Bacterial aspect of Food Poisoning

Azza.S. M. Abu Elnaga[1]; Riham H, Hedia; Nagwa S, Ata[1] and Mona S , Zaki [2]

[1]Dept. Microbiology and Immunology, National Research Center, Giza, Egypt.
[2]Dept. Hydrobiology, National Research Center, Giza, Egypt.
Dr_mona_zaki@yahoo.co.uk

Abstract: Food poisoning syndrome results from ingestion of water and wide variety of food contaminated with pathogenic microorganisms (bacteria and / or their toxins). There are two kinds of food poisoning: poisoning by toxic agent or by infectious agent. The present literature view types of bacterial food poisoning, symptoms, prevention and treatment. The present study concluded that proper diagnosis of bacterial cause of food poisoning is necessary for the right way to treat and control either the best choice of antibiotic drugs or antitoxins. This will be achieved by the modern diagnostic immunological and molecular techniques
[Azza.S. M. Abu Elnaga; Riham H, Hedia; Nagwa S, Ata and Mona S, Zaki. **Bacterial aspect of Food Poisoning.** *Life Sci J* 2014;11(3):290-298]. (ISSN:1097-8135). http://www.lifesciencesite.com. 43

Key words: *Clostridium botulinum, Listeria monocytogene,* Shigellae, neurological signs, food-borne botulism

1. Introduction

Food poisoning (FP) is defined as any syndrome of an infectious or toxic nature caused by the consumption of food or drink. To date, around 250 different food-borne diseases have been described **(Gould *et al*., 2013).** The term is most often used to describe the illness, usually diarrhea and /or vomiting caused by bacteria, FP must be suspected when an acute illness with gastrointestinal or neurological manifestation affect two or more persons, who have shared a meal during the previous 72 hours **(Friedman and Rasooly, 2013).** Foods are particularly susceptible to contamination if not handled, stored or cooked properly include; raw meat and poultry, raw eggs, raw shellfish, unpasteurized milk, 'ready to eat' foods, such as cooked sliced meats, soft cheeses and pre-packed sandwiches **(Bodhidatta *et al*., 2013).**

The importance of foodborne diseases as a public health problem is often overlooked because their true incidence is difficult to be evaluated and the severity of their health and economic impact is often not fully understood **(Hassanain *et al.*, 2013)**.

Bacteria are considered the most common cause of foodborne illness representing two thirds of food-borne disease outbreaks and wide variety of microbes with much common and less specific clinical symptoms, so this review gives a light on these bacteria and their diagnosis, especially the application of new methods as molecular characterization of the bacteria or their toxins.

Bacterial FP is more commonly occurs after eating at picnics, school cafeterias, large social functions, or restaurants. The bacteria may get into the food you eat (called contamination) in different ways as Meat or poultry can come into contact with bacteria from the intestines of an animal that is being processed or Water that is used during growing or shipping can contain animal or human waste or Food handling or preparation in grocery stores, restaurants, or homes.

Food poisoning often occurs from eating or drinking any food prepared by someone who does not wash their hands properly, using cooking utensils, cutting boards, and other tools that are not fully cleaned. Dairy products that have been out of the refrigerator too long. Raw fish or oysters. Undercooked meats or eggs.Water from a well or stream, or city or town water that has not been treated **(Sodha *et al*., 2009).**

Classification of Bacterial Food Poisoning

I- According to The Cause

A- Food infection: refers to the presence of bacteria which infect the body after consumption

B- Food intoxication: refers to the ingestion of readily produced exotoxins within the food.

II- According to The duration of onset

A - Rapid within six hours: e.g. *Staphylococcus aureus* and *Bacillus cereus* emetic form.

B - Rapid within 8-16 hours: e.g. *Clostridium perfringens* and *Bacillus cereus* diarrheal form

C – Slow within 16-48 hours: e.g. Salmonellae, Shigellae, *Vibrio parahemolyticus* ,enteroinvasive *E.coli*, *Yersinia enterocolitica* and *Clostridium botulinum* .

D - Slow within 3-4 days: e.g. *Campylobacter jejun*i, and Enterohemorrhagic *E.coli*.

III- According to pathogenesis

A - Ingestion of preformed bacterial toxins: *S. aureus*, *B. cereus*, *C. botulinum* and *C. perfringens*

B-Noninvasive bacteria that secrete toxins while adhering to the intestinal wall: Enterotoxigenic *E. coli* and *C. jejuni*.

C - Follow an intracellular invasion of the intestinal epithelial cells: *Shigella* spp. and *Salmonella* spp.

D - Bacteria that enter the blood stream via the intestinal tract: *L. monocytogenes*.

IV- According to temperature

A - Psychrotrophic bacteria: Bacteria grow at 0 - 7°C but their optimum is from 20° to 30°C and cause food poisoning stored under refrigeration e.g. *Listeria,* and *Yersinia*.

B- Mesophilic bacteria: Most bacteria are capable of growing at 15 - 43°C and most pathogenic bacteria grow at these temperatures.

C-Toxins which can persist the high temperature degrees e.g. *Staphylococcal enterotoxins*.

V- According to oxygen requirement

A - Anaerobic: grow in the absence of oxygen: *Clostridia* spp.

B - Facultative anaerobic: Salmonellae

C - Microaerophilic: low oxygen concentration: Campylobacter *jejun*i (Porter *et al*., 2013).

