Bacillus cereus

Bacillus cereus is a spore forming bacterium that produces toxins that cause vomiting or diarrhoea. Symptoms are generally mild and short-lived (up to 24 hours). *B. cereus* is commonly found in the environment (e.g. soil) as well as a variety of foods. Spores are able to survive harsh environments including normal cooking temperatures.

Description of the organism

B. cereus is a Gram-positive, motile (flagellated), spore-forming, rod shaped bacterium that belongs to the *Bacillus* genus. Species within this genus include *B. anthracis, B. cereus, B. mycoides, B. thuringiensis, B. pseudomycoides and B. weihenstephanensis* (Rajkowski and Bennett 2003; Montville and Matthews 2005). Genomic sequencing data has shown *B. anthracis, B. cereus* and *B. thuringiensis* to be very closely related (Rasko et al. 2004) with their 16S rRNA gene sequence sharing more than 99% similarity (Ash et al. 1991).

B. cereus is widespread in nature and readily found in soil, where it adopts a saprophytic life cycle; germinating, growing and sporulating in this environment (Vilain et al. 2006). Spores are more resistant to environmental stress than vegetative cells due to their metabolic dormancy and tough physical nature (Jenson and Moir 2003).

B. cereus produces two types of toxins – emetic (vomiting) and diarrhoeal – causing two types of illness. The emetic syndrome is caused by emetic toxin produced by the bacteria during the growth phase in the food. The diarrhoeal syndrome is caused by diarrhoeal toxins produced during growth of the bacteria in the small intestine (Ehling-Schulz et al. 2006).

Growth and survival characteristics

Strains of *B. cereus* vary widely in their growth and survival characteristics (refer to Table 1). Isolates from food and humans can be subdivided as either mesophilic or psychrotrophic strains. Mesophilic strains grow well at 37°C but do not grow below 10°C; psychrotrophic strains grow well at refrigeration temperatures but grow poorly at 37°C (Wijnands et al. 2006a). All isolates of *B. cereus* associated with emetic toxin production have been found to be mesophilic in nature (Pielaat et al. 2005; Wijnands et al. 2006b).

The maximum salt concentration tolerated by *B. cereus* for growth is reported to be 7.5% (Rajkowski and Bennett 2003). *B. cereus* growth is optimal in the presence of oxygen, but can occur under anaerobic conditions. *B. cereus* cells grown under aerobic conditions are less resistant to heat and acid than *B. cereus* cells grown anaerobically or microaerobically (Mols et al. 2009).

Mesophilic strains of *B. cereus* have been shown to have greater acid resistance than psychrotrophic strains (Wijnands et al. 2006b). The observed average decimal reduction value or D-value (the time required to reduce the initial concentration of bacterial cells or spores by 1 $\log_{10}$ unit) was 7.5 min for mesophilic strain stationary phase cells (pH 3.5, 37°C). In comparison the D-value for psychrotrophic strains under the same conditions was 3.8 min (Wijnands et al. 2009; Augustin 2011).

There is considerable strain variability in the heat resistance of *B. cereus* spores. The D-values of some strains is up to 15 to 20 times greater than the more heat sensitive strains. The D-value at 85°C is 33.8–106 min in phosphate buffer, and at 95 °C is 1.5–36.2 min and

1.8–19.1 min in distilled water and milk, respectively (ICMSF 1996). Heat resistance is increased in high fat and oily foods, for example in soybean oil the D-value at 121°C is 30 min. Spores are more resistant to dry heat than moist heat, with heat resistance usually greater in foods with lower water activity. Spores are also more resistant to radiation than vegetative cells (Jenson and Moir 2003).

Nisin is a preservative that is used to inhibit the germination and outgrowth of spores. Antimicrobials which inhibit the growth of *B. cereus* include benzoate, sorbates and ethylenediaminetetraacetic acid (Jenson and Moir 2003).

