Journal of the Egyptian Society of Parasitology, Vol. 53, No.1, April 2023
J. Egypt. Soc. Parasitol. (JESP), 53(1), 2023: 165– 184
Online: 2090-2549

ASSESSMENT ON FOODBORNE DISEASES (GASTROENTERITIS) WITH INCREASED CLIMATIC CONDITIONS: A REVIEW ARTCILE

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Abstract

Foodborne illness is caused by consuming contaminated foods or beverages. Others are caused by harmful toxins or chemicals that have contaminated food. Commonly recognized foodborne infections are: campylobacteriosis, cryptosporidiosis, cyclosporiasis, *Escherichia coli* O157:H7, and hemolytic uremic syndrome, giardiasis, listeriosis, norovirus (aka norwalk virus, calicivirus, viral gastroenteritis), salmonellosis, scombroid fish poisoning, shigellosis, toxoplasmosis, vibrio infection, and yersiniosis.

Key words: Food-poisoning, Bacteria, Chemicals, Parasites, Virus, Diagnosis, Treatment, Nursing.

Introduction

Food-borne illness accounts for about 76 million cases, 323,000 hospitalizations, and 5000 deaths annually in the United States (Mead *et al,* 1999). Clinical manifestations ranged from mild gastroenteritis to life-threatening neurologic, hepatic, and renal syndromes (Santosham *et al,* 1997). Although food-borne illnesses attack patients in all age groups, young children, the elderly and immunocompromised hosts are at highest risk for serious consequences from these diseases. The hospitalized rate for bacterial enteric diseases in children, for example, rose during 1990 to 1994 compared with 1985 to 1989: 100.8 from 57.5/100,000 hospital discharges in children <1 year & 21.5 from 7.5/100,000 in those one to four years (Frost *et al,* 1998). An estimated 300 to 400 children in the United States died annually from diarrheal illness (Glass *et al,* 1991).

Definitions: The CDC defines a food-borne disease outbreak as any cluster of two or more individuals who develop similar symptoms following ingestion of a common food. Laboratory or clinical guidelines to confirm an etiology of a food-borne disease outbreak vary for bacterial, chemical, parasitic, & viral agents (Bean *et al,* 1996). If the agent of a food-borne outbreak can't be determined, incubation times are used i.e., <1hr = probable chemical ingestion; 1 to 7hrs = probable *Staphylococcus aureus* or *Bacillus cereuus* toxin mediated; 8 to 14hrs = others, especially bacteria; ≥15hrs = others, especially viruses (WHO, 2017).

Review and Discussion

Epidemiology: CDC (1973) maintained a collaborative surveillance program for food-borne diseases but recognizes that this system results in underestimate incidences. During 1998-2002, a total of 6647 outbreaks affected more than 128,000 people were reported. The annual number of outbreaks reported to the CDC increased during this period compared with previous years (Olsen *et al,* 2000), probably due to the implementation of measures to enhance outbreak surveillance. The following findings were noted (Lynch *et al,* 2006): 1- Among 2167 outbreaks of determined etiology (only about a third have a defined cause), bacterial pathogens caused largest outbreaks and cases (55%), 2- Of bacterial pathogens, *Salmonella* accounted for the largest number of outbreaks, and *Listeria monocytogenes* accounted for the majority of any pathogen deaths, 3- Viral pathogens, predominantly noro-virus, caused 33% of outbreaks, 4- Outbreaks attributed to viral agents increased from 16% in 1998 to 42%

in 2002, probably reflecting the increased availability of improved viral diagnostics tests, 5- Chemical agents caused 10% of outbreaks, & 6- Parasites caused 1%of outbreaks

Contributing factors: Many factors contribute to the emergence of food-borne disease in the United States and worldwide. They include changes in human behavior and demographics, advances in industry and technology, lack of adequate public health infrastructure, and changing antimicrobial susceptibility patterns of organisms. Commercially or institutionally prepared food causes 79% of cases of food poisoning. Increasing number of meals eaten either away from outdoors or brought to the home for immediate consumption after being purchased elsewhere created greater demand for food from catering services, restaurants, and institutional kitchens. Burgeoning market for quick, convenient, and inexpensive food renders careful handling and storage of foods mandatory (Altekruse *et al,* 1997).

Improper food storage: Holding and food storage at improper temperatures were the commonest contributors to foodborne disease from 1983 through 1992, with (CDC) up to 97% (Shewmake and Dillon, 1998). It included leaving prepared food at temperatures that allow bacteria to grow, improperly heating or reheating food, and allowing cross food contamination, which occur either through direct contact or following contact with contaminated food preparation surfaces (Kirchner *et al,* 2021)

Food handlers: Personal hygiene practices of food handlers were a common cause of foodborne illness (Zanin *et al,* 2017). Fecal-oral transmission resulted from poor hygiene, particularly failure to wash hands after using bathroom facilities. The most commonly reported contamination factor for foodborne outbreaks during 1998-2002 was barehanded contact by handler/worker/preparer (Collins, 1997).

Widespread distribution of foods: Small farms, factories, and dairies replaced with large factories supplying millions of individuals in far-reaching parts of the world, magnifying the impact of a single contamination (Nightingale, 1987). Small outbreaks might be unrelated, and tracing the source of contamination becomes a greater challenge. All of these factors added to the opportunity for outbreaks of food-borne illness that could affect hundreds to thousands of people.

Emerging microbial agents: A wide variety of microorganisms can cause food poisoning. *Salmonella* sp. produces the outbreaks majority of food-borne disease when an etiology can be established. However, *Escherichia coli* have become frequent food-borne pathogens.

Salmonella: Salmonella is the number-one cause of food-borne illness in the United States, accounted to 585 outbreaks and more than 16,000 cases between 1998 & 2002. It was the second commonest cause of death from foodborne pathogens there. Non-typhoidal *Salmonellae* are associated with animal reservoirs and therefore with agricultural products, especially eggs and poultry. Clinical illness spectrum ranges from acute gastroenteritis to occult bacteremia and typhoid fever (Udall, 1996).

Non-typhoidal salmonellosis refers to illnesses caused by all serotypes of *Salmonella* except for *Typhi, Paratyphi* A, *Paratyphi* B (tartrate negative), and *Paratyphi* **C**. non-typhoidal salmonellae are one of the leading causes of bacterial diarrhea worldwide; approximately 153 million cases of gastroenteritis and 57,000 deaths globally each year. Salmonellosis risk was among travelers returned to the USA varied by visited regions; highest risk was among those back from Africa (incidence of 25.8 cases /100,000 air travelers), Latin America and the Caribbean (7.1 cases/100,000), & Asia (5.8cases/100,000). A systematic review of travelers' diarrheal salmonellosis (included typhoidal serotypes) was detected in <5% of patients who traveled to Latin America, the Caribbean, and South Asia & in 5%-15% of patients who traveled to Africa or South-east Asia. *Salmonella* infection and carriage was among intern-

ationally adopted children (CDC, 2019).

