**Staphylococcus aureus**

*Staphylococcus aureus* is a bacterium that causes staphylococcal food poisoning, a form of gastroenteritis with rapid onset of symptoms. *S. aureus* is commonly found in the environment (soil, water and air) and is also found in the nose and on the skin of humans.

**Description of the organism**

*S. aureus* is a Gram-positive, non-spore forming spherical bacterium that belongs to the *Staphylococcus* genus. The *Staphylococcus* genus is subdivided into 32 species and subspecies. *S. aureus* produces staphylococcal enterotoxin (SE) and is responsible for almost all staphylococcal food poisoning (Montville and Matthews 2008; FDA 2012). *S. intermedius*, a *Staphylococcus* species which is commonly associated with dogs and other animals, can also produce SE and has been rarely associated with staphylococcal food poisoning (Talan et al. 1989; Khambaty et al. 1994; Le Loir et al. 2003).

**Growth and survival characteristics**

The growth and survival of *S. aureus* is dependent on a number of environmental factors such as temperature, water activity (aw), pH, the presence of oxygen and composition of the food (refer to Table 1). These physical growth parameters vary for different *S. aureus* strains (Stewart 2003).

The temperature range for growth of *S. aureus* is 7–48°C, with an optimum of 37°C. *S. aureus* is resistant to freezing and survives well in food stored below -20°C; however, viability is reduced at temperatures of -10 to 0°C. *S. aureus* is readily killed during pasteurisation or cooking. Growth of *S. aureus* occurs over the pH range of 4.0–10.0, with an optimum of 6–7 (ICMSF 1996; Stewart 2003).

*S. aureus* is uniquely resistant to adverse conditions such as low aw, high salt content and osmotic stress. In response to low aw, several compounds accumulate in the bacterial cell, which lowers the intracellular aw to match the external aw (Montville and Matthews 2008). As such, most *S. aureus* strains can grow over a aw range of 0.83 to >0.99 (FDA 2012). *S. aureus* is a poor competitor, but its ability to grow under osmotic and pH stress means that it is capable of thriving in a wide variety of foods, including cured meats that do not support the growth of other foodborne pathogens (Montville and Matthews 2008).

*S. aureus* is a facultative anaerobe so can grow under both aerobic and anaerobic conditions. However, growth occurs at a much slower rate under anaerobic conditions (Stewart 2003).

For a non-sporing mesophilic bacterium, *S. aureus* has a relatively high heat resistance (Stewart 2003). The observed average decimal reduction value (D-value, the value at which the initial concentration of bacterial cells would be reduced by 1 log10 unit) was 4.8–6.6 min at 60°C when heated in broth (Kennedy et al. 2005). The bacteria has a higher heat resistance when it is encapsulated in oil, with a D-value at 60°C of 20.5 min for *S. aureus* in fish and oil (Gaze 1985). An extremely heat resistant strain of *S. aureus* (D-value at 60°C of >15 min in broth) has been recovered from a foodborne outbreak in India (Nema et al. 2007).
Several chemical preservatives, including sorbates and benzoates, inhibit the growth of *S. aureus*. The effectiveness of these preservatives increases as the pH is reduced. Methyl and propyl parabens are also effective (Stewart 2003; Davidson and Taylor 2007).

**Table 1:** Limits for growth of *S. aureus* and enterotoxin production when other conditions are near optimum (ICMSF 1996)

<table>
<thead>
<tr>
<th></th>
<th>Bacterial Growth</th>
<th>Enterotoxin Production</th>
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<tbody>
<tr>
<td></td>
<td>Optimum</td>
<td>Range</td>
</tr>
<tr>
<td><strong>Temperature (°C)</strong></td>
<td>37</td>
<td>7–48</td>
</tr>
<tr>
<td><strong>pH</strong></td>
<td>6–7</td>
<td>4–10</td>
</tr>
<tr>
<td><strong>Water activity</strong></td>
<td>0.98</td>
<td>0.83–&gt;0.99</td>
</tr>
</tbody>
</table>

**Symptoms of disease**

Staphylococcal food poisoning symptoms generally have a rapid onset, appearing around 3 hours after ingestion (range 1–6 hours). Common symptoms include nausea, vomiting, abdominal cramps and diarrhoea. Individuals may not demonstrate all the symptoms associated with the illness. In severe cases, headache, muscle cramping and transient changes in blood pressure and pulse rate may occur. Recovery is usually between 1–3 days (Stewart 2003; FDA 2012). Fatalities are rare (0.03% for the general public) but are occasionally reported in young children and the elderly (4.4% fatality rate) (Montville and Matthews 2008).

*S. aureus* can cause various non-food related health issues such as skin inflammations (e.g. boils and styes), mastitis, respiratory infections, wound sepsis and toxic shock syndrome (Stewart 2003; Montville and Matthews 2008).

**Virulence and infectivity**

Staphylococcal food poisoning is an intoxication that is caused by the ingestion of food containing pre-formed SE (Argudin et al. 2010). There are several different types of SE; enterotoxin A is most commonly associated with staphylococcal food poisoning. Enterotoxins D, E and H, and to a lesser extent B, G and I, have also been associated with staphylococcal food poisoning (Seo and Bohach 2007; Pinchuk et al. 2010).

SEs are produced during the exponential phase of *S. aureus* growth, with the quantity being strain dependent. Typically, doses of SE that cause illness result when at least \(10^5 – 10^6\) cfu/g of *S. aureus* are present (Seo and Bohach 2007; Montville and Matthews 2008). Most genes for SEs are located on mobile elements, such as plasmids or prophages. As such, transfer between strains can occur, modifying the ability of *S. aureus* strains to cause disease and contributing to pathogen evolution (Argudin et al. 2010; Pinchuk et al. 2010).

*S. aureus* produces SEs within the temperature range of 10–48°C, with an optimum of 40–45°C (refer to Table 1). As the temperature decreases, the level of SE production also decreases. However, SEs remain stable under frozen storage. SEs are extremely resistant to heating and can survive the process used to sterilise low acid canned foods. SE production can occur in a pH range of 4.5–9.6, with an optimum of 7–8. Production of SE
can occur in both anaerobic and aerobic environments; however, toxin production is optimum in aerobic conditions (ICMSF 1996; Stewart 2003).

SEs are resistant to the heat and low pH conditions that easily destroy *S. aureus* bacteria. The SEs are also resistant to proteolytic enzymes, hence SEs retain their activity in the gastrointestinal tract after ingestion. SEs range in size from 22–28 kDa and contain a highly flexible disulphide loop at the top of the N-terminal domain that is required for stable conformation and is associated with the ability of the SE to induce vomiting (Argudin et al. 2010).

It has been suggested that SEs stimulate neuroreceptors in the intestinal tract which transmit stimuli to the vomiting centre of the brain via the vagus nerve (Montville and Matthews 2008; Argudin et al. 2010). In addition, SEs are able to penetrate the lining of the gut and stimulate the host immune response. The release of inflammatory mediators, such as histamine, causes vomiting. The host immune response also appears to be responsible for the damage to the gastrointestinal tract associated with SE ingestion, with lesions occurring in the stomach and upper part of the small intestine. Diarrhoea that can be associated with staphylococcal food poisoning may be due to the inhibition of water and electrolyte re-absorption in the small intestine (Argudin et al. 2010).

**Mode of transmission**

Staphylococcal food poisoning occurs when food is consumed that contains SE produced by *S. aureus*. Food handlers carrying enterotoxin-producing *S. aureus* in their noses or on their hands are regarded as the main source of food contamination via direct contact or through respiratory secretions (Argudin et al. 2010).

**Incidence of illness and outbreak data**

Staphylococcal food poisoning is not a notifiable disease in Australia or New Zealand. There were two reported outbreaks of staphylococcal food poisoning in Australia in 2011 and two outbreaks reported in 2010. In New Zealand there were no outbreaks of staphylococcal food poisoning in 2011 and two outbreaks reported in 2010 (Lim et al. 2012; OzFoodNet 2012a; OzFoodNet 2012b). It is generally recognised that there may be significant under reporting of staphylococcal food poisoning due to the short duration of illness and self-limiting symptoms. In Australia it is estimated that *S. aureus* accounts for 1% of foodborne illness caused by known pathogens (Hall et al. 2005).

In the European Union there were 0.07 reported cases of staphylococcal food poisoning per 100,000 population in 2011 (ranging from <0.01–0.45 per 100,000 population between countries). This was similar to the 2010 rate of 0.06 cases per 100,000 population (EFSA 2012; EFSA 2013).

In the United States (US) the notification rate for vancomycin-intermediate *S. aureus* was 0.04 cases per 100,000 population in 2010, which was an increase from the 2009 rate of 0.03 (CDC 2012). It is estimated that in the US, *S. aureus* accounts for 2.6% of foodborne illness caused by 31 major pathogens (Scallan et al. 2011).

The incidence of staphylococcal food poisoning is seasonal. Most cases occur in the late summer when temperatures are warm and food is stored improperly (Montville and Matthews 2008).
Foods associated with outbreaks of staphylococcal food poisoning include meat and meat products, poultry and egg products, milk and dairy products, salads, cream-filled bakery products and sandwich fillings. Foods that require extensive handling during preparation and are kept above refrigeration temperature (4°C) for extended periods after preparation are often involved in staphylococcal food poisoning (Argudin et al. 2010; FDA 2012) (refer to Table 2). Foods high in starch and protein are believed to favour SE production (Stewart 2003).

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

<table>
<thead>
<tr>
<th>Year</th>
<th>No. of cases (fatalities)</th>
<th>Food</th>
<th>Country</th>
<th>Comments</th>
<th>References</th>
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</thead>
<tbody>
<tr>
<td>2007</td>
<td>400 (1)</td>
<td>UHT milk</td>
<td>Paraguay</td>
<td>Production line operator identified as source of post-pasteurisation contamination</td>
<td>(Weiler et al. 2011)</td>
</tr>
<tr>
<td>2006</td>
<td>113</td>
<td>Chicken and rice</td>
<td>Austria</td>
<td>Kitchen worker positive for <em>S. aureus</em></td>
<td>(Schmid et al. 2007)</td>
</tr>
<tr>
<td>2000</td>
<td>13,420</td>
<td>Powdered skim milk</td>
<td>Japan</td>
<td>Production of powdered skim milk stopped midway and delayed for 9 hours due to power cut, this permitted <em>S. aureus</em> growth and SE production. SE survived the pasteurisation process that destroyed <em>S. aureus</em> bacteria</td>
<td>(Asao et al. 2003)</td>
</tr>
<tr>
<td>1998</td>
<td>4000 (16)</td>
<td>Chicken, roast beef, rice and beans</td>
<td>Brazil</td>
<td>Food preparation began over 48 hours before food served, food left at room temperature for one day. All food handlers positive for <em>S. aureus</em></td>
<td>(Do Carmo et al. 2004)</td>
</tr>
<tr>
<td>Year</td>
<td>No. of cases (fatalities)</td>
<td>Food</td>
<td>Country</td>
<td>Comments</td>
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<tr>
<td>1990</td>
<td>100</td>
<td>Ham</td>
<td>US</td>
<td>Food handler positive for <em>S. aureus</em> removed ham casings without gloves. Improper refrigeration, prolonged handling and inadequate reheating of ham</td>
<td>(Richards et al. 1993)</td>
</tr>
<tr>
<td>Mid-1980s</td>
<td>&gt;850</td>
<td>Chocolate milk</td>
<td>US</td>
<td>Milk stored for several hours in inadequately cooled tank prior to pasteurisation, conditions permitted <em>S. aureus</em> growth and SE production</td>
<td>(Evenson et al. 1988)</td>
</tr>
</tbody>
</table>

**Occurrence in food**

Despite *S. aureus* colonising a wide range of animals, people are the main reservoir of food contamination (Montville and Matthews 2008). Prevalence of enterotoxigenic *S. aureus* in food handlers is variable between industries and countries. Prevalence estimates from several small studies range from 2% of food handlers in Italy (n=545) (Talarico et al. 1997), 12% of flight-catering staff in Finland (n=136) (Hatakka et al. 2000), 19% of restaurant workers in Chile (n=102) (Figueroa et al. 2002) to 62% of fish processing factory workers in India (n=87) (Simon and Sanjeev 2007).
The udders and teats of cows are known sources of enterotoxigenic S. aureus, and the occurrence of S. aureus in unpasteurised milk and cheese is common. The tonsils and skin of pigs, chickens and turkeys often harbour S. aureus, and are also potential sources of S. aureus contamination (Stewart 2003).

A survey of food from retail markets and dairy farms in Turkey was performed between 2007 and 2008. Enterotoxigenic S. aureus was found in 11.3% of meat (n=115), 10.2% of unpasteurised milk (n=303), 8.0% of dairy products (n=452), 3.5% of bakery products (n=141) and 2.3% or ready-to-eat products (n=44) (Aydin et al. 2011).

An Italian survey performed between 2003 and 2005 indicated that 9.2% of dairy products (n=641) and 5.0% of meat products (n=993) were positive for enterotoxigenic S. aureus (Normanno et al. 2007). In Japan a retail survey performed between 2002 and 2003 found 17.6% of raw chicken meat (n=444) were positive for enterotoxigenic S. aureus (Kitai et al. 2005).

Host factors that influence disease

All people are believed to be susceptible to staphylococcal food poisoning. However, the severity of symptoms may vary depending on the amount of SE consumed in the food and the general health of individuals. The young and elderly are more likely to develop more serious symptoms (FDA 2012).

Dose response

A human feeding trial in the 1960s demonstrated that 20–25 µg (0.4 µg/kg body weight) of enterotoxin B caused illness (Raj and Bergdoll 1969). However, more recently it has been reported that less than 1.0 µg of SE is sufficient to cause staphylococcal food poisoning (FDA 2012). In fact evidence from outbreaks indicates that ingestion of less than 200 ng of enterotoxin A is sufficient to cause illness in susceptible individuals (Evenson et al. 1988; Asao et al. 2003).

Recommended reading and useful links


References


Khambaty FM, Bennett RW, Shah DB (1994) Application of pulsed-field gel electrophoresis to the epidemiological characterization of *Staphylococcus intermedius* implicated in a food-related outbreak. Epidemiology and Infection 113:75–81


OzFoodNet (2012b) OzFoodNet Quarterly report, 1 July to 30 September 2011. Communicable Diseases Intelligence 36(2):E188–E195


Last updated May 2013