Staphylococcus aureus

S. aureus is a facultative anaerobic Gram-positive spherical single or paired or form grape-like clusters, non-motile, catalase and coagulase positive. Staphylococcus bacteria are found on the skin and in the nose and throat of most people; people with colds and sinus infections are often carriers. The most commonly reported food sources are red meats, sausages, poultry, cheeses, milk, dairy products, egg, and fermented dairy products, such as soft cheeses and creams **(Pinchuk *et al.,* 2010)**.Staphylococcal food poisoning occurs most often in foods that require hand preparation. Sometimes these types of foods are left at room temperature for long periods of time. Although cooking destroys the bacteria, the toxin produced is heat stable and may not be destroyed. *S. aureus* produces a number of enterotoxins (SEs) that cause hyperemia and inflammation of the gastric mucosa. Symptoms include early nausea, vomiting, and abdominal cramping, with rare diarrhea. These usually appear within one to eight hours after eating staph-infected food and last one or two days. The illness seldom is fatal **(Kérouanton *et al.,* 2007)**. Genes encoding SEs have different genetic supports, most of which are embedded in mobile genetic elements. For example; *sea* is carried by a family of temperate phages. *seb* is chromosomally located in pathogenicity island in some clinical isolates, whereas it has been found in a 750-kb plasmid in other *S. aureus* strains as SEA gene amplified at 270 bp while SEB amplified at 165 bp **(Ataee *et al.,* 2011)**. Antibiotics are not recommended, as this illness occurs via pre-formed toxins, and not from direct infection with *S. aureus*.

Clostridium botulinum

C. botulinum is a Gram-positive, spore forming, non-motile anaerobic rod shape, causing death in approximately 30 % of the cases and occurs mostly in home-canned foods. *C. botulinum* can exist as a heat-resistant spore, and can grow and produce a neurotoxin (BoNT) in under processed, home-canned foods. An affected food may show signs of spoilage such as a bulging can or an off-odor. This is not true in all cases, so canned foods should not be tasted before heating. The BoNT is one of the most toxic substances known to the humankind. From primate experiments, the toxin has an extremely low minimal lethal dose (LD50), *i.e.*, 1 ng per kg body weight but destroyed by boiling the food for 10 minutes **(Barash and Arnon 2013)**.

The neurological signs in food-borne botulism may include abdominal cramps, nausea, and vomiting. The neurological form is characterized by symmetrical, descending, and flaccid paralysis of parasympathetic nerves. Symptoms of intoxication generally occur within 12 to 72 hours (range 2 hours to 8 days) after exposure; the symptoms usually begin with cranial nerve (face) paralysis, including drooping of the upper eyelids, double vision, blurred vision, difficulty in speaking, and difficulty in swallowing (dysphagia). The paralysis then develops to general weakness of several muscles such as arms, legs, and diaphragm which may lead to fatality if not treated properly and promptly. The severity of the disease varies from individual to individual and depending upon the amount of toxins consumed and absorbed **(Jeeraphong and Wanpen, 2011)**.

The identification of botulinum toxin in and around *C. botulinum* colonies grown on agar plates would facilitate the identification and isolation of *C. botulinum* and neurotoxin-producing *C. butyricum* and *C. baratii* among competitive microflora. Procedures based on immune-diffusion or immune-blotting have been published. As a single *C. botulinum* colony may produce as much as 10^5 minimal lethal doses (MLD) of toxin within 24 h , the colony immunoblot assay, which detects 10 to 25 50% MLD (MLD50) of toxin per spot, would be sufficiently sensitive for the identification of *C. botulinum* on an agar plate **(Lindström and Korkeala, 2006)** .

The treatment of botulism is based on supportive measures including artificial respiration and passive administration of human and animal (mainly horse) derived anti-BoNT immune globulin polyclonal antibodies (PAb) to the afflicted individual. Research efforts have been directed towards the development of a vaccine to prevent botulism. While there has not yet been a vaccine for botulism approved by the Food and Drug Administration (FDA), botulinum toxoids, subunit

and recombinant vaccines have been studied as potential vaccine candidates for botulism in both animals and humans **(Andrew *et al.*, 2012)**.

Clostridium perfringens

C. perfringens is a Gram-positive, rod-shaped, spore-forming, anaerobic bacterium found in soil, dust and the gastrointestinal tracts of animals and man. When food containing a large number of *C. perfringens* is consumed, the bacteria produce a toxin in the intestinal tract that causes illness but is not as severe as botulism and few deaths have occurred. *C. perfringens* can exist as a heat-resistant spore, so it may survive in cooking and grow to large numbers if the cooked food is held. Meat and poultry dishes, sauces and gravies are the foods most frequently involved. *C. perfringens* causes a toxin-mediated disease represents the 3rd most commonly reported food-borne illness **(Grass *et al.*, 2013)**. Some *C. perfringens* strains produce important toxin named *C. perfringens* enterotoxin (CPE), which is responsible for several human gastrointestinal diseases, therefore, detection of CPE produced by *C. perfringens* in feces specimens of ill individuals is a criterion for clinical diagnosis **(Lahti *et al.*, 2008)**.

Enteric toxins form pores or channels in host cell membranes and disrupt tight junctions. Symptoms occur within eight to 24 hours after contaminated food is eaten. They include acute abdominal pain and diarrhea. Nausea, vomiting and fever are less common. Recovery usually is within one to two days, but symptoms may persist for one or two weeks **(Bos *et al.*, 2005)**.