Antimicrobial resistance among *Salmonella* isolates became a major problem, partially due to extensive antimicrobial drugs use in livestock (Su *et al,* 2004) Multidrug-resistant *S. typhimurium* DT104 has emerged as a cause of salmonellosis in Europe and Americas. In an outbreak, Mexican-style soft cheese made with unpasteurized milk was an important vehicle for children (Villar *et al,* 1999). CDC (2023) estimated *Salmonella* bacteria cause about 1.35 million infections, 26,500 hospitalizations, & 420 deaths in the United States annually. Food is the source for most of these illnesses. Most people who get ill from *Salmonella* have diarrhea, fever, and stomach cramps; usually begin 6 hours to 6 days after infection and last 4 to 7 days. Most people recover without specific treatment and should not take antibiotics. Antibiotics are typically used only to treat people who have severe illness or who are at risk for it. But, some people's illness was so severe that they need to be hospitalized. While occult non-typhoidal *Salmonella* bacteremia in children accounted for <0.2% of fevers (≥39.0ºC) in outpatients 3 to 36 months of age, severe complications resulted in a case was neither recognized nor treated. Persistent bacteremia, osteomyelitis, meningitis, sepsis, and death were reported much more common in infants less than three months or immunocompromised older ones (Kuppermann, 1999).

Campylobacter: Campylobacter jejuni and *C. coli* commonly cause diarrhea in man, and 15 species of *Campylobacter* were identified (Yang *et al,* 2008) *Campylobacter* sp. caused < 1% of food-borne outbreaks in the United States, but foodborne illness estimated to be responsible for more than 2 million cases of diarrhea (Scallan *et al,* 2011).

The organism is transmitted primarily via contaminated food. Risk factors for disease include handling raw chicken, eating raw or undercooked chicken or ducks, or contact with farm animals (cattle and pigs) or cats and dogs, or consuming untreated water, raw milk, or eggs with possible zoonoses (Cornick and Gorbach, 1988). Transmission occurs via fecal-oral route by ingestion of contaminated food and/or water (Facciolà *et al,* 2017). It was isolated most commonly in the first life year with rates of 15/100,000 with male to female ratio was 1.3:1. It caused disease ranged from asymptomatic secretion to frank dysentery. It was unique in that constitutional symptoms such as coryza, headache and general malaise accompanied with diarrhea persist for two weeks (Tauxe *et al,* 1988). *Campylobacter* infection is diagnosed when a laboratory diagnosed *Campylobacter* bacteria in stool (poop), body tissue, or fluids. It could be a culture to isolate the bacteria or a rapid diagnostic test to detect genetic material of the bacteria. Most people recover from *Campylobacter* infection without antibiotic treatment. The patients must drink extra fluids as long as diarrhea lasts. However, some people with, or at risk for, severe illness might need antibiotic treatment. These people include those who are 65 years or older, pregnant women, and people with weakened immune systems, such as those with a blood disorder, with AIDS, or receiving chemotherapy (CDC, 2023a).

Most of *C. jejuni* infected patients have a self-limited illness. But, a substantial proportion of them are treated with antibiotics.

Escherichia coli: E. coli is a prominent constituent of normal stool flora. Diarrheagenic *E. coli* were defined by the specific virulence genes and include enteropathogenic *E. coli* (EPEC), enteroinvasi ve *E. coli* (EIEC), enterotoxigenic *E. coli* (ETEC), enteroaggregative *E. coli* (EAggEC also called EAEC), enterohemorrhagic *E. coli* (EHEC), & diffusely adherent *E. coli* (DAEC). ETEC, EPEC, & EAEC were commonly associated with diarrhea in the Developing World but, were infrequently recognized in the Developed World. DAEC and EIEC were uncommon in both developed and developing world. The precise mechanisms of the illness associated with EAEC, EPEC, & DAEC were not defined. Enterotoxigenic and enterohemorrhag-

ic are still the most common *E. coli* strains to produce food-borne illness worldwide.

Enterohemorrhagic *E. coli:* Enterohemorrhagic *E. coli* (EHEC) infection is characterized by bloody stool with little or no fever. EHEC was first recognized as a human pathogen in 1982, after an outbreak of severe bloody diarrhea in individuals who had consumed hamburgers from a fast-food chain (Lederberg, 1997). EHEC, also known as the *Shiga* toxin-producing *E. coli* (STEC), or verotoxin-producing *E. coli* (VTEC), produce Shiga toxins (Stx1, Stx2, & variants of Stx2); *E. coli* O157:H7 was the prevalent strain in the United States (Bower, 1999). But, this prevalence might be secondary to the fact that only this strain identified in most clinical laboratories. *E. coli* O157:H7 was the only enterohemorrhagic stool strain for testing in biological laboratories. An outbreak of hemorrhagic colitis and hemolytic uremic syndrome (HUS) in Europe that began in May 2011 was unusual in that the organism was an EAEC with Shigatoxin 2 genes.

Improperly cooked beef and unpasteurized milk traditionally have been the source of EHEC ingestion. Healthy cattle, dogs, deer, sheep, horses, and goats serve as reservoirs. However, *E. coli* O157:H7 can be transmitted by food or water or from person to person. Young children carry the organism longer than do older children or adults. The causative organism was isolated from manure, as well as water troughs. Outbreaks were traced back to tainted fruits and vegetables. Radish sprouts, lettuce, unpasteurized apple juice and cider (CDC, 1999a), alfalfa sprouts, and spinach have been implicated.

Because of difficulties in organism detection, since *E. coli* is a prominent constituent of normal stool flora, the number of cases probably was an underestimation. In many subclinical disease and lack of appropriate test in sorbitol-MacConkey agar led to great underreporting (Mead and Griffin, 1998). The best way to identify all STEC infections was to screen stools for Shiga toxins by enzyme immunoassay (EIA) or PRC, but, laboratory must be STEC cultured (CDC, 2005)

A multistate outbreak of *E. coli* O157:H7 infections from fresh spinach were reported in the United States in September-October 2006, nearly 200 individuals from 26 states who were infected with the outbreak strain were reported to the CDC (2011). Approximately 50% of cases were hospitalized and 16% HUS. Among those developed HUS, 29% were children (<18 years old), 3% were between 18 & 59 years of age, and 14% were ≥60 years. At least three confirmed deaths were associated with the outbreak. The 2011 European outbreak with the Stx2 producing EAEC strain serotype O104:H4 was unusual in that life threatening HUS commonly occurred in adults.

