

A Primer on Food and Water Borne Illnesses When Dining Out: A Case Study on Norovirus Outbreak on a Cruise Ship

Abstract

A recent outbreak of norovirus occurred approximately 24 hours into a cruise, while passengers were en route to a northern destination aboard a holiday cruise ship on a scheduled week-long journey. The symptoms of classic norovirus including watery diarrhea, severe abdominal cramps and nausea were quickly recognized, and the illness causation confirmed via on-board laboratory testing in the ship's infirmary. Appropriate treatment prescribed by the ship's physician, and containment was realized within a few days. Because of the limited number of passengers affected (12 out of over 1,000), and the timing of the incident, it was concluded that the origin of the infection likely occurred prior to or soon after the passengers boarded, possibly linked to a welcoming ceremony hosted by the ship's captain during which a variety of food and beverage items including some seafood items were made available during a buffet dinner. Following the initial outbreak, no additional cases were reported or identified, no on-board origin was confirmed, and all passengers recovered without incident and continued the cruise.

Keywords

Norovirus, Food Borne Illness, Episode, Cruise Ship, Outbreak.

Introduction

Foodborne and waterborne illness (FWBI) represent a never-present potential risk at home and abroad and present a significant cause of morbidity and mortality in the United States and other countries [1,2]. FWBI can be caused by numerous entities, including heavy metals, toxins of biological or other origin, bacteria, viruses, and parasites [3]. According to the CDC, approximately 48 million people become ill from foodborne and waterborne diseases each year, stemming from 10 or more commonly reoccurring noxious or pathogenic agents. The most common vector is unintentional consumption of contaminated food, water, or beverage that have been consumed and thus

introduced into the food chain of the unsuspecting host or hosts via the fecal-oral route of transmission. Of the 48 million or more cases annually, approximately 128,000 require hospitalization, and unfortunately, some 3,000 or more may succumb to a food or waterborne illness in any given year [1-4]. Food safety is a major concern in most Westernized nations, where the food processing systems and retail providers typically employ an unparalleled level of scrutiny during manufacture, testing, storage and final table presentation, but when breaches in the system occur, the unintended consequences may be substantial.

Despite the precautions, as many as one in six restaurant diners may experience a food-borne related illness in any given year according to the CDC reports [3-5].

untreated water sources including rivers, streams and unprotected

Contributing to this high incidence are factors that may occur as the consumable items become contaminated at any stage of the origination or intermediate processes leading up to the serving of the dining clientele [4]. Because potential sources such as uneaten left-over food items are often discarded and thus no longer available for survey by the time symptoms are first reported, positive confirmation of the origin may not always be readily attainable. In addition, some patients and experienced travelers experiencing less severe symptoms may self-medicate unreported episodes without reporting to their primary care provider for confirmation or documentation of their illness. Accordingly, it sometimes poses difficulty in determining where and at what stage of food production the contamination may have occurred, and what specific steps must be taken to contain the impending illness as was the situation in this outbreak. A brief overview of some common causes of FWBI is summarized below.

Typical common causes of FWBI include viral, microbial, parasitic, and chemical agents [1,3]. These include such items as *Listeria monocytogenes*, a microbial contamination often found in cantaloupe fruit that may have been surface contaminated from natural fertilizers including manure, and inadequately cleaned and decontaminated melons prior to cutting and serving [6-8]. Over 8,000 reports of 138,000 cases of listeriosis were reported from 2011 to 2020, nearly 10,000 hospitalizations, and 241 deaths. As with many types of infectious illness, those individuals who are in their later years, and those with compromised immunity and other comorbidities and infants typically present the greatest risk of morbidity and mortality from the consequences of FWBIs. Globally, over a billion individuals may be infected with a parasitic illness of one kind or another at any given time, often by way of compromised industrial hygiene and contaminated foods, beverages, and public water sources [2,3,9].

