to acidic conditions with many STEC strains able to survive at pH 2.5–3.0 for over 4 hours. *E. coli* O91:H21 was able to survive at pH 3.0 for more than 24 hours. Arnold and Kaspar (1995) found that *E. coli* O157:H7 is more tolerant to acid when it is in stationary phase or starved during log-phase growth. Therefore STEC may be able to survive and grow in food products previously considered too acidic to support the survival of foodborne pathogens. The effect of pH on *E. coli* survival, however, is dependent on the type of acid present. For example, *E. coli* O157:H7 can survive in a growth medium adjusted to pH 4.5 with hydrochloric acid but not when adjusted to the same pH with lactic acid (ICMSF 1996).

The minimum water activity ( $a_w$ ) required for growth of *E. coli* is 0.95, or approximately 8% sodium chloride. In sub-optimal temperature or pH conditions, a higher  $a_w$  value is required for growth of *E. coli* (Desmarchelier and Fegan 2003).

*E. coli* are facultative anaerobic organisms so do not require oxygen for growth, however, they grow better in aerobic conditions (Meng and Schroeder 2007).

**Table 1: Limits for growth when other conditions are near optimum (ICMSF 1996; Desmarchelier and Fegan 2003)**

	Minimum	Optimum	Maximum
Temperature (°C)	7–8	35–40	46
pH	4.4	6–7	10.0
Water activity	0.95	0.995	–

## Symptoms of disease

Infection with STEC can lead to no clinical symptoms (asymptomatic infection) or can cause abdominal cramps, diarrhoea (may progress to bloody diarrhoea), vomiting and fever. The onset of illness is 3–8 days (median of 3–4 days), with most patients recovering in 10 days (WHO 2005; Meng and Schroeder 2007). In some cases, patients develop haemolytic uraemic syndrome (HUS). HUS is characterised by haemolytic anaemia, thrombocytopenia (decrease in blood platelets) and kidney failure. HUS can also have neurological effects and cause seizures, stroke and coma (WHO 2005; Meng and Schroeder 2007). Approximately 6.3% of patients develop HUS with a fatality rate of 4.6%. Children are more susceptible, with 15.3% of children under five years of age developing HUS (Gould et al. 2009).

STEC are shed in the faeces of infected individuals for several weeks. In children the median shedding time is 13 days (range of 2–62 days) for individuals with diarrhoea. In people who develop HUS, bacterial shedding occurs for 21 days (range 5–124 days) (Meng and Schroeder 2007; Pennington 2010).

## Virulence and infectivity

STEC strains produce two types of Shiga toxins (Stx1 and Stx 2). Stx1 is virtually identical to the toxin produced by *Shigella dysenteriae* 1. Stx2 is significantly associated with human disease (Spears et al. 2006). Stx are toxic to Vero cells (African green monkey kidney cells) and so are also known as verotoxins (VT). The term STEC is used interchangeably with VTEC. In the laboratory, Vero cells can be used to detect Stx activity, as Stx causes Vero cell death (Desmarchelier and Fegan 2003; Meng and Schroeder 2007).

Due to the acid resistance of STEC, when ingested it is able to survive the stomach environment and attach to the cells of the intestine. Some STEC strains form a characteristic attaching and effacing lesion on the intestinal cells. The presence of these lesions is a risk factor for the development of HUS (Gyles 2007). Once STEC has colonized the intestinal track, if sufficient Stx is produced it will bind to the vascular endothelial cells in the colon, resulting in the death of these cells. This damage to the blood vessels of the colon causes bloody diarrhoea. If sufficient Stx is taken up by the blood and circulated through the body, this can lead to impaired kidney and neurological function and the development of HUS (Desmarchelier and Fegan 2003; Gyles 2007).

## Mode of transmission

STEC are transmitted by the faecal-oral route by either consumption of contaminated food or water, from direct contact with infected animals or via person-to-person contact (Gyles 2007).

## Incidence of illness and outbreak data

Infection with STEC is a notifiable disease in all Australian states and territories. The incidence of STEC infections notified in Australia in 2008 was 0.5 cases per 100,000 population (106 cases), which includes both foodborne and non-foodborne cases. This is a slight increase from the 2003–2007 mean of 0.4 cases per 100,000 population per year (ranging from 0.2–0.5 cases per 100,000 population per year). *E. coli* O157 was the most common STEC identified in Australia in 2008 (26% of cases), the next most common were *E. coli* O111 and O26. There were 16 cases of STEC-associated HUS reported in Australia in 2008 (OzFoodNet 2009; NNDSS 2010).

The notification rate for STEC in New Zealand in 2008 was 3.0 cases per 100,000 population (128 cases). This was higher than the 2007 rate of 2.4 cases per 100,000 population. There were 3 cases of HUS associated with STEC reported in New Zealand in 2008 (ESR 2009).

In the US the notification rate for STEC in 2008 was 1.76 cases per 100,000 population. This was a slight increase from the 2007 rate of 1.62 cases per 100,000 population (CDC 2010). In the EU there were 0.7 cases of STEC infection per 100,000 population in 2008 (ranging from 0–4.8 cases per 100,000 between countries). This was a 8.7% increase in the number of cases from 2007 (EFSA 2010).