C. perfringens strains carrying the *cpe* gene on the chromosome, whether as vegetative cells or spores, usually possess much higher resistance properties against heat, cold, pH, and nitrites than type A strains carrying *cpe* on a plasmid. Proving that *C. perfringens* as the etiologic agent of an outbreak, epidemiologically link isolates obtained from patients with those found in suspected food vehicles undergo classical serotype assays; however, many *cpe*-positive food poisoning strains cannot be serotyped using existing antisera **(Li and McClane, 2008)**. **Udompijitkul et al. (2013)** developed a strategy to inactivate *C. perfringens* spores on stainless steel surfaces by inducing spore germination and killing of germinated spores with commonly used disinfectants.

Bacillus cereus

B. cereus is a Gram-positive, aerobic-to-facultative, spore-forming straight or slightly curved slender bacilli with square ends singly or in short chains widely distributed environmentally; found in dust, soil and spices. It can survive normal cooking as a heat-resistant spore, and then produce a large number of cells if the storage temperature is incorrect. The spores may be present on raw foods, and their ability to survive high cooking, meats, and dairy products are also reported as food sources. *B. cereus* may be the single most common cause of foodborne illnesses worldwide, but is rarely diagnosed in the clinical setting or reported to health agencies because symptoms tend to be short-lived and self-limited**(Hoffmaster *et al.*, 2006)**.

Victims often complain of transient abdominal discomfort, accompanied by 1-2 episodes of vomiting or diarrhea within 1-24 hours after exposure. Distinct toxins are responsible for two forms of gastrointestinal disease. Some serotypes of *B. cereus* produce a mild emetic illness mediated by a peptide gastric irritant called cereulide, while other serotypes produce a mild diarrheal illness mediated by hemolysin BL, which punches pores or channels into intestinal cell membranes and disrupts tight junctions. Vomiting and diarrhea rarely occur together with *B. cereus* **(Shiota *et al.*, 2010)**.

Severe intoxications caused by the *B. cereus* emetic toxin cereulide can hardly be prevented due to the ubiquitous distribution and heat resistance of spores and the extreme thermal and chemical stability of cereulide. It would therefore be desirable to inhibit cereulide synthesis during food manufacturing processes or in prepared foods, which are stored under time-temperature abuse conditions, the inhibition impacts of three long-chain polyphosphate (polyP) formulations on growth and cereulide production were established. Quantitative PCR (qPCR) monitoring at sub-lethal concentrations revealed that polyPs reduced the transcription of *ces*; nonribosomal peptide synthetase (NRPS) genes by 3- to 4-fold along with a significantly reduced toxin production level. At lower concentrations, toxin synthesis was decreased, although the growth rate was not affected **(Frenzel *et al.*, 2011).**

Majority of *B. cereus* strains were susceptible to many antibiotics but showed resistant to amoxyclav and cephalosporins. Rehydration and bowel rest are sufficient, and further treatment is rarely needed, given the short duration of symptoms. Young children may experience mild dehydration requiring intravenous fluids, especially if oral intake is poor. Cooked foods might be served hot or cooled rapidly to prevent the growth of this bacterium **(Banerjee *et al.*, 2011)**.

Salmonellae

Salmonella are Gram-negative rods, facultative anaerobic bacteria in the family of *Enterobacteriaceae*. It includes more than 2300 serotypes, but two types, *S. enteritidis* and *S. typhimurium* are the most common and account for half of the infections. The gastrointestinal tracts of animals and man are common sources of Salmonella with association of high protein foods such as meat,

poultry, fish and eggs, however, any food that becomes contaminated and is then held at improper temperatures can cause salmonellosis. Foods commonly involved include eggs or any egg-based food, salads (such as tuna, chicken, or potato), poultry, beef, pork, processed meats, meat pies, fish, cream desserts and fillings, sandwich fillings, raw sprouts, and milk products. These foods may be contaminated at any of the many points where the food is handled or processed from the time of slaughter or harvest until it is eaten. Salmonellae are spread through indirect or direct contact i.e., they may be spread to food by hands that are not washed after using the toilet. They also may be spread to raw meat during processing so that it is contaminated when brought into the kitchen **(Coburn et al., 2007)**. **Ahmed and Shimamoto (2014)** investigated the incidence of *Salmonella* among the Egyptian meat and dairy products (800 samples from each) collected from street vendors, butchers, retail markets and slaughterhouses as *S. enterica* serovars were detected in beef, raw milk, cheese and chicken meat 4.9,1.5, 0.5 1.8, % respectively.

Once eaten, the bacteria may continue to live and grow in the intestine, set up an infection and cause illness. *Salmonella* is phagocytosed by lympho-follicular M cells located in the follicle-associated epithelium in the Payer's patches, allowing invasion. Salmonella excretes a heat-labile enterotoxin causing an inflammatory diarrhea by invading the mucosa. The possibility and severity of the illness depends in large part on the size of the dose, the resistance of the host and the specific strain of *Salmonella* causing the illness. Symptoms of salmonellosis include headache, abdominal pain, nausea, chills, fever, vomiting, and diarrhea ranging from a few loose stools to a severe watery diarrhea. The majorities of stools are non-bloody and moderate in volume, although gross blood may occur. These occur within 8 to 72 hours after eating contaminated food and may last four to seven days. Arthritis symptoms may follow three to four weeks after onset of acute symptoms. Infants, young children, pregnant women, the elderly or people already ill have the least resistance to disease effects **(Sandt *et al.*, 2013)**.