Of common parasitic illnesses, helminthic tapeworms reign among the supreme [3,8,9]. The beef/pork tapeworm *Taenia saginata* (beef origin), *Taenia solium* (pork origin) and *Taenia asiatica* (Asian tapeworm) are classed as cestodes, causing mostly intestinal illness with significant gastrointestinal distress, while *Taenia solium* can also cause cysticercosis. In addition, prolonged duration of cestode infection may also be marked with nutritional deficiencies, as the rapidly developing parasites consume vitamin B12 and other essential nutrients during their life cycle, thereby preventing their intestinal absorption by their unsuspecting host. The protozoal agent *Giardia lamblia* is often found in contaminated, unprotected and

springs due to human and animal contamination, and also cause typical gastroenteritis illness [9,10]. While both intestinal cestodes and protozoal infestations are readily treated with anti-parasitic agents, care must be taken to ensure clothing and linens are adequately cleaned and decontaminated to prevent re-exposure that could predispose to fecal-oral reinfection.

In addition, infection with cryptosporidium species organisms (due to *C. hominis* or *C. parvum*) ranks among the most common recreational waterborne parasitic infections to occur in the United States, and also occurs via consumption or exposure to water sources including public pools that have become contaminated [11,12]. The parasite causes gastroenteritis and watery diarrhea that may persist for up to two weeks following infection, and often fails to fully resolve in the absence of definitive pharmacotherapy. The parasite is relatively resistant to common water chlorination as is commonly applied in private or public pools as a disinfection agent with proven effectiveness for many other potential contaminating organisms. Symptoms of acute diarrhea and enteritis typically commence about a week after exposure to infective oocytes of cryptosporidium species if consumed via ingestion of the polluted water or by person-to-person direct contact via the fecal-oral route [9].

Entamoeba histolytica is another infectious parasite that can cause amoebiasis followed by severe intestinal inflammation [9,13,14]. It is an anaerobic parasitic amoebozoan organism that can survive outside the body for a considerable time, and can infect humans and other primates including canines. Because *Entamoeba histolytica* can survive outside the body for a time, it can be readily transmitted by the fecal-oral ingestion route from contaminated food or fingers. Amoebiasis has been estimated to infect about 35-50 million people worldwide, resulting in approximately 55,000 deaths annually due to the severity of the pathobiologic consequences of the organism. The trophozoite form of the parasite can migrate from infected intestinal tissues where it multiplies and can travel via the circulatory system to infect other tissues distant from its origin including lungs, brain, and liver [9,13,14].

Bacillus cereus species are yet another occasional culprit in the area of food-borne illnesses [9,15]. These bacteria are Gram-positive rod-shaped, spore-forming microbes that may be commonly found in soil, food, and marine sponges, and because they are capable of forming persistent biofilms on glass surfaces they may pose complications of contamination risk during pharmaceutical manufacturing processes. The spores are easily spread via air-borne droplets and can also cause moderate, typically non-life-threatening gastrointestinal infection when present in improperly stored or left-

over foods. The bacterium readily utilizes glucose as its primary energy source, which represents approximately 98% of its metabolizable energy pathways [9]. While ordinary cooking temperatures can kill the bacteria, the spores are typically somewhat hardier, require a longer and higher temperature to inactivate, and often survive lower cooking and warming temperatures and emerge as active bacterial colonies in stored or warmed foods.

Their unique contributions to FWBI remain an ongoing threat to pharmaceutical manufacturers, restaurants, and other instances where foods may be left open to airborne spore contamination on warming tables, serving trays, and other suitable environments [15].

The pathogenic O157:H7 serotype of *Escherichia coli* also forms a constant threat for FWBI illness in raw or undercooked beef, milk, salads, and other vegetables that may have become inadvertently contaminated by direct or indirect exposure to bovine manure residues during vegetable propagation, or to meat processing [9]. The contamination occurs from storm or water run-off during heavy rains or irrigation processes. This serotype strain produces a Shiga-

like toxin that may result in hemorrhagic diarrhea, hematology, and hemolytic uremic syndrome (HUS) in which red blood cells are destroyed, resulting in kidney failure. The most susceptible include young children, the elderly, and immunocompromised adults. The mode of transmission is via the fecal-oral route, and most reported illnesses

have occurred via commercial distribution of contaminated raw leafy green vegetables, undercooked ground meat and raw milk. In the US, 2-

7% of children develop HUS, which is the leading cause of acute kidney failure among young children. Children under the age of 5 years are at significant risk for the direst of consequences when exposed to this microbe [3,9].