The incidence of STEC infections has a seasonal association, with the number of cases increasing during the warmer months. In Australia STEC is most prevalent from November to April (OzFoodNet 2009).

Outbreaks of STEC have been associated with undercooked hamburger meat, fresh produce, unpasteurised juices, salami, game meat, cheese and raw (unpasteurised) milk (Yoon and Hovde 2008; FDA 2009) (refer to Table 2).

Table 2: Selected major foodborne outbreaks associated with STEC (&gt;50 cases and/or ≥1 fatality)

Year	Strain	Total no. cases (fatalities)	Food	Country	Comments	References
2009	O157:H7	80	Raw pre-packaged cookie dough	USA	STEC isolated from sample of cookie dough at the factory, however, it was different to the outbreak strain	(CDC 2009)
2006	O157:H7	205(3)	Pre-packaged spinach	USA	STEC isolated from river, cattle and wild pig faeces near spinach field	(California Food Emergency Response Team 2007)
1996-1997	O157:H7	490(20)	Cooked meat products	Scotland	Either inadequate cooking or cross-contamination from raw meat to cooked products	(Bell and Kyriakides 1998)
1996	O157:H7	7966 (3)	White radish sprouts	Japan	Suspected product supplied in school meals	(Montville and Matthews, 2005)
1995	O111:H-	161(1)	Uncooked fermented mettwurst	Australia	No starter culture used, pH drop during fermentation and water activity during drying not monitored. Product released before maturation was completed.	(South Australia Coroner 1995)
1992–1993	O157:H7	731 (4)	Hamburgers	USA	Insufficient cooking of hamburgers	(Doyle et al. 1997)

## Occurrence in food

The major animal reservoir of STEC is ruminants, in particular cattle and sheep (Gyles, 2007). Individual animals can carry more than one serotype of STEC (Barlow and Mellor 2010). Meat derived from these animals may become contaminated with STEC organisms if the meat is exposed to faecal material during processing. A study of Australian beef cattle faecal samples showed 10% of samples (n=300) were STEC positive, *E. coli* O157 was isolated in 1.7% of all samples (Barlow and Mellor 2010). Barlow et al. (2006) isolated STEC from 16% of ground beef (n=285) and 40% of lamb cuts (n=275) sampled in Australia, although of serotypes not associated with reported human cases in Australia. The detection of STEC at a substantially higher rate in lamb is consistent with the higher concentration and prevalence of *E. coli* on sheep carcasses compared to beef carcasses (Phillips et al. 2001a; Phillips et al. 2001b). The reported prevalence of STEC in bulk tank milk internationally is 0–33.5% (FSANZ 2009).

STEC outbreaks have occurred due to the consumption of fruits and vegetables. Fresh produce may be contaminated due to irrigation with contaminated water or the use of soil treated with farm effluent (Fremaux et al. 2008). The presence of STEC on seafood and poultry at retail may be due to cross-contamination or harvesting seafood from contaminated waters (Desmarchelier and Fegan 2003). STEC has been found to survive for months in soil, manure, water trough sediments. It can survive for long periods of time in water and has been isolated from ponds, streams, wells and water troughs. Waterborne transmission of STEC has been reported, both from contaminated drinking water and from recreational water (e.g. swimming) (WHO 2005; Yoon and Hovde 2008; Fremaux et al. 2008).



## Host factors and immunity

People of all ages are susceptible to infection with STEC. However, the young and the elderly are more likely to develop the more serious symptoms (FDA 2009).

## Dose response

The dose response relationship for STEC is complicated by the number of serotypes and the association of STEC with a variety of foods.

Dose response models have been developed for *E. coli* O157:H7. Teunis et al. (2004) used data from an *E. coli* O157:H7 outbreak at a school in Japan to estimate the dose required to cause disease. In children the estimated ingested dose was 31 organisms, with 25% of exposed children becoming ill. In adults the estimated ingested dose was 35 organisms, with 16% of exposed adults becoming ill. Haas et al. (2000) used data from a prior animal study undertaken by Pai et al. (1986) and validated their model by comparison with two human outbreaks, one foodborne and the other waterborne, that occurred in the US. This model estimated that the dose required for 50% of the exposed population to become ill was  $5.9 \times 10^5$  organisms. The corresponding probability of illness for the ingestion of 100 organisms was  $2.6 \times 10^{-4}$ . The US Food and Drug Administration (FDA) has suggested that from the compilation of outbreak data and taking into consideration the ability of *E. coli* O157:H7 to be passed from person-to-person, the infective dose may be similar to that of *Shigella* spp. (as few as 10 organisms (FDA 2009).

Human feeding trial data has been used to generate a dose response model for non-O157:H7 *E. coli* (*E. coli* O111 and O55) (Haas et al. 2000). The model estimated the dose required for 50% of the exposed population to become ill was  $2.55 \times 10^6$  and the probability of illness for ingestion of 100 organisms was  $3.5 \times 10^{-4}$ .

## Recommended reading and useful links

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Parts of this document have been published in previous FSANZ microbiological risk assessments for poultry meat and dairy (including raw milk products) – these are available on the FSANZ website [www.foodstandards.gov.au](http://www.foodstandards.gov.au)

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