Bacterial Food Intoxication: An Overview

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ABSTRACT

Food Contamination, food spoilage, foodborne diseases, bacterial foodborne disease, food infections, food poising, bacterial food poisoning, infective bacterial food poisoning and toxic bacterial food poisoning (bacterial food intoxication) have been all defined.

This review concentrates on bacterial food intoxication. Characteristics of the bacteria causing food intoxication, properties of toxins, symptoms of food intoxication, foodstuffs associated with food intoxication, preventive measures of food intoxication and pathogenesis of each food intoxication were reviewed. The following bacteria were included: *Bacillus cereus*, *Clostridium botulinum*, *Clostridium perfringens*, enterotoxigenic *Escherichia coli*, enterohaemorrhagic *Escherichia coli* and *Staphylococcus aureus*.

Kew words: intoxication, food poisoning, Bacillus cereus, Clostridium botulinum, Clostridium perfringens, enterotoxigenic Escherichia coli, enterohaemorrhagic E. coli, Staphylococcus aureus.

INTRODUCTION

Food contamination can be defined as the undesired occurrence of harmful microorganisms or substances in food. Food contamination results in food spoilage and/or foodborne diseases. A foodborne disease is considered to be any illness associated with or in which the causative agent is obtained by the ingestion of food. If the causative agent is bacteria, the disease called bacterial foodborne disease (Frazier & Westhoff, 1988, Janseen *et al*., 1997).

Bacterial illnesses in which the food does not support growth of the pathogens are called food infections (i.e. pathogens such as those causing tuberculosis, diphtheria, dysenteries, typhoid fever, brucellosis, cholera, etc.). Bacterial food poisoning is an acute condition, usually presenting as gastroenteritis, the first symptoms of which normally arise within a few hours or few days of consumption of food containing pathogenic bacteria and/or their products. In most cases the food in question will have supported rapid growth of pathogens. Food poisoning that is not caused by bacterial by-products (toxins) but through ingestion of infectious bacteria, is referred to as infective bacterial food poisoning. Food poisoning caused by bacterial toxins is called toxic bacterial food poisoning or bacterial food intoxication (Eley, 1996, Mines *et al*., 1997, Matsui 2004, Marriott & Gravani, 2006, Thompson, 2007, Todar, 2008).

The global incidence of foodborne disease is difficult to be estimated, but it has been reported that in 2005 alone 1.8 million people died from diarrheal diseases. A great proportion of these cases can be attributed to contamination of food and drinking water. Additionally, diarrhea is a major cause of malnutrition in infants and young children. In industrialized countries, the percentage of the population suffering from foodborne diseases each year has been reported to be up to 30%. In the United States of America (USA), for example, around 76 million cases of foodborne diseases, resulting in 325,000 hospitalizations and 5,000 deaths, are estimated to occur each year. Number of cases of foodborne diseases in Australia is 5.4 million yearly. In France there are an estimated 750,000 cases of foodborne illness every year, while in Japan there were 9666 cases in 2007. Unfortunately, reliable comparable figures from the developing world are not available (Granum, 2006, Farthing & Kelly, 2007, WHO, 2007, IASR, 2008, Hamer & Gorbach, 2008, Acheson, 2009, Anonymous, 2009, Senior, 2009). According to the European Food Safety Authority (EFSA), the numbers of cases caused by bacterial toxins represent 16% of the total cases caused by foodborne diseases. In EU, 93% of the outbreaks caused by bacterial toxins were verified (EFSA, 2009).

The present overview deals with the most important bacterial food intoxication. Bacteria that

cause food intoxication may be divided into two groups. The first is responsible for pre-formed toxin in foods, i.e. *Bacillus cereus* (emetic), *Clostridium botulinum* and *Staphylococcus aureus*. The second group forms toxin in the intestine, i.e. *Bacillus cereus* (diarrheal), *Clostridium perfringens*, enterotoxigenic *E. coli* (ETEC) and enterohaemorrhagic *E. coli* (EHEC).

Characteristics of the bacteria causing food intoxication, properties of toxins, symptoms of food intoxication, foodstuffs associated with food intoxication and preventive measures of food intoxication are summarized in Tables 1, 2, 3, 4, and 5, respectively. Pathogenesis of each food intoxication will be further discussed.

Bacillus cereus intoxication

Bacillus cereus has been recognized as an agent of food poisoning since 1955. There are only a few outbreaks a year reported. It is not a reportable disease and usually goes undiagnosed. *Bacillus cereus* is associated with two distinct toxinmediated types of food poisoning: the emetic syndrome, which has a short incubation period (range 1 to 6 hr), and the diarrheal syndrome, which has a longer incubation period (range 8 to 16 hr). The type of toxin elaborated depends on the type of food contaminated with *B. cereus*, rather than the strain of the organism. The emetic toxin is produced in foods such as fried rice left at room temperature, whereas proteinaceous foods are usually associated with the diarrheal syndrome (Batt, 2004, Matsui, 2004, Thompson, 2007, Todar, 2008, Acheson, 2009).

The emetic disease follows the ingestion of food contaminated with a low–molecular weight toxin. It is a thermo-stable protein associated with and/or produced during spore formation. Outbreaks have occurred as a result of consuming fried rice served in restaurants. Spores in rice are not always killed during the cooking process. When the rice cools and following spore germination, vegetative cell growth may be rapid, especially at room temperature. Of the new large number of vegetative cells in the food some may sporulate and lead to toxin formation, especially if the rice is left for more than a few hours at room temperature (Ouoba *et al*., 2008, Ankolekar *et al*., 2009, Cronin & Wilknosn, 2009).

The longer incubation period and large inoculum 10^6 organisms per gram of food required to induce the diarrheal syndrome suggest intestinal colonization, rather than performed toxin alone, as the likely mechanism of disease. In diarrhetic type, a diarrheal toxin is formed as follows: spores can survive boiling in foods, and these spores can germinate in the gastrointestinal tract after consumption of the contaminated food. Toxin is produced on germination (Eley, 1996, Al-Khatib *et al*., 2007, Arnesen *et al*., 2007).

Botulism

In botulism, the food item becomes contaminated with spores from the environment, which are not destroyed by the initial cooking or processing. If the food is then kept in conditions appropriate for growth, the spores may germinate, leading to production of toxin. If not destroyed by heating before serving, the toxin can be ingested with the food item and absorbed. Once the toxin is absorbed, it attaches to the neuromuscular junction of affected nerves and prevent the release of acetylcholine, thus causing muscular paralysis. In sever case the paralysis can be profound, with death resulting from respiratory failure within 24 hours.

Clostridium botulinum produces neurotoxins which bind to the pre-synaptic nerve endings of peripheral cholinergic nerves after entering the vascular system. The binding between botulinum toxin and peripheral cholinergic nerves ending blocks the release of acetylcholine which induces weakness of the innervating muscles. Associated symptoms include neurological, autonomic and gastro-intestinal symptoms (Eley, 1996, Aureli *et al*., 2008, Draghici *et al*., 2008, Johnson & Montecucco, 2008, Acheson, 2009, Tseng *et al*., 2009).

Infants can be affected by this disease through the ingestion of as few as 10 to 100 spores that germinate in the intestinal tract and produce toxin. Death occurs in approximately 60% of the cases from respiratory failure. Infantile botulism, first discovered in the late 1970s, is a rare neuroparalytic disease caused by the absorption of *Clostridium botulinum* found in dust, soil, corn/maple syrups, and honey containing products-in the infant's intestinal tract. *Clostridium botulinum* spores are nontoxic when ingested by adults, however, the spores may germinate, multiply, and produce toxin within the intestinal cavity when consumed by infants younger than 1 year of age. The ensuing illness may range in severity from failure to thrive to death. Botulism should be suspected in any infant who presents with difficulty feeding, constipation, paralysis, autonom-

Table 1: Characteristics of bacteria causing food intoxication

Characteristics \ Bacteria	*Bacillus cereus*	*Clostridium botulinum*	*Clostridium perfringens*	*Escherichia coli*	*Staphylococcus aureus*
Cell shape	Rods	Rods	Rods	Rods	Cocci
Gram stain	+	+	+	–	+
Spore-forming	+	+	+	–	–
Spore-heat resistance	+	+	±		
Air requirements	Obligate aerobes	Anaerobes	Anaerobes	Facultative anaerobes	Facultative anaerobes
Temperature range (°C)	15–55*	10–50**	15–50	10–44	7–46
Optimal temperature (°C)	30	30–40	43–46	37	37
Growth at 4°C	±	±	–	–	–
pH range	5–8.8	4.8–8.9	5–9	4–8.5	4–9.8
Large numbers of active bacteria are needed to cause intoxication	+	–	+	+***	+

* Psychrotrophic strains are able to grow at 4-6°C.
** Type E thrives at 3.3 to 45°C.
*** The infectious dose is 100-2000 for enterohaemorrhagic *E. coli.*,
References: Eley, 1996, Janssen *et al*., 1997, Marriott & Gravani, 2006, Arnesen *et al*., 2007, Roy *et al*., 2007, Ankolekar *et al*., 2009.

Table 2: Properties of toxins produced by bacteria causing food intoxication

	M.W. (Da)	Proteinaceous	Heat stability***	Neuro-toxicity	Increase cAMP/ GMP	No. of toxins
Bacillus cereus						
Emetic	5000	+	+	–	–	1
Diarrheal	50,000	+	–	–	+	1
Clostridium botulinum	150,000	+	–	+	–	8 (A, B, C_1, C_2, D, E, F, G)
Clostridium perfringens	35,000	+	–	–	–	4 (α, β, γ, σ)
E. coli						
ETEC : ST_a*	2000	+	+	–	+	
ST_b*	5000	+	+	–	–	
LT*	86,000	+	–	–	+	
EHEC : VT1**	30,000	+	–	–	–	
VT2**	32,000	+	–	–	–	
S. aureus	28,000–35,000	+	+	+	–	8 (A, B, C_1, C_2, C_3, D, E, F)

* ST: thermo -stable, LT: thermo -labile. ** VT1, VT2: verotoxin 1 and verotoxin 2, also referred to as shigatoxin 1 and 2.
*** Heat-stability: 100°C for 30 minutes.
References: Eley, 1996, Smedley, *et al.*, 2004, Basak *et al.*, 2006, Apetroaie-Constantin *et al.*, 2008, Aureli *et al.*, 2008, Boynukara *et al.*, 2008, Fu & Wang, 2008, Johnson & Montecucco, 2008, Ankolekar *et al.*, 2009, Tseng *et al.*, 2009.

Table 3: Symptoms of bacterial food intoxication

	Incubation time (hr)	Duration of illness (hr)	Vomiting	Nausea	Diarrhea	Abdominal pain	Fever
Bacillus cereus							
Emetic	1–6	6–24	+	+	±	+	–
Diarrheal	8–16	6–24	–	+	+	+	–
*Clostridium botulinum**	12–35	(1 week–6 months)	±	±	±	±	–
Clostridium perfringens	8–24	12–24	–	±	+	+	–
E. coli							
ETEC	(1–3 days)	(1–7 days)	–	+	+	+	+
EHEC	(2–12 days)	(3–8 days)	–	+	+	+	–
S. aureus	1–6	6–24	+	+	+	+	–

* Impaired swallowing, speaking, respiration, and coordination. Dizziness, double vision, weariness and weakness.
References: Eley, 1996, Mines *et al.*, 1997, Nishina, 1997, Marriott & Gravani, 2006, Karageanes, 2007, Draghici *et al.*, 2008.

Table 4: Foodstuffs associated with bacterial food intoxication

	Meat products	Poultry	Eggs	Raw milk	Dairy products	Seafood Shellfish	Vegetables	Cereals	Others
Bacillus cereus									
Emetic	–	–	–	–	–	–	–	+	
Diarrheal	+	–	–	–	+	–	+	–	
Clostridium botulinum	+	–	–	–	–	+	+	–	+*
Clostridium perfringens	+	+	–	–	–	+	–	–	
E. coli									
ETEC	+	–	–	–	–	–	–	–	
EHEC	+	–	–	+	+	–	–	–	+**
S. aureus	+	+	+	–	+	–	–	–	+***

* Home-canned vegetables

** Potato salad, custard-filled pastries

*** Water, radish sprouts in Japan 1996

References: Lee *et al.*, 1996, Agata *et al.*, 2002, Vahdani *et al.*, 2002, Balaqué *et al.*, 2006, Soejima *et al.*, 2007, Draghici *et al.*, 2008, Fu & Wang, 2008, Van *et al.*, 2008, Chokesajjawatee *et al.*, 2009, Preira *et al.*, 2009.

Table 5: Preventive measures of food poisoning caused by toxin forming bacteria

	B. cereus		*C. botulinum*	*C. perfringens*	*E. coli*		*S. aureus*
	Emetic	**Diarrheal**			**ETEC**	**EHEC**	
Food should be cooked and eaten immediately							+
Handling of cooked food should be at minimal							+
Keep foods below 4°C or above 55°C	+	+		+			+
Avoid or minimize contamination			+				
Good personal hygiene and good sanitation through production	+	+	+	+	+		
Killing vegetative cells and spores	+	+	+	+			
Destroy of toxins			+				
Control spore germination	+	+					
Avoid proliferation of vegetative cells in cooked foods	+	+					
Untreated sewage should not be used in fertilization of crops or vegetables					+		
Avoid cross-contamination between raw and cooked food					+	+	
Proper or effective cooking or canning			+		+	+	

References: Eley, 1996; Nishina, 1997, Brackett, 1999, Aureli *et al.*, 2008, Johnson & Monteucco, 2008.

ic involvement, or respiratory failure (Marriott & Gravani, 2006, Famularo, 2009).

Clostridium perfringens intoxication

This form of food poisoning is caused by a thermo-labile, 35 kDa enterotoxin produced by *C. perfringens* type A. The enterotoxin is a structural protein of the spore coat and is produced during sporulation. Unlike cholera toxin, the *C. perfringens* enterotoxin has greatest activity in the ileum and can inhibit glucose transport, damage intestinal epithelial cells, and induced protein loss. Outbreaks usually are associated with large group gatherings and institutional settings in which precooked meat (beef, chicken, or turkey) that requires reheating is served. When food is cooked in large batches for these gatherings, spores of *C. perfringens* may survive and germinate as the food is cooled. If the food is not reheated to a temperature sufficiently high to kill the organisms, food poisoning may develop among individuals who ingest a large number of organisms and a large amount of toxin. (Petit *et al*., 1999, Matsui, 2004, Basak *et al*., 2006).

In contrast, *C. perfringens* type C causes a more severe illness called enteritis necroticans or pigbel, which has a high mortality rate (40%). Historically, eating rancid meat (post-World War II Germany) and large amounts of poorly cooked pork (New Guinea) has been associated with this illness. Although rare in the United States, outbreaks have been associated with eating chitterlings (hog intestines). Although the enterotoxin elaborated by *C. perfringens* type C is similar to that produced by type A, it induces a much more severe clinical course that includes bloody diarrhea, necrotizing intestinal damage, and intestinal perforation. The enteric toxins of *C. perfringens* share two common features: (1) they are all single polypeptides of modest (~25-35 kDa) size, although lacking in sequence homology, and (2) they generally act by forming pores of channels in plasma membranes of host cells (Matsui, 2004, Smedley *et al*., 2004, Basak *et al*., 2006).