Additional causes of gastrointestinal infection of bacterial origin include *Shigella* (shigellosis), *Salmonella* (salmonellosis), and *Cholera* (cholera) and are caused by facultative, anaerobic gram negative bacteria that may inhabit the intestinal tract of humans and occasionally a few other primates [9]. Shigellosis species comprises of different 4 strains of the organism that result in

varying degrees of illness from a mild traveler's diarrhea to severe diarrhea. *Shigella* bacteria appear to be genetically related to enterohemorrhagic *E. coli* (*E. coli* O157:H7) and represent a common cause of diarrheal illness. The organisms produce variable strain-specific syndromes of diarrhea and bacillary dysentery, gastroenteritis, and is typically characterized by abdominal pain and discomfort, bloody diarrhea (<20 bowel movements/day), nausea, fever, and vomiting. The onset of symptoms may occur from 12 hours to two weeks following exposure,

which may occur with an inoculum of only 10 or more of the most virulent infective bacteria. The most virulent strain (*S. dysenteriae*) produces an inflammatory Shiga toxin

that attaches to M cells in the epithelial lining of the small intestine, where the organism multiplies rapidly, and can cause breaks in rRNA and intestinal lining, and enhance the release of inflammatory cytokines. The inflammatory cytokines can also damage renal epithelium and contribute to hemolytic uremic syndrome. Shigella organisms are notoriously resistant to macrophage inactivation, and usually require antimicrobial and rehydration therapy. The CDC reports that Shigellosis may affect up to 450,000 individuals per year and mortality can be as high as 20% in those most severely affected. Young children and the elderly/immunocompromised are among the most susceptible groups and is easily spread via the fecal-oral route via contaminated foods and fingers [1-3,9].

Salmonellosis is caused by another group of facultative, anaerobic gram-negative bacterial organisms, resulting in strains specific illnesses including gastroenteritis (S. enteritidis; dysentery) and typhoid fever (S. typhi) in the most pathogenic strains [9]. Of the approximately 2,000 serovars of Salmonella organisms, only about 50 are commonly isolated from human feces. They are common asymptomatic inhabitants in poultry and are difficult to control in the commercial poultry industry and thus mandate thorough sanitation measures and cooking temperatures during preparation. That rare celebratory delicacy of "pheasant under glass" or holiday "turkey w/gravy" banquet may be just the opportunity your salmonella has been waiting for! The Salmonella organisms infect the intestinal lining of humans, where they can multiply with intestinal epithelial cells and M cells and can enter the circulation via the intestinal vascular/lymphatic network, and travel to many tissues distant from the intestinal tract, including the spleen, liver, and other organs. Once in the circulation, the bacteria may cause bacteremia and septic shock [1-3,9]. Of the 1.4 million cases reported annually in the US, less than 1% (~400) prove to be fatal. The organisms may survive asymptotically in the human intestinal tract in 1 to 3% of previously infected individuals for many months which likely contribute to their persistence among human populations. Classic symptoms of salmonella infection include diarrhea and moderate fever and may require rehydration and antimicrobial therapy to resolve the problem.

Cholera infection commonly follows environmental disasters and can persist for many years following an outbreak [9,16,17]. Cholera outbreaks can occur wherever contaminated water can infiltrate the domestic water and food supply including typical home or commercial usage of unclean water. Cholera is caused by the 0.1 AndO:139 strains of Vibrio cholerae, a motile, morphologically curved gram-negative organism. An active infection usually requires a large number of organisms (~100,000) to be consumed as stomach acid can inactivate the organism; of those that survive the gastric environment, they readily

multiply in the intestinal tract, where they produce a potent toxin capable of causing extraordinary and profound loss of water and electrolytes via vomiting and diarrhea, with especially severe loss of potassium along with sodium within only hours after infection. The extraordinary loss of water and electrolytes can cause hypovolemic shock and become fatal in up to 50% of previously healthy individuals, but early intervention with both oral and intravenous rehydration can prevent most deaths. Because of the sodium loss, oral rehydration should contain 5% glucose in order to support sodium-glucose cotransport across the luminal epithelium. The recent epidemic of Cholera in Haiti was caused by fecal contamination of a community water supply and persisted for nearly a decade (2010-2019) and resulted in XXXX infections XXX deaths [16, 17].