Fever should be treated promptly with appropriate antibiotics (fluoroquinolones, ceftriaxone, azithromycin). Antimotility agent (e.g., loperamide) may decrease the number of stools, and probably does not prolong course or increase incidence of carrier state, but should be used with caution because of the increased potential for bacteria invasion and systemic spread **(Rodriguez *et al.*, 2009)**.

Salmonella bacteria grow at temperatures between 5 and 45oC. They are readily destroyed by cooking to 71oC and do not grow at refrigerator or freezer temperatures. They do survive refrigeration and freezing, however, and will begin to grow again once warmed to room temperature. It is important to make sure hands and working surfaces are thoroughly washed after contact with raw meat, fish and poultry and before working with foods that require no further cooking **(Sandt *et al.*, 2013)**.

Finally, it will be critical for public health systems worldwide to continue and expand systems that monitor emergence and frequency of different *Salmonella* serotypes. These continued efforts will be critical not only to identify emerging new *Salmonella* strains, but also to allow for an improved understanding of various factors that may be responsible for or contribute to emergence of new strains of *Salmonella*, which continues to be responsible for most deaths due to known foodborne pathogens in many countries around the world **(Raguenaud *et al.*, 2012)**.

Escherichia coli

E. coli are Gram- negative rods, facultative anaerobic bacteria of microorganisms called coliforms, belong to family *Enterobacteriaceae*., over 170 serogroups of *E. coli* are distinguished by the expression of O (somatic), H (flagellar), and/or K (capsular) antigens. Many strains of *E. coli* live peacefully in the gut, helping keep the growth of more harmful microorganisms in check. However, one public health strain, *E. coli* O157:H7, causes a distinctive and sometimes deadly disease. *E. coli* is a significant cause of diarrhea in developing countries and localities of poor sanitation **(Hartland and Leong, 2013).**

Food left outdoors for extended periods of time is particularly vulnerable to contamination by *E. coli*. Ground beef is the food most associated with *E. coli* O157:H7 outbreaks, but other foods also have been implicated. These include raw milk, dry-cured salami, cheese curds, raw unpasteurized cow's milk, semi-soft cheeses, raw seafood, meat, poultry, and untreated water. Infected food handlers and diapered infants with the disease likely help spread the bacteria **(Jaros et al., 2013).**

E. coli O157:H7 was detected in Egyptian beef, chicken meat, raw milk and cheese as 2.6, 0.5, 2.5 and 1.1% respectively and all *E. coli* O157:H7 isolates were positive for *stx*1 and/or *stx*2 virulence toxin genes **(Ahmed and Shimamoto, 2014)**. **Abd El-Aziz (2010)** recorded *E. coli* as higher percentage in soft cheese 20% and hard cheese16% while the organism was isolated from chicken luncheon, chicken sausage, beef burger and kofta as 12, 4, 8 and 12%, respectively. The *E. coli* serotypes isolated include *E. coli* O157:H7 in kofta as 4%, O125:H7 in all samples types except kofta and soft cheese, other

isolated serotypes are O8:H6, O159:H19, O27:H19, O55:H6 and O44:H4. It is found that the isolated *E. coli* O157:H7 has *stx*1 gene.

Symptoms begin with watery non-bloody diarrhea one to five days after eating contaminated food, dehydration, and severe cramps, and progress to bloody diarrhea, with occasional vomiting and fever. Gastrointestinal symptoms resolve within 14 days with supportive care. 10-15% of pediatric patients progress to hemolytic uremic syndrome (HUS), manifested by hemolytic anemia, and oliguric renal failure within 2 weeks of resolution of gastrointestinal symptoms. Mortality with intensive care is 3-5%. In adults, the complications sometimes lead to thrombocytopenic purpura (TPP), which characterized by cerebral nervous system deterioration, seizures and strokes **(Bloch *et al.*, 2012).**

Supportive care with rehydration and bowel rest is sufficient for the gastrointestinal phase of illness. Antibiotic therapy with a second- or third-generation cephalosporin (e.g., ceftriaxone) is generally recommended until stool cultures return. Definitive diagnosis is often delayed by EHEC serotyping, thus close communication with the laboratory regarding bacteria identification is recommended in cases in which empiric antibiotic therapy has been initiated. Once *E. coli* has been identified, discontinuation of antibiotics should be strongly considered **(Jensen *et al.*, 2006)**.

Preventive strategies for *E. coli* infections include thorough washing and other measures to reduce the presence of the microorganism on raw food, thorough cooking of raw animal products, and avoiding recontamination of cooked meat with raw meat. To be safe, cook ground meats to 75o C as well as personal hygiene of food handlers **(Infante *et al.*, 2005)**.