EHEC strains (particularly O157:H7, but also O26:H11, O103, O111: NM, as well as less common serotypes) were linked with HUS development. HUS is the leading cause of renal failure in children; it was characterized by the microangiopathic hemolytic anemia, thrombocytopenia and renal failure. The HUS complicated 5 to 15% of EHEC infections started during first few days of diarrhea or ten days after onset (Lutter, 1999).

In multivariate analyses, a retrospective study identified use of anti-motility agents within the first three days of the development of diarrhea and vomiting for patients under the age of five years as risk factors for HUS (Bell *et al,* 1997). A prospective study of 71 children younger than 10 years of age with *E. coli* O157:H7 isolated from stool found that those receiving antibiotics were more likely to develop HUS (5/9 versus 5/62, P = 0.002). It is unclear whether these associations reflect therapy given to more severe diarrheal illnesses or whether these agents are actually contributing to the pathogenesis of HUS (Wong *et al,* 2000). The genes for toxin production are carried on phages. It is possible that early administration of some antibiotics (e.g., trimethoprim-sulfamethoxazole) to these children may promote the HUS development by enhancing release of Shiga toxins via induction of ph-

age-mediated lysis. Hemolytic uremic syndrome (HUS) is defined by occurrence of microangiopathic hemolytic anemia, thrombocytopenia, and acute kidney injury, mainly due to Shiga toxin-producing *Escherichia coli* (STEC), which is one of the main causes of acute kidney injury in children less three years (Noris and Remuzzi, 2005)

Enterotoxigenic *E. coli* (ETEC) is one of the commonest causes of dehydrating diarrhea in children less than two years of age in the developing world (Black *et al,* 1982). These strains also frequently produce diarrhea in travelers to tropical regions as a result of exposed to contaminated food and water. ETEC, in contrast to EHEC, usually induces watery diarrhea caused by secretory toxins production (Nataro and Kaper, 1998). The illness duration varied, but generally it was 24 to 72 hours. Appropriate treatment is oral rehydration therapy, unless the diarrhea is severe and complicated by shock, coma, or ileus; in such cases, intravenous therapy is required. Priorities in the management of dehydration must include early recognized symptoms, identifying the degree of dehydration, stabilization, and rehydration strategies (Falszewska *et al,* 2018).

Differential diagnosis: 1- Hypernatremia in emergency, 2- Hyperosmolar Hyperglycemic Nonketoic coma, 3- Hypokalemia in emergency, 4- Hyponatremia in emergency, 5- Hypovolemic shock, 6- Metabolic acidosis in emergency, 7- Pediatric gastroenteritis in emergency, and 8-Pediatric pyloric stenosis (Vega and Avva, 2022)

Shigella is a member of the Gram-negative Enterobacteriaceae family, and current classification divides genus into four species based on serological typing: *S. dysenteriae, S. boydii*, *S. flexneri* and *S. sonnei*. Ingestion, which typically has a low infectious dose, commonly results in an aggressive watery or mucoid/bloody diarrhea (Baker and Rutter, 2023). *Shigella* causes classic bacillary dysentery and acute watery diarrhea. Shigellosis is still a major cause of diarrhea in the developing world; shigellosis can be acquired by fecal-oral contact or ingestion of contaminated food or water (Ahmed *et al,* 1997). World incidence of shigellosis was about 165 million cases; but mortality decreased over the past three decades (Kotloff *et al,* 1999).

Shigella sonnei is the commonest species of laboratory-confirmed *Shigella* infection in the United States and usually causes an acute, self-limited diarrheal illness. *S. dysenteriae* 1 produces Shiga toxin, sometimes complicated by HUS (Jalal *et al,* 2022). Seizures occurred in children with risk factors of fever, dehydration, hypoglycemia, hyponatremia, and meningitis (Khan *et al,* 1999).

Numerous outbreaks of multidrug-resistant *S. sonnei* infection have been associated with day-care centers (CDC, 2006a). As few bacteria are required to transmit shigellosis from person to person via fecal-oral route, shigellosis can propagate in settings with the less hygiene practices. Sometimes ingestion of as few as 10 organisms can cause illness. Consumption of cold salads, such as potato or macaroni salads, has been implicated in several common source outbreaks.

Shigella flexneri was a leading diarrhea agent in low socioeconomic countries with various serotypes in ***S****. flexneri* from the different regions of the world (Nisa *et al,* 2020). Treatment as *Shigella* infection, irrespective of dysentery status, is associated with death; antibiotics might play a critical role in treating non-dysenteric *Shigella* infection. But, an antibiotic resistance developed quickly in *Shigella* infections (Gu *et al,* 2015). The early dependable diagnosis and efficacy treatment could minimize mortality and morbidity in non-dysenteric children and decrease antimicrobial resistance among *Shigella* isolates (Tickell *et al,* 2017). Since the *Shigella* manifestations can include the watery diarrhoea, dysentery, or complications such as encephalopathy, WHO (2005) diarrhea guidelines focused on rehydration, and the provision of zinc, and they specifically addressed *Shigella* infections by recommending ciprofloxacin be given to children with dysentery, defined as observed presence or caregiver-

report of blood in the patient's stool.

Staphylococcus aureus: S. aureus can frequently colonize the skin and mucosa of a high percentage of healthy humans: about 50% of the healthy adults can spread *S. aureus* (i.e., nosocomial) throughout their nasal mucosae (Kluytmans and Wertheim, 2005). Illness is caused by toxins elaborated by the organism. Symptoms may begin as early as 30 minutes or as late as eight hours after eating contaminated food; illness is characterized by sudden onset of nausea, vomiting, and diarrhea. The incubation period and disease severity depend upon individual host factors and the amount of inoculum. The disease usually is self-limited, but deaths were reported. Foods containing staphylococcal enterotoxin usually look and taste normal (Gallina *et al,* 2013).

Cooking didn't destroy either the organism or toxin. The organism and toxin proliferate in temperatures between 68ºF & 99ºF. Precooked, prepackaged meats with high salt contents are best vehicles for Staphylococci. Staphylococcal disease was reported most often as a result of ham ingestion (CDC, 1983). An outbreak of community-acquired foodborne illness by methicillin-resistant *S. aureus* occurred (Jones *et al,* 2002).

Cyclospora: Cyclosporiasis is an intestinal illness caused by a protozoan parasite *C. cayetanensis*. Patients became infected with *Cyclospora* by the consuming of contaminated food or water. People living or traveling to countries where cyclosporiasis was endemic may be at increased risk for infection. *C. cayetanensis* was one of emerging parasites causing diarrhea that were not known until 1993 (Ortega *et al,* 1993). It gained more attention as a food-borne pathogen after large outbreaks of illness in the United States in 1996 & 1997 were linked to raspberry farms in Guatemala (Herwaldt and Ackers, 1997). The exact source for contamination of raspberries was unknown, and rinsing didn't prevent disease (Herwaldt, 2000). In Guatemala, cyclosporiasis was relatively uncommon among children <18 months of age. Infection was common in children between the ages of 1.5 & 9 years. Disease was more severe in children < five years of age, who were more likely to have fever and tended to have diarrhea for up to 15 days, a higher median number of stools per day, and a higher probability of mucoid stools. Soil contact was a strong risk factor for disease in those less than two years (Bern *et al,* 1999).