Table 1: Limits for growth of *B. cereus* and toxin production when other conditions are near optimum (Kramer and Gilbert 1989; Sutherland and Limond 1993; ICMSF 1996; Fermanian et al. 1997; Finlay et al. 2000)

	Bacterial Growth		Emetic Toxin Production		Diarrhoeal Toxin Production	
	Optimum	Range	Optimum	Range	Optimum	Range
Temperature (°C)	30–40	4–55	12–15	12–37	32	10–43
pH	6.0–7.0	4.9–10.0	-	-	8.0	5.5–10
Water activity	-	0.93–0.99	-	-	-	-

Symptoms of disease

B. cereus causes two types of foodborne illness – emetic (vomiting) and diarrhoeal syndromes. The emetic syndrome is an intoxication that is caused by ingestion of a cyclic peptide toxin called cereulide that is pre-formed in the food during growth by *B. cereus*. This syndrome has a short incubation period and recovery time. The symptoms of nausea, vomiting and abdominal cramping occur within 1–5 hours of ingestion, with recovery usually within 6–24 hours (Schoeni and Wong 2005; Senesi and Ghelardi 2010).

The diarrhoeal syndrome is caused by enterotoxins produced by *B. cereus* inside the host. The incubation period before onset of disease is 8–16 hours and the illness usually lasts for 12–14 hours, although it can continue for several days. Symptoms are usually mild with abdominal cramps, watery diarrhoea and nausea (Granum 2007).

In a small number of cases both types of toxin are produced, and emetic and diarrhoeal symptoms occur (Montville and Matthews 2005). Neither form of illness is considered life-threatening to normal healthy individuals, with few fatal cases reported (Jenson and Moir 2003). *B. cereus* has been associated with non-food related illness, although this occurs rarely. The bacterium has been found in postsurgical and traumatic wounds and can cause opportunistic infections, especially in immunocompromised individuals, such as septicaemia, meningitis and pneumonia. *B. cereus* has also been known to occasionally cause localised eye infections in humans (Schoeni and Wong 2005).

Virulence and infectivity

The pathogenic mechanism for the *B. cereus* emetic illness has been well characterised. The emetic toxin (cereulide) causes vacuole formation in HEp-2 cells in the laboratory (Agata et al. 1994; Schoeni and Wong 2005). Using an animal model, Agata et al. (1995) showed that cereulide causes vomiting, potentially by binding to the 5-HT$_3$ receptors in the stomach/small intestine to stimulate the vagus nerve and brain.

Cereulide is produced by a non-ribosomal peptide synthetase (NRPS) complex (Horwood et al. 2004; Toh et al. 2004). The entire NRPS cluster has been characterised (Ehling-Schulz et al. 2006) resulting in a highly specific method for detection of cereulide producing *B. cereus* strains (Fricker et al. 2007).

Production of the emetic toxin has been shown to occur in skim milk within the temperature range of 12–37°C, with more toxin produced at 12 and 15°C compared to higher temperatures (Finlay et al. 2000). The emetic toxin is highly resistant to environmental factors, showing stability from pH 2–11 and during heating to 100°C for 150 minutes (pH 8.7–10.6) (Jenson and Moir 2003; ESR 2010).

Three types of enterotoxins are associated with the diarrhoeal form of disease. These are: the three component enterotoxin haemolysin BL (HBL), the three component non-haemolytic enterotoxin (NHE) and the single component enterotoxin cytotoxin K. After consumption of food containing *B. cereus*, the enterotoxins are released into the small intestine during vegetative growth following spore germination, and by any surviving vegetative cells (Wijnands et al. 2009).

The diarrhoeal enterotoxins can be produced in the temperature range of 10–43°C, with an optimum of 32°C (Kramer and Gilbert 1989; Fermanian et al. 1997). Production occurs between pH 5.5–10, with an optimum of pH 8 (Sutherland and Limond 1993). The diarrhoeal enterotoxins are stable at pH 4–11 and inactivated by heating to 56°C for 5 minutes (Jenson and Moir 2003). Maltodextrin is known to stimulate growth of *B. cereus* and to aid diarrheal enterotoxin production in reconstituted and stored infant milk formulae (Rowan and Anderson 1997). It has also been shown that *B. cereus* produces more HBL and NHE under conditions of oxygen tension (low oxygen reduction potential) that simulate the anaerobic, highly reducing fermentative conditions encountered in the small intestine (Zigha et al. 2006).

Up to 26% of *B. cereus* vegetative cells can survive conditions that simulate passage through the stomach. The survival rate of the vegetative cells is dependent on the strain type, phase of vegetative cell growth and the gastric pH (Wijnands et al. 2009). As diarrhoeal enterotoxins are unstable at low pH and are degraded by digestive enzymes, any enterotoxins pre-formed in food would be destroyed during passage through the stomach and so not cause illness if ingested (Jenson and Moir 2003).

In contrast, spores of *B. cereus* are able to pass unaffected through the gastric barrier. The spores contain receptors that need triggering by certain low molecular weight substances to commence germination. These inducers may be present in the food as well as the intestinal epithelial cells. In the small intestine the spores germinate, grow and produce enterotoxins (Wijnands 2008).

A crucial virulence factor required for causing the diarrhoeal symptoms is the ability of the vegetative cells and spores of *B. cereus* to adhere to the epithelial cell wall of the small intestine. The adhesion efficiency of spores and cells has been shown to be low, approximately 1% (Wijnands 2008).

The ability of the enterotoxins to act as tissue-destructive proteins and damage the plasma membrane of the epithelial cells of the small intestine suggests a role for these enterotoxins in causing diarrhoea (Senesi and Ghelardi 2010). Beecher et al. (1995) showed HBL causes fluid accumulation in ligated rabbit ileal loops, implicating a role in diarrhoea. However, direct involvement of NHE and cytotoxin K in causing diarrhoea is yet to be demonstrated (Senesi and Ghelardi 2010).

Efficient horizontal DNA transfer systems are present within the *B. cereus* group, enabling plasmids to be transferred among strains of different species of this group (*B. cereus, B. anthracis* and *B. thuringiensis*). The plasmids are known to be important determinants of virulence properties of *B. cereus* strains, since they contain genes responsible for virulence such as the *ces* gene cluster required for cereulide formation and emetic disease (Arnesen et al. 2008). Furthermore, chromosomal DNA contains genes associated with the diarrhoeal disease, and is therefore present in all strains. In view of the homogeneity of the *B. cereus* group, an online tool has been developed for ascertaining the foodborne virulence potential of strains (Guinebretière et al. 2010).

Mode of transmission

B. cereus food poisoning can be caused by either ingesting large numbers of bacterial cells and/or spores in contaminated food (diarrhoeal type) or by ingesting food contaminated with pre-formed toxin (emetic type). Transmission of this disease results from consumption of contaminated foods, improper food handling/storage and improper cooling of cooked foodstuffs (Schneider et al. 2004).

Incidence of illness and outbreak data

B. cereus related food poisoning is not a notifiable disease in most countries, including Australia and New Zealand, and therefore incidence data is extremely limited. It is recognised that there may be significant under reporting of *B. cereus* illness due to the generally mild, short duration and self-limiting symptoms, in addition to it being infrequently tested for in routine laboratory analyses of stool samples.

There was one reported outbreak of *B. cereus* foodborne illness in Australia in 2011 and one outbreak reported in 2010 (OzFoodNet 2012a; OzFoodNet 2012b). It has been estimated that *B. cereus* accounts for 0.5% of foodborne illness caused by known pathogens in Australia (Hall et al. 2005). In New Zealand there was one foodborne *B. cereus* outbreak reported in 2011, there were no outbreaks reported in 2010 (Lim et al. 2012).

