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A Review on Major Food Borne Bacterial Illnesses

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Abstract

Food borne illnesses are defined by the world health organization as diseases of infectious or toxic nature caused by consumption of contaminated foods or water. Food borne illnesses are classified in to two broad groups namely intoxication and infection. Intoxication is caused by ingestion of toxin produced by pathogens, while infection is caused by ingestion of food containing viable pathogens. Toxin can be present even when the bacteria or other causative agents are not and it is possible to develop food intoxication by eating animals that have consumed toxin producing organisms. Onset of illness is very rapid with food intoxication and people become very sick. The main causes of food borne illness are bacteria which constitutes 66% of the problems. *Botulism*, *Clostridium perfringen* gastro enteritis, *E. coli* infection, Salmonellosis and staphylococcal food poisoning are the major food illness caused by bacteria. Microorganisms present in food will grow under favorable conditions and will produce toxin in food. Following ingestion, toxins are absorbed through gastro intestinal epithelial lining and cause local tissue damage. In some cases toxins are translocated to distant organs or tissues such as kidney, liver, central nervous system or peripheral system where they can cause damage. The most common clinical symptoms of food borne illnesses are diarrhea, vomiting, abdominal cramps, headache and nausea. Food borne illnesses are usually diagnosed based on patient's history and the symptoms. Food borne illness prevention system will depend on the extent of food safety control in place through food production, processing and distribution keeping food clean, separation of raw and cooked, and cooking thoroughly, keeping food at safe temperature and using safe water and raw materials are some of the important points especially for safety of food of humans. The high level of bacteria which could cause food borne illness in various foods presents public health risk to the consumer. This suggests the need to implement strict hygienic control measures along the food chain to improve the hygienic conditions during manufacturing, handling, storage and commercialization of foods.

Keywords: Food; Bacteria; Food borne illnesses; Food borne infection; Food borne intoxication

Introduction

Food poisoning syndrome results from ingestion of water and wide variety of food contaminated with pathogenic organisms (bacteria, viruses, parasites, and fungi), their toxin and chemicals. Food poisoning must be suspected when an acute illness with gastrointestinal or neurological manifestation affect two or more persons or animals who have shared a meal during the previous 72 hours. The term generally used encompasses both food related infection and food related intoxication. Some microbiologists consider microbial food poisoning to be different from food born infections. In microbial food poisoning, the microbes multiply readily in the food prior to consumption, whereas in food born infection, food is merely the vector for microbes that do not grow on their transient substrate. Other considers food poisoning as intoxication of food by chemicals or toxins from bacteria or fungi [1]. Food borne illness (FBI) often called food poisoning, it's caused by pathogens or certain chemicals present in ingested food bacteria, viruses, molds, worms and protozoa that cause diseases are all pathogens, although there are also harmless and beneficial bacteria that are used to make yogurt and cheese. Some chemicals that causes food borne illness are natural components of food, while other may be accidentally added during production and processing, either through carelessness or pollution. The main causes of food borne illness are bacteria (66%), chemicals (26%), virus (4%) and parasites (4%). The two most common types of food borne illness are intoxication and infection. Intoxication occurs when toxin produced by the pathogens cause food poisoning, while infection is caused by the ingestion of food containing pathogens [1,2]. Some microorganisms can use our food as a source of nutrients for their growth. By growing on the food, metabolizing them and producing by-products, they not only render the food inedible but also pose health problems upon consumption. Many of our foods will support the growth of pathogenic microorganisms or at least serve as vector for their transmission. Food can get contaminated from plant surfaces, animals, water, sewage, air, soil or from food handlers during handling and processing [1,3]. The symptoms of food borne illness often resemble intestinal fluke and they may last a few hours or several days. Typical symptoms include diarrhea, vomition, abdominal cramps, headaches, nausea, dry mouth, and difficulty swallowing and fluke-like symptoms (such as fever, chills, backache). The consumption of poisonous mushroom leads mycetism, while consumption of food contaminated with toxin producing fungi leads to mycotoxicosis [4]. Most cases of food borne illness can be prevented through proper cooking and processing of food which kill bacteria such as adequate refrigeration, improve personal hygiene, adequate cooking or heat processing of food at higher temperature and prevent holding of food in warming device at bacterial growth temperatures [5]. The safety of food with respect to FBI is of great concern around the world. This is especially true in developing countries like Ethiopia, where production of food often takes place under unsanitary conditions. In spite of the aforementioned prevailing situation and the presence of a number of public health problems due to FBI resulting from the consumption of different food items in Ethiopia, there is paucity of well-documented information on the occurrence of FBI. Therefore, this seminar paper was designed to review and give background information on major food borne bacterial illnesses [2,5].