Shigellae

Shigella spp is a genus of Gram-negative, facultative anaerobic, non- spore forming, non-motile, rod-shaped bacteria closely related to *Salmonella*. Shigellae are found in undercooked meat and contaminated water, milk, or drinking water source with exposure to fecal contamination including cold food on commercial airlines. Handling of food by asymptomatic carriers may also transfer *Shigella* spp. to food. Symptoms include a brief nonspecific prodrome of anorexia, malaise, weakness and osmotic, watery diarrhea that may precede dysentery – followed by the very sudden onset of severe cramping, tenesmus, fever, myalgias, and frequent, small bloody, mucoid stools. Seizures may occur, particularly in children. *S. dysenteriae* usually produces very severe disease, in which patients may pass >20 dysenteric stools in one day. less severe cases, usually coming from other species, such as *S. sonnei* and symptoms may resolve spontaneously after 7 days **(Kim *et al.*, 2013).**

A molecular-based diagnostic assay which amplifies simultaneously four specific genes to identify *inv*C for *Shigella* genus, *rfc* for S. flexneri, *wbg*Z for *S. sonnei*, and *rfp*B for *S. dysenteriae*, as well as one internal control (*omp*A) gene, was developed in a single reaction to detect and differentiate *Shigella* spp., which yielded 100% specificity **(Ojha *et al.*, 2013).**

All cases of confirmed or suspected shigellosis should be treated with antimicrobials to avoid complications (especially seizures), disease recurrence, and secondary transmission. Adults may receive ceftriaxone, azithromycin, or quinolones in susceptible strains (resistance to sulfonamides, penicillins, and tetracyclines is prominent) **(Xiao *et al.*, 2012)**.

Yersinia enterocolitica

Yersinia enterocolitica are Gram-negative facultative anaerobic non- spore forming rod-shaped bacteria that belong to the family *Enterobacteriaceae*. Yersiniosis, infection caused by this microorganism, occurs most commonly in the form of gastroenteritis. Children are most severely affected. *Y. enterocolitica* is commonly present in foods and this organism is also can grow at refrigeration temperatures. It is sensitive to heat (5%) and acidity (pH 4.6), and will normally be inactivated by environmental conditions that will kill salmonellae **(Ong *et al.*, 2012).**

Symptoms of pseudoappendicitis have resulted in many unnecessary appendectomies. Death is rare and recovery is generally complete in 1-2 days. Arthritis has been identified as an infrequent but significant sequel of this infection **(Rosner *et al.*, 2010)**.

Campylobacter jejuni

Campylobacter spp. are motile, non-spore-forming, comma-shaped, Gram-negative rods reported as the most common cause of human acute bacterial entero –colitis. Raw and inadequately cooked foods of animal origin and non-chlorinated water are the most common sources of human infection (e.g., raw milk, undercooked chicken, raw hamburger, and raw shellfish). *C. jejuni* enteritis is primarily transferred from animal origin foods to humans in developed countries. However, fecal contamination of food and water and contact with sick people or animals predominates in developing countries. Although milk has been most frequently identified throughout the world to be a vehicle for Campylobacter, one anticipates that future investigations will identify poultry and its products

and meats (beef, pork, and lamb) as major reservoirs and vehicles **(Shimizu *et al.*, 2012)**.

C. jejuni colonizes in the colon and distal ileum, and releases a heat-labile, cytolethal-distending toxin (CDT), which: 1) attacks intestinal endothelial cell DNA, 2) recruits protein kinases to block cells from entering into mitosis, and 3) induces a local inflammatory reaction, cell lysis. Watery or bloody diarrhea, nausea, abdominal cramps, muscle pain, headache and fever are common symptoms. Onset usually occurs two to ten days after eating contaminated food. Duration is two to seven days, but can be weeks with such complications as urinary tract infections and reactive arthritis. Meningitis, recurrent colitis, acute cholecystitis, and Guillain-Barre syndrome are rare complications. Deaths, also rare, have been reported **(Jay-Russell *et al.*, 2013)**.

Treatment with macrolides (azithromycin) is recommended for 5-7 days to reduce illness duration by 5-10 days and to attenuate subsequent fecal shedding of bacteria **(Wimalarathna *et al.*, 2012)**.

Vibrio parahaemolyticus

Vibrio parahaemolyticus is a Gram-negative, halophilic (like saline media) asporogenous rod that is straight or has a single, rigid curve. It has a single polar flagellum and is motile when grown in liquid medium. *V. parahaemolyticus* is found on sea foods, and requires the salt environment of sea water for growth. *V. parahaemolyticus* is very sensitive to cold and heat. Proper storage of perishable sea foods below 4^{o}C, and subsequent cooking and holding above 60^{o}C, will destroy all the *V. parahaemolyticus* on sea foods. Food poisoning caused by this bacterium is a result of insufficient cooking and/or contamination of the cooked product by a raw product, followed by improper storage temperature. It is a major problem in East Asian countries where many sea foods are consumed raw. *V. vulnificus* is truly an emerging pathogen, but it can be controlled with proper cooking and refrigeration **(Shinoda ,s., 2011).**

V. parahaemolyticus strains showed the typical multidrug-resistance phenotype and were resistant to sulfamethoxazole, trimethoprim, cotrimoxazole, chloramphenicol, streptomycin, ampicillin, tetracycline, nalidixic acid, and gentamicin. The antibiotic resistance genes detected includes *dfr*18 and *dfr*A1 for trimethoprim; *flo*R, *tet*A, *str*B, *sul*2 for chloramphenicol, tetracycline, streptomycin and sulfamethoxazole respectively. Some of these genes were only recently described from clinical isolates, demonstrating genetic exchange between clinical and environmental *V. parahaemolyticus* species **(Okoh and Igbinosa, 2010).**