Intoxication caused by enterotoxigenic *E. coli*

After ingestion, bacteria that survive the hostile environment of the stomach must penetrate the mucous layer of the small intestine, where they adhere to mucosal cells. There, they produce either or both of two types of enterotoxin: LT (thermolabile) and ST (thermo -stable), which may result in profuse watery diarrhea, typically this is less sever than in patients with cholera. The LT increases adenylate cyclase activity. Two types of ST have been described, ST_a and ST_b. The mode of action of ST_b is uncertain. The ST_a binds to a receptor leading to increase the activity of guanylate cyclase with conversion of GTP to cyclic GMP. The later acts as a messenger like cAMP to cause a physiological response. The increase of cAMP or cGMP alters the balance of Na^+ and Cl^- ions entering and leaving the enterocytes which directly affect water absorption and secretion. The net result is an efflux of Na^+ and Cl^- with more water being secreted than is absorbed (Eley, 1996, Kaper, 2005, Rappelli *et al*., 2005, Vicente *et al*., 2005).

Intoxication caused by enterohaemorrhagic *E. coli*

Disease is due to the production of one or more verocytotoxins (VTS) called VT1, VT2, VT3. These toxins are closely related to shiga toxin produced by *Shigella dysenteriae* serotype 1. Pathological effects include morphological changes in epithelial cells, increased mitotic activity in crypts, mucin depletion and an infiltration of polymorphnuclear cells into mucosa. These changes result in either watery and/or bloody diarrhea. The infective dose is thought to be low and is estimated to be fewer than 100 cells (Eley 1996, Kaper, 2005, Rappelli *et al*., 2005, Chassagne *et al*., 2009).

Escherichia coli O157: H7 is a bacterium belongs to this group and causes approximately 73,000 cases of foodborne illness each year in U.S. Also, this bacterium resulted in an outbreak in south Wales in 2005 and in Scotland in 1996 (Marriott & Gravani, 2006, CDC, 2008).

Staphylococcus aureus intoxication

Staphylococcal toxins cannot be considered as classical enterotoxins like cholera toxin, since they do not act directly on intestinal cells. Instead, the toxins act on receptors in the intestine, with the stimulus reaching the vomiting centre in the brain via the vagus nerve and should, therefore, be considered as neurotoxins. *S. aureus* was the leading cause of food poisoning in the United States until 1973, but now ranks third. The illness is associated with coagulase-positive strains that elaborate thermo-resistant enterotoxins A, B, C_1, C_2, C_3, D, E and/or F (28 to 35 kDa) (Chaubeau-Duffour, 1992, Eley, 1996, Bergdoll & Wong, 2003, Matsui, 2004, Soejima *et al*., 2007, Thompson, 2007, Boynukara *et al*., 2008, Acheson, 2009).

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التسمم الغذائي الناتج من سموم البكتريا: نظـرة شـاملة

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تم تعريف كل من تلوث الغذاء، فساد الغذاء، الأمراض الناتجة من تناول الغذاء، الأمراض الناتجة من تناول غذاء ملوث بالبكتريا، أمراض العدوى المنقولة عن طريق الغذاء، التسمم الغذائي، التسمم الغذائي من البكتريا، التسمم الغذائي من بكتريا معدية والتسمم الغذائي من سموم البكتريا.

ركزت هذه المقالة على التسمم الغذائي الناتج من سموم البكتريا وقد تناولت كل من صفات البكتريا المسببة للتسمم، صفات السموم، أعراض التسمم، الأغذية التي تنقل هذا النوع من التسمم، منع حدوث التسمم وميكانيكية حدوث المرض وقد شملت المقالة كلا" من البكتريا التالية:

Bacillus cereus, *Clostridium botulinum*, *Clostridium perfringens*, enterotoxigenic *E. coli*, enterohaemorrhagic *E. coli* and *Staphylococcus aureus.*