Food-Borne Microbial Diseases and Control: Food-Borne Infections and Intoxications

Sait Aykut Aytac and Birce Mercanoglu Taban

1 Introduction

World Health Organization (WHO) defined the term food-borne disease as any disease of an infectious or toxic nature caused by, or thought to be caused by, the consumption of food or water. Regardless of the latest advances in food hygiene and food manufacturing and processing, in addition with increase in consumer awareness, food-borne microbial diseases are the most widespread problem and still represent a significant threat to public health in the contemporary world, especially to the very young, the old, the very sick, and the immunocompromised people who are all more at risk of them.

Each year in the United States of America (the USA), for example, food-borne diseases result in an estimated 76 million illnesses, 325,000 hospitalizations and 5,000 deaths (WHO World Health Organization 2007). In 2011, Centers for Disease Control and Prevention (CDC) estimates the numbers of food-borne diseases caused by the main known pathogens and unspecified agents transmitted by food as 47.8 million/year, resulting in 127,839 hospitalizations and 3,037 deaths in the USA (Scallan et al. 2013). It has been also estimated that the risk of becoming ill as a result of microbial contamination of food is 100,000 times greater than the risk from pesticide contamination (Adams and Moss 2008).

Changes in farming practices, increase in international trading of foods, changes in food manufacturing and processing, increase in international movement of people, changing lifestyle of the population, and microbial evolution are the factors that contribute to food-borne microbial diseases (Adams and Moss 2008).

S.A. Aytac
Department of Food Engineering, Hacettepe University, 06532, Beytepe, Ankara, Turkey

B.M. Taban (✉)
Department of Dairy Technology, Ankara University, 06110, Diskapi, Ankara, Turkey
e-mail: btaban@ankara.edu.tr

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Food-borne microbial diseases are caused by the consumption of either food or water contaminated with alive pathogenic bacterial cells (or bacterial spores as in infant botulism) or food that include toxins produced by the toxigenic bacteria or moulds (Ray and Bhunia 2008). In other words, there are three types of food-borne microbial diseases: intoxication or poisoning, infection, and toxico-infection (Dhama et al. 2013). Food intoxication arises as a result of the consumption of preformed bacterial or mould toxin (mycotoxins) which has to be in active form in food. In this type of disease, there is no need of alive cells during the consumption of the food after the toxigenic bacteria or moulds have grown and produced the regarding toxin under favourable conditions in the food. In other words, toxins, not bacteria, cause disease. On the other hand, food infection arises due to the consumption of food or water contaminated with alive enteropathogenic bacteria or viruses. Lastly, toxico-infection arises as a result of the ingestion of many alive pathogenic bacterial cells via contaminated food and water and then they sporulate, colonize, and produce toxin(s) in the body (Ray and Bhunia 2008).

2 Food-borne Bacterial Pathogens

2.1 Bacterial Infections

2.1.1 *Campylobacter* spp.

The genus *Campylobacter* belongs to the family of Campylobacteriaceae that comprises 18 species of *Campylobacter* and 4 species of *Arcobacter* (Nachamkin 2001). *Campylobacter jejuni* subsp. *jejuni* (referred to subsequently as *C. jejuni*) is the most important member of this genus since it is known as one of the causative agents of food-borne microbial diseases. However, other *Campylobacter* species such as *C. coli* and *C. fetus* also cause infectional diseases called campylobacteriosis (FDA 2012).

Campylobacter spp. are Gram-negative, oxygen-sensitive microaerophilic (85 % nitrogen, 5–10 % carbon dioxide, and 3–5 % oxygen: oxygen concentrations lower than the atmospheric oxygen level), oxidase-positive, non-spore forming curved rods, or spiral-shaped bacteria and are motile with polar flagella on one or both ends that produce the corkscrew-like motility. They cannot ferment or oxidize sugars. All *Campylobacter* species grow at 37 °C, however, the optimum growth temperature for *C. jejuni* and *C. coli* is around 42–45 °C (thermoduric). They stay alive weakly at room temperature and do not grow below 30 °C. Under unfavourable conditions, they cannot be isolated by cultural detection methods but still remain infective [viable but non-culturable (VBNC) state] (Adams and Moss 2008; FDA 2012).

Campylobacter spp. are responsible for an estimated 2.5 million food-borne illness per year in the USA which means the 17 % of hospitalizations resulting from food-borne infections and an estimated 5 % of food-borne-related deaths in the USA (Mead et al. 1999). The infective dose of *Campylobacter* spp. given in the literature varies from 5×10^2 to 10^5 organisms due to the type of contaminated food consumed

and the general health of the exposed person (Robinson 1981; Black et al. 1988; FDA 2012). Campylobacteriosis which is a generic term for a variety of syndromes caused by infection with *Campylobacter* spp., usually occurs in 2–5 days after ingestion of the contaminated foodstuff. Their symptoms include abdominal pain (which may mimic appendicitis), watery and/or bloody diarrhoea, malaise, fever, myalgia, and sometimes vomiting that last less than 1 week (FDA 2012). They also cause the serious neurological disease, Guillain–Barre syndrome (Adams and Moss 2008).

Since the optimum growth temperature of *C. jejuni* and *C. coli* is high as the body temperature of birds, the primary reservoir of *Campylobacter* spp. is the gastro-intestinal tract of wild and domesticated birds (Adams and Moss 2008). The main food sources are improperly handled or raw poultry products, raw or inadequately pasteurized milk, contaminated water, and cheeses made from unpasteurized milk (Horrocks et al. 2009; Jore et al. 2010; Schnider et al. 2010; Pitkänen 2013). In addition to these, they can form biofilm on the stainless steel surfaces over the time (Nguyen et al. 2010).

The ideal way to control the number of human infections by *Campylobacter* spp. would be to maintain hygienic conditions on farm and to avoid cross-contamination during slaughtering which lead to decrease in the number of *Campylobacter* spp. on poultry products. Raw or under-cooked foods, untreated drinking water, and unpasteurized milk should not be consumed. Hence, foods should be cooked completely. Post-pasteurization contamination should be avoided since it reintroduces these bacteria into milk. The personnel of the catering industry should not handle food if he/she suffers from campylobacteriosis or handle cooked food or ready-to-eat food with bare hands. Cutting boards and utensils used in handling uncooked poultry or other foods should be washed before being used for preparation of other foods that are eaten raw (Allos 2001).

2.1.2 *Cronobacter sakazakii*

The genus Cronobacter, which belongs to the family of Enterobacteriaceae, includes seven recognized species: *C. condimenti*, *C. dublinensis* (subsp. *dublinensis*, *lausannensis*, and *lactaridi*), *C. malonaticus*, *C. muytjensii*, *C. sakazakii* (formerly named *Enterobacter sakazakii* in honour of the eminent Japanese bacterial taxonomist Riichi Sakazaki), *C. turicensis*, and *C. universalis* (Adams and Moss 2008; Tall et al. 2013). Among them; *C. sakazakii* has recently considerable concern, although it is responsible for only sporadic individual cases or relatively small outbreaks of infections.

C. sakazakii is a Gram-negative, facultatively anaerobic, catalase-positive, oxidase-negative, non-spore forming rods, and is motile with peritrichous flagella that can grow at temperatures ranging from 6 to 47 °C with an optimum at 37 °C (mesophilic). Pasteurization at 72 °C for 15 s ensures more than a 10 log reduction in the number of survivor cells. It is moderately acid resistant compared to other Enterobacteriaceae. The minimum pH for its growth is 3.89 but the optimum pH ranges from 5.0 to 9.0. It produces yellow pigmented colonies on non-selective agar media (Lambert and Bidlas 2007; Adams and Moss 2008; Tall et al. 2013).

C. sakazakii is an emerging opportunistic pathogen that represents a significant risk to the health of neonates in hospital nurseries and neonatal intensive care units since it is related with occasional but life-threatening cases of meningitis, cerebritis, necrotizing enterocolitis (NEC), and septicaemia in neonates and infants with weakened immune systems, particularly premature and full-term infants with mortality rates of 40–80 % being reported. Even in the case of recovery, long-term neurologic sequela have been reported in affected infants. Wound infections, urinary tract infections, septicaemia, vaginitis, and aspiration pneumonia are the symptoms of this infection in adults and elderly people (Lai 2001; Giovannini et al. 2008; Healy et al. 2010; Tall et al. 2013).

C. sakazakii is regarded as ubiquitous organism that has been isolated from various foods. A high tolerance to osmotic and dry stress and high temperatures than other Enterobacteriaceae ensure the survival of it in powdered infant formulae (PIF) more than 2 years in which the contamination can occur during the manufacturing process or during post-manufacture reconstitution. Follow-on formula, weaning foods, milk and sodium caseinate powders, rice seed, dried herbs and spices, spiced meats, dried corn, soy, potato, wheat, and rice flours, dried infant and adult cereals, dried vegetables, grains, tofu, powdered herbal tea, mixed salad vegetables, tomato harvesting bins, chocolate and candied cough drops, and pastas are the other foods from which it has been isolated (Adams and Moss 2008; Healy et al. 2010; Tall et al. 2013).

The most important prevention and control way is to avoid poor hygiene during preparation and feeding of infants by applying regularly hand washing and proper washing of teats, bottles, cups, blender, and spoons with boiled water. In addition to these, it is better to avoid storage of reconstituted formulae over a prolonged period of time at refrigerated and at room temperatures and to discard unused formulae (Adams and Moss 2008; Tall et al. 2013).

2.1.3 Pathogenic *Escherichia coli*

Escherichia coli, which belongs to the family of Enterobacteriaceae, was firstly described by the German bacteriologist Thedor Escheric in 1885 during its isolation from childrens' faeces (Adams and Moss 2008).

E. coli is a Gram-negative, facultatively anaerobic, catalase-positive, oxidase-negative, urease-negative, H_2S-negative, non-spore forming rod, and is motile with peritrichous flagella that can grow at temperatures ranging from 7–10 °C to 50 °C with an optimum at 37 °C (mesophilic). However, some ETEC strains can grow below 4 °C. In IMViC tests, most of the *E. coli* strains are indole and methyl red positive and VP and citrate negative. It is heat sensitive and so is killed by pasteurization process (62.8 °C for 30 min or 71.7 °C for 15 s) but can survive during refrigerated or frozen storage for extended times. The optimum pH for its growth is 7.0 but can grow at pH 4.4. It ferments lactose with producing gas. Since it normally lives in the intestines of humans, warm-blooded animals, and birds at a very high level, it has been used as an index organism of possible faecal contamination and the presence of enteric pathogens in water and food (Adams and Moss 2008; Lund 2008; Ray and Bhunia 2008).

E. coli is one of the major enteric species in the gut of humans, warm-blooded animal. As being an important part of a healthy human intestinal tract, most *E. coli* strains are harmless and in fact provide many health benefits such as preventing colonization of the gut by harmful pathogens and producing vitamin K_2. However, some *E. coli* strains, which are described as enterovirulent *E. coli*, diarrhoeagenic *E. coli*, or more commonly, pathogenic *E. coli,* have the potential to be pathogenic to humans and can cause severe diarrhoeal diseases. Currently, pathogenic *E. coli* strains are categorized into six recognized pathotypes which are enteroinvasive *E. coli* (EIEC), enteropathogenic *E. coli* (EPEC), enterotoxigenic *E. coli* (ETEC), enterohaemorrhagic *E. coli* (EHEC), enteroaggregative *E. coli* (EaggEC), and diffusely adherent *E. coli* (DAEC) (Adams and Moss 2008). Among them, EHEC is distinguished from others by the production of Shiga-like toxins (Stx) (also known as verotoxin). There are more than 200 serotypes of Shiga toxin-producing *E. coli* (STEC) or Vero (so-called because of its ability to kill Vero that is African Green Monkey Kidney cells) toxin-producing *E. coli* (VTEC), but not all have been involved in human illness. Thus, *EHEC* is a subset of the STEC that has been implicated in haemorrhagic colitis (HC), the potentially fatal haemolytic uremic syndrome (HUS) or thrombotic thrombocytopaenic purpura (TTP). However, EHEC not only produce the shiga toxin coded by the *Stx* gene but can also attach to the intestinal wall due to the protein intimin (an adhesin) coded by the *eae* gene, and also includes the *ehx*A gene that encodes the enterohemolysin. Although several serotypes, including O111:H8, O26:H11, O103:H2, O113:H2, O104:H21, have caused human illness and are recognized as EHEC, serotype O157:H7 is the most common and widely recognized EHEC serotype. *E. coli* are also categorized based on their serotype, defined by the O antigen determined by the polysaccharide portion of cell wall lipopolysaccharide (LPS) and the H antigen due to flagella protein. The following six STEC O serogroups (non-O157 STEC), in addition to O157, represent the greatest public health risk: O26, O111, O102, O121, O145, and O45 (Feng and Monday 2006). Besides, many of the virulence genes carried by pathogenic *E. coli* exist in mobile genetic elements and can be transferred. As an example, serotype O104:H4 which have been known to cause persistent diarrhoea in under-developed countries, but rarely have been associated with major food-borne incidents, caused a large outbreak in Germany in 2011 by producing Shiga toxin, a characteristic of EHEC, although it was genetically EAEC. Thus, the O104:H4 strain that caused this outbreak appeared to be an EAEC strain that had acquired the ability to produce Shiga toxin (FDA 2012).

Enteroaggregative *E. coli* (EAggEC)

EAggEC, which was formerly categorised as EPEC, differs from this group due to their aggregative adherence on Hep 2 cells and produce a heat-stable enterotoxin called EAST 1. EAggEC causes chronic diarrhoea in children (Duffy 2006).

Enterohaemorrhagic *E. coli* (EHEC)

The symptoms of infection from this group of organisms includes severe abdominal cramps, watery diarrhoea which may develop into bloody diarrhoea (haemorrhagic colitis-HC), HUS, and TTP that may result in a fatality rate of approximately 5 % in patients. HC lasts 4–10 days. HUS is characterized by the triad of haemolytic anaemia, thrombocytopaenia, and renal insufficiency and mostly affects children under 5 years of age. TTP is a serious condition describing the presentation with HUS, fever and neurologic symptoms. The infective dose for O157:H7 is estimated to be 10–100 cells (Duffy 2006; Adams and Moss 2008; Scannell 2011). Although O157:H7 is currently the predominant strain and accounts for approximately 75 % of the EHEC infections in the world, other non-O157 STEC are also emerging as a cause of food-borne illnesses (FDA 2012).

Many cases of O157:H7 infection have been related to consumption of raw or under-cooked ground beef and beef products and raw milk. O157:H7 can develop acid tolerance and so also isolated from yogurt, mayonnaise, fermented sausages, cheeses, and unpasteurized fruit juices. Various water sources, including potable, well, and recreational water, have also caused O157:H7 infections. The other food sources are salad vegetables (especially lettuce, spinach, and alfalfa sprouts contaminated with faeces and manure) (Duffy 2006; Adams and Moss 2008).

The most important control way is to avoid contamination of carcasses during slaughtering. Besides, treating of animal waste applied to salad vegetables, adopting a hazard analysis and critical control point (HACCP) approach in food manufacturing, processing, and service sectors; preventing cross-contamination from raw to cooked meat; not drinking raw milk; and cooking minced or ground meat thoroughly can also prevent and control the illness caused by this pathogen (Adams and Moss 2008; FDA 2012; Lund 2008; WHO 1997).

Enteroinvasive *E. coli* (EIEC)

Infection with EIEC results in an illness with the classical symptoms of invasive bacillary dysentery similar to that caused by *Shigella* species. Illness caused by EIEC usually occurs 24 h after ingestion and symptoms are fever, severe abdominal pains, malaise, and often a watery diarrhoea, followed by the passage of bloody stools (Ewing and Gravatti 1947; Duffy 2006; Adams and Moss 2008).

Enteropathogenic *E. coli* (EPEC)

While EPEC was a frequent causative agent of infantile diarrhoea in the USA in the 1940s and 1950s, its infections are less important in developed countries, but continue to be a major cause of diarrhoea in developing countries, especially in infants nowadays (FDA 2012). Symptoms generally appear about 12–36 h after ingestion and include vomiting, malaise, and diarrhoea with stool containing mucus, but rarely blood (Adams and Moss 2008). The major O groups within this group which are linked to human illness include O55, O86, O111, O119, O126, O127,

O128, and O142. Food and water are the vectors of this pathogen and cases are seen mostly in under-developed countries where there is inadequate sanitation and poor water quality (Duffy 2006).

Enterotoxigenic *E. coli* (ETEC)

ETEC, which is also a common cause of infantile diarrhoea and serious dehydration in developing countries, is best known as the causative agent of travellers' diarrhoea. It produces both heat-labile (LT) and heat-stable (ST) toxins and has several colonization-factor antigens (FDA 2012). Illness caused by ETEC usually occurs between 12 and 36 h after ingestion and symptoms can range from mild diarrhoea to a severe cholera-like illness with diarrhoea characterised by watery stools accompanied by high fever or vomiting and severe stomach pain. Serotypes which cause illness in humans include O6, O8, O15, O25, O78, O148, O159, and O167 (Duffy 2006; Adams and Moss 2008; FDA 2012). Foods implicated include turkey, mayonnaise, imported French cheese, and salad vegetables. Besides, food handlers with diarrhoea are also the vehicles for this illness (Lund 2008).

2.1.4 *Listeria monocytogenes*

L. monocytogenes, which was firstly described as *Bacterium monocytogenes* due to its causal of mononuclear leucocytosis in rabbits by Murray in 1926 (Murray et al. 1926), has been known as the causative agent of meningoencephalitis in sheeps and cattles, stillbirth, abortion, and septicaemia in monogastrics and young ruminants (Adams and Moss 2008). The role of *L. monocytogenes* as a causative agent in food-borne disease had not been confirmed until the large-scale outbreak involving Mexican-style soft cheese (Linnan et al. 1988).

The genus *Listeria which was formerly in the family of* Corynebacteriaceae *and has been in the family of Listeriaceae* since 2001, includes eight recognized species which are *L. monocytogenes*, *L. innocua*, *L. welshimeri*, *L. seeligeri*, *L. ivanovii*, *L. grayi*, *L. Marthii*, and *L. rocourtiae*, the latter two were described in 2009. However, *L. monocytogenes* which is closely related to *L. innocua* and *L. marthii*, and *L. ivanovii* which is closely related to *L. seeligeri* have been identified as the pathogens of warm-blooded hosts (den Bakker et al. 2010). *L. monocytogenes* has 13 serotypes, including 1/2a, 1/2b, 1/2c, 3a, 3b, 3c, 4a, 4ab, 4b, 4c, 4d, 4e, and 7 in which serotypes 1/2a, 1/2b, and 4b have been associated with the majority of food-borne infections (FDA 2012).

L. monocytogenes is a Gram-positive, facultatively anaerobic, catalase-positive, oxidase-negative, non-spore forming, motile rod that can grow at temperatures ranging from 0 to 45 °C with an optimum between 30 and 35 °C (mesophilic) and a pH range from 6.0 to 9.0, or in nutrient broth with up to 10 % (w/v) NaCl. It is an acid-tolerant pathogen owing to its glutamate decarboxylase (GAD) enzyme system. It cannot ferment xylose but ferments rhamnose and glucose but the latter one

without producing any gas. It is sensitive to pasteurization temperature (Adams and Moss 2008; Ray and Bhunia 2008; FDA 2012; Wang and Orsi 2013).

Listeriosis is a generic term for a variety of syndromes caused by infection with *Listeria* spp. *L. monocytogenes* causes two types of diseases: invasive listeriosis, which occurs predominantly in the elderly, the pregnant women, the neonates, the cancer patient, and the immunocompromised people, has an infectious dose in the range of 10^2–10^4 cells and an incubation period ranging from 24 h to 91 days. However, more severe form of this type of listeriosis has a very long incubation period (e.g. 3 days to 3 months) ending with septicaemia, meningitis, encephalitis, endocarditis, liver abscess, foetal loss, and death with a fatality rate of 20–30 %. On the other hand, febrile gastroenteritis, which is mostly associated with healthy individuals, has an infectious dose in the range of 10^8–10^{10} cells and a relatively short incubation period (e.g. a few hours to 2 or 3 days) and its mild flu-like symptoms include slight fever, nausea, vomiting, and diarrhoea (Farber and Peterkin 2000; WHO/FDA 2004; Ray and Bhunia 2008; FDA 2012; Wang and Orsi 2013).

L. monocytogenes is widespread in the environment and so can be found in soil, water, silage, sewage, slaughterhouse waste, milk of both normal and mastitic cows, human and animal faeces, and foods. Almost all (99 %) of human listeriosis are food-borne (Scallan et al. 2011). Although food-borne listeriosis in humans is an opportunistic rare disease and is mostly sporadic, outbreaks were reported from the consumption of raw milk, inadequately pasteurized milk and dairy products (e.g. ice cream), soft cheeses, meat pate, turkey franks, cold cut meats, improperly cooked chicken, contaminated coleslaw, and smoked mussels. Besides, *L. monocytogenes* outbreaks are mainly associated with ready-to-eat (RTE) foods. It is quite resistant to curing ingredients and so can be found in delicatessen meats such as salami, ham, and corned beef. The infective dose of *L. monocytogenes* is estimated higher than 10^3 CFU/g food, but is believed to vary with the strain and the susceptibility of the host, and the food matrix (WHO/FDA 2004; Adams and Moss 2008; Lund 2008; Ray and Bhunia 2008; FDA 2012; Wang and Orsi 2013).

The ability of *L. monocytogenes* to colonize on food processing niches which acts for an important source of contamination in the finished food products and the ability to grow in many foods at refrigerated temperature due to its resistance to the freezing effect which helps the organism to reach to a level of infective dose during storage of refrigerated foods, make this organism an important problem for the food industry. On the other hand, it is inactivated in meats at 70 °C for 2 min, giving an estimated 7 log inactivation (Farber and Peterkin 2000). Since the majority of listeriosis result from the consumption of contaminated food, foods especially the RTE ones, are in the high risk food group. Because they can become contaminated after cooking and before packaging, or during fermentation and ripening of certain cheeses, in other words before the RTE food is eaten (Tompkin 2002). Therefore the most effective control way of this pathogen is to eliminate the post-processing contamination (PPC) by using lethal or post-lethality treatments and/or growth inhibitors, to prevent in-plant contamination by performing good hygiene and manufacturing practices, testing and sanitation of food contact surfaces, and pre- and post-packaging interventions, and to control measures in the processing facilities (e.g. storage temperatures and periods) (Wang and Orsi 2013).

2.1.5 *Salmonella* spp.

The genus *Salmonella*, which was created in 1900 by Ligniéres and named in honour of the American veterinary pathologist Dr. Salmon, belongs to the family of Enterobacteriaceae. It contains a single species, *S. enterica* (earlier named as *S. choleraesuis*), which consists of seven subspecies. However, subspecies I (*S. enterica* subsp. *enterica*), which accounts for more than 59 % of the 2,400 serovars known, is responsible for nearly all infections in humans and warm-blooded animals, while six other subspecies are isolated principally from cold-blooded animals. The taxonomic nomenclature of this genus is relatively different from that of other genera. Until 2005, most of the different serovars were named as if they were different species. However now, for the taxonomic integrity, the non-italicized serovar name is used after the species name so that *S. enteritidis* becomes *S. enterica* subsp. *enterica* ser. Enteritidis or, shortly, *Salmonella* Enteritidis. For the other subspecies, the serovar formula is used after the name of the subspecies, e.g. *S. fremantle* would be *S. enterica* subsp. *salamae* ser.42;g, t:- (Adams and Moss 2008; Desai et al. 2013).

Salmonellas are Gram-negative, facultatively anaerobic, catalase-positive, oxidase-negative, urease-negative, H_2S-positive, non-spore forming rods, and are generally motile with peritrichous flagella that can grow at temperatures ranging from 5 to 47 °C with an optimum at 37 °C (mesophilic). They are heat sensitive and so are killed by pasteurization process. The minimum pH for their growth changes with the acidulant from 5.4 with acetic acid to 4.05 with hydrochloric and citric acids. However, the optimum pH for their growth is 7.0 (Adams and Moss 2008).

Salmonellosis, which is described as a zoonotic infection, is the second ranking food-borne disease in humans after campylobacteriosis in most European countries. The most common diseases caused by *Salmonella* in human are enteritis, septicaemia, and abortion. The symptoms of *Salmonella* infection usually appear 12–72 h after infection and include fever, abdominal pain, cramps, chills, diarrhoea, nausea, and sometimes vomiting. The illness usually lasts 4–7 days, and most people recover without treatment. Although most infections cause mild to moderate self-limited gastroenteritis, serious infections, especially the ones in the very young and the elderly, and in cases when the bacteria enter the bloodstream, leading to death do occur. The mortality rate for the most of the serovars is usually low (<1 %) but it is 20–30 % for the host-adapted serovars such as *S. Dublin* and *S. Choleraesuis* (Clarke and Gyles 1993; Mølbak et al. 2006; Adams and Moss 2008; Hald 2013).

Salmonella species enjoy widespread occurrence in the environments, including a wide range of domestic and wild animals and a variety of foodstuffs of both animal and plant origin and cause salmonellosis in humans and animals. Transmission is by the faecal–oral route whereby the intestinal contents from an infected animal are ingested with the foodstuff or water. Human infections also result from eating raw or under-cooked foods, including meats, poultry and poultry products, eggs, and dairy products (Adams and Moss 2008; Hald 2013).

In spite of the efforts to prevent and control food-borne salmonellosis during the last decades, it continues to be one of the leading causes of human gastroenteritis. Preharvest food safety is the most important component of an effective prevention

and control strategy for *S.* Enteritidis infection. HACCP plans, disinfection protocols, and preventing the recycling of offal and inedible raw materials into animal feeds may reduce direct horizontal transmission of *Salmonella* spp. within and between herds and flocks. In the EU, the Zoonosis Directive (92/117/EEC) starts an EU-wide control effort against *Salmonella*, especially in broiler and layer breeders (Hald 2013).

2.1.6 *Vibrio parahaemolyticus* and *Vibrio vulnificus*

Among the vibrios, *V. parahaemolyticus, V. vulnificus*, and *Vibrio cholerae* which belong to the family of Vibrionaceae, are responsible for most cases of the water-borne and food-borne illnesses (Oliver and Kaper 2001; Morris 2003, Nair et al. 2006; Ray and Bhunia 2008; Wright and Harwood 2013). The first two will be discussed in this section and the last one will be included in Sect. 2.2.

V. parahaemolyticus which was firstly described as the cause of an outbreak of food-borne illness in Japan in 1950 (Lund 2008), is a Gram-negative, facultatively anaerobic, catalase-positive, oxidase-positive, non-spore forming, motile curved rod that can grow at temperatures ranging from 5 to 42 °C with an optimum between 30 and 37 °C (mesophilic), or in media with 3–5 % (w/v) NaCl (halophilic). However, it cannot tolerate 10 % (w/v) NaCl. It is slowly inactivated at temperatures below 10 °C and so the cultures should never be stored in refrigerators. It hardly grows at pH 5.0 or below. It ferments glucose without producing gas, but cannot ferment lactose and sucrose. It is highly sensitive to pasteurization temperature, low pH, drying, and freezing. All *V. parahaemolytic* strains are not pathogenic; the pathogenic ones are haemolytic which can be detected on human blood agar plates, known as the Kanagawa positive (KP) since they produce thermal stable direct hemolysin (TDH) or TDH-related hemolysin (TRH). However, although the most environmental isolates do not produce TDH or TRH, they have also been associated with food-borne outbreaks. On the other hand; *V. vulnificus* is a lactose-positive and salicin-positive and a highly invasive bacterium that produces a haemolytic cytotoxin (Nair et al. 2006; Ray and Bhunia 2008; FDA 2012; Wright and Harwood 2013).

The infective dose of *V. parahaemolyticus* and *V. vulnificus* given in the literature varies from 10^5 to 10^7 organisms due to the type of contaminated food consumed and the general health of the exposed person. The most common symptoms of *V. parahaemolyticus* and *V. vulnificus* gastroenteritis are abdominal pain, nausea, vomiting, and rapid onset non-bloody diarrhoea, fever, chills which are much milder symptoms than of *V. cholera* and so recovery without treatment normally occurs after several days without any long-term consequences. However; *V. vulnificus* can infect the bloodstream, causing a severe and life-threatening illness characterized by decreased blood pressure (septic shock) in immunocompromised persons. In addition to these symptoms, septicaemia and severe necrotizing infections of soft tissues that typically result from exposure of open wounds to water harbouring *V. vulnificus*, occur with a very high fatality rate (40–60 %) (Ray and Bhunia 2008; Jones and Oliver 2009; Wright and Harwood 2013).

V. parahaemolyticus and *V. vulnificus* are ubiquitous in the natural flora of coastal marine, in estuarine and freshwater environments, especially being present in the highest numbers during the summer months when water gets warmer and have been isolated from water, sediments, and various seafood such as shrimps, squid, octopus, fish, crabs, oysters, lobsters, and clams (Ray and Bhunia 2008; Jones and Oliver 2009, FDA 2012). *V. parahaemolyticus* and *V. vulnificus* gastroenteritis is commonly attributed to the consumption of raw, under-cooked, or post-heat-contaminated seafood (primarily raw oysters) (Jones and Oliver 2009; Lipp et al. 2002; Morris 2011).

The most important control way is to harvest seafood only from approved waters free from faecal contamination and to chill promptly. In addition to this; avoiding exposure of vegetables to contaminated irrigation water, cooking seafood thoroughly before serving, applying proper refrigeration to raw and heated products, avoiding cross-contamination of cooked seafood and other foods with raw seafood, applying employee health, and hand washing policy are necessary for prevention and control of this bacteria (Lund 2008; FDA 2012).

2.1.7 *Yersinia* spp.

The genus *Yersinia*, which was firstly described as the causative agent of the bubonic plague by the French bacteriologist Alexandre Yersin in 1894, killed an estimated 25 % of the European population in the fourteenth century (Adams and Moss 2008). Among the 11 species within the genus, four are pathogenic but only *Y. enterocolitica* and *Y. pseudotuberculosis* are food-borne diarrhoeagens. Although *Y. pestis* is closely related with *Y. pseudotuberculosis* with a gene homology of almost 97 %, it infects humans by routes aside from food and causes plague (Scannell 2011; FDA 2012; Nesbakken 2013). *Y. enterocolitica* strains have been differentiated into biotypes and serotypes. Most of the pathogenic strains are included in Biotype 1B (O:4, 32; O:8; O:13a; O:13b; O:18; O:21), Biotype 2 (O:5,27; O:9), Biotype 3 (O:1, 2, 3; O:3; O:5,27), Biotype 4 (O:3), and Biotype 5 (O:2,3); however, among them Biotype 4/serotype O:3 is the most frequently isolated pathogen in the world, especially in Europe, Canada, Japan, and South Africa. In the USA, Biotype 1/serotype O:8 most commonly causes human yersiniosis. On the other hand; *Y. pseudotuberculosis* is divided into five serotypes (I to V) among which Serotype I is the most common serotype associated with human and animal infections in Europe (Robins-Browne 2001; Lund 2008; Nesbakken 2013).

Y. enterocolitica, which is a member of the family Enterobacteriaceae, is a Gram-negative, facultatively anaerobic, catalase-positive, oxidase-negative, urease-positive, H_2S-negative, non-spore forming rod (occasionally coccoid) that can grow at temperatures ranging from −1 to +40 °C, with an optimum around 29 °C (psychrotrophic) and over a pH range of 4–10, with an optimum 7.0–8.0, or at NaCl concentrations greater than 7 % (w/v). It is non-motile at 35–37 °C, but motile with peritrichous flagella at 22–25 °C. It ferments mannitol and glucose but the latter one without producing any gas. It multiplies more rapidly at 0–5 °C than any other food-borne pathogenic bacterium and can grow in a range of foods at

refrigeration temperature. It can tolerate freezing and so can survive in frozen foods for extended time. It is sensitive to pasteurization (Bercovier and Mollaret 1984; Adams and Moss 2008; FDA 2012; Nesbakken 2013).

Yersiniosis, which is a generic term for gastroenteritis and other syndromes caused by infection with *Y. enterocolitica*, occurs most commonly in children less than 14 years of age, especially infants under 1 year being at greatest risk. It has an incubation period of 4–7 days and may last for 1–3 weeks or for several months. The symptoms of gastroenteritis which dominates in children and young people are low-grade fever, abdominal pain, watery or mucoid diarrhoea, bloody stools (rarely), and acute lower right quadrant abdominal pain that may be mistaken diagnosis of appendicitis. However; various forms of reactive arthritis and erythema nodosum (red skin lesion) are most common in adults, especially in adult females (Robins-Browne 2001; Sutherland and Varnam 2002; Adams and Moss 2008; FDA 2012).

Pigs are the main reservoir and chronic carriers for the human pathogenic types of *Y. enterocolitica*, however, pathogenic serotypes have also been found in cattle, sheep, deer, rodents, cats, and dogs (Adams and Moss 2008; Nesbakken 2013). *Y. enterocolitica* strains can be found in pork, beef, and lamb meats, oysters, fish, crabs, raw milk, and contaminated water. However, poor sanitation and improper sterilization techniques by food handlers, including improper storage offer many opportunities for *Yersinia* to enter the food supply (FDA 2012).

Most of the preventive and control measures against salmonellosis are also valid against this zoonotic infection, *yersiniosis.* Cooking meat products, particularly pork, poultry, and seafood thoroughly before serving; avoiding drinking unpasteurized milk, avoiding exposure of vegetables to contaminated irrigation water; following good hygienic practices; proper sanitation of food contact surfaces (avoiding cross-contamination); applying employee health and hand washing policy are necessary for prevention and control of this bacteria (Lund 2008).

2.2 *Bacterial Intoxications and Toxico-Infections*

2.2.1 *Bacillus cereus*

The taxonomy of the genus *Bacillus*, which is in the family of Bacillaceae, has undergone considerable revision in recent years; however it still contains about 80 species, including *B. cereus* which is best known as one of the causative agent of food-borne microbial diseases (Jay et al. 2005; Adams and Moss 2008).

B. cereus was first recognized as a food-borne pathogen in 1949 (Hauge 1950) and then in 1955 (Hauge 1955). It is a Gram-positive, facultatively anaerobic, catalase-positive, and endospore-forming rod that grows over a temperature range from 8 to 55 °C, optimally around 28–35 °C (mesophilic) and a pH range from 4.5 to 9.5, optimally around 6.0–7.0 (Ehling-Schulz et al. 2004; Adams and Moss 2008; Lund 2008; FDA 2012).

B. cereus is responsible for two different types of food-borne illnesses which are relatively late-onset toxico-infection, "diarrhoeal syndrome" and rapid-onset intoxication, "emetic syndrome (vomiting illness)". The symptoms of diarrhoeal syndrome which resemble those of *Clostridium perfringens* food poisoning, are profuse watery diarrhoea, abdominal cramps, and pain that last for 12–24 h. The emetic syndrome which resembles the illness caused by *Staphylococcus aureus*, is characterized by nausea, malaise, and vomiting that subside within 6–24 h (Kramer and Gilbert 1989; Granum and Baird-Parker 2000; Kotiranta et al. 2000; Adams and Moss 2008; Lund 2008).

B. cereus, like many other bacilli, is common in soil and as a result of its spore-forming ability, it is distributed in the environment thus can easily contaminate foodstuffs, especially the ones with plant origin. Since spores of *B. cereus* are resistant to drying and heat treatment, a wide variety of foods, including spaghetti, pasta, rice, dairy and dried milk products, spices, and other dried foodstuffs as well as meat, chicken, vegetables, fruits, grain, and seafood allow the survival of *B. cereus* cells and their spores (Kamat et al. 1989; FDA 2012). Also the emetic toxins stay active after a heat treatment of 100 °C for 150 min (Ehling-Schulz et al. 2004). The diarrhoeal syndrome is mainly associated with the consumption of milk products, vegetables, fish, and meats, while the emetic syndrome is transmitted by rice and pasta (FDA 2012). Most *B. cereus* food poisoning cases are related to cereal-based or protein-based foods, slowly cooled and stored between 10 and 50 °C which allows the spores to germinate and cause the illness (Adams and Moss 2008).

Cleaning and disinfection of food manufacturing and production equipments and devices are important for preventing a build-up of *B. cereus* cells and their spores. On the other hand, most heating and cooking treatments as well as, steaming under pressure, frying, grilling, and roasting, generally kill the vegetative cells, and probably the spores (ICMSF 1996). However, only canning can guarantee complete destruction of *B. cereus* spores. In addition to these, cooked foods should be consumed promptly, or kept above 63 °C for a short time, or cooled rapidly and kept below the temperatures of about 7–8 °C (ideally below 4 °C). Dried milk products and similar powdered foods should be used promptly after reconstitution and holding them at room temperature for several hours should be avoided (EFSA 2005).

2.2.2 *Clostridium botulinum*

Thirteen people became ill and six of them later died after consuming a type of sausage called Blunzen that was made by packing blood and other ingredients into a pig's stomach in Wildbad in 1793. However, the causative agent of this illness which was later named botulism (derived from the Latin word; *botulus*: sausage), was primarily isolated in 1897 by Dr. Van Ermengem, Professor of Bacteriology at the University of Ghent. He found that botulism resulted from the consumption of food containing a heat-labile toxin produced by an obligate anaerobic, spore-forming bacillus firstly named *Bacillus botulinum* (Adams and Moss 2008).

C. botulinum which is a member of the family Bacillaceae, is a Gram-positive, obligate anaerobic, catalase-negative, spore-forming straight or slightly curved rod that produces central or subterminal oval spores and is motile with peritrichous flagella. The vegetative cells are sensitive to low pH (<4.6), low A_w (0.93), and quite high salt concentrations (5.5 %) and can be killed by pasteurization, whereas their spores are highly heat resistant and thus can only be killed at the temperatures above 115 °C (Adams and Moss 2008; Ray and Bhunia 2008).

C. botulinum strains have been divided into seven types which are A, B, C, D, E, F, and G according to the type of toxin that they produce. However, only the types A, B, E, and F (rarely) strains are associated with human food-borne intoxications since types C and D strains cause botulism in animals and no outbreaks of type G strain have been reported till now. Types C and E (psychrotrophic) strains also cause botulism in birds. Type A strains are proteolytic and can grow between 10 and 48 °C, with the optimum at 35 °C (mesophilic). Type E strains are non-proteolytic and grow optimally at 30 °C (mesophilic), with a range between 3.3 and 45 °C. However, types B and F strains can be either proteolytic or non-proteolytic. Actually type C is recognized as C_1 and C_2 toxin (though C_2 is not a neurotoxin) (Adams and Moss 2008; Ray and Bhunia 2008; Hill et al. 2009; FDA 2012).

There are 5 types of botulism which are food-borne botulism, infant or infectious botulism, hidden botulism, wound botulism, and inadvertent botulism. Food-borne botulism which results from the ingestion of an exotoxin produced by *C. botulinum* growing in the food, usually occurs within 12–36 h of ingestion of toxin, but may occur within 6 h or after 10 days. The first effect of the toxin is often on neuromuscular junctions in the head and neck, causing symptoms such as double vision, failure in focusing, drooping eyelids (ptosis), dry mouth, failure in speaking clearly (dysphonia), and difficulty in swallowing (dysphagia) accompanying with some gastrointestinal disorders (e.g. nausea, vomiting, diarrhoea, constipation, and urine retention). Subsequently, paralysis occurs in the muscles of arms, legs, trunk, and heart which results in death. The mortality rate is high (20–50 %), but will depend on the type of toxin (type A usually produces a higher mortality than B or E), the amount of ingested toxin, the type of food and the speed of treatment (Adams and Moss 2008; Lund 2008; Ray and Bhunia 2008). Infant botulism has been caused by the ingestion of spores of *C. botulinum* that colonize and form neurotoxin in the intestinal tracts of infants, especially babies under the age of 1 year (i.e. intestinal toxaemia botulism) (Lund 2008; Glass and Marshall 2013). Constipation, lethargy, inability to suck, weak cry, and difficulty in swallowing are the initial symptoms continuing with paralysis. Honey and corn syrup have been associated with infant botulism cases (Lund 2008; Ray and Bhunia 2008). Hidden botulism is seen in adults suffering from chronic gastrointestinal disorders (i.e. adult variant of infant botulism) (Ray and Bhunia 2008). Wound botulism which is caused by a subcutaneous infection with *C. botulinum*, has been more commonly associated with accidental cut and intravenous drug usage (Adams and Moss 2008; Ray and Bhunia 2008; Glass and Marshall 2013) whereas inadvertent botulism has been associated with therapeutic or cosmetic usage of botulism neurotoxins (Ray and Bhunia 2008).

Almost any type of food that is not very acidic (pH>4.6) can support growth and toxin production of *C. botulinum* (FDA 2012). In other words, both the vegetative cells and the spores can be isolated from a wide range of foods, including fish, meat, vegetables (e.g. green beans, corn, spinach, asparagus, pepper, and mushrooms), fruits (e.g. figs and peaches), honey, mushrooms, cheese, and nuts. The main reason of outbreaks is improper home canning of the contaminated foodstuffs (Adams and Moss 2008; Ray and Bhunia 2008; Glass and Marshall 2013).

C. botulinum spores usually enter the food chain via direct contamination of the animal or plant during production or via cross-contamination during harvest, handling, or manufacturing and processing. Foods that allow the growth of *C. botulinum* should either undergo a processing that will inactivate the spores, or that the composition and storage conditions are controlled so as to prevent growth. The most important control way is to use proper temperature and time in home canning of low-acid products Cooked foods in which spores may survive should be stored at low temperatures (≤3 °C) Prepared ready meals, vacuum-packed vegetables, pasteurized, chilled foods and herbs/spices/vegetables stored in oil, should be controlled. Honey should not be given to babies under the age of 1 year, or to adults with recent abdominal surgery or gastrointestinal abnormalities (Lund 2008; Glass and Marshall 2013).

2.2.3 *Clostridium perfringens*

C. perfringens (earlier named as *C. welchii*), which was firstly described by the American bacteriologist Welch in 1892, has been known as the causative agent of gas gangrene (Adams and Moss 2008). The role of *C. perfringens* as a causative agent in food-borne disease had not been confirmed until the large-scale outbreak of a food poisoning among schoolchildren in Leicester, England, due to the consumption of gravy contaminated by *C. perfringens* (Knox and MacDonald 1943).

C. perfringens which is a member of the family Bacillaceae, is a Gram-positive, anaerobic (but can tolerate some oxygen), catalase-negative, non-motile, blunt-ended, and oval subterminal spore-forming rod that can be characterized by the reduction of nitrate, the liquefaction of gelatin, the production of lecithinase (α-toxin), and the fermentation of lactose. It is intolerant to low temperatures and thus cannot grow below the temperature of 12 °C. It grows optimally around 43–45 °C (thermoduric) and continues growing up to 50 °C. It grows at a pH range from 5.0 to 9.0, optimally around 6.0–7.0, but cannot grow at pH<5.0 and also in the presence of 6 % (w/v) NaCl. The vegetative cells can be killed by pasteurization, whereas their spores are extremely heat resistant, and may survive even boiling for several hours (de Jong et al. 2004; Adams and Moss 2008; Lund 2008; Ray and Bhunia 2008; Labbé and Juneja 2013).

C. perfringens strains have been classified into 5 types which are A, B, C, D and E according to the production of four major lethal enterotoxins, α, β, ε, and ι. However, Type A strains are mainly associated with food borne toxico-infections and gas gangrene and produce only the α-toxin which has lecithinase (phospholipase

C) activity. Type B strains produce α, β, and ε toxins, whereas Type C strains produce α and β toxins which cause more severe, but more rare enteric disease, necrotic enteritis (NE). Type D strains produce α and ε toxins and Type E strains produce α and ι toxins (de Jong et al. 2004; Adams and Moss 2008; Ray and Bhunia 2008).

Because of its almost ubiquitous distribution in the environment, including the intestinal tract of animals, soils, and retail foods, the common form of poisoning is *C. perfringens* Type A poisoning (CPTA) (Heikinheimo et al. 2006). The vegetative cells survive in the acidity of stomach while passing through it and then enter into the small intestine where they multiply, sporulate, and release their enterotoxins. The enterotoxin which is synthesized almost by the sporulating cells causes only gastroenteritis. Diarrhoea and lower abdominal cramps typically occur usually 8–24 h after the consumption of food containing large numbers of the vegetative cells of the organism (or 10^6 spores/g foods). Nausea, vomiting, and fever are less common. Mortality occurs mostly in elderly people as a result of dehydration. Symptoms lessen within 1–2 days, but cramps can continue a little longer (Ray and Bhunia 2008; FDA 2012; Labbé and Juneja 2013).

High-protein foods such as meat (cooked beef which is prepared with its gravy, meat pies, sauces, roasts, casseroles) and poultry are the most related vehicles involved in *C. perfringens* food-borne toxico-infection. In addition to this, temperature-abused meat and poultry products (i.e. inadequate holding temperature and cooking, slow cooling, or prolonged storage at room temperature after cooking) allow the spores to germinate and multiply rapidly (Kalinowski et al. 2003; Adams and Moss 2008; Ray and Bhunia 2008; Labbé and Juneja 2013).

The major factors in prevention of *C. perfringens* toxico-infection are rapid cooling, refrigerated storage of cooked foods, adequate reheating of cooked and cooled foods which inhibit germination of surviving spores and growth of vegetative bacteria. In other words, cooked foods should be eaten immediately or kept above 63 °C for a short time, or be cooled rapidly and uniformly and maintained below 7–8 °C (ideally below 4°) and reheated to at least 72 °C before consumption (Lund 2008).

2.2.4 *Staphylococcus aureus*

The genus *Staphylococcus*, which belongs to the family of Staphylococcaceae, includes over 40 species (Jay et al. 2005). Among them, *S. aureus* is the most important member of this genus since it is an enterotoxin-producing species. However; *S. intermedius* and *S. hyicus* have also been reported with the enterotoxin production (Adams and Moss 2008; Landgraf and Destro 2013).

The Staphylococci (derived from the Greek word: *staphyle* : bunch of grapes and *coccus*: a grain or berry) (Adams and Moss 2008) were firstly described by the Scottish surgeon, Sir Alexander Ogston during his observation of pyogenic abscesses in humans (Ogston 1882). In 1884, while the investigation of an outbreak in Michigan by Vaughan and Sternberg, the staphylococci had been found as the causative agents of a food poisoning regarding the consumption of cheddar cheese (Dack 1956).

In 1914, Barber found out that a toxin produced by staphylococci was responsible for staphylococcal food poisoning (SFP) (i.e. intoxication) (Bhunia 2008).

S. aureus is a Gram-positive, facultatively anaerobic, catalase-positive, oxidase-negative, non-spore forming, non-motile coccus that occurs in pairs, tetrads, short chains, or bunched in grape-like clusters and can grow at temperatures ranging from 7 to 48 °C with the optimum at 37 °C (mesophilic) and a pH range from 4.0 to 9.8–10.0 with the optimum 6.0–7.0. The optimum temperature for enterotoxin production is between 35 and 40 °C. It is one of the most salt-tolerant pathogenic microorganisms in foods, grows easily in media containing 5–7 % (w/v) NaCl and some strains can grow in media with up to 20 % (w/v) NaCl. Optimum growth of *S. aureus* occurs at A_w of >0.99. However, it can grow at a A_w as low as 0.83, depending on the temperature, pH, type of humectants, and other parameters. The minimum A_w for enterotoxin production is 0.86. It ferments glucose which is used for distinguishing it from the strictly aerobic genus *Micrococcus* (Bannerman and Peacock 2007; Adams and Moss 2008; Schelin et al. 2011; Landgraf and Destro 2013).

The genus *Staphylococcus* includes both coagulase-negative and coagulase-positive strains which can produce highly heat-stable enterotoxins [known as staphylococcal enterotoxins (SEs)] that cause gastroenteritis in humans. However, the bacterium itself can be destroyed by pasteurization. There are 21 different SEs or enterotoxin-like proteins (SE-*l*) described (from SEA to SEV, except SEF which was the original name of toxic shock syndrome toxinTSST-1), excluding molecular variants. The first five (SEA; SEB; SEC1,2,3; SED; and SEE) have emetic activity and are called classical SEs which cause 95 % of human SFP. Among them, SEA is the most often reported toxin involved in SFP followed by SEB, SEC, or SED, depending on the district of the world. There is a high correlation between the coagulase activity and SEs. In other words; the detection of coagulase activity is important in distinguishing *S. aureus*-related food-borne illness from other strains. However other species that are coagulase-negative can also produce SEs. SEs are also resistant to proteolytic enzymes, such as trypsin and pepsin, which allows them to pass through the digestive tract without any denaturation. In addition to SEs, *S. aureus* produces a variety of extracellular products such as adhesion proteins, coagulase, superantigens, thermostable nuclease (TNase), ADP-ribosylating toxins, and hemolysins, many of which play a role as virulence factor (Bhunia 2008; Ono et al. 2008; RůŽičkova et al. 2008; FDA 2012; Hennekinne et al. 2012; Landgraf and Destro 2013).

CDC estimates that SFP causes approximately 241,188 illnesses, 1,064 hospitalizations, and 6 deaths each year in the USA (FDA 2012). Nausea, stomach cramps, retching, vomiting, and diarrhoea, which are the most common symptoms of SFP, usually occur within 2–4 h of ingestion because of fast-acting ability of SEs, depending on the amount of toxin ingested and host-specific factors. Breathing of SEs may cause rapid onset of fever, chills, cough, and difficulty in breathing. However; headache, muscle cramping, dehydration, temporary changes in blood pressure, and prostration can also be experienced in more severe cases. Ingested bacteria do not produce toxin, and the symptoms therefore normally subside after 24 h. Besides, infections caused by methicillin-resistant (MRSA) and vancomycin-resistant strains may be fatal because of lack of suitable antibiotics. The intoxication dose of SE is

less than 1.0 μg which is barely reached when there are more than 10^5cells of *S. aureus* in 1 g of foodstuff (Scannell 2011; Schelin et al. 2011; FDA 2012).

S. aureus is natural inhabitant of human and animal skin so it occurs commonly on the skin, nostrils, and mucous membranes of warm-blooded animals. Staphylococcal contamination may be introduced into foods by direct contact such as through the hands of the workers, or indirectly such as through skin fragments and also by coughing and sneezing which is seen when there is a respiratory infection. Creamy food prepared with milk, custard (pudding), mashed potato made with raw milk, deli foods, salad dressings, meats, hams, fish, shellfish, raw milk, and cheeses made from unpasteurized milk are the most related foods involved in SFP. It is better to remind that the foodstuffs may not smell terrible or look spoiled in order to produce SEs (Bhunia 2008; Landgraf and Destro 2013).

The most important control way is to avoid time and temperature abuse of food products, inadequate refrigeration, inadequate heating or cooking, and preparation or serving of food by a worker who have wounds or skin infections on his hands or wrists (poor personal hygiene). Hygienic practices are crucial in preventing SFP (Bhunia 2008; Landgraf and Destro 2013).

2.2.5 *Vibrio cholerae*

V. cholerae which is a member of the family Vibrionaceae, was firstly isolated in pure culture by Robert Koch in 1883. It is classified on the basis of its somatic (O) antigens into serovars or serogroups, and there are at least 206 serogroups. *V. cholerae* serogroup O1 and O139 include all the strains responsible for epidemic and endemic cholera. Serogroup O1 has two major serotypes, Ogawa and Inaba, and rarely reported serotype Hikojima. These serotypes can be further divided into two biotypes named as "Classical" and "El Tor", based on antibiotic resistance and hemolysin expression. All strains that were identified as *V. cholerae* but cannot agglutinate with O1 antiserum belonged to the non-O1*V. cholerae* until 1993 in which a new bacterium was determined as the causative agent of the epidemic cholera-like disease in Bangladesh (8th great pandemic). This bacterium did not agglutinate with O1 antiserum and therefore did not belong to any of the O serogroups previously described for *V. cholerae* but to a new serogroup O139 (Ramamurthy et al. 1993; Shimada et al. 1993; Faruque et al. 1998; Maheshwari et al. 2011; Wright and Harwood 2013).

V. cholerae is a Gram-negative, facultatively anaerobic, catalase-positive, oxidase-positive, non-spore forming, motile curved rod that is capable of respiratory and fermentative metabolism (*V. cholerae* serogroup O1 ferment sucrose and mannose but not arabinose and they produce acid but not gas) and can grow at temperatures ranging from 5 to 42 °C with an optimum between 30 and 37 °C (mesophilic), or in media with 3–5 % (w/v) NaCl (halophilic). It tolerates alkaline media that kill most of the intestinal commensals, but it is sensitive to acid and so dies rapidly in solutions below pH 6. It is killed by pasteurization but can persist in raw milk as long as 4 weeks, even if refrigerated (Maheshwari et al. 2011).

Worldwide, between the year 1817 and the present time eight great pandemics caused by toxigenic *V. cholerae* have been recorded (Lund 2008). Water from public supplies was implicated in the first six pandemics. *V. cholerae* causes a water-borne disease called cholera that is characterized by a harmful watery diarrhoea which leads to rapid dehydration, and death occurs in 50–70 % of untreated people (Faruque et al. 1998). Infection due to *V. cholerae* begins with the ingestion of contaminated water or food contaminated directly or indirectly with faeces of infected individual. Infected people excrete between 10^7 and 10^8 cells of *V. cholerae* per gram of stool and total output of *V. cholerae* by a patient can be in the range of 10^{11}–10^{13} CFU (Nair et al. 2006). After its passage through the acid barrier of the stomach, *V. cholera* colonizes in the small intestine and produces an enterotoxin called cholera toxin (CT) which has been shown to have biological similarities with the *Escherichia coli* enterotoxin (LT). In addition to CT, several other toxins such as neuraminidase, disulphide isomerase, protease, haemolysin-cytolysin toxin, ZO toxin, accessory cholera enterotoxin, Shiga-like toxin, and thermostable direct hemolysin (TDH) can be produced by the *V. cholera* (Faruque et al. 1998; Maheshwari et al. 2011).

Likewise *V. parahaemolyticus* and *V. vulnificus*, *V. cholerae* is found in coastal waters, especially is widely distributed in temperate and tropical aquatic environments in association with a wide range of aquatic life, including cyanobacteria, diatoms, oysters, water hyacinths, and blue crab. Faecal–oral spread is the primary mode of cholera transmission. It can be isolated from areas where poor environmental sanitation is coupled with poor personal hygiene. Using polluted water for irrigation and inadequately treated sewage sludge make it spread to foods such as leafy green vegetables. However, cholera is commonly attributed to the consumption of faecally contaminated water followed by human-to-human transmission and can be rapidly fatal due to massive dehydration (Lipp et al. 2002; Ray and Bhunia 2008; Maheshwari et al. 2011; Morris 2011; FDA 2012; Wright and Harwood 2013).

The application of main sanitary principles and ensuring safe drinking-water would go a long way toward controlling the disease cholerae. A number of approaches to the control of *V. cholera* have been proposed which are generally similar to those proposed for the control of *V. parahaemolyticus* and *V. vulnificus*, such as avoiding exposure of vegetables to contaminated irrigation water, cooking seafood thoroughly before serving, applying proper refrigeration to raw and heated products, avoiding cross-contamination of cooked seafood and other foods with raw seafood, applying employee health and hand washing policy (Lund 2008; FDA 2012).

3 Toxigenic Fungi

Fungi are organisms made up of eukaryotic cells that have cell walls containing chitin and are heterotrophs that obtain their nutrients by extracellular digestion based on the activity of secreted enzymes, followed by absorption of the solubilized breakdown products, which all distinguish them from animals. They are not capable

of forming true tissues like complex plants and animals and they can reproduce both sexually and asexually (Moss 2000; Brock 2006; Webster and Weber 2007).

Fungi are ubiquitous in nature and there are probably over 1.5 million species of fungi in which about 80,000 to 120,000 species of fungi have been identified up to date (Hawksworth 2001; Brock 2006). They have colonized in a wide range of ecosystems and they have the ability to produce many extracellular chemicals that are known as secondary metabolites, in which some may be very useful for pharmaceutical usages, and the others are toxic and so called mycotoxins (Magan and Aldred 2007).

Mycotoxins which are produced by filamentous fungi (molds) and contaminate agricultural commodities pre- or postharvest and so foods and foodstuffs, pose important food safety risks and public health hazards, and result in economic losses by reducing the commercial value of crops and so limiting the marketability of grain supplies (Atanda et al. 2011; Gnonlonfin et al. 2013; Woloshuk and Shim 2013). Although hundreds of mycotoxins have been identified up to now, only few are known to impact global agriculture (Bennett and Klich 2003). Aflatoxins, ochratoxins, trichothecenes, zearelenone, fumonisins, tremorgenic toxins, and ergot alkaloids are the mycotoxins that have the greatest agro-economic importance (Atanda et al. 2011) (Table 1). The toxicity and acute and chronic disorders caused by these mycotoxins in humans and animals have been comprehensively documented many times before (Bennett and Klich 2003; Richard 2007).

Although a very diverse range of fungi can produce mycotoxins, some of them are the most carcinogenic compounds in nature and are toxic to vertebrates. Besides, there are three genera of mycotoxigenic moulds that are especially important in foods: *Aspergillus*, *Penicillium* and *Fusarium* (Moss 2000). Therefore, the following section will deal only with these major fungi and their characteristics.

3.1 Aspergillus *and Related Teleomorphs*

The genus *Aspergillus* and related 12 teleomorph genera (*Chaetosartorya*, *Dichotomomyces*, *Emericella*, *Eurotium*, *Fennellia*, *Neocarpenteles*, *Neopetromyces*, *Neosartorya*, *Penicilliopsis*, *Petromyces*, *Sclerocleista*, and *Warcupiella*) belong to the family Trichocomaceae of the class Euascomycetes in the phylum Acsomycota. Among the teleomorps, *Eurotium*, *Neosartorya*, and *Emericella* are significant in foods (Pitt and Hocking 2009; Samson and Varga 2010; Public Health Agency of Canada 2013). The genus *Aspergillus* includes eight subgenus (subgenus *Aspergillus* with the sections *Aspergillus* and *Restricti*; subgenus *Fumigati* with the sections *Fumigati*, *Clavati*, and *Cervini*; subgenus *Circumdati* with the sections *Circumdati*, *Nigri*, *Flavi*, and *Cremei*; subgenus *Candidi* with the section *Candidi*; subgenus *Terrei* with the sections *Terrei* and *Flavipedes*; *subgenus Nidulantes* with the sections *Nidulantes*, *Usti* and *Sparsi*; subgenus *Warcupi* with the sections *Warcupi* and *Zonati* and subgenus *Ornati* with the section *Ornati*), each containing several species (Samson and Varga 2010).

Table 1 Some of the mycotoxins and their producers [Adapted from Moss (2000), Frisvad et al. (2007)]

Mycotoxin name	Producer name
Aflatoxin	*Aspergillus bombycis, A. flavus, A. nomius, A. ochraceoroseus, A. parasiticus, A. parvisclerotigenus, A. pseudotamarii, A. rambellii, A. toxicarius, Emericella astellata, E. olivicola, E. venezuelensis*
Citreoviridin	*A. terreus, Eupenicillium cinnamopurpureum, Penicillium citreonigrum, P. manginii, P. miczynskii, P. smithii*
Citrinin	*A. terreus chemotype II, A. carneus, A. niveus, Blennoria sp., Clavariopsis aquatica, Monascus ruber, P. chrzaczszii, P. citrinum, P. expansum, P. manginii, P. odoratum, P. radicicola, P. verrucosum, P. westlingii*
Cyclopiazonic acid	*A. flavus, A. lentulus, A. oryzae, A. parvisclerotigenus, A. pseudotamarii, A. tamarii, P. camemberti, P. commune, P. dipodomyicola, P. griseofulvum, P. palitans*
Ergot alkaloids	*Claviceps fusiformis, C. paspali, C. purpurea*
Fumonisins	*Fusarium anthophilum, F. dlamini, F. napiforme, F. nygamai, F. proliferatum, F. thapsinum, F. verticillioides*
Moniliformin	*F. avenaceum, F. napiforme, F. nygamai, F. oxysporum, F. proliferatum, F. subglutinans, F. tricinctum, F. thapsinum, F. verticillioides*
Ochratoxin A	*A. carbonarius, A. cretensis, A. flocculosus, A. lacticoffeatus, A. niger, A. ochraceus, A. pseudoelegans, A. roseoglobulosum, A. sclerotioniger, A. sclerotiorum, A. steynii, A. sulphureus, A. westerdijkiae, Neopetromyces muricatus, P. nordicum, P. verrucosum, Petromyces albertensis, Petromyces alliaceus*
Patulin	*A. clavatonanica, A. clavatus, A. giganteus, A. longivesica, Byssochlamys nivea, P. carneum, P. clavigerum, P. concentricum, P. coprobium, P. dipodomyicola, P. expansum, P. formosanum, P. gladioli, P. glandicola, P. griseofulvum, P. marinum, P. paneum, P. sclerotigenum, P. vulpinum*
Penicillic acid	*A. auricomus, A. bridgeri, A. cretensis, A. flocculosus, A. insulicola, A. melleus, A. neobridgeri, A. ochraceus, A. ostianus, A. persii, A. petrakii, A. pseudoelegans, A. roseoglobulosus, A. sclerotiorum, A. sulphureus, A. westerdijkiae, Neopetromyces muricatus, P. aurantiogriseum, P. brasilianum, P. carneum, P. cyclopium, P. fennelliae, P. freii, P. matriti, P. polonicum, P. radicicola, P. tulipae, P. viridicatum*
Penitrem A	*P. clavigerum, P. crustosum, P. glandicola, P. janczewskii, P. melanoconidium, P. tulipae*
Sterigmatocystin	*A. multicolor, A. ochraceoroseus, A.rambellii, A. versicolor, Chaetomium thielavioideum, Chaetomium spp., Emericella nidulans, Emericella spp., Humicola fuscoatra, Monocillium nordinii*
Tenuazonic acid	*Alternaria citri, Alternaria japonica, Alternaria kikuchiana, Alternaria longipes, Alternaria mali, Alternaria oryzae, Alternaria solani, Alternaria tenuissima, Phoma sorghina*
Trichothecenes	*F. crookwellense, F. culmorum, F.equiseti, F. graminearum, F. langsethiae, F. poae, F. pseudograminearum, F. sambucinum, F. sporotrichioides, F. venenatum*
Zearalenone	*F. crookwellense, F. culmorum, F. equiseti, F. graminearum*

The genus *Aspergillus* is characterized by unbranched, aseptate conidiophores with usually swollen spherical vesicles which are covered with phialides, or metulae and phialides that are borne simultaneously. This character definitely distinguishes

the genus *Aspergillus* from the genus *Penicillium.* Compared to *Penicillium*, *Aspergillus* spp. have ability to grow at higher temperatures and/or lower water activities and usually grow more rapidly although they take longer to sporulate. In addition to these, their spores are more resistant to light and chemicals (Pitt and Hocking 2009). However, *Aspergillus* spp. are susceptible to sodium hypochlorite solutions. Although conidia of *A. fumigates (*the most pathogenic species) are generally heat-resistant, the conidia of *A. niger* and *A. flavus* are easily inactivated at 60 °C for 45 min (Public Health Agency of Canada 2013).

Aspergillus spp. contain approximately 184 species, 40 of which have been implicated in human or animal infections, a group of diseases termed as aspergillosis. Aspergillosis which is mostly caused by *A. fumigatus*, *A. Flavus*, and *A. niger*, include illnesses that usually affect the respiratory system: clinical allergies [allergic bronchopulmonary aspergillosis (ABPA), rhinitis, Farmers's lung], superficial and local infections (cutaneous infections, otomycosis, tracheobronchitis), damaged tissue infections (aspergilloma and osteomyelitis), and chronic invasive pulmonary infections (CPA) (Public Health Agency of Canada 2013). These illnesses are common among people who work in the farming industry, and are considered an occupational hazard.

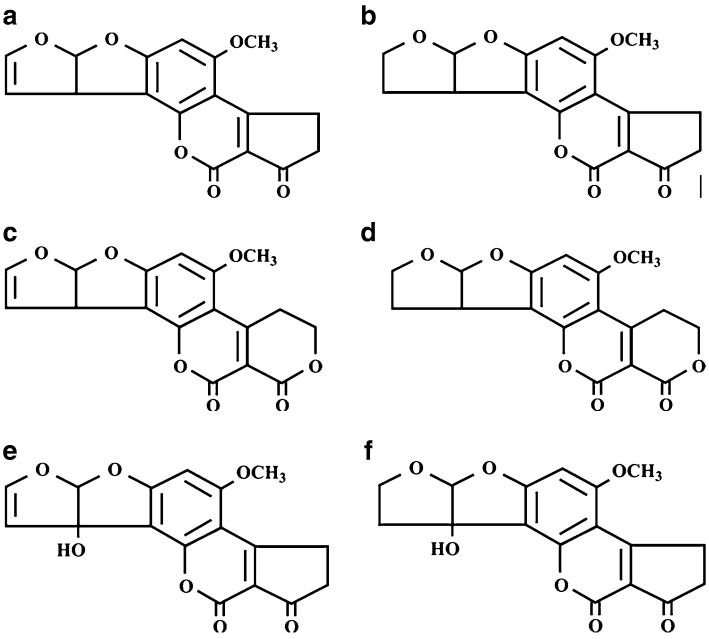

Fig. 1 Structures of (**a**) AFB_1, (**b**) AFB_2, (**c**) AFG_1, (**d**) AFG_2, (**e**) AFM_1, (**f**) AFM_2

Although *Aspergillus* spp. can produce several mycotoxins (Table 1), we deem giving detailed information on aflatoxins more appropriate in the following lines. The structural formulas of all some types of aflatoxins are given in Fig. 1. The genus *Aspergillus* includes notorious pathogens that produce aflatoxins B_1, B_2, G_1, or G_2, which are one of the most effective naturally produced mycotoxins in the world "B" and "G" refer to the blue and green fluorescent colours, respectively, produced under UV light on thin layer chromatography (TLC) plates and the subscript numbers 1 and 2 indicate major and minor compounds, respectively (Pitt and Hocking 2009).

AFM_1 which is the major metabolite of AFB_1 (4-hydroxy derivative of AFB_1) in mammals can partially excreted into milk. The carryover of AFB_1 in feeds as AFM_1 in milk varies from animal to animal and day to day changing 1–2 % (Moss 2000). The maximum permitted concentration of AFM_1 in cows' milk is 0.05 μg/kg in the European Union (EU) (Britzi et al. 2013). Consequently limiting AFB_1 in animal feeds is the most effective way of controlling AFM_1 in milk (FAO 2003).

Aspergillus spp. are widely distributed in the environment, including soil, plant debris, wood, and air. Water and inadequately harvested and stored grains and other foodstuffs such as peanuts, almonds, rye, wheat, and maize kernels, or whole-meal products are also act as reservoirs of transmission of *Aspergillus* spp. (Public Health Agency of Canada 2013). Even non-mouldy foods may contain aflatoxins since aflatoxins excreted by the mycelium can penetrate several centimetres into foods during a few days. Relative humidity <65 %, temperature <10 °C and moisture content <12 % are found as the conditions for preventing mould from growing and toxin production in the stored grains and other foodstuffs (http://pac.iupac.org/publications/pac/pdf/1973/pdf/3503x0239.pdf). The usage of stress and aflatoxin-tolerant cultivars, irrigation practices and management of insect pests in the field before harvest; avoiding direct contact of grains with soil after harvest; rapid grain drying and moisture control during storage may also given as effective ways to control aflatoxins from field to fork (http://www.aflasafe.com/aflatoxins).

3.2 Penicillium *and Related Teleomorphs*

The genus *Penicillium* which is known as opportunistic saprophytes is more diverse than the genus *Aspergillus*. Although most of the species in this genus are soil fungi, some species are directly related with food spoilage. Pitt and Hocking (2009) stated the taxonomy of this genus as difficult due to the similar colour and general colony appearance of the species occurring in foods.

The genus *Penicillium* has branched, septate conidiophores with a relatively slight, apparent stipe ending with a penicillus which helps to determine the subgenera of this genus. In other words, the number of branch points between phialide or conidial chain and stipe down the penicillus helps to find out the subgenera belonging to this genus. *Aspergilloides* is the simplest subgenus with only one branch point

between conidial chain and stipe. Species with two branch points are classified in one of two subgenera named *Furcatum* or *Biverticillium*, while species with three or four branch points are classified in subgenus *Penicillium* (Pitt and Hocking 2009).

The genus *Penicillium* is associated with two teleomorph genera; *Eupenicillium* and *Talaromyces*. *Penicillium* species associated with *Talaromyces* teleomorphs are all classified in the *Penicillium* subgenera *Biverticillium* while the species associated with *Eupenicillium* teleomorphs are classified in the *Penicillium* other subgenera.

Penicillium spp. can produce several mycotoxins (Table 1). The growth of *P. citreonigrum* in rice resulted in acute cardiac beriberi due to the production of a neurotoxin named as citreoviridin; *P. citrinum*'s and *P.verrucosum's* production of citrinin in various cereals which is a renal toxin to domestic animals, causes kidney degeneration in chickens and turkeys, teratogenic effects in rats and adverse effects on human T cells; *P. crustosum's* production of penitrem A which is a powerful neurotoxin, causes tremor syndrome in humans; *P. expansum*'s production of patulin which is also an indicator of the use of poor quality raw materials in fruit juice manufacturing and processing plants, causes damages in the DNA of mammalian cells; *P. verrucosum*'s production of ochratoxin A which is a chronic nephrotoxin and also produced by several *Aspergillus* species, has immunosuppressive, teratogenic, genotoxic, and carcinogenic effects in animals and in humans (Frisvad et al. 2007; Pitt and Hocking 2009). This list can be extended.

Although there is not any data on human toxicity of patulin, an upper limit as 50 mg patulin in 1 L of fruit juices, especially in apple juice (FAO 2003) and a limit as 10 mg patulin in 1 kg food products produced for babies and infants, have been approved in some countries (EU 2004). The maximum level of ochratoxin A is set to 5 mg/kg for raw cereal grains whereas it is set to 3 mg/kg for cereal products and cereal grains intended for direct human consumption. In addition to these, its level is set to 10 mg/kg for currants and raisins (Adams and Moss 2008).

3.3 Fusarium *and Related Teleomorphs*

The genus *Fusarium* is well known for taxonomic difficulties, comprising numerous members which can be recovered from plants, plant debris, and cultivated soils worldwide as pathogens, endophytes and harmless saprobes. Therefore, the *Fusarium* species are mostly regarded as soil-borne fungi due to their abundance in soil and plant roots (Nelson et al. 1994). It can be characterized by the production of macroconidia which are in crescent shapes, microconidia which are smaller one to two-celled conidia in various shapes (oval, ampulliform, ovoid, clavate, falcate, apiculate, reniform, and etc.), and chlamydospores which are thick-walled spores (formed singly, doubles, in clumps, or in chains) filled with lipid-like material that serves to carry the fungi over winter in soil when a suitable host is not available. Some species produce all these three types of spores although other species do not (Nelson et al. 1994; Pitt and Hocking 2009).

A number of *Fusarium* species have teleomorphs belonging to the genera *Gibberella*, *Haematonectria* (which includes *F. solani complex*), and *Albonectria* (which includes *F. decemcellulare*). Among these telemorph genera, *Gibberella* includes most of the significant pathogens such as *F. graminearum* (*G. zeae*) which commonly infects barley if there is rain late in the season and *F. verticillioides* (*G. moniliformis*) (Summerell and Leslie 2011).

Fusarium spp. are opportunistic fungi that can affect humans mainly in immunocompromised hosts, by producing either mycotoxicosis or invasive diseases. The main toxins produced by these *Fusarium* species are fumonisins and trichothecenes (a family of sesquiterpenes). During the closing years of World War II, *F. sporotrichoide* and *F. poae*-contaminated over-wintered wheat (wheat which had been left in the fields throughout the winter) the flour of which was baked into bread caused alimentary toxic aleukia (ATA) with the deaths of hundreds of thousands of people in the USSR (Nelson et al. 1994; Pitt and Hocking 2009). The active ingredient was found to be trichothecene T-2 mycotoxin. Two to three weeks after the consumption of toxic grain, ATA appears with an abdominal pain, diarrhoea, vomiting and prostration, and continues with fever, chilling, bloody diarrhoea, myalgias, and bone-marrow depression with granulocytopenia and secondary sepsis within a few days.

In Japan, *F. graminearum* or *F. sporotrichioides*-infected cereal grains (barley, oats, rye, rice, and wheat) are commonly associated with outbreaks of akakabi-byo (red mould disease or scabby grain intoxication) which is characterized by symptoms such as abdominal pain, diarrhoea, nausea, vomiting, headache, chills, giddiness, and anorexia. *Fusarium* isolates isolated from these scabby cereal grains are found to produce the trichothecenes: deoxynivalenol, nivalenol, fusarenon-X, diacetoxyscirpenol, neosolaniol, and T-2 mycotoxins in culture (Nelson et al. 1994).

F. moniliforme is one of the most prevalent fungi associated with mouldy corn toxicosis which causes human oesophageal cancer.

As an example, the highest rate of human oesophageal cancer occurs in the southwestern districts of the Transkei in southern Africa, where corn is consumed as the main foodstuff. The ingestion of *F. moniliforme*-contaminated corns gave rise to outbreaks of a neurotoxic disease called equine leukoencephalomalacia (LE) in horses, donkeys, and mules and pulmonary oedema (PPE) in swines (Nelson et al. 1994).

Zearalenone, which is not a true mycotoxin but is an oestrogen analogue, is mostly produced by *F. graminearum* and *F. culmorum* in cereals. Limits for zearalenone in maize and other cereals vary from 50 to 1,000 mg/kg (FAO 2003).

4 Non-bacterial and Non-fungal Food-borne Microbial Diseases

Viruses, protozoa, toxigenic algae, nematodes, and helminths are also the causative agents of some other food-borne microbial diseases. However, the following section will not explain them as in the same detail as food-borne bacterial pathogens and

toxigenic fungi but it will give the readers a brief significance and occurrence of important food-borne viruses and protozoa.

4.1 Food-borne Viruses

Viruses (derived from the Latin word; *poisons*) are non-cellular, strict intracellular parasites that have only one type of nucleic acid (either DNA or RNA) wrapped in a protein coat or capsid. They require a live host for replication and cannot multiply in foods, and so foods only play passive roles in the transmission of viral infection. Sensory characteristics of foods containing these viral pathogens and those of non-contaminated foods are identical (Koopmans and Duizer 2004). Food-borne viral infections can occur only from human enteric pathogenic viruses due to contamination of the fresh produce or processed foods by virus-containing faecal material. Almost more than 100 of them have been identified as the causative agents of food-borne diseases in humans. They enter the body via the gut but they differ in their target tissues. As an example; gastroenteritis viruses stay, multiply, and cause illnesses in gut while polio and hepatitis viruses cause illnesses when they have migrated to and multiply in the other organs of the host (Adams and Moss 2008; Scallan et al. 2011).

Poliovirus, which is in the genus *Enterovirus* of the family Picornaviridae, is a single-stranded RNA virus that causes an illness characterized by headache, fever, and sore throat. When it spreads to neurons of the spinal cord, cell destruction, and paralysis occur. Before the 1940s, contaminated milk had been the main source of it; however, this problem is overcome by the application of good sanitation and personal hygiene habits (Adams and Moss 2008; Ray and Bhunia 2008).

Several types of hepatitis viruses exist but only hepatitis A (mostly) which is in the genus *Hepatovirus* of the family Picornaviridae, and hepatitis E (very rare) which is classified in a separate genus *Hepevirus* of the family Hepeviridae are generally associated with food-borne illnesses and are major public health concerns (Schlauder and Mushahwar 2001; Vasickova et al. 2005). Hepatitis A virus (HAV) is a single-stranded RNA virus that causes an illness characterized by anorexia, fever, malaise, nausea, and vomiting, followed after a few days by abdominal discomfort, inflammation of liver, and jaundice. It causes the vast majority of mortality associated with food-borne viral disease. In 1988, the largest outbreak (approximately 300,000 people) of HAV, which was resulted with 47 deaths, occurred in Shanghai, China due to consumption of contaminated dairy clams (Bhunia 2008). On the other hand, Hepatitis E virus (HEV) can cause life-threatening infections in women in the later stages of pregnancy. HEV has been isolated in swine and rats, and in pig livers sold in the local grocery stores in the USA. Under-cooked pork and deer meat were the sources of infection in the major HEV outbreaks in Japan (Feagins et al. 2007). Faecal–oral spread is the primary mode of HAV transmission. RTE foods which are contaminated by infected food handlers and workers are the

main source of it however; sewage contaminated-drinking water, milk, fruits (especially strawberries and raspberries when contaminated water is used to rinse them), salad vegetables contaminated with polluted water and raw or improper-cooked shellfish are also the other sources of it. It survives in water and sewage for months and survives in foods for several days, even at refrigeration temperatures but can be inactivated by boiling or cooking to 85 °C. Besides, only HAV has available vaccines to prevent illness associated with food-borne viruses (Atreya 2004; Adams and Moss 2008; Ray and Bhunia 2008).

Norovirus (NoV) [earlier named as Norwalk-like viruses (NLV) due to the virus which caused an outbreak of gastroenteritis in elementary school children in Norwalk, Ohio, in the USA in 1968 (Adler and Zickl 1969)] or small round structures viruses (SRSVs) which is in the family Caliciviridae and is a plus-strand RNA virus, causes gastroenteritis characterized by vomiting, diarrhoea, and abdominal pain. They are estimated to be responsible for over 95 % of non-bacterial epidemic gastroenteritis outbreaks, and 50 % of all gastroenteritis outbreaks, worldwide (Jones and Karst 2013). However, asymptomatic infections are common and may contribute to the spread of the infection. Large numbers of virus particles are excreted by diarrhoeal stools during the illness since it multiplies in the gut. It is spread by the faecal–oral route. Foods can be contaminated with NoVs at the source (where the food is produced) by contaminated irrigation water or during handling and preparation processes. RTE foods and shellfish are the common sources of NoVs (Adams and Moss 2008; Glass et al. 2009; Jones and Karst 2013).

Norovirus is not the only virus that causes diarrhoeal disease. Astrovirus (AstV), rotavirus, sapovirus, and parvovirus are the other viruses that cause gastroenteritis. Among them, AstV and rotavirus which affect primarily the infants have more importance in food-borne viral infections. AstV [named for its star-like appearance (derived from the Greek word *astron*: star)] which is a single-stranded RNA virus including the only members of the family Astroviridae, and rotavirus [named for its wheel-like appearance (derived from the Latin word *rota* : wheel)] which is a double-stranded RNA virus belonging to the family Reoviridae, show the same symptoms such as vomiting for up to 48 h which is followed by 24 h of diarrhoea. If dehydration and electrolyte imbalances occur, death may occur. Poor water quality (using polluted water for irrigation), inadequately treated sewage sludge, and oysters, clams, mussles, and cockles, lettuce, and other vegetables are the main sources of AstVs and rotaviruses (Vasickova et al. 2005; Gastañaduy et al. 2013; Karlsson and Schultz-Cherry 2013).

Consequently, good agriculture practice (GAP), good manufacturing practice (GMP) and HACCP should be applied in order to avoid introduction of viruses onto the raw food material and into the food-manufacturing and processing environment. Good personal hygiene, improved sanitation and provision of clean drinking water should be provided. Since viruses remain infectious in most foods for several days or weeks, especially if kept cooled (at 4 °C), more attention should be given to personal hygiene during handling, preparation and service of foods (Koopmans and Duizer 2004).

4.2 Food-borne Protozoa

Amongst the protozoa, the flagellate *Giardia lamblia*, the amoeboid *Entamoeba histolytica*, and three sporozoid (*Cryptosporidium parvum, Toxoplasma gondii* and *Sarcocystis*) are of special concern from the point of view of food-borne microbial diseases.

Giardia lamblia which is also known as *G. intestinalis*, *G. lamblia*, or *G. duodenalis*, survives as cysts in soil, food, water, or on surfaces that has been contaminated with faecal material from infected humans or wild animals, such as beavers, for weeks or months although it is a parasite that feeds off a live host to survive. After the ingestion of its cysts, excystation occurs and so two active flagellate protozoa known as trophozoites which are characterized by eight flagella, two nuclei and tumbling motility, are released from each of the cyst into the small intestine. The main food sources are contaminated water, salad vegetables and fruits (especially lettuce in sandwiches, strawberries and raspberries when contaminated water is used to rinse them), improperly handled (by infected persons not observing good hygiene practices—poor personal hygiene) and improperly cooked foods. However, swallowing water during swimming in water where *Giardia* may live and having contact with a person who is ill also cause a disease called giardiasis which is characterized by recurrent abdominal cramps, nausea, acute or chronic dysentery type diarrhoea, with malabsorption and failure of children to thrive, greasy stools that tend to float and dehydration. The cysts are resistant to chlorine but are killed by cooking (Adams and Moss 2008; Bhunia 2008; CDC 2012).

Entamoeba histolytica cysts enjoy widespread occurrence in the environments wherever there is poor hygiene and inadequate sanitation facilities (endemic especially in the tropical areas with poor sanitary conditions), including contaminated water, sewage, insects, a wide range of domestic and wild animals, and a variety of foodstuffs of both animal and plant origin, and cause amoebiasis which is characterized by stomach pain, stomach cramping, and loose faeces in humans and animals. In severe forms, amoebic dysentery associated with bloody stools and fever occurs. However, asymptomatic infections are common and may contribute to the spread of the infection. Transmission is by the faecal–oral route. A person with amoebic dysentery may pass up to 50 million cysts per day. Following the ingestion of its cysts, excystation occurs and each cyst gives rise to eight daughter trophozoites. The cysts can survive outside the host for several weeks to months although it is a parasite that feeds off a live host to survive. The cysts are sensitive to temperature below −5 °C or over 40 °C (Adams and Moss 2008; Bhunia 2008; Nagata et al. 2012).

Cryptosporidium parvum which causes a waterborne diarrhoeal disease called cryptosporidiosis that is characterized by a cholera-like illness with stomach and muscle pain, nausea, vomiting, low-grade fever, dehydration, weight loss, and anorexia. Some people have no symptoms at all. After the ingestion of its oocysts, excystation occurs and so four sporozoites which parasitize epithelial cells of the gastrointestinal or respiratory tract, are released from each of the cyst into the small intestine. The oocysts complete their life cycles in one host (homoxenous). Vegetables or foods which are exposed to contaminated water, serve as the major

sources of it. The oocysts are resistant to disinfectants usually used to treat water. In the midst of 1990, a large outbreak of *Cryptosporidium* occurred among HIV patients in Wisconsin, the USA due to the consumption of water from municipal water supply, which was contaminated with cattle manure (Adams and Moss 2008; Bhunia 2008). Between October and December 2011, an outbreak of 26 cases (24 children under 2 years of age and two caregivers) of cryptosporidiosis occurred in a day-care centre in Gipuzkoa, Spain (Artieda et al. 2012).

Toxoplasma gondii which causes a zoonotic disease called toxoplasmosis is mostly transmitted by the members of the family Felidae (domestic cats and their relatives). It not transmitted by person-to-person contact, except in cases of mother-to-child (congenital) transmission. After the ingestion of its oocysts, they dissolve in the gut and tachyzoites are released which penetrate the intestinal epithelial cells, reach to blood circulation and invade muscle tissues, and develop into tissue cyst bradyzoites. Although food-borne infection in humans is uncommon, it could occur by drinking water contaminated with cat faeces and consumption of raw or under-cooked meat, especially mutton or pork. It is normally symptomless or associated with a mild influenza-like illness (fatigue, joint, and muscle pain) in healthy humans. However, spontaneous abortion or stillborn child usually occurs in pregnant women (Montoya and Liesenfeld 2004; Adams and Moss 2008; Bhunia 2008).

On the other hand, *Sarcocystis* (derived from the Greek words; *sarx*: flesh and *kystis*: bladder) species are intracellular protozoan parasites with a requisite two-host life cycle based on a prey–predator (intermediate-definitive) host relationship: an intermediate host such as cattle, sheep, or pigs in the tissues of which the asexual cysts are formed, and the definitive host such as cats, dogs, or humans, in which sexual reproduction of this parasite takes place. In other words, sarcocysts in meat eaten by humans initiate sexual stages in the intestine which terminate in oocysts excreted in the faeces. *S. hominis* and *S. suihominis* can infect humans who eat raw or under-cooked meat from cattle and pigs containing mature sarcocysts, respectively. The most likely source of sarcocysts is water contaminated with faeces or foods washed or irrigated with contaminated water. They usually cause mild illness starting with nausea and diarrhoea. They can be prevented by thoroughly cooking or freezing meat to kill bradyzoites (spindle- or crescent-shaped bodies) in the sarcocysts (Fayer 2004; Adams and Moss 2008).

5 Conclusion

A significant amount of foodstuff in our daily life goes to the garbage since it is spoiled by different microorganisms. Among these microorganisms, pathogens have received abundant attention because of the illnesses and diseases that they or their toxins cause. Although there are certainly many more unrecognized microbial pathogens awaiting identification, a great deal is known about pathogens. However; we are still not able to control them and thus food-borne diseases caused by pathogens still occur at unacceptably high rates even in industrialized and developing countries.

Food-borne microbial diseases are important causes of personal suffering, illness, death, and economic burden. Increased public awareness of the health-related and economic impact of food-borne microbial contamination and disease has resulted in greater efforts to develop of new methods of food manufacturing, processing and preservation in order to prevent and control the contamination during the flow of food from the farm to table. This technological improvement together with the good hygienic and good agricultural and manufacturing practices and the knowledge on these pathogens, supported by research, help us control the contamination and the incidence of food-borne microbial pathogens.

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