Unlike most infectious agents, histamine poisoning via contaminated fish is a heat-stable chemically mediated agent and thus is not destroyed by ordinary cooking temperatures [18]. Decomposing fish (most often in decomposing mackerel, grouper, and amberjack species) may enzymatically convert the endogenous amino acid histidine to histamine, resulting in acute sympathetic, histaminergic reactions, and occasional hospitalizations. In addition, Ciguatera fish poisoning, or CFP, represents yet another form of chemical fish poisoning. This FWB agent is found in herbivorous fish found in reef waters. The ciguatera toxins are originally made by, *Gambierdiscus toxicus*, a small marine organism that grows on and around coral reefs in tropical and subtropical waters. The suspect fish flesh becomes contaminated with the ciguatera toxins produced via bioconcentration as larger, carnivorous fish dine on the herbivores. The agent causes diarrhea, vomiting, numbness, heat sensitivity to hot and cold, cardiovascular, and other symptoms including hypotension, which may persist for several weeks or longer following recovery from the acute effects. The diagnosis of histamine or ciguatera poisoning is typically based on a person's symptoms and recent history of fish consumption [9, 18, 19]. If a number of individuals consumed the same fish during the same time frame also develop symptoms, the diagnosis becomes more probable without further testing, but if some of the questionable fish is still available it can be tested to further confirm the presumptive diagnosis of histamine or CFP poisoning [9, 18, 19].

Aflatoxins, ergot alkaloids, and botulinum poisoning are additional chemical entities that can cause serious illness in man and animals [8, 20-23]. Aflatoxins consist of a group of poisonous, mutagenic, mitotoxic, and carcinogenic chemicals that are produced by various species of aerobic *Aspergillus* fungi. The notorious fungi are also sometimes referred to as molds that normally grow in soil and decay in vegetation, and they can easily thrive in virtually any

nonrefrigerated or inadequately stored foodstuff, including vegetables, grains, nuts, and spices. Of the various chemical

forms including AF B₁, B₂, G₁, G₂, M₁, M₂, L, and Q₁. Aflatoxin B₁ is produced by both *A. flavus* and *A. parasiticus* strains and is considered the most toxic form to affect humans and animals, where it causes often fatal carcinomas of the liver and other organs [20,22].

Ergotism is caused by ergot alkaloids, toxic chemicals that are formed by molds that often grow on rye plants and inadvertently become carried over to rye flour

during its manufacture [9,22]. When ergots are accidentally consumed in sufficient quantities, they typically cause a variable, dose-related range of vasoconstrictions in the CNS and peripheral tissues, causing hallucinations, peripheral vasoconstriction, vascular collapse and often death.

The toxic alkaloids may be blended in such that use of the flour in baking bread and other baked goods resulted in ergot toxicity in the baked goods primarily intended for human consumption. The earliest historical report of what now appears to have been ergot poisoning in rye bread extends back for many centuries, over 1000 years, until the source and composition of the active principle was ultimately discovered [8,22-25].

Scientific study of the ergot alkaloid eventually gave rise to the development of LSD (lysergic acid diethylamide) by Hofman, and continue to be studied for possible therapeutic benefits for certain psychological and psychiatric disorders including PTSD [26].