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Salmonellosis

Etiology: The *salmonella* are small, gram negative, non-spore forming rods that are indistinguishable from the *Escherichia coli* under the microscope or ordinary nutrient media. They are widely distributed in the nature with humans and animals being their primary reservoirs. *Salmonella* food poisonings results from ingestion of food containing appropriate strains of this genus in significant numbers. Some significant changes have occurred in the taxonomy of salmonella [6]. The genus *salmonella* are considered to have a two species named *salmonella enteric* and *salmonella bongori*. Serotyping differentiates the strains and they are referred as to by, for example *S. enterica* serotype-*typhimurium* or as *S. typhimurium* [7].

Epidemiology: The primary habitat of *salmonella* species is the intestinal tract of the animals such as farm animals, humans, birds, reptiles and insects. Although their primary habitat is intestinal tract, they may be found in other parts of the from time to time. As an intestinal form, the organisms are excreted in feces from which they may be transmitted by insects and other living creatures to large number of places [8-10]. For epidemiological purposes, the salmonella can be placed into three groups; the first are those that infect humans only. This includes, *S. Typhi*, *S. Paratyphi* A, *S. Paratyphi* C. this group includes the agents of typhoid and paratyphoid fevers, which are the most severe of disease caused by *salmonella*. The second was the host-adapted serovars (some of which are human pathogens and may be contracted from food), included are *S. gallirinum* (poultry), *S. dublin* (cattle), *S. abortus-equi* (equine), *S. abortus-ovis* (sheep) and *S. choleraesuis* (swine). The third is unadapted serovars (no host preference). There are pathogenic for humans and other animals. The epidemiology of the salmonella is complex, which often make animals control of the disease is difficult. Animals are the reservoir of food born disease of salmonella [11,12].

Pathogenesis: *Salmonella* often enter the host by ingestion, and even with several system to mediate acid resistance, few survive the stomach and move into the small intestine. Normal flora protects against colonization of administration of oral antibiotics facilitates establishment of infection. Entry of salmonella usually occurs without mucosal damage in systemic infections, but enteric infection is characterized by local damage without septicemia-*salmonella* infection with M cells in payer's patches is facilitated by fimbrial adhesions. This is followed by ruffling of the target cell membrane which result in internalization of the bacteria in membrane bound vacuoles [13]. The ruffles facilitates uptake of the bacteria in membrane bound vacuoles or vesicles which often coalesce. The organisms replicate in these vesicles and are eventually released from the cells, which sustains only mild or transient damage. The complex invasion process is mediated by the product of a number of chromosomal genes, whereas growth within host a cell depends on the presence of virulence plasmids [14].

Symptoms: The incubation period of the salmonella is 12-36 hours. The clinical sign includes diarrhea, which may be watery, greenish and foul smelling. This may be preceded by headache and chills. Other findings include prostration, muscle weakness and moderate fever. In most cases the symptoms resolve in 2-3 days without any complication [3]. The bacterium induces responses in the animal that is infecting, this is what typically causes symptoms, rather than any direct toxin product. Symptoms are usually gastrointestinal, including nausea, vomiting, abdominal cramps and bloody diarrhea with mucous, headache, fatigue and rose spots are also possible. These symptoms can be severe, especially in young children and elderly. Symptoms last generally up to a week, and can appear 12-72 hours after ingesting the bacterium. Reactive arthritis, sickle-cell anemia and osteomyelitis due to salmonella infection is much more common that in the general population [9,10].

Detection of pathogens: Detection of *salmonella* can be provided only by isolation of the agent from stool or vomit in human, feed samples in cases of animals, and samples concerned food items like milk and milk-products samples. In general, for culture and isolation, the use of selective enrichment media such as *salmonella-shigella*, agar, hektoen enteric agar or deoxycholate agar and broth for enrichment before sub-culture to these sugar agars after 24 hours is usual procedure. Selenite enrichment broth or tetrathionate broth can be used to isolate highly selective for salmonella, especially *S. enterica serovar Typhi*. Agar and plates are incubated at 37°C overnight and growth identified by biochemical tests and slide agglutination tests [13].