Treatment by trimethoprim/sulfamethoxazole (TMP/SMX), under trade names Bactrim®, Septra®, and Cotrim® is used for *Cyclospora* infection, without highly effective alternative antibiotic regimen was identified yet for patients who didn't respond to standard treatment or have a sulfa allergy (CDC, 2018).

Cryptosporidiosis: The protozoan *Cryptosporidium parvum* is one of the prominent cause of water-borne diarrheal illness (Fricker and Crabb, 1998). A largest waterborne outbreak occurred, when 403,000 residents of Milwaukee developed gastrointestinal symptoms after their drinking water became contaminated (Levy *et al,* 1998). Oocysts outbreaks associated with apple cider contaminated were reported (Millard *et al,* 1994).

Symptoms (most commonly, profuse, watery diarrhea) begin within 2 weeks (typically 5-7 days) after infection and are generally self-limited. Other symptoms can include abdominal pain, flatulence, urgency, nausea, vomiting, and low-grade fever. In immunocompetent people, symptoms typically resolve within 2-3 weeks; patients may experience a recurrence of symptoms after a brief recovery period and before complete symptom resolution (Adamu *et al,* 2014). Cryptosporidiosis clinical picture in immunocompromised patients varied with level of immuno-suppression, ranged from no symptoms or transient disease to relapsing or chronic diarrhea or even cholera-like diarrhea that led to dehydration and life-threatening wasting and malabsorption. Extraintestinal infection (in biliary or respiratory tract, but rarely pancreas) was in children and immunocompromised hosts (Kotloff *et al,* 2013)

Diagnosis: Clinicians must specifically request testing when cryptosporidiosis was suspected. New molecular enteric panel assays generally include *Cryptosporidium* as a target pathogen. Intermittent excretion of *Cryptosporidium* in stool necessitated collection of multiple samples, on 3 successive days, to increase test sensitivity. Others include microscopy with the gold standard direct fluorescent antibody (DFA), enzyme immunoassay (EIA) kits, molecular assays; microscopy using modified acid-fast staining, and rapid immunochromatographic cartridge assays (El-Bahnasawy *et al,* 2018).

Rapid immunochromatographic cartridge assays can generate false-positive results; consider confirmation microscopy for specimens identified as positive by this method. Infections caused by the different *Cryptosporidium* species and subtypes can differ clinically (Garcia *et al,* 2017). However, despite all variations in clinical presentation caused by this protozoan, most *Cryptosporidium* species, all with multiple subtypes, are indistinguishable by traditional diagnostic tests. To better understand cryptosporidiosis epidemiology and track infection sources, CDC (2019) has launched Crypto-Net, which gave *Cryptosporidium* genotyping and subtyping services in collaboration with state public health agencies. CryptoNet recommends not using formalin to preserve stool for *Cryptosporidium* testing because formalin impedes reliable genotyping and subtyping. Cryptosporidiosis is a nationally reportable disease.

Treatment most immunocompetent people recover without treatment. US/FDA approved nitazoxanide as a treatment for cryptosporidiosis in immunocompetent people aged ≥1 year. Nitazoxanide didn't show efficacy cryptosporidiosis treatment in immunocompromised patients. However, dramatic clinical and parasitologic responses without specific treatment were reported in such patients following reconstitution of immune system. Protease inhibitors have direct anti-*Cryptosporidium* activity. Oral rehydration proved to be the best effective supportive therapy in both immunocompetent and immunocompromised patients. Foodborne outbreaks were not common. However, 88 students & four university employees became ill in one outbreak associated with consumption of food in a university cafeteria. Genotyping linked a *C. parvum* genotype 1 isolate to an infected food handler who prepared raw produce (Quiroz *et al,* 2000). Watery diarrhea, nausea, vomiting, and fever are common symptoms with cryptosporidiosis. Illness usually is self-limited in immunocompetent hosts, but it can cause severe, protracted diarrhea in immunocompromised hosts.

Clostridium perfringens: Acute self-limiting gastroenteritis caused by contaminated food products often is associated with *C. perfringens*. Primary meat contamination with spores is common when the food is allowed to stand at a temperature between 80ºF and 120ºF; temperatures of cooked meat must exceed 210ºF to ensure that spores were killed. Symptoms are caused by the production of enterotoxins by the organism once it resides in the gut. Clinical manifestations typically include cramp abdominal pain and watery diarrhea without fever. *C. perfringens* type C produces beta toxins that can cause enteritis necroticans (pigbel), a hemorrhagic necrosis of the jejunum. Pigbel was an uncommon health problem in developing countries and very rare in developed countries. Factors associated with enteritis necroticans include decreased GI acidity, motility, and chronic disease or malnutrition. A case of pigbel in a 12-year-old diabetic associated with the consumption of pig intestines (chitterlings). Simultaneous ingestion of sweet potatoes contained trypsin inhibitors prevented intestinal degradation of the toxin can be a potentiating factor (Petrillo *et al,* 2000).

Botulism: Botulism is a rare but potentially life-threatening neuroparalytic syndrome resulted from the neurotoxin elaborated by the spore-forming microorganism *Clostridium botulinum*. Spores are heat resistant and survive in food preservation and preparation.

Botulism in children occurs in two ways:

Food-borne botulism; ingestion of food contaminated by preformed botulin's toxin, and infant botulism; clostridial spores ingestion that then colonize in gastrointestinal tract of the host and release toxin produced in vivo.

Infant botulism has been recognized as a separate disease entity since 1976. Since that time, over 900 cases of infant botulism have been reported in the United States; making it the most frequently reported form of botulism in the world. Despite the frequency of infant botulism among all cases of botulism, the CDC notes little change in the number of cases of infant or food-borne botulism in recent years (Werner *et al,* 2000). Unlike most of the other agents, infant botulism the bacteria can be indoors and outdoors characterized by constipation, rather than diarrhea. From 1990 to 2000, 263 cases from 160 food-borne botulism events were reported in the USA, 39% of cases occurred in Alaska, all attributable to traditional Alaska Native foods. In the lower 49 states, a noncommercial food item was implicated in 91% of events; most commonly home-canned vegetables were 44% (Sobel *et al,* 2004). After gaining entrance to the cell's cytoplasm, the toxin produces an irreversible disruption in stimulation-induced acetylcholine release by that presynaptic nerve terminal. Return of synaptic function requires sprouting of a new presynaptic terminal with subsequent formation of a new synapse, a process that requires approximately six months. Adrenergic synapses are not affected by the toxin, nor does the toxin appear to permeate the blood brain barrier, therefore limiting involvement to the peripheral cholinergic nervous system (Jin *et al,* 2006).