Botulinum poisoning represents yet another potential source of severe adverse effects, as the toxin is formed by *Clostridium botulinum* [27]. *C. botulinum* is an obligate anaerobic endospore-forming gram-positive bacillus

that is commonly found in recently tilled soil and aquatic drainage sediments, where the heat stable endospores can become transmitted to food crops nourished by the nutrients found in the soil and aquatic resources [9]. When present in anaerobic environments such as sealed canned

goods, the microorganisms release a potent neuroinhibitory exotoxin that is highly specific for synaptic acetylcholine receptors, thereby blocking the parasympathetic

element of neuromuscular transmission. Toxicity with the exotoxin results in a flaccid paralysis of muscle fibers, which may persist for 1 to 10 days, dependent on the magnitude of the inhibition [9]. The neuromuscular blockade

becomes clinically apparent with the secondary symptoms such as nausea and generalized weakness within the first day of exposure but left untreated can result in fatal respiratory distress and cardiac failure within a few days of exposure. The pharmacologic effects of botulinum toxin gave way to the pharmaceutical development of Botox, where extremely small doses are injected into specific muscle groups as a cosmetic procedure to control unsightly wrinkles [27].

Clostridium tetani is another strain of *Clostridium* that

presents as an obligate anaerobic endospore-forming gram-positive bacillus that is commonly found in recently tilled soil and aquatic drainage sediments [9,28]. *C. tetani*

produce the neurotoxin *tetanospasm*, the definitive cause of tetanus, which like botulinum toxin, also affects skeletal muscles, but by blocking muscle relaxation rather than excitatory pathways. Early symptoms produce the condition known as lockjaw, a condition in which the mouth can no longer open, and swallowing is inhibited, followed by spasms of muscles of the back, head, extremities, and heels to bow in a backward position (*opisthotonos*), and death via spasms of the respiratory muscles. The organism flourishes in anaerobic environments including deep puncture wounds, where it can continue to produce the neurotoxin, and unlike botulinum poisoning, although phylogenetically related, *C. tetani* does not commonly occur in edible foods and tetanus is easily prevented by vaccination early in life as part of the DPT vaccine followed by periodic boosters for most individuals, thereby preventing most cases nowadays [9,28].

Commonly occurring viral agents that cause FWBI include both norovirus and rotavirus, RNA viruses that pose significant risks to infants, young children and older adults [9,29]. Norovirus infection is caused by highly transmissible strains of the RNA-norovirus and typically presents as a foodborne illness that is responsible for approximately 50% of the FWBI illnesses causing nonbacterial foodborne gastroenteritis that are reported in the United States; norovirus is responsible for over 80% of FWBI episodes that occur on board of International Cruise ships, a staggering incidence [2,4,5]! Norovirus infection typically produces symptoms of nausea, intestinal cramps, diarrhea, malaise, and dehydration ± a low-grade fever within 18-48 hours post exposure to the infectious viral agent. The diagnosis is readily confirmed via sensitive, rapid RIA or PCR analysis of stool specimens, which facilitates an early onset of an appropriate treatment regimen [9]. Norovirus infection is considered the most common non-bacterial cause of FWBI infection in the US, where it is linked to approximately 20 million cases annually, with approximately 300 deaths resulting from the infection [29]. Norovirus infection can be transmitted via food, water and aerosol contaminated surfaces, thus accounting for the high incidence of FWBI infections on International cruise ships (95 outbreaks between 2011-2019) [2]. Infection with norovirus causes injury and inflammation to the microvilli lining the small intestine, resulting in the classic symptoms of watery diarrhea, severe cramps, low grade fever, and generalized abdominal distress. Cleanup following an outbreak is challenging due to the aerosol microdroplet nature that contributes to its dissemination [4,5]. Understandably, cruise ships that are bound for international destinations take considerable time to prepare for the intended journey, spend many days away from port while at sea, and

must load enormous quantities of food and other edible consumables prior to departure, since while on board dining options are limited to only what the vessel dining services can provide. Onboard storage and preparation

may not always occur under opportune environmental or working conditions and may be further compromised by the loading schedules and restocking options at ports of call, which may lead to interruptions in refrigeration or optimal transit conditions. Vessels often travel great distances *en route*, where unforeseen environmental and human conditions that could compromise food storage and preservation may also occur. The overall complexity of the procurement, monitoring and delivery of the dining options poses opportunities for the weakest link in the system to materialize.