Prevention and control: The principal sources of infection are carrier animals and contaminated feeds containing food stuff of animal origin. There is a critical need to develop method to control the spoilage or poisoning of food by salmonella ordinary farms by instituting bio-security and bio-containment practices in addition to enhanced food processing method, preparation and storage practices [12]. Effective heat processing of food of animal origin, which includes pasteurization of milk and eggs, irradiation of meat and poultry thermal processing; good hygiene practices during production of food; vaccination of egg-producing flocks and food producing animals. Food service establishment, safe food preparation practices, including through cooking and reheating of food and boiling of milk, adequate refrigeration, prevention of cross contamination; cleaning and disinfection of food preparation surfaces; exclusion of pets and other animals from food handling areas. Consumer particularly vulnerable groups should avoid reward of under cooked meat and poultry, raw milk, eggs and foods contain raw egg and uncleaned vegetables. The control is also based on reducing the risk of exposure to infection. Intensively-reared, food producing animal are more likely to acquire infection and are also major source of human infection [9].

Staphylococcus aureus

Etiology: *Staphylococcus aureus* is gram positive cocci that occur in singles, short chains, tetrads and irregular grape like cluster. Only the strains that produce enterotoxin can cause food poisoning. The food handler with an active lesion or carriage later initiates infection [12,14].

Epidemiology: The *Staphylococcus* species are host adapted with about one-half of the known species inhabiting humans solely and other animals. The largest numbers tent to be found near opening of the body surface such as the anterior nares, inguinal and perineal areas where in moist habitats, numbers per square centimeter may reach 1000-1000000 and in dry habitats 10-1000. The two most important sources to foods are nasal carriers and individuals whose hands and arms are inflicted with boils and carbuncles, who are permitted to handle foods [12].

Pathogenesis: If food is stored for some times in room temperature the organism may in the food and can produce toxin. The bacteria produce enterotoxin while multiplying in food. *S. aureus* is known to produce six serologically different types of enterotoxins (A, B, C1, C2, D and E) that differ in toxicity. Most food poisoning is caused by enterotoxin A followed by type D. These enterotoxins are heat stable, with type B being most heat resistant. Enterotoxin stimulates Central Nervous Systems (CNS) vomiting center and inhibit water and sodium absorption in the small intestine. Staphylococcal enterotoxins, along with the toxic syndrome toxin and others, are classed as bacterial super antigens relative to in vivo antigen recognition in contrast to

conventional antigens [12,15].

Symptoms: Food poisoning by *S. aureus* is characterized by a short incubation period typically 2-4 hours. The onset is sudden and is characterized by vomiting and diarrhea but no fever. The illness lasts less than 12 hours. In severe cases dehydration, masked pallor and collapse may require treatment (intravenously) infusion. The short incubation periods are the characteristics of intoxication where illness is the results of ingestion of the preformed toxin in the food [1].

Detection of the organisms: The presence of a large number of *S. aureus* organism in a food indicate poor handling or sanitation. The dilution is placed on baird-parker agar or mannitol salt agar. The enterotoxin can be detected and identified by gel diffusion [10,12].

Prevention and control: Food born bacterial illness by bacteria are most commonly prevented and controlled by proper cooking and preparing of food as well as storing. For examples adequate refrigeration of food, improve personal hygiene, adequate cooking and heating processing. The control method or measures also includes; a) education of those who prepare the food at home and other food handlers, so that they have to take proper personal measures; b) prohibiting individuals with absences or other skin lesions from handling food; c) placing of food in cold place at 4 degree centigrade or lower of all food in order to prevent bacterial multiplication and the formation of toxin. Foods must be kept at room temperature for as little time as possible [16].

Clostridium botulinum

Etiology: *Clostridium botulinum* is gram positive anaerobic spore bearing bacilli that is widely distributed in soil, sediments of lakes, ponds and decaying vegetations. Seven different strains of the organisms (A-G) are classified based on serologic specificity and other neurotoxin. Most reported human outbreaks are associated with fish and sea food products. Botulism in animals is predominantly due to type C and D and rarely type A and B. All toxin-producing strains have placed in to one of four groups; I, II, III and IV. Group I contains the proteolytics, group II the non proteolytic and group IV serological type G. group III consists of type C and D [17].

Epidemiology: Botulism has no geographical limitations. Sporadic outbreaks occur in the most countries. The sources of exposure to the toxin and risk for the disease differ between regions because of difference in the food storage, feeding and management practices. Outbreaks associated with ingestion of toxin in observed feeds are common in northern United States of America and Europe where as outbreaks in animals on pasture are reported primarily from South Africa, Australia and Gulf coast are the USA [10]. Geographic distribution varies considerably. In a study conducted in the USA, the type A was found in the neutral and alkaline soil in the west while type B and C in damp or wet soil all over, except that B was not found in south. Type C was found in soils in Gulf coast and type D in alkaline soil in west. A common source in Australia has been reported to be from hay made at a time of mouse plague. At such time even good and fresh hay can contain a great deal of carrion. In another record incident 427 of 444 dairy cattle died after ingesting feed contaminated with botulism type C toxin from cat carcass. Spores of *C. botulinium* are present throughout the world, although most of recorded outbreaks of botulism have reported in north of the tropic of cancer with exception of Argentina. The geographical prevalence of the disease necessitates some important observations such as home canning fruits and vegetables in most tropical countries [9,10].

Pathogenesis: During their growth *C. botulinum* strains produce high potent neurotoxin that cause neuroparalytic disease known as botulism in humans and animals without the development of histological lesion. Botulism may lead to death due to respiratory muscle paralysis unless treated properly [9]. Botulism toxin is absorbed from the glandular stomach and anterior small intestine or the wound and carried through the blood stream bind to receptor and enters the nerve cell after receptor mediated endocytosis to peripheral cholinergic nerve terminals including neuromuscular junction, postganglionic parasympathetic nerve ending and peripheral ganglion. The heavy chain of the toxin is responsible for binding to receptor and translocation into the cell and the light chain of the toxin for the resultant blockage of the release of the acetylcholine at neuromuscular junction then causing flaccid paralysis due to lack of neurotransmitters (acetylcholine) and affect muscle of respiration, then death due to respiratory failure [9,18].

Symptoms: The incubation of *C .botulinium* is 12-36 hours. The most common features include vomiting, thirst, dryness of mouth, constipation, ocular paresis (blurred-vision), difficulty in speaking, breathing and swallowing. Death occurs due to respiratory paralysis within 7 days). Clinically, botulism recognized as a lower motor neuron disease resulting progressive flaccid paralysis. Although deficient's in somatic neuro-muscular transmission are the most prominent effects, the motor deficient's in cranial nerve function, as well as the autonomic nervous system have also been reported [19].

Detection of toxin: Using bioassay in mice coupled with toxin neutralization with polyvalent antitoxins used but the sensitivity is found to be low in both ruminants and horse as they are substantially more sensitive than mice to botulism toxin. Protection with monovalent toxin allows type identification of the toxins involved. Toxin detection by ELISA test appears less sensitive than mouse bioassay. Diagnosis of botulism requires demonstration of toxin in plasma or tissue before death or from fresh carcass. Demonstration of the toxin in feedstuff, fresh stomach content or vomitus supports diagnosis of botulism. The spoilage of food or swelling of cans or presence of bubbles inside the can indicate *clostridial* growth. Food is homogenized in broth and incubated in Robertson cooked meat medium and blood agar or egg-yolk agar, which are incubated anaerobically for 3-5 days at 37°C. The toxin can be demonstrated by injecting intra peritoneal the extract of food or culture into mice or guinea pig [18,19].

Prevention and control: Preformed toxin in food can completely destroyed by exposure to a temperature of 80 d/c for 30 minutes or boiling for 10 minutes. Therefore all homo canned low acid foods should be boiled before tasting for consumption. Never taste food if it has an odor shows gas formation. Prevention of food born botulism also depends on ensuring effective control of commercially and home canned foods are destroying all *C. botulism* spores. This requires cooking at 121°C or higher. Vegetables that are home canned should be boiled and stirred for at least 3 minutes prior to serving to destroy botulism toxins. Foods with apparent off odors or suspected odor should not be opened [9].

Clostridium perfringens

Etiology: *Clostridium perfringens* is gram-positive anaerobic spore bearing bacilli that is present abundantly in the environment, vegetation, sewage and animal feces. *Perfringens* food poisoning is most commonly caused by organisms producing type A enterotoxin. Other types of enterotoxin (B to G) do not normally cause food born disease [19].

Epidemiology: *Clostridium perfringen* type A food poisoning remains one of the most prevalent food born disease in western countries. In the US, between 1983 and 1997 there were 121 confirmed outbreaks, with 9316 cases and a total of 13 deaths. The food poisoning strains of *C. perfringens* exist in soils, water, food, dust, spices and intestinal tract of humans and other animals. Various investigators have reported that the incidence of the heat-resistant, non-hemolytic stains to range from 2% to 6% in the general population. Between 20% and 30% of healthy hospital personnel and their families have been found to carry these organisms in their feces, and the carrier rate of victims after 2 weeks may be 50% or as high as 88% [9].

Pathogenesis: Spores in food may survive cooking and then germinate when they are improperly stored. When these vegetative cells form endospore in the intestine, they release enterotoxins. The bacterium is known to produce at least 12 different toxins. Food poisoning is mainly caused by type A strains which produce alpha and theta toxins. The toxin results in excesive fluid accumulation in the intestinal lumen [2]. The *C. perfringens* enterotoxin (CPE) is not a super antigen as are staphylococcal enterotoxins. Enterotoxigenesis begins when CPE bind to one or more protein receptors on epithelial cells in the gastro intestinal tract. It does not affect cyclic adenosine mono phosphate levels as do enterotoxigenic strains of *E. coli*. It localizes in small plasma membrane complex and apparently associated with a membrane protein to form a larger complex, which coincides with the onset of CPE-induced membrane permeability alterations that leads to cell death from lysis or metabolic disturbances [10].

Symptoms: The incubation period is 8-24 hours. The illness characterized by acute abdominal pain, diarrhea and vomiting, illness it self-limiting and patient recovers within 8-24 hours. The classic symptoms of *C. perfringens* type A food poisoning are diarrhea with lower abdominal cramps. Vomiting is not common, and fever is rare. Symptoms typically occur in 8-24hours after ingestion of temperature abused foods containing large number of vegetative cells of organism. Mortality is low, and such cases have been associated with elderly patients. It subsides within 1-2days, although cramps can continue a little longer [20].

Detection of the organism and enterotoxin: A number of criteria have been proposed for establishing an outbreak of *C. perfringens* type A food poisoning. These includes; a) more than 10^6spores/gram feces from ill individuals; b) more than 10^5 cells/gram incriminated food; c) the presence of the same serotypes of *C. perfringens* in both contaminated food and feces; d) the presence of the same serotype in all ill individuals in an outbreak or detection of enterotoxin in feces of individuals. Homogenized food is diluted and plated on selective medium as well as Robertson cooked meat medium and incubated anaerobically. The isolated bacteria must be shown to produce enterotoxin [12].

Control and prevention: The *C. perfringens* gastroenteritis syndrome may be prevented by proper attention to the leading causes of food poisoning of bacterial intoxication .because this syndrome often occurs in institutional cafeterias, some special precautions should be taken. Up on investigation, *C. perfringens* food poisoning outbreaks in school lunchroom 88% of students and teachers became ills in USA [13]. As means of preventing reoccurrence of such problems, these investigators suggested the following points; a) cook meat until the internal breast temperature reaches at least 74°C, preferably higher; b) thoroughly wash and sanitation of all containers and equipment that previously had contact with raw meat/eggs. c) wash hands and use disposable plastic gloves when handling raw or uncooked foods; d) separate meat and other food stock before chilling; e) chill meat rapidly after cooking; f) use refrigeration for storage. It's not possible to prevent carries from handling food, because most people harbor *C. perfringens* in their intestinal tract. Since the organism is present in animals, it is not surprising that *C. perfringens* is found in raw meat and poultry. The spores will also survive indefinitely in dust and in environmental nooks. Cooking at temperatures not exceeding 100°C will allow the survival of the spores. The cooking process drives off oxygen's, creating real anaerobic conditions in foods such as rolls of cooked meat, pies, strew and gravies and in poultry carcass. Therefore, prevention of vegetative cells in cooked and this is the most practical way of preventing *C. perfringens* food borne illness [20].

Escherichia coli

Etiology: *Escherichia coli* (*E. coli*) are bacterium which belongs to family enterobactereriae and are gram negative rod up to 3 um in length, ferment glucose and wide range of sugars. These lactase fermenters produce pink colonies on Mcconkey agar. Hemolytic activity on blood agar is characteristics of certain strain of *E. coli*. It's motile with peritrichous flagella and often fimbriate [9,12]. The O_{157}: H_7 is the major serotype that was recognized as a cause of human illness. *E. coli* O_{157}:H_7 is one of the more than 60 serotypes of verotoxin producing *E. coli* that cause that a variety of human illness such as mild diarrhea, hemorrhagic colitis and hemolytic-uremic syndrome (HUS) [13].

Epidemiology: Enterohemorrhagic *E .coli* and verocytotoxin producing *E. coli* are being recovered in humans and animals and they constitute major food borne illness. *E. coli* O_{157}:H_7 is an important of serotype and it seems to predominate in most areas. The strains producing verotoxin are shiga-like toxin (SLT) which produces diarrhea in humans and animals. In most cases cattle are represents the main reservoir of *E. coli*. *E. coli* O_{157}:H_7 is transient inhabitant of gastrointestinal tract of normally ruminant. Source of infection is contamination of food by human and animal feces. The organism can persist in manure, water trough and other farm location. The association of *E. coli* O_{157}:H_7 with raw meat, under cooked ground beef and raw milk lead to investigation of the role of cattle as a reservoir of the pathogens [21].

Pathogenesis: Enterohemorrhagic *E .coli* (EHEC) strain may produce one or more types of cytotoxins which are collectively referred as shiga-like toxins (SLTs) since they are antigenically and functionally similar to shiga toxin produced by *shigella dysenterica*. SLTs were previously known as verotoxin. The toxins provoke cell secretion and kill colonic epithelial cells [9]. Enterohemorrhagic *E. coli* are characterized by presence of SLTs genes, locus for enterocyte effacement (LEE) and higher molecular-weight plasmid that encodes for a hemolysin. These three virulence factors are present in most *E. coli* associated with bloody diarrhea and hemolytic uremic crisis in humans [10]. The most virulent factor of *E. coli* O_{157}:H_7 is the production of cytotoxic SLT. *E. coli* O_{157}:H_7 likely gained ability to produce the SLT1 and SLT2 as a result of ingestion with a bacteriophage carrying SLT1 and SLT2 genes [20]. The SLTs of *E. coli* O_{157}:H_7 are cytotoxic to human colon and ileum cells. In animals, toxin has been shown to cause localized fluid accumulation and colonic lesion characterized by sloughing of surface and crypt epithelial cell [9].

Symptoms: The incubation period is 72-120 hours. The clinical sign initially may be diarrhea with abdominal cramps, which may turn into grossly bloody diarrhea in a few days. There is however, no fever. The symptoms of *E. coli* septicemia are mainly referable to bacteremia, end toxemia and the effect of bacteria localization in a variety of tissue spaces throughout the body [5].

Detection of toxin: Laboratory diagnoses involve culturing the food on Macconkey's agar or sorbitol. Macconkey's agar, where they do not ferment sorbitol. Strains can be then identified by serotyping using specific antisera. SLTs can be detected by ELISA and gene coding for them can be detected by DNA hybridization techniques [7]. Sorbitol Mcconkey agar is recommended for isolation of *E. coli* O_{157}:H_7 from food and feces samples. Various immunoassay techniques can be used to detect SLT in food and fecal matter or cultures. Isolation becomes difficult beyond 1 week after onset of symptom [10,12].

Control and prevention: The prevention/avoidance of food borne illness caused by *E. coli* can be prevented by the same method as prevention of other food borne illness caused by bacteria. However, because of the consequences to young children, special precaution needed to observe. The heat sensitivity of this organism is such that cases should not occur when food properly cooked. In the cases of ground beef, the recommendation is that it cooked to 160°F or that the core temperature be brought to a minimum of 155°C for at least 15 second and that the juices are clear. Because of unevenness of food cooking at 155-160°F provides a measure of safety. Once cooked, the food meats should not be held between 40°F and 140°F for more than 3-4 hours [16].

Shigellosis

Etiology: *Shigella* is a species of enteric bacteria that causes disease in humans and other primates (*Shigella* is gram-negative rods that are non-motile and non-spore forming. The bacterias are primarily a human disease, but has been found in some primates. *Shigellas* are facultative anaerobes, similar to enterics such as *E. coli* [9,10].

Epidemiology: *Shigella* transmission can occur through direct person-to-person spread or from contaminated food and water. The minimal infectious dose can be transmitted directly from contaminated fingers, since intermediate bacterial replication is not required to achieve the low infectious dose. In developed countries, most cases are transmitted by fecal-oral spread from people with symptomatic infection. In developing countries, both fecal-oral spread and contamination of common food and water supplies are important mechanisms of transmission symptom [10].

Pathogenesis: *Shigella* attachs to and penetrate intestinal cell walls of the small intestines by producing toxins that may promote the diarrhea characteristic of the disease. The Shiga toxin enables the bacteria to penetrate the epithelial lining of the intestines, leading to a breakdown of the lining and hemorrhage. *Shigella*s also have adhesins that promote binding to epithelial cell surfaces and invasion plasmid antigens that allow the bacteria to enter target cells, thus increasing its virulence [4].

Symptoms: Abdominal pain, cramps, diarrhea, fever, vomiting, blood, pus or mucus in stools and tenesmus are the common symptoms. Mild infections cause low-grade fever (about 100.4 to 102°F [38 to 38.9°C] and watery diarrhea 1 to 2 days after people ingest the bacteria. Abdominal cramps and a frequent urge to defecate are common with more severe infections. Severe infections may cause low-grade or moderate fever and watery diarrhea that progress to dysentery. In dysentery, bowel movements are frequent and contain blood, pus, and mucus. Children, particularly young children, are most likely to have severe complications High fever (up to 106°F [41°C], sometimes with delirium. Severe dehydration with weight loss up to 20 bowel movements a day with severe diarrhea, protrusion of part of the rectum out of the body (rectal prolapsed) rarely, marked swelling of the intestine and tearing (perforation) of the large intestine [22,23].

Detection of toxin: *Shigella* infection is diagnosed through testing of a stool sample**.** First a stool sample must be obtained from the potentially infected person, and then the sample is placed on a medium to encourage the growth of bacteria. If and when there is growth, the bacteria are identified, usually by looking at the growth under a microscope [4].

Control and prevention: *Shigella* is heat-sensitive and will be killed by thorough heating (over 70°C). Raw or undercooked foods and cross-contamination, when cooked material comes into contact with raw produce or contaminated materials (cutting boards), are the main causes of infection. Proper cooking and hygienic food handling thus can prevent *Shigella* infections to a large extend. There is currently no vaccine for Shigellosis prevention, but there is current research that appears promising. The most effective method for prevention is frequent and vigorous hand washing with warm, soapy water and insuring clean drinking water sources and proper sewage disposal in developing nations [23].

Campylobacteriosis

Etiology: Campylobacteriosis is an infection caused by bacteria of the genus *Campylobacter*. There are approximately sixteen species associated with *Campylobacter*, but the most commonly isolated are *C. jejuni, C. coli*, and *C. upsaliensis*. The most prevalent species associated with human illness is *C. jejuni. Campylobacter* is also responsible for 15% of foodborne illness-related hospitalizations, and 6% of foodborne illness-related deaths [9,10].

Epidemiology: *Campylobacter* is one of the most common causes of human bacterial gastroenteritis. A large animal reservoir is present as well, with up to 100% of poultry, including chickens, turkeys, and waterfowl, having asymptomatic infections in their intestinal tracts. Infected chicken feces may contain up to 10^9 bacteria per 25 grams, and due to the installations, the bacteria are rapidly spread to other chickens. This vastly exceeds the infectious dose of 1000-10,000 bacteria for human [12].

Pathogenesis: Bacterial motility, mucus colonization, toxin production, attachment, internalization, and translocation are among the processes associated with *C. jejuni* virulence. Infection begins with ingestion of the *C. jejuni* in contaminated foods or water. Gastric acid provides a barrier, and the bacteria must reach the small and large intestines to multiply; *C. jejuni* invades both epithelial cells and cells within the lamina propria [12].

Symptoms: The symptoms associated with this disease are usually flu-like: fever, nausea, abdominal cramping, vomiting, enteritis, diarrhea, and malaise. Symptoms begin within 2-5 days after ingestion of the bacteria, and the illness typically lasts 7-10 days. Recurrence of this disease can occur up to three months after pathogen ingestion [14]. Other complications can include meningitis, urinary tract infections and short-term reactive arthritis. Some individuals may develop *Guillain Barré* (GB) syndrome, a nerve disorder that causes muscle weakness and paralysis of the limbs, about 2-4 weeks after infection (NIAID, 2007). GB symptoms can last several weeks to many years. About 1 in 1,000 people with campylobacteriosis are expected to develop GB, and it is estimated that 40% of GB cases in the US may have been triggered by campylobacteriosis [4].