The vast majority of wound botulism cases in California since 1988 were among injection drug users, particularly those using black tar heroin by the subcutaneous or intramuscular (not intravascular) route. How the heroin becomes contaminated with the clostridial organisms or their toxins is unknown. Wound botulism has also been reported in patients who inhale cocaine. Patients presented with sinusitis, with one patient growing *C. botulinum* from a sinus aspirate (Roblot *et al,* 2006). Food-borne cases of botulism were mainly recognized as small outbreaks, usually involving home canned foods such as fruits, vegetables, and fish. The commercial products and restaurants are occasionally sources (Sheth *et al,* 2008).

Honey was directly linked to infant botulism. Corn syrup, chopped garlic in oil, canned cheese sauce, canned tomatoes, carrot juice, baked potatoes wrapped in foil although implicated, has never been linked with botulism (Moharrami *et al,* 2022). Honey can contain the bacteria cause infant botulism, so don't feed honey to children less than 12 months, but safe for people 1 year of age & older (CDC, 2018). Breast-fed infants older than two months of age have an increased risk for being hospitalized with botulism. However, because breast-fed infants typically present at a later age than do formula-fed infants, these children may have a slower disease progression, although initially thought to be involved in some cases of sudden infant death syndrome, a 10-year prospective study didn't show association (Golding *et al,* 1997).

Treatment: infantile botulism was of nutritional and respiratory supports until motor endplates are regenerated, which results in spontaneous recovery. Neurologic sequelae are seldom reported. Some children require outpatient tube feeding and may have persistent hypotonia (Cox and Hinkle, 2002). If left untreated, mortality rate of 40% to 50% (Dembek *et al,* 2007). US FDA approved human botulinum immune globulin intravenous (BIG-IV) for the treatment of infant botulism. This blood product is derived from the pooled plasma of human adults immunized with pentavalent botulinum toxoid. It was presented as having a far lower risk of anaphylaxis when compared to trivalent equine antitoxin (Robinson and Nahata, 2003). Medical therapies include guanidine hydrochloride, a derivative of the nucleic acid guanine, & 3, 4-diaminopyridine, a presynaptic

potassium channel blocker, both promote presynaptic release of acetylcholine at neuromuscular junction. Also, there was an increasing advocate's number of plasma exchange for botulism (Sato *et al,* 2000).

Listeria monocytogenes: There was several historical foodborne illness breakouts by *L. monocytogenes* that proved to be a foodborne illness linked to a variety of foods. In 1985, a massive outbreak of *L. monocytogenes* was traced to a brand of soft cheeses involving 142 cases, 28 deaths, and 20 fetal losses (Jackson *et al*, 2018). But, *L. monocytogenes* was not the most common foodborne illness; it had the highest mortality rate secondary to its unique virulence factors (Jordan and McAuliffe, 2018).

L. monocytogenes typically produces a febrile illness, sepsis, or central nervous system infection, principally in immunocompromised patients. Pregnant women also can be affected and should avoid unpasteurized dairy products. *L. monocytogenes* can cause febrile gastroenteritis in immunocompetent individuals. Pregnant women are at high infection risk and occult or overt bacteremia caused chorioamnionitis producing early-onset neonatal listeriosis (Girard *et al,* 2014). Infants have characteristic clinical features, including prematurity, sepsis at birth, fever, a diffuse maculopapular cutaneous eruption, and evidence of significant hepatic involvement with jaundice (Mylonakis *et al,* 2002). Mortality rate of early-onset listeriosis, even with treatment was highly common stillbirth. Autopsy in early-onset listeriosis showed significant chorioamnionitis in placental remnants and granulomas in many organs, particularly liver and spleen, in infected infants. Original descriptions from East Germany entirely characterized the syndrome as granulomatosis infantiseptica (Potel, 1952).

Treatment: *L. monocytogenes* remains susceptible to most β-lactam antibiotics except cephalosporins usually resistant (Hof *et al,* 1997). Empiric therapy for bacterial meningitis with ampicillin is recommended for older adults but may not be necessary for children beyond the neonatal period (Okike *et al*, 2015). When listeriosis is a likely diagnosis; ampicillin or, in penicillin-allergic patients, vancomycin provides empiric coverage for *L. monocytogenes* until the diagnosis done by culture. A combination of ampicillin and gentamicin is the current therapy of choice for all listeriosis forms (Temple and Nahata, 2000). Listeriosis treatment was usually with a combination of ampicillin and an aminoglycoside but other regimens have been used (Schlech, 2019). But, the duration of treatment for invasive listeriosis has not been studied. Relapses appear to be uncommon, and 2 to 3 weeks of therapy with ampicillin and gentamicin is sufficient for most forms of listeriosis. Rhombencephalitis with abscess formation in the central nervous system may require more prolonged therapy, but data without available to support treatment beyond 4 weeks (Lorber, 1997). An outbreak at primary schools, 1566 out of 2189 people (72%) had symptoms of gastroenteritis traced to the consumption of a corn salad in cafeterias (Aureli *et al,* 2000). The median time from meal consumption to onset of symptoms was 24 hours.

Yersinia enterocolitica: Yersinia enterocolitica can cause both outbreaks and sporadic cases of food-borne gastroenteritis. Commonest vehicle was undercooked pork products, such as chitterlings. An outbreak among children in Vermont and was linked to consumption of milk from one dairy (Ackers *et al,* 2000). The commonest picture of children's ienterocolitis diagnosed by PCR-based DNA fingerprinting was fever and diarrhea (Gray *et al,* 2001). In Congo diarrhea were found in 121 patients (69.41%) dominated by intestinal agents associated sickle cell anemia with in 58 patients were *Yersinia enterocolitis, Entamoeba histolytica, Giardia lamblia* & *Clostridium difficile). Y. entericolitica* were adapted in a dairy pig, the suggested source of contamination (Taylor, 2012), and various domestic and wild animals worldwide, as well as from contaminated surface waters (Ragno *et al,* 2019). Septic shock

was identified with increased frequency as a causative agent of post-transfusion, globally led to an estimated incidence of one case per 6.5 million RBC units distributed in France (Leclercq *et al,* 2005).

Enterobacter sakazakii: Contamination of powdered infant formula preparations by *E. sakazakii*, or *Cronobacter* (Iversen *et al*, 2007) was linked to invasive infections included meningitis, brain abscess, and necrotizing enterocolitis; without diarrhea (Bowen *et al,* 2006). The environmental contamination with *E. sakazakii* isolated the pathogen from sweeping surfaces or vacuum cleaner bags in eight of nine food factories, and from vacuum cleaner bags in 5 of 16 households (Kandhai *et al,* 2004). They concluded that this organism was widespread in the environment, which should be taken into account in designing measures for preventing infant formula contamination.

