Salmonella (non-typhoidal)

Salmonella spp. are bacteria that cause salmonellosis, a common form of foodborne illness in humans. Outcomes from exposure to *Salmonella* spp. can range from mild symptoms to 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 (into serovars) based on the presence and/or absence of O (somatic) and H (flagella) antigens. Phage typing is used to subtype *Salmonella* serovars. 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* serovar Typhimurium. For practical reasons, the abbreviated versions of these names using just the serovar are commonly used, such as *S.* Typhimurium (Crum-Cianflone 2008).

Some *Salmonella* serovars 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). In Australia 97.9% of notified cases of typhoid fever were likely to have been acquired overseas in 2010 (OzFoodNet 2012).

Growth and survival characteristics

Salmonella spp. 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 of *Salmonella* spp. 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 of 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 a_w (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 a combination of several preservative factors, such as the use of a preservative in conjunction with reduction in pH and temperature (ICMSF 1996; Ha et al. 2004; Banerjee and Sarkar 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 of *Salmonella* spp. 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 develops, predominantly in immunocompromised individuals. This occurs when *Salmonella* spp. enter the bloodstream, leading to symptoms such as high fever, lethargy, abdominal and chest pain, chills and anorexia; and can be fatal. A small number of individuals develop a chronic condition or sequelae such as arthritis, appendicitis, meningitis or pneumonia as a consequence of infection (Hohmann 2001; WHO/FAO 2002; FDA 2012).

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. It is estimated that 0.5% of individuals with non-typhoid salmonellosis 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 spp. possess a number of structural and physiological virulence factors, enabling them to cause acute and chronic disease in humans. The virulence of *Salmonella* spp. varies with the length and structure of the O side chains of lipopolysaccharide molecules at the surface of the bacterial cell. Resistance of *Salmonella* spp. 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* spp. to attach to host 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 bacterial invasion into intestinal epithelial cells, while systemic infections and intracellular accumulation of *Salmonella* spp. 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 *Escherichia 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 consumption of contaminated food or water, person-to-person contact, 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 2012 of 49.8 cases per 100,000 population (11,273 cases). This was an increase on the previous 5 year mean of 46.9 cases per 100,000 population per year (ranging from 38.6–54.2 cases per 100,000 population per year) (NNDSS 2013).

The salmonellosis notification rate varied between jurisdictions from 40.5 cases per 100,000 population in New South Wales to 180.1 cases per 100,000 population in the Northern Territory in 2012 (NNDSS 2013). Children between 0–4 years had the highest notification rate, with 218.3 and 160.2 notifications per 100,000 population for males and females, respectively, in 2010 (OzFoodNet 2012) 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 2010, representing 44% of all notified *Salmonella* infections in Australia. Internationally, *S.* Enteritidis is frequently reported as causing human illness; however it is not endemic in Australia. In 2010, 93% of *S.* Enteritidis cases reported in Australia were acquired overseas (Greig and Ravel 2009; OzFoodNet 2012).

The notification rate for salmonellosis in New Zealand in 2011 was 24 cases per 100,000 population (1,056 cases). This was a slight decrease from the 2010 rate of 26.2 cases per 100,000 population (Lim et al. 2012). In the United States (US) 17.73 cases of salmonellosis were notified per 100,000 population in 2010. This was a slight increase from the 2009 rate of 16.18 cases per 100,000 population (CDC 2012). In the European Union the notification rate for salmonellosis was 20.7 cases per 100,000 population in 2011 (ranging from 1.6–80.7 cases per 100,000 population between countries). This was a 5.4% decrease in the number of cases from 2010 (EFSA 2013).

Outbreaks attributed to *Salmonella* spp. have predominantly been associated with animal products such as eggs, poultry, raw meat, milk and dairy products, but also include 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 outbreaks associated with *Salmonella* spp. (>50 cases and/or ≥1 fatality)

Year	Serovar	Total no. cases (fatalities)	Food	Country	Comments	Reference
2010	*S.* Typhimurium PT9	170	Aioli	Australia	Aioli was made with raw eggs. *S.* Typhimurium PT9 isolated from aioli and chopping boards	(OzFoodNet 2010)
2009-2010	*S.* Montevideo	272	Salami containing red or black pepper	US	Pepper was added to the salami after the kill step, pepper samples were positive for *S.* Montevideo	(CDC 2010)
2006-2007	*S.* Tennessee	628	Peanut butter	US	Environmental samples from the plant were positive for *S.* Tennessee	(CDC 2007)
2005-2006	*S.* Oranienburg	126	Alfalfa	Australia	Alfalfa at the production facility were positive for *S.* Oranienburg	(OzFoodNet 2006)
2005	*S.* Typhimurium PT135	63	Eggs used in bakery products	Australia	*S.* Typhimurium 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)

Year	Serovar	Total no. cases (fatalities)	Food	Country	Comments	Reference
2001-2002	*S.* Oranienburg	>439	Chocolate	Germany	The high fat content of chocolate increases the heat resistance of *Salmonella* spp.	(Werber et al. 2005)
1999	*S.* Typhimurium PT135a	507	Unpasteurised fruit juice	Australia	*S.* Typhimurium 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	*S.* Typhimurium	16,284 (7)	Pasteurised milk	US	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* spp. 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 foods that have been subject to sewage pollution (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. have been detected 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 accounted for a large proportion of isolates, it appears to be a non-virulent serovar and has been rarely associated with human or animal illness (Gan et al. 2011). The predominance of *S.* Sofia in poultry is unique to Australia 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 that influence disease

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 2012).

Dose response

Human feeding trials were undertaken during the 1950s to determine the relationship between the dose of *Salmonella* spp. ingested and whether illness occurred. These studies showed that ingestion of $1.3 \times 10^5 – 2.4 \times 10^7$ organisms could cause illness; however, for some strains 1×10^{10} organisms were required for illness to occur (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). Investigations of salmonellosis outbreaks have estimated a wide range in the dose of organisms that has caused disease. Ranges reported vary from <10 to 10^9 depending on the food. As such, doses resulting in illness 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 and estimated a 13% probability of illness from consumption of 100 *Salmonella* organisms.

Recommended reading and useful links

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