Detection of toxin: Because of the unique growth characteristics of Campylobacter, isolation of these organisms from field samples requires the use of special media and culture conditions. *Campylobacter jejuni* and *Campylobacter coli* can be isolated from the intestines of healthy

farm animals, poultry, pets, zoo animals, and wild birds. Diagnosis of *C. jejuni* is based on isolation of the organism on selective media under micro aerophilic conditions. PCR-based methods are effective in identifying infection especially if cultivation is difficult or if the sample has been somewhat mishandled. However, a positive test is not sufficient evidence to determine causation and must be considered in conjunction with clinical signs [9].

Control and prevention: Control depends on sanitation and hygiene in livestock barns to reduce the bacterial populations in the environment of the animals. The number of organisms can be reduced and controlled in meat processing plants by using Hazard Analysis of Critical Control Points including the washing, handling and freezing of carcasses. Improvement of food-handling skills in restaurants and in the home kitchen will reduce transmission of the organism and adequate cooking of raw meat such as poultry to an internal temperature of 82°C will eliminate the organism [16].

Listeriosis

Etiology: *Listeria monocytogenes* is the bacterium that causes the infection listeriosis. It is a facultative anaerobic bacterium, capable of surviving in the presence of oxygen. It can grow and reproduce inside the host's cells and is one of the most virulent food-borne pathogens, with 20 to 30 percent of clinical infections resulting in death [9].

Epidemiology: *Listeria* species are widely distributed in the environment and can be isolated from soil, plants, decaying vegetation and silage (pH 5.5) in which the bacteria can multiply. Asymptomatic fecal carrier occurs in man and animal species. They can grow at temperature range of 3-4 [5].

Pathogenesis: *Listeria* originally evolved to invade membranes of the intestines, as an intracellular infection, and developed a chemical mechanism to do so. This involves a bacterial protein "internalin" which attaches to a protein on the intestinal cell membrane "cadherin". *L. monocytogenes* has also D-Galactose residues on its surface that can attach to D-Galactose receptors on the host cell walls. These host cells are generally M cells and Payer's patches of the intestinal mucosa. Once attached to these cells, *L. monocytogenes* can translocate past the intestinal membrane and into the body. *L. monocytogenes* may invade the gastrointestinal epithelium. Once the bacterium enters the host's monocytes, macrophages, or polymorphonuclear leukocytes, it becomes blood-borne (septicemic) and can grow. Its presence intracellularly in phagocytic cells also permits access to the brain and probably transplacental migration to the fetus in pregnant women [4].

Symptom: The symptoms of listeriosis usually last 7-10 days, with the most common symptoms being fever, muscle aches, and vomiting. Diarrhea is another, but less common symptom. If the infection spreads to the nervous system it can cause meningitis, an infection of the covering of the brain and spinal cord [1,3].

Detection of toxin: Enrichment procedures are required for this organism. This involves inoculation of selective or non-selective broths that are incubated at 4°C for up to 8 weeks. An ELISA, using monoclonal antibodies, has been developed to identify *listeria* in food, and also DNA probe for detection of bacterium in dairy products [14].

Control and prevention: The main means of prevention is through the promotion of safe handling, cooking and consumption of food. This includes washing raw vegetables and cooking raw food thoroughly, as well as reheating leftover or ready-to-eat foods like hot dogs until steaming hot (CDC, 2011). Preventing listeriosis as a food illness requires effective sanitation of food contact surfaces. Alcohol and Quaternary ammonium is an effective topical sanitizer against *Listeria*. Refrigerated foods in the home should be kept below 4°C (39.2°F) to discourage bacterial growth. Preventing listeriosis also can be done by carrying out an effective sanitation of food contact surfaces [9].

Conclusion

Bacterial food borne illnesses are among the most wide spread global public health problems of recent times, and their implication for health and economy is increasingly recognized. However, the true incidence of bacterial food borne illnesses are unknown for a number of reasons, including poor responses from victims during interviews with health officials, misdiagnosis of the illness, inadequate collection of samples for laboratory analysis and improper laboratory examination. The presence of various pathogenic bacteria in different foods poses a health hazard and rise concerns about the safety of these food products. In addition, there is a need to implement strict hygienic measures in the manufacturing, handling, storage and selling of food in order to guarantee the quality of these foods so as to minimize or eliminate the risk of FBI. To the best of our knowledge, this is the first review on major bacteria that causes FBI.

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