Brucella: Brucellosis is caused by bacteria of the genus *Brucella*, with *Brucella abortus*, *B. melitensis,* and *B. suis;* infecting cattle, small ruminants, and swine, respectively, being species of particular importance in human and livestock infections worldwide. Other species of concern include *B. canis* infecting dogs, and *B. ovis* infecting sheep of worldwide significant highly contagious disease in domesticated animals; humans serve an incidental hosts (Olsen and Palmer, 2014). The direct losses include reduced herd fertility and the costs associated with changing the herd structure to compensate for the overall reduction in productivity and fertility, mainly in cattle are caused by post-abortion metritis and retained placentas (Franc *et al,* 2018). Humans' brucellosis typically manifests as a range of non-specific clinical signs including malaise, fatigue, arthritis, and fever (Mohamad *et al,* 2011). Chronicity and recurring febrile conditions with joint pain are common sequelae; the acute and chronic symptoms can result in a significant loss of work days and consequential disparity in the socioeconomic status of infected persons and their families (El-Metwally *et al,* 2011). Human-to-human transmission occurs transplacentally, via breast-feeding, but rarely via sexual intercourse, organ transplantation or blood transfusions (Tuon *et al,* 2017). In the Eastern Mediterranean Countries, incidence ranged between 12.2% in sheep, 11.3% in goats, 41.3% true prevalence of sheep and 32.2% of goats in infected Egyptian villages (Hegazy *et al,* 2011), to 2.2% at the individual animal level and 45% at the herd level in Jordan (Al-Talafhah *et al,* 2003). Brucellosis a worldwide health and economic problem, its diagnosis by clinical pictures, otherwise true incidence and/or prevalence were not available.

Seafood ingestions: Seafood is a frequent source of food-borne illness, which can arise from consumption of fish, especially shellfish, contaminated by viruses such as Norwalk or Hepatitis A; ingestion of a toxin such as scombroid, found in certain finfish; or poisoning from fish or shellfish contaminated with toxins from algae and other marine life forms, such as dinoflagellates that produce toxins causing ciguatera fish poisoning and paralytic shellfish poisoning, or *Gymnodinium breve*, which causes the red tide (Morris, 1999). Human calicivirus and other viruses: Noroviruses (NoVs) are the leading cause of foodborne disease outbreaks worldwide, and may soon eclipse rotaviruses as the most common cause of severe pediatric gastroenteritis, as the use of rotavirus vaccines becomes more widespread. Genetic mutations and re-combinations contribute to the broad hetero-genetic of noro-viruses and the emergence of new epidemic strains. But, typically a self-limited disease, noro-virus gastroenteritis can cause significant morbidity and mortality among children, the elderly, and immunocompromised (Koo *et al,* 2010).

Viruses such as caliciviruses, the prototype of which are noroviruses (formerly known as Norwalk-like viruses), probably account for a sizable proportion of foodborne illness (Estes and Leparc-Goffart, 1999). Application of new molecular diagnostic methods showed that the calicivirus family was the co-

mmonest cause of acute viral gastroenteritis outbreaks in the United States (Glass *et al,* 2000). Between 1998 & 2002, total 657 outbreaks by noro-virus resulting in more than 27,000 cases (CDC, 2007a). Calicivirus was responsible for 42% outbreaks of shellfish-associated illness with identified pathogen (Wallace *et al,* 1999). Norovirus outbreaks occur in a range of other institutional settings including schools, child care centers, colleges, prisons, and military encampment. Gastroenteritis outbreaks in child care centers were caused by other pathogens, including rotavirus, sapo-virus, and astro-virus, due to lack of viruses' immunity in them (Lyman *et al,* 2009)

Viruses can be transmitted from contaminated food or water, and food handlers often are suspected as the source of food-borne outbreaks. Genomic characterization of viruses has allowed the development of reverse transcriptase polymerase chain reaction assays that can be used in the testing of clinical stool samples. The assays were used in an outbreak of gastroenteritis to trace the source to a food handler whose infant was sick with the infection (Daniels *et al,* 2000). The incubation period generally is 24 to 48 hours, with a range from 18 to 72hrs. Clinical manifestations include nausea, vomiting, abdominal pain, and diarrhea. Generally, disease manifestations last 48 to 72hrs with a full and rapid recovery.

Vibrio species: *Vibrio* species that include *Vibrio vulnificus*, *V. parahaemolyticus*, & *V. cholera* are major causes of seafood & fish-acquired food poisoning (Munoz, 1999).

1. *V. cholera* serogroups 01 and 0139 cause pandemic and epidemic cholera, a disease characterized by voluminous watery diarrhea (Basu *et al,* 2000). An isolate indistinguishable from the epidemic Latin American strain was identified in oysters from Mobile Bay, Alabama in 1991 &1992, but no clinical cases of cholera resulted (CDC, 1973). In the United States, three outbreaks of *V. cholera*, affecting 12 people. Although non-choleragenic illnesses were reported in 22 residents of Louisiana and Mississippi after Hurricane Katrina, without cholera epidemic or evidence of increased risk to residents of Gulf-Coast (CDC, 2006b).

V. vulnificus most frequently is implicated in outbreaks from the United States. *V. vulnificus* infection can be acquired by eating raw or undercooked shellfish, and directly by contaminating open wounds during swimming or cleaning shellfish (CDC, 1993b). *V. vulnificus* infection usually causes cellulitis, wound infection, or septicemia. of *Vibrio* illnesses (236) associated with recreational water were due to cholera exposure and other vibrio illness surveillance during 2007-2008, *V. vulnificus* caused the highest rate 75% hospitalization and 13% mortality (Hlavsa *et al,* 2011). Symptoms of both *V. parahaemolyticus* and *V. vulnificus* infections include cramps, nausea, fever, and bloody diarrhea. *V. vulnificus* skin infections can be more severe and lead to rapid septicemia and death if an open wound encounters salt or 2-brackish water; embedded within a self-secreted matrix gave protection from the outside environment (Jamal *et al,* 2018).

Vibrio parahaemolyticus is a curved, rod-shaped; Gram-negative bacterium found in sea & in estuaries, on ingestion caused gastrointestinal illness (Ryan and, Ray, 2004). Outbreaks were associated with raw oysters and clams from the northeastern, as well as Pacific northwestern, coasts of USA (CDC, 2006c). Many people enjoy eating raw oysters, and raw oyster bars are popping up at some of trendiest restaurants. Swimming or working in affected areas can lead to infections of the eyes, ears, or open cuts and wounds (Penland *et al,* 2000). But eating raw oysters and other undercooked seafood can put man at risk for infections, including vibriosis result in only diarrhea and vomiting. More risky illness, including bloodstream infections and severe blistering skin lesions, *V. vulnificus* infections patients' require intensive care or limb amputations, and 15-30% was fatal (CDC, 2020).