In the European Union there were 0.04 reported cases of *B. cereus* foodborne illness per 100,000 population in 2011 (ranging from <0.01–0.24 per 100,000 population between countries). This was an increase from the 2010 case rate of 0.02 cases per 100,000 population (EFSA 2012; EFSA 2013).

B. cereus was reported as a major causative agent of foodborne illness in the Netherlands in 2006 (causing 5.4% of the foodborne outbreaks) and in Norway in 2000 (causing 32% of foodborne outbreaks) (Wijnands 2008). Scallen et al. (2011) estimated that in the United States (US), *B. cereus* caused 0.7% of foodborne illness caused by 31 major pathogens.

Meat, milk, vegetables and fish have been the predominant food types associated with the diarrhoeal syndrome. In contrast, rice products, potato, pasta and cheese products have been the predominant foods associated with the emetic syndrome (FDA 2012) (refer to Table 2).

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

Year	No. of cases (fatalities)	Food	Syndrome type	Country	Comments	References
2008	1 (1)	Spaghetti with tomato sauce	Emetic	Belgium	Food stored at room temperature for 5 days after preparation. *B. cereus* and cereulide isolated from pasta	(Naranjo et al. 2011)
2007	2 (1)	Asparagus sauce	Emetic	Australia	Prior to serving, the sauce was stored for 2 hours in a hot kitchen (up to 37°C), permitting *B. cereus* growth	(NSW Food Authority 2013)
2003	4 (1)	Pasta salad	Emetic	Belgium	Food stored for 3 days in fridge at 14°C, permitting *B. cereus* growth. *B. cereus* isolated from food	(Dierick et al. 2005)
2000	173	Cake	Diarrhoeal	Italy	*B. cereus* isolated from food and rolling board. Rolling board likely source of contamination	(Ghelardi et al. 2002)
1998	44 (3)	Vegetable puree	Diarrhoeal	France	Cytotoxin K produced by *B. cereus* involved	(Jenson and Moir 2003)
1991	139	Barbequed pork	Diarrhoeal	US	*B. cereus* spores from dried foods, slaughtered animals or worker hands likely source of contamination. Unrefrigerated storage of cooked pork for >18 hours permitted *B. cereus* growth	(Luby et al. 1993)
1989	55	Cornish game hens	Diarrhoeal	US	Inadequate thawing and cooking, cross-contamination from basting brush used before and after cooking, inadequate refrigeration	(Slaten et al. 1992)

Occurrence in foods

As *B. cereus* is found in soil, raw plant foods such as rice, potatoes, peas, beans and spices are common sources of *B. cereus*. The presence of *B. cereus* in processed foods results from contamination of raw materials and the subsequent resistance of spores to thermal and other manufacturing processes. During the cooling processes, spores may germinate, enabling *B. cereus* to multiply in the food and/or produce high levels of the emetic toxin cereulide, depending on the strain(s) present (Wijnands 2008).

B. cereus has been recovered from a wide range of food types. A survey carried out in Brisbane on 1,263 retail food products reported the prevalence of *B. cereus* as 1.6% on unbaked pizza bases (n=63), 4.5% on ready-to-reheat frozen cooked meat pies (n=157), 0.3% on processed meats (n=350) and 5.5% on raw diced chicken (n=55) (Eglezos et al. 2010).

In the Netherlands, an investigation was carried out on the prevalence of potentially pathogenic strains of *B. cereus* in retail food samples (Wijnands et al. 2006b). The strains containing potential toxin producing genes were classified as psychrophilic, intermediate and mesophilic in nature. It was found that 89.9% of the isolates were mesophilic, with psychrophilic and intermediate strains amounting to 4.4% and 5.7%, respectively (n=796). Of the isolates found in flavourings, 98.9% were mesophilic (n=92). Prevalence of mesophilic isolates was also high in ready-to-eat products (92.7%, n=384), vegetables and vegetable products (91.4%, n=115) and pastry (90.1%, n=81). A higher prevalence of psychrophilic strains of *B. cereus* have been reported in meat and meat products (20.8%, n=24) and in fish and fish products (40%, n=40) (Wijnands et al. 2006b). A study by Ankolekar et al. (2009) undertaken in the US reported that 93.3% of the *B. cereus* strains isolated from retail uncooked (raw) rice (n=83) were positive for NHE or HBL, representing diarrhoeal strains.

Agata et al. (2002) performed an investigation into cereulide in foods. When an emetic type strain of *B. cereus* was added to food products and the food was stored under conditions designed to simulate temperature abuse (30°C, 24h), cereulide production occurred in rice dishes and other starchy foods. Addition of ingredients such as mayonnaise, vinegar and other condiments retarded the growth of bacteria and the quantity of cereulide formed. Bacterial growth and/or cereulide production was inhibited in egg and egg products, meat and meat products, milk and soybean curd. Shaking (aeration) of milk and soymilk resulted in increased cereulide production (Agata et al. 2002).

Host factors that influence disease

All people are believed to be susceptible to *B. cereus* food poisoning. However, some individuals, especially young children, are particularly susceptible and may be more severely affected (ICMSF 1996). Individuals vary in their response to cereulide dosages; this may be associated with differences in the number of $5-HT_3$ receptors in the stomach/small intestine of individuals (Wijnands 2008).

The risk of illness after ingestion of vegetative cells is influenced by the strain, composition of the food, the liquid nature of the food and the age of the individual. Liquid foods are transported faster to the small intestine and therefore are protected from the influence of gastric conditions, providing more opportunity for survival of the pathogen (Wijnands 2008).

Dose response

No human dose response relationship is available for either the emetic or diarrhoeal toxin produced by *B. cereus*. Epidemiological evidence suggests that the majority of outbreaks worldwide due to *B. cereus* have been associated with concentrations in excess of 10^5 cfu/g in implicated foods. Rare cases of both emetic and diarrhoeal illness have been reported involving 10^3 –10^5 cfu/g of *B. cereus* in food. These cases occurred in infants or aged and infirm individuals (Kramer and Gilbert 1989; Becker et al. 1994). Laboratory studies on the formation of emetic toxin in boiled rice cultures support this finding, with >10^6 cfu/g of *B. cereus* required for toxin production to occur (Finlay et al. 2002). The use of a threshold is analogous to the No Observed Adverse Effects Level (NOAEL), commonly used in the assessment of risk from chemical substances in food. The threshold of 10^5 cfu/g is at any point after cooking, and not just the final concentration as used by McElroy et al. (1999) (described below).

A surrogate approach using vegetative cell concentrations has been used to estimate dose response for the emetic toxin. McElroy et al. (1999) developed a two-step approach that linked the probability of illness, based on *B. cereus* concentrations from outbreaks and an attack rate (assumed to be independent of dose). A weakness of this approach is that the probability of illness is based on outbreaks where toxins may have been pre-formed in the food vehicle and subsequently cooked. As a result of the cooking, the total *B. cereus* concentration in the food vehicle may be reduced and therefore not directly related to the presence or concentration of toxin. This may account for disease outbreaks being reported where *B. cereus* concentrations in the food have been as low as 10^3 cfu/g.

Epidemiological data gathered during outbreaks in the Netherlands has been used to estimate that a dose of cereulide of approximately 9.5 µg/kg of bodyweight is required to cause the onset of the emetic syndrome (Finlay et al. 1999).

Recommended reading and useful links

Arnesen SLP, Fagerlund A, Granum PE (2008) From soil to gut: *Bacillus cereus* and its food poisoning toxins. FEMS Microbiology Reviews 32:579-606

FDA (2012) Bad bug book: Foodborne pathogenic microorganisms and natural toxins handbook, 2nd ed, US Food and Drug Administration, Silver Spring, p. 93–96. http://www.fda.gov/Food/FoodborneIllnessContaminants/CausesOfIllnessBadBugBook/ucm2006773.htm

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