In addition to noroviruses summarized above, infants and young children are also susceptible to rotavirus infection, another viral species phylogenetically related to the norovirus [9,29-34]. Rotavirus infection can also cause an acute gastroenteritis mostly in children. Rotavirus infections are likely the most common cause of viral gastroenteritis in children, contributing to an approximately three million cases annually in the US, and sadly are linked to approximately 100 fatalities in the under-3y.o. population, whereas 90% show immunologic evidence of exposure. Natural immunity occurs following infection and is believed to protect individuals from repeat infections following an initial episode. Typical symptoms include vomiting, diarrhea, and dehydration that persist for a week or more, and necessitate prompt attention to rehydration early in the course of illness. In contrast to norovirus where an infective dose may be as few as 10 viral particles, rotavirus infection typically requires a larger infective dose, and occurs following an infective exposure of up to 100 viral particles. In both viral infections, the duration of incubation is 2 to 3 days in otherwise healthy individuals [9].

Case Study: Dining Out on a Cruise Ship

Ms. J.T. was a 62 Y.O. retired female who presented at the Ship infirmary on the second day of her voyage. Upon arrival at the infirmary, she presented with complaints of severe abdominal cramps, watery diarrhea, and nausea with an onset of early symptoms of nausea approximately 18 hours after embarking on the cruise, which she was unable to control by rest and fluids, and which became emergent within 24 hours of travel. Upon further investigation, she reported attending a buffet dinner hosted by the Ship's Captain the evening before, during which she reported consuming portions of various seafood items, including what appear to be uncooked shrimp and oysters. Ms. J.T.'s vital signs were generally normal with the exception of a mild fever typically not exceeding 102°F, abdominal tenderness upon palpation, lethargy, mild dehydration, and non-life-threatening hypotension. Ms. J.T. was followed by 11 additional passengers on the same day who presented with similar symptoms and chief complaints of mild fever, lethargy, watery diarrhea, and hypotension.

Comment [V1]: Explain the methods of diagnosing the cause of the disease

and abdominal tenderness upon palpitation, and who had attended and consumed the same buffet dinner, including these seafood entrees. The timing and cluster of patients was suggestive of an on-board food or water borne illness,

and stool specimens were collected from the entire patient cluster for laboratory analysis in the infirmary laboratory. Initial on-

board RIA rapid tests for norovirus were positive for all 12 patients, including Ms. J. T. The medical team administered supportive therapeutic measures including 1) administering intravenous fluids to restore hydration; 2) immediate administration of Compazine (10 to 20 mg, I.M.); Sachets of 20.5 mg of WHO low

osmolar formulae oral rehydration solutions (low osmolar Oral Rehydration Solution consisting of 2.6 g/L NaCl, 2.9 g/L Na-disodium citrate dihydrate, 1.5 g/L KCl, and 13.5 g/L anhydrous glucose) as needed to restore hydration [31].

3) In addition, patients were administered two 20 mg tablets of loperamide initially and an additional tablet after

each subsequent watery bowel movement thereafter until symptoms subsided; and 4, an 8 mg tablet of ondansetron was administered every 8 hours for symptoms of nausea until gastrointestinal symptoms subsided. Patients were released to their quarters within 4 hours to continue

their oral treatment regimen and returned to the infirmary periodically (~ 8 hours) to continue to monitor vital signs for up to 3 days thereafter. All patients continued the voyage and experienced a full recovery by the final port of call.

As a result of the outbreak, the on-board medical team implemented strict infection control measures, including isolation of affected passengers in the infirmary or their quarters, enhanced cleaning protocols in the food preparation and dining areas, care to dispose of

uneaten leftovers, and promoted hand hygiene measures for all food service personnel, with recommendations for shipwide attention to periodic hand washing especially before dining. In a Public Health Response, the ship's medical staff promptly reported the outbreak to relevant health authorities at the next port of call. Passengers were advised on proper hygiene practices, and the ship implemented additional sanitation measures to contain the spread of the virus.