Listeria monocytogenes

L. monocytogenes is a Gram-positive ubiquitous (i.e. found in everywhere) bacterium which is widely distributed in the environment and is the most consistently pathogenic species causing diseases named listeriosis. It has been isolated from a variety of sources including soil, vegetation, silage, fecal material, sewage and water. The organism has survived the pH 5 environment of cottage cheese and ripening cheddar. It is salt tolerant surviving concentrations as high as 30.5 % and survives for 100 days at 4 ^{o} C, but only 5 days if held at 37 ^{o} C **(Goulet *et al.*, 2013)**.In humans, ingestion of the bacteria may be marked by a flu-like illness or symptoms may be so mild that they go unnoticed. A carrier state can develop. Death is rare in healthy adults; however, the mortality rate may approximate 30 % in the immunocompromised, new born or very young and among pregnant women, intrauterine or cervical infections may result in spontaneous abortion or still birth. Infants born alive may develop meningitis. The mortality rate in diagnosed cases is 20 to 25 percent. The incubation period is a few days to several weeks **(Mook *et al.*, 2011)**. Since high heat, greater than 80^{o} C, will inactivate *Listeria* organisms, post-process contamination from environmental sources then becomes a critical control point for many foods. Since *Listeria* will grow slowly at refrigeration temperatures, product rotation becomes even more important. Preventive measures for listeriosis include maintaining good sanitation, turning over refrigerated ready-to-eat foods quickly, pasteurizing milk, avoiding post-pasteurization contamination, and cooking foods thoroughly **(Chahad *et al.*, 2012)**.

Expectations (prognosis)

Most people fully recover from the most common types of food poisoning within 12 - 48 hours. Serious complications can occur, however, from certain types of food poisoning. Death from food poisoning in people who are otherwise healthy is rare in the United States.

Complications

Dehydration is the most common complication. This can occur from any causes of food poisoning. Less common, but much more serious complications depend on the bacteria that are causing the food poisoning. These may include: Arthritis Bleeding Damage to the nervous system Kidney problems swelling or irritation in the tissue around the heart **(Craig and Zich 2009).**

Treatment

The goal is to make you feel better and make sure your body has the proper amount of fluids. Getting enough fluids and learning what to eat will help keep you or your child comfortable. You may need to manage the diarrhea , control nausea and

vomiting and get plenty of rest. If you have diarrhea and are unable to drink or keep down fluids, you may need fluids given through a vein (by IV) especially for young children. If you take diuretics, ask your health care provider if you need to stop taking the diuretic while you have diarrhea. Never stop or change medications without first talking to your doctor.

Do not use these medicines without talking to your health care provider if you have bloody diarrhea, a fever, or the diarrhea is severe.Do not give these medicines to children **(Schiller and Sellin 2010).**

Prevention

The first step in preventing food poisoning is to assume that all foods may cause food-borne illness. Follow these steps to prevent food poisoning:

1. Wash hands, food preparation surfaces and utensils thoroughly before and after handling raw foods to prevent recontamination of cooked foods.
2. Keep refrigerated foods below 4°C.
3. Serve hot foods immediately or keep them heated above 60°C.
4. Divide large volumes of food into small portions for rapid cooling in the refrigerator. Hot, bulky foods in the refrigerator can raise the temperature of foods already cooled.
5. Remember the danger zone is between 4°C and 60°C.
6. Follow approved home-canning procedures. These can be obtained from the Extension Service or from USDA bulletins.

Infants, older persons, women who are pregnant and anyone with a compromised immune system are especially susceptible to food-borne illness. These people should never consume raw fish, raw seafood, or raw meat type products. You are the key to preventing food-borne illness. By observing the simple rules of good handling, food poisoning can be eliminated **(FDA, 2012).**

The general strategy of prevention of foodborne diseases require the application of surveillance and information systems for these diseases and Hazard Analysis and Critical Control Points (HACCP) system in the food production chain from farm to consumers and import/export regulations. Reducing microbiological contamination in meat can be achieved through environmental hygiene, best quality of livestock feed, animal vaccination, mandatory inspection of livestock as well as sanitary standards for slaughter-houses **(Hassanain *et al.*, 2013)**.

References

1. Abd El-Aziz, A.E. (2010): Epidemiological studies on bacterial food poisoning from animal products. M.V.Sc. (Zoonoses) Faculty of Veterinary Medicine Cairo Univerisity.
2. Ahmed, M. A and Shimamoto T. (2014): Isolation and molecular characterization of *Salmonella enterica*, *Escherichia coli* O157:H7 and *Shigella* spp. from meat and dairy products in Egypt. International Journal of Food Microbiology 168–169: 57–62.
3. Andrew, P. Karalewitz, A. and Joseph, T. B. (2012): Vaccines against botulism. Current Opinion in Microbiology 15:317–324.
4. Ataee, R.A.; Karami, A.; Izadi, M.; Aghania, A. and Ataee, M.H. (2011): Molecular screening of staphylococcal enterotoxin B gene in clinical isolates. Cell J. Fall 13(3):187-92.
5. Banerjee, M.; Nair, G.B. and Ramamurthy, T. (2011): Phenotypic & genetic characterization of *Bacillus cereus* isolated from the acute diarrheal patients. Indian J Med Res. 133:88-95.
6. Barash, J.R. and Arnon, S.S.(2013): A Novel Strain of *Clostridium botulinum* That Produces Type B and Type H Botulinum Toxins. J Infect Dis. Under publishing
7. Bloch, S.K.; Felczykowska, A. and Nejman-Faleńczyk, B.(2012): Escherichia coli O104:H4 outbreak--have we learnt a lesson from it?. Acta Biochim Pol. 59(4):483-8.
8. Bodhidatta, L.; Srijan, A.; Serichantalergs, O.; Bangtrakulnonth ,A.; Wongstitwilairung, B.; McDaniel, P. and Mason, C.J. (2013): Bacterial pathogens isolated from raw meat and poultry compared with pathogens isolated from children in the same area of rural Thailand. Southeast Asian J Trop Med Public Health. 44(2):259-72.
9. Bos, J.; Smithee, L.; McClane,B.; Distefano, R.F.; Uzal, F.; Songer, J.G.; Mallonee, S. and Crutcher, J.M.(2005): Fatal necrotizing colitis following a foodborne outbreak of enterotoxigenic *Clostridium perfringens* type A infection. Clin Infect Dis. 40(10):e78-83.
10. Chahad, O.B.; El Bour, M.; Calo-Mata, P.; Boudabous, A. and Barros-Velàzquez, J. (2012): Discovery of novel bio-preservation agents with inhibitory effects on growth of food-borne pathogens and their application to seafood products. Res Microbiol. 163(1):44-54.
11. Coburn, B.; Grassl, G.A. and Finlay, B.B. (2007): *Salmonella*, the host and disease: a brief review. Immunol Cell Biol. 85(2):112-8.
12. Craig, S.A. and Zich, D.K. (2009): Gastroenteritis. In: Marx JA, ed. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th ed. Philadelphia, Pa: Mosby Elsevier; chap 92.
13. Food and Drug Administration (2012): U.S. Department of Health and Human Services. *Bad*

Bug Book – Foodborne Pathogenic Microorganisms and Natural Toxins Handbook, 2nd ed.

14. Frenzel, E.; Letzel, T.; Scherer, S. and Ehling-Schulz, M. (2011): Inhibition of cereulide toxin synthesis by emetic *Bacillus cereus* via long-chain polyphosphates. Appl Environ Microbiol. 77(4):1475-82.
15. Friedman, M. and Rasooly, R. (2013): Review of the inhibition of biological activities of food-related selected toxins by natural compounds. Toxins 5(4):743-75.
16. Gould, L.H.; Walsh, K.A.; Vieira, A.R.; Herman, K.; Williams, I.T.; Hall, A.J. and Cole, D. (2013): Surveillance for foodborne disease outbreaks - United States, 1998-2008.Centers for Disease Control and Prevention. MMWR Surveill Summ. 62(2):1-34.
17. Goulet, V.; King, L.A.; Vaillant, V. and de Valk, H. (2013): What is the incubation period for listeriosis? BMC Infect Dis. 10(13):11.
18. Grass, JE.; Gould, L.H. and Mahon, B.E. (2013): Epidemiology of food borne disease outbreaks caused by *Clostridium perfringens*, United States, 1998-2010. Food borne Pathog Dis. 10(2):131-6.
19. Hartland, E.L. and Leong, J.M.(2013): Enteropathogenic and enterohemorrhagic *E. coli*: ecology, pathogenesis, and evolution. Front Cell Infect Microbiol. 3:15.
20. Hassanain, N. A.; Hassanain, M. A.; Ahmed, W. M.; Shapaan, R. M.; Barakat, A. M. and El Fadaly, H. A.M. (2013): Public Health Importance of Foodborne Pathogens. World J Med Sci 9 (4): 208-222.
21. Hoffmaster, A. R.; Hill, K. H.; Gee, D. J. E.; Marston, C. H. and Jackson. P. J. (2006): Characterization of *Bacillus cereus* isolates associated with fatal pneumonias: strains are closely related to *Bacillus anthracis* and harbor *B. anthracis* virulence genes. J Clin Microbiol. 44 (9):3352-60.
22. Infante, B.; Grape, M. and Larsson, M. (2005): Acquired sulphonamide resistance genes in fecal *Escherichia coli* from healthy children in Bolivia and Peru. Int J Antimicrob Agents 25:308-12.
23. Jaros, P.; Cookson, A.L.; Campbell, D.M.; Besser, T.E.; Shring, S. and French, N.P.(2013): A prospective case control and molecular epidemiological study of human cases of Shiga toxin-producing *Escherichia coli* in New Zealand. BMC Infect Dis. 13:450.
24. Jay-Russel, l M.T.; Mandrell, R.E.; Yuan, J.; A. and Miller, W.G.(2013): Using major outer membrane protein typing as an epidemiological tool to investigate outbreaks caused by milk borne *Campylobacter jejuni* isolates in California. J. Clin. Microbiol. 51(1):195-201.
25. Jeeraphong, T. and Wanpen, C. (2011): Botulinum Neurotoxins and Botulism: A Novel Therapeutic Approach. Toxins, 3, 469-488.
26. Jensen, V.F.; Jakobsen, L.; Emborg, H.D.; Seyfa rth, A.M. and Hammerum, A.M. (2006): Correlation between apramycin and gentamicin use in pigs and an increasing reservoir of gentamicin-resistant *Escherichia coli*. J Antimicrob Chemother 58:1017.
27. Kérouanton, A.; Hennekinne, J.A.; Letertre, C.; Petit, L.; Chesneau, O.; Brisabois, A. and De Buyser, M.L.(2007): Characterization of *Staphylococcus aureus* strains associated with food poisoning outbreaks France. Int J Food Microbiol. 115(3):369-75.
28. Kim, H.J.; Youn, S.K.; Lee, S. and Choi, Y.H. (2013): Epidemiological characteristics of imported shigellosis in Korea, 2010-2011. Osong Public Health Res Perspect. 4(3):159-65.
29. Lahti, P.; Heikinheimo, A.; Johansson, T. and Korkeala, H. (2008): *Clostridium perfringens* type A isolates carrying the plasmid-borne enterotoxin gene (genotype IS1151-cpe or IS1470-like-cpe) are a common cause of food poisoning. J. Clin. Microbiol. 46:371–373.
30. Li, J. and McClane, B.A. (2008): A novel small acid soluble protein variant is important for spore resistance of most *Clostridium perfringens* food poisoning isolates. PLoS Pathog. 4:e1000056.
31. Lindström, M. and Korkeala, H. (2006): Laboratory diagnostics of botulism. Clin Microbiol Rev. 19 (2):298-314.
32. Mook, P.; O'Brien, S.J. and Gillespie, I.A.(2011): Concurrent conditions and human listeriosis, England, 1999–2009. Emerg Infect Dis 17:38–43.
33. Ojha, S. C.; Yean, Yean C.; Ismail, A. and Singh, K.K. (2013): A pentaplex PCR assay for the detection and differentiation of *Shigella* species. Biomed Res Int. 2013:412370.
34. Okoh, A.I. and Igbinosa, E.O.(2010): Antibiotic susceptibility profiles of some *Vibrio* strains isolated from wastewater final effluents in a rural community of the Eastern Cape Province of South Africa. BMC Microbiol. 10:143.
35. Ong, K.L.; Gould, L.H.; Chen, D.L.; Jones, T.F. and Mahon, B.E. (2012): Changing epidemiology of *Yersinia enterocolitica* infections: markedly decreased rates in young black children, Foodborne Diseases Active Surveillance Network (FoodNet), 1996-2009. Clin Infect Dis. 54 Suppl 5:S385-90.