Scombroid fish poisoning, or histamine fi-

sh poisoning, is an allergic-type reaction that occurs within a few hours of eating fish contaminated with high levels of histamine. When certain types of fish are not properly refrigerated, bacteria in the fish can multiply, break down the flesh of fish, and produce high amounts of histamine. The commonest illness sources were finfish such as tuna, mackerel, amberjack and bonito. Other fish, such as notably tuna mahi-mahi, bluefish, marlin, and escolar, can also cause scombroid fish poisoning (CDC, 1997). The illness is caused by histamine and other products from bacteria that propagate on the fish in warm water or when they are inadequately refrigerated. Prompt harvesting and refrigeration until the fish is cooked are the best way of poisoning prevention (CDC, 1999b). One of the largest reported outbreaks of scombroid fish poisoning in the United States was associated with a rare vehicle; escolar fish (Feldman *et al,* 2005). CDC reported 118 outbreaks of scombroid from 1998 to 2002, resulting in 163 cases. One Pennsylvania outbreak was associated with consumption of a tuna-and-spinach salad in a restaurant. Flushing, nausea, sweating, diarrhea, and headache occurred from five minutes to two hours after ingestion and resolved within hours. Histamine levels were found to be elevated in fish, caught in the Gulf of Mexico by a long-line method that kept fish suspended for 12 to 24hrs on the line in relatively warm water prior to harvesting (CDC, 2007b).

Ciguatera fish poisoning (or ciguatera) is an illness caused by eating fish that contain toxins produced by a marine microalgae called *Gambierdiscus toxicus*. People who have ciguatera may experience nausea, vomiting, and neurologic symptoms such as tingling fingers or toes. They also may find that cold things feel hot and hot things feel cold. Ciguatera has no cure. Symptoms usually went away in days or weeks but can last for years. People who have ciguatera can be treated for their symptoms (CDC, 2018). Several outbreaks were described in Australia (Karalis *et al,* 2000). The fish appear and taste norm-al; cooking does not destroy the toxin, and, thus, avoidance of these fish was the only preventive means. Most resort areas in the Caribbean and Hawaii don't serve implicated species of local reef fish. Patients develop gastrointestinal symptoms generally within three to six hours of eating the fish. Neurologic symptoms, including circumoral paresthesias and weakness in lower extremities, can occur later; occasionally a reversal of hot-cold taste sensation; symptoms often persist for several weeks, but in some patients can last for years (L'Herondelle *et al,* 2020).

Mushroom ingestions: Poisonous mushrooms are eaten by mushroom hunters out of ignorance, after misidentification as edible mushrooms, or as a psychoactive drug. Mushroom poisoning commonly leads to consultation with a poison information center and to hospitalization. Mushroom ingestions are discussed separately (Pegler, 1989). Of the vast number of mushroom species only about 100 that are toxic, and about 6000 ingestions annually in the United States. Of these, over half of the exposures were in children under six years. Most poisonings cause symptoms only of gastrointestinal upset, as a common feature across several toxidromes and most likely occurred by ingestions of small quantities of toxic mushrooms. Severe poisonings are primarily a consequence of misidentification by adults foraging for wild mushrooms who consume them as a food source (Beuhler *et al,* 2009) The most reliable means of diagnosis is still macroscopic identification of ingested mushrooms or un-ingested leftovers (gills current or decurrently; pores; stem shape; cap color), or microscopic identification of spores in cooked mushroom remains, vomit, or feces, by a qualified mushroom expert contact details may be obtained from relevant poison center (Wennig *et al,* 2020). Mushroon poisoning must be differenced from 1- Gastroenteritis, 2- Foodborne toxin, 3- Acute viral hepatitis, 4- Acetaminophen overdose/toxicity, 5- He-

patitis ischemic or alcoholism, 6- Isoniazid toxicity (Tran and Juergens, 2022), and 7- Insecticides as organophosphate or carbamate toxicity (El-Bahnasawy *et al,* 2015).

Treatment of vast array of possible symptoms primarily consists of supportive care. Acute gastrointestinal effects may benefit from rehydration and antiemetic added to correction of any electrolyte derangements. For those patients with adverse hallucinations, benzodiazepines may provide anxiolysis. Cholinergic toxicity was benefit from given anticholinergic agents such as glycopyrrolate or atropine. Consider Atropine 0.5-1mg IV adults or 0.01mg/ kg for children. Specifically, for patients with refractory seizures secondary to gyromitra-ingestion, pyridoxine (B6) should be administered. Pyridoxine at 25mg/kg IV can be given as treatment or prophylactically for seizure control. Benzodiazepines may help adjunct, for patient ingested amatoxin, consider N-acetylcysteine (NAC), silibinin, & penicillin (Diaz, 2018).

Prevention through food irradiation: Irradiation is extremely effective in reducing pathogens associated with food products. Irradiation of flour, fruits, vegetables, meats, and spices was approved by US/FDA. Meat, pork, and poultry also have been approved by the USDA for irradiation up to 3.0 Grays. This irradiation dose destroys *S. aureus, Salmonella, Shigella*, *Campylobacter*, and *Cyclospora*. *E. coli* O157:H7 required much higher doses. Food irradiation at approved levels didn't cause significant change in the amino acid or fatty acid content of food, with minimal thiamine reduction (Štajner *et al,* 2007). Irradiating food prolongs shelf life, delays ripening time for fruit, and destroys the bacteria that cause food-borne illnesses. Many countries already use irradiation widely. In the United States, many foods are irradiated. Although food irradiation is a safe and useful technology, it remains underutilized (Parnes and Lichtenstein, 2004). Also, there were other new non-thermal technologies for microbial inactivation, such as high hydrostatic pressure, pulsed electric field, and ultrasound under pressure. Non-thermal methods allow micro-organisms to be inactivated at sub-lethal temperatures, thus better preserving the sensory, nutritional and functional properties of foods (Mañas and Pagán, 2005).

Prevention via safe food handling: Proper food storage and preparation also can reduce the risk of food poisoning. The United States Department of Agriculture (USDA) identified several critical control points in food safety: 1- Purchase, 2- Home storage, 3- Preparation, 4- Cooking, 5- Serving, & 6- Storage of leftovers

Major control measures include: 1- Avoid contact between cooked and uncooked foods, 2- Refrigerate foods promptly after purchase, preparation, or eating, 3- Wash hands and preparation surfaces after handling raw meats, poultry, fish, and eggs before contact with other items, 4- Cook meats, poultry, fish, and eggs thoroughly, & 5- Maintain proper refrigerator and freezer temperatures, and never leave cooked foods at room temperature for more than two hours (shorter if ambient temperatures are higher).