The outcome of the on-board incident was chalked up to experience. Over the following days, the number of reported cases decreased progressively as the implemented measures took effect. Mrs. J. T. and other affected passengers showed gradual improvement with supportive care. The cruise line faced scrutiny and implemented changes to food handling practices to prevent future outbreaks of norovirus or other common food borne agents that could negatively

impact ocean going voyages. In our experience, ORS treatment may lower the mortality rate of diarrhoea by as much as 93%. Case studies in four developing countries also have demonstrated an association between increased

use of ORS and reduction in mortality. ORT using the original ORS formula has no effect on the duration of the diarrhoeal episode or the volume of fluid loss [12], although reduced osmolarity solutions have been shown to reduce both the magnitude of vomiting and stool volume.

Conclusion

In conclusion, this clinical case and the accompanying overview of agents linked to food and water borne illnesses underscores the challenges of managing infectious outbreaks not only on cruise ships but also on excursions. Diligence during casual dining out at your favorite restaurant, swift identification, isolation, and public health interventions are crucial in preventing further spread of F/WBI and ensuring the well-being of passengers, crew members, and casual diners especially while enjoying travel abroad.

References

1. Centers for Disease Control and Prevention. (2018). Estimates of foodborne illness in the United States. CDC. Available at: <https://www.cdc.gov/foodborneburden/estimates-overview.html>.
2. Centers for Disease Control and Prevention. (2022). National Outbreak Reporting System (NORS). CDC. Available at: <https://www.cdc.gov/nors/dashboard/>.
3. Centers for Disease Control and Prevention. (2020). Parasites - Cysticercosis. CDC. Available at: <https://www.cdc.gov/parasites/cysticercosis/index.html>.
4. Centers for Disease Control and Prevention. Bacillus cereus food poisoning associated with fried rice at two child day care centers - Virginia, 1993. MMWR Morb Mortal Wkly Rep. 1994;43(10):177-178.
5. Centers for Disease Control and Prevention. (2012). Pathogens Causing US Foodborne Illnesses, Hospitalizations, and Deaths, 2000-2008. Available at: <https://www.cdc.gov/foodborneburden/pdfs/pathogens-complete-list-01-12.pdf>.
6. Centers for Disease Control and Prevention. (2023). *Listeria* outbreaks. Available at: <https://www.cdc.gov/listeria/outbreaks/index.html>.
7. Centers for Disease Control and Prevention. Multistate outbreak of listeriosis associated with Jensen Farms cantaloupe - United States, August-September 2011. MMWR Morb Mortal Wkly Rep. 2011;60(39):1357-1358.
8. Weber AC, Levison AL, Srivastava SK, Lowder CY. A case of *Listeria monocytogenes* endophthalmitis with recurrent inflammation and novel management. J Ophthalmic Inflamm Infect. 2015;5(1):28.
9. Tortola GJ, Funke BR, Case CL (Eds) Ch 25. In: Textbook of Microbiology, an Introduction. 11th Edn. Pearson Pubs, NY, USA. ISBN 10-0-321-79310-2. ISBN 13-978-0-321-79310-2.

10. Flanagan PA. Giardia--diagnosis, clinical course and epidemiology. *A review. Epidemiol Infect.* 1992;109(1):1-22.
11. Stanley SL Jr. Amoebiasis. *Lancet.* 2003;361(9362):1025-1034.
12. Centers for Disease Control and Prevention. (2020). Parasites- Cysticercosis. CDC. Available at: <https://www.cdc.gov/parasites/cysticercosis/index.html>.
13. Brailita DM, Lingvay I, Aung K, Ojha AK. (2019). Amebic liver/hepatic abscesses. *Medscape Drugs & Diseases from WebMD*. Available at: <https://emedicine.medscape.com/article/183920-overview>.
14. Yamamoto K, Yanagawa Y, Oka S, Watanabe K. Two cases of endoscopically diagnosed amoebic colitis treated with paromomycin monotherapy. *PLoS Negl Trop Dis.* 2020;14(3):e0008013.
15. Centers for Disease Control and Prevention. Bacillus cereus food poisoning associated with fