Biographies

Geoff Hogg is Director of the Microbiological Diagnostic Unit Public Health Laboratory. His role includes ensuring that laboratory services meet the needs of all those relying on them. He has a diverse background leading to a firm belief that where organisms and the diseases they cause involve multiple sectors, such as is evident for Listeria and Listeriosis, that a One-health approach is required to meet the needs of all stakeholders, including mitigating risks to patients.

Agnes Tan is a Senior Scientist at the Microbiological Diagnostic Unit. She has assisted public health colleagues in outbreak investigations over many years.

Joy Gregory is the OzFoodNet epidemiologist employed by the Victorian Department of Health. She has been with OzFoodNet since its inception in 2000. Joy has a keen interest in surveillance and epidemiology of enteric diseases, especially those with a foodborne mode of transmission, and has been involved in investigating numerous foodborne outbreaks including multijurisdictional outbreaks of listeriosis. Joy has a strong commitment to working in multifaceted teams during investigations to achieve the best public health outcomes.

Food poisoning due to Clostridium perfringens

Classical Type A Clostridium perfringens are typically β-haemolytic on Horse Blood Agar (HBA) and the spores are oval and subterminal with no swelling of the sporangium. The bacilli are 1–1.5 μm wide and 4–8 μm long. The organism has complex nutritional requirements for growth, and therefore their frequent involvement in meat and curry-type dishes is hardly surprising. Depending on which text is consulted, the incubation period for Cl. perfringens food poisoning can vary from 6 to 24 hours following consumption of contaminated food. However, in practice, cases usually manifest in 8–22 hours with a mean of around 15 hours. Unlike Staphylococcal or Bacillus cereus emetic food poisoning, the enterotoxin of Cl. perfringens is not preformed in the food during multiplication. Rather, the food, laden with high numbers of vegetative cells, has to be ingested first, and after passing through the acid barrier of the stomach to the alkaline environment of the jejunum, the vegetative cells start to sporulate, and as the spores are released into the lumen of the gut, enterotoxin is also released. The enterotoxin binds to brush border membrane receptors of intestinal epithelial cells, which then induces a calcium ion-dependant breakdown of permeability, leading to a massive influx of fluid into the gut and producing the profuse diarrhoea associated with this syndrome. The peak for enterotoxin production is just before lysis of the cell sporangium. This is why the time intervals for onset of symptoms are considerably longer, and it also means that testing the food for enterotoxin is not warranted.

The main criteria for diagnosis of food poisoning caused by Cl. perfringens include the detection of at least $1 \times 10^5$ vegetative cfu/g in incriminated food, and/or a faecal spore count of at least $1 \times 10^6$ cfu/g in faeces from ill complainants. However, high counts need to
be interpreted very carefully in geriatric patients, as counts of at least $1 \times 10^5$ cfu/g are common in this age group. There is a Reversed Passive Latex Agglutination (Oxoid PET-RPLA, Basingstoke, England) kit available for detection of enterotoxin in faeces, but it is essential to test samples within 24–48 hours of onset of symptoms as the toxin is rapidly eliminated from the gut. Of more use is a molecular approach which can determine the serotype of the organism and the presence of the cpe (enterotoxin) gene, and a separate PCR to determine if the cpe gene is carried on a plasmid or chromosomally (see below).1

The literature is replete with reports of large outbreaks of food poisoning resulting from catered events. Most outbreaks occur in large eating establishments where large quantities of food are prepared. In particular, Cl. perfringens was a leading cause of food poisoning in hospitals in the United Kingdom up until the late 1980s.2 Careful reading of many of these reports will usually reveal that food had been kept warm (<47°C) for extended periods of time, allowing germination of spores and subsequent proliferation of vegetative cells. A recent article from the United States estimates that Clostridium perfringens is the second most common bacterial cause of food poisoning.3 Likewise in Australia, Clostridium perfringens is a common cause of food poisoning and outbreaks are often quite large because they occur at mass-catering events.4–8

Studies on food poisoning strains have demonstrated that chromosomal carriage of the cpe gene actually confers heat resistance on the organism, a handy trait for an organism that grows in cooked food. One of the key features of food poisoning strains is their ability to grow at elevated temperatures. The optimum growth temperature ranges from 43°C to 47°C. For this reason, many of the outbreaks in Australia are associated with the use of Bain Maries (Figure 1), particularly in restaurants serving curries and other meat dishes, where the Bain Marie is turned down to prevent the food from drying out too much. With a generation time of only seven minutes at 43°C, foods do not have to be temperature-abused for long before the vegetative cell count becomes significant. In particular, the practice of using the Bain Marie to heat the food from scratch is highly prone to proliferation of this organism. Investigation of outbreaks will invariably uncover a critical control point (CCP) that has not been implemented correctly. In the case of some restaurants in Brisbane, curries have been cooked and then placed in bins to allow them to cool down slowly out the back of the restaurant. This is particularly a problem with food preparation personnel who translate their home food practices to a catering premise where the standards of food handling are necessarily expected to be much higher. Often, a change in practices resulting from the breakdown of a key piece of equipment used to keep food hot will be uncovered. In some cases, outbreaks will occur simply because the food has not been cooked properly in the first place and is then kept warm for an extended period, allowing ample time for spores to germinate and proliferate, eg. spit-roast caterers.

**Other manifestations of toxigenic Clostridium perfringens**

In addition to its traditional role of food poisoning, Cl. perfringens has also been associated with a couple of different syndromes. In Papua New Guinea, the highlanders suffered for many years from a frequently fatal human necrotic enteritis syndrome called Pig-Bel, which was associated with consumption of Type C Clostridium perfringens in pig feasts. The highlanders’ staple diet throughout the year is the sweet potato, which contains a trypsin inhibitor which predisposes them to intoxication by this strain.9

More recently, there have been outbreaks in nursing homes in a couple of Australian States where person to person spread over a number of days is suspected, rather than the traditional point-source outbreaks associated with food poisoning. These outbreaks are caused by Antibiotic-Associated Diarrhoea (AAD) strains of Cl. perfringens, in which the cpe gene that codes for enterotoxin production is carried on a plasmid, rather than chromosomally as seen with food poisoning strains.10

There have also been reports in the literature of fatal cases of Cl. perfringens food poisoning in elderly patients in psychiatric hospitals who have been medicated with anti-cholinergic drugs. These drugs have been linked to faecal impaction in the large bowel, leading to retention of toxin in cases of food poisoning and massive necrosis to the lining of the colon, resulting in death.11,12
Conclusion

Food poisoning caused by *Cl. perfringens* is a preventable condition. If the right checks and balances are built into the food preparation process, then the organism should not have an opportunity to germinate and proliferate. Even if this does occur, thorough reheating of the food at temperatures in excess of 60°C will eliminate the organism and prevent an outbreak of food poisoning. This highlights the importance of appropriate education and compliance of personnel involved in food preparation so that they understand what can happen when they mishandle food or essential equipment breaks down.

References


Biography

John Bates is the Chief Scientist in charge of the QHFSS Public Health Microbiology laboratory. This laboratory provides a support arm to the Queensland Health Public Health Units in the investigation of outbreaks of human, water and foodborne disease, as well as providing phenotypic and genotypic data on a wide range of notifiable bacteria in Queensland.

**Salmonella** and egg-related outbreaks

Non-typhoidal *Salmonella* infections are a significant public health issue in Australia, with record numbers of both disease notifications and outbreaks being reported in recent years. Epidemiology plays an important role in *Salmonella* outbreak investigation, helping to identify raw and minimally cooked eggs as an increasingly common cause for these events. Of particular relevance to disease caused by Australian eggs is *Salmonella enterica* subsp. *enterica* serovar Typhimurium. A crucial element in demonstrating this serovars presence throughout the food chain is the ability to trace suspect eggs to their source. High product turnover makes this challenging but through the adoption of integrated surveillance practices and harmonised laboratory methods, a more effective response may emerge.

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