36. Pinchuk, I.V.; Beswick, E.J. and Reyes, V.E. (2010): Staphylococcal enterotoxins. Toxins 2(8):2177-97.
37. Raguenaud ,M.E.; Le Hello, S.; Salah, S.; Weill, F.X.; Brisabois, A.; Germonneau, P. and Delmas, G. (2012): Epidemiological and microbiological investigation of a large outbreak of monophasic *Salmonella Typhimurium* 4, 5, 1 i: in schools associated with imported beef in Poitiers, France, October 2010. Euro Surveill. 17(40):20289.
38. Rodriguez, I.; Barownick, W.; Helmuth, R.; Mendoza, M.C.; Rodicio, M.R.; Schroeter, A. and Guerra, B. (2009): Extended-spectrum -lactamases and *Amp*C -lactamases in ceftiofur-resistant *Salmonella enterica* isolates from food and livestock obtained in Germany during 2003–07. J. Antimicrob. Chemother. 64:301–309.
39. Rosner, B.M.; Stark, K. and Werber, D. (2010): Epidemiology of reported *Yersinia enterocolitica* infections in Germany, 2001-2008. BMC Public Health. 14(10):337.
40. Sandt, C.H.; Fedorka-Cray, P.J.; Tewari,D.; Ostroff, S.; Joyce, K.; M'ikanatha, N.M. (2013): A comparison of nont-typhoidal *Salmonella* from humans and food animals using pulsed-field gel electrophoresis and antimicrobial susceptibility patterns. PLoS One. 8(10):e77836.
41. Schiller, L.R. and Sellin, J.H. (2010): Diarrhea. In: Feldman M, Friedman LS, Brandt LJ, eds. *Sleisenger & Fordtran's Gastrointestinal and Liver Disease*. 9th ed. Philadelphia, Pa: Saunders Elsevier; chap 15.
42. Shimizu, Y.; Ishii, A.; Takahata, A.; Kajiyama, T.; Yamahatsu, A. and Tomino,Y.(2012): Campylobacter bacteremia in hemo-dialysis patients by eating raw meat the importance of sanitary education. Case Rep Nephrol Urol. 2(2):145-51.
43. Shinoda, S. (2011): Sixty years from the discovery of *Vibrio parahaemolyticus* and some recollections. Biocontrol Sci. 16(4):129-37.
44. Shiota, M.; Saitou, K.; Mizumoto, H. and Matsusaka, M. (2010): Rapid detoxification of cereulide in *Bacillus cereus* food poisoning. Pediatrics 125:e951–e955.
45. Sodha, S.V.; Griffin, P.M. and Hughes, J.M. (2009): Foodborne disease. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and Practice of Infectious Diseases*. 7th ed. Philadelphia, Pa: Elsevier Churchill Livingstone; chap 99.
46. Udompijitkul, P.; Alnoman, M; Paredes-Sabjaa, D; Sarker, M.R. (2013): Inactivation strategy for *Clostridium perfringens* spores adhered to food contact surfaces. Food Microbiol. 34(2):328-36.
47. Wimalarathna, H.M.; Richardson, J.F.; Lawson, A.J.; Elson, R.; Meldrum, R. and Sheppard, S.K. (2013): Widespread acquisition of antimicrobial resistance among *Campylobacter* isolates from UK retail poultry and evidence for clonal expansion of resistant lineages. BMC Microbiol. 15 (13):160.
48. Xiao, G.G.; Fan, J.; Deng, J.J.; Chen, C.H.; Zhou, W. and Li, X.H.(2012) : A school outbreak of *Shigella sonnei* infection in China: clinical features, antibiotic susceptibility and molecular epidemiology. Indian Pediatr. 49(4):287-90.

2/13/2014