Nursing role: Despite the introduction of vaccination against rotavirus, and even though it can often be treated on an outpatient basis, acute infectious gastroenteritis is however the second commonest non-traumatic cause of emergency hospitalization in children aged 1 to 5 years (Posovszky *et al,* 2020)

What is gastroenteritis? Infectious diarrhea is commonly known as gastroenteritis.

1. Although often considered a benign disease, acute gastroenteritis remains a major cause of morbidity and mortality in children worldwide, accounting for 1.34 million deaths annually in children younger than 5 years, or roughly 15% of all child deaths, 2- Diarrhea may be mild, accompanied by slight dehydration, or it could be extremely severe, required the prompt and effective treatment (Yang *et al,* 2019).

Nursing management of a child with gastroenteritis includes: Lack of a specific diagnosis can hinder best management, treatment

and lead to secondary infection to others, including health care workers (Guerrant *et al,* 2001). 1- Assess stool characteristics. In addition to basic information about the child, the interview with family must include specific information about history of bowel patterns and the onset of diarrheal stools, with details on number and type of stools/day; suggest terms to describe the color and odor of stools to assist the caregiver with descriptions. 2- Assess for vomiting: Inquire about recent feeding patterns, nausea, and vomiting. 3- Assess for illness presence: Ask the caregiver about fever and other signs of illness in the child and signs of illness in any other family members (Kawata *et al,* 2019), & 4- Physical examination: This is observation of skin turgor and condition, including excoriated diaper area, temperature, anterior fontanelle, apical pulse rate, stools, irritability, lethargy, vomiting, urine, lips and mucous membranes of mouth, eyes, and any not-able physical signs.

Nursing diagnoses: Based on assessment data, major diagnoses are: 1- Risk for infection related to inadequate secondary defenses or insufficient knowledge to avoid exposure to pathogens. 2- Impaired skin integrity related to constant presence of diarrheal stools. 3- Deficient fluid volume related to diarrheal stools. 4- Imbalanced nutrition less than body needs related to nutrients' mal-absorption. 5- Hyperthermia related to dehydration, & 5- Risk for delayed development related to decrease sucking when infant is NPO (Yang *et al,* 2019).

Pathophysiology: Two primary mechanisms responsible for acute gastroenteritis are: 1- Damage to villous brush border of intestine, causing mal-absorption of its contents and leads to an osmotic diarrhea. 2- Release of toxins that bind to specific enterocyte receptors and cause the chloride ions release into intestinal lumen causing to secretory diarrhea. Even in severe diarrhea, however, various sodium-coupled solute co-transport mechanisms remain intact, allows for efficient reabsorption of salt and water. By givin a 1:1 proportion of sodium to glucose, classic oral rehydration solution (ORS) takes adv antage of a specific sodium-glucose transporter (SGLT-1) to increase the sodium reabsorption, which leads to passive reabsorption of water (Field, 2003).

Apart from foodborne diseases (gastroenteritis), climate changes have a complex impact influences human and animal health by alters the pathogens and vectors borne diseases (VBD) conditions (Morsy, 2023). Insects are, by far, the commonest living creatures on our planet with more than 1.5 million species of Phylum Arthropoda, vastly exceeds the number of all vertebrates kinds. They live in the widest range of habitats and eat the greatest varieties of food. They can transmit several infectious pathogens (bacteria, parasites, protozoa and viruses) resulting in more than 700,000 deaths annually worldwide (Belluco *et al,* 2013). Pests, as insects damage vegetables, animals and humans via different mechanisms (Morsy, 2012). First, insect pests can seriously damage forests trees, wood products, cultivated crops and agricultural products by eating leaves or digging burrows in stems, fruit, or roots. This action always leads to contamination (e.g., body parts, exoskeletons, eggs, off-odors) of produce. Furthermore, to the loss of food and feed products must be added economic losses associated with growing, transporting, and storage. In addition to direct damage to the plant by feeding, insects can indirectly affect plant health by delivering plant pathogens to wounded sites, from where pathogens spread via plants (Islam *et al,* 2020). Secondly, some insects, such as house flies live in the disease agents' places as dumps, sewers, and garbage heaps, feed on fecal matter, discharges from wounds and sores, sputum, and all moist decaying matters such as spoiled fish, eggs and meat. These flies are incriminated in transmitting at least 65 diseases to humans, including typhoid fever, dysentery, cholera, gangrene, poliomyelitis, yaws, anthrax, tularemia, leprosy & tuberculosis. They regurgi-

tate and excrete wherever come to rest and thereby mechanically transmit diseases. Cockroaches can transmit: Salmonellosis (*Salmonella* spp.), typhoid fever (*S. typhi* by consuming contaminated feces), Cholera (*V. cholerae* with feces and vomit), Dysentery (dysentery bacillary or shigellosis and amoebic one by *Entamoeba histolytica*), Leprosy (*M. leprae* via feces), campylobacteriosis (*Campylobacter* spp. by ingesting contaminated food and drink such as unpasteurized milk and undercooked and poorly handled poultry), Listeriosis (*L. monocytogenes* by consuming contaminated food & drink) as well as asthma especially children (Hareza *et al,* 2020). The VBD are increasing in recent years due to increasing human population expanding into animal-populated territories, and changes in geographic distribution and pet owners mainly in tropic and subtropics areas (Fasolato *et al,* 2018) Despite prevention and control measures, VBD are still emerging with climatic changes with multiplications and spreading amongst the major public health concerns worldwide (Garofalo e*t al*, 2017).

Conclusion

1. CDC defines a food-borne disease outbreak as any cluster of two or more individuals developed similar symptoms following ingestion of a common food. Laboratory or clinical guidelines to prove the etiology of a foodborne disease outbreak vary for bacterial, chemical, parasitic, and viral agents.
2. Factors contribute to foodborne diseases include improper food storage and handling and lack of adequate public health infrastructure.
3. Many microorganisms cause food poisoning. *Salmonella* species cause majority of outbreaks when an etiology was established.
4. Food poisoning by seafood ingestion cause human caliciviruses (noro-viruses), others (hepatitis A), *Vibrio* species, scombroid, and ciguatera fish poisoning.
5. Prevention of mushroom poisoning are to avoid absorption of toxins from digestive tract into the humans.
6- Food poisoning can be prevented by food irradiation and safe food handling.
7- Food handlers must be medical checkups, trained, educated on food hygienic practices.
8- Appropriate hand hygiene is the most important method to prevent infection and control transmission by thorough washing with running water and plain or antiseptic soap.
9- Clean toilet and bathroom regularly, especially the toilet seat, door handles and taps.
10- Good hygiene, tidy housekeeping and biosafety effective insect, birds and rodents' exclusion will generally discourage vermin.

Authors' declaration: The authors declared that they have neither competing interests nor received any funds. Also, they equally contributed in this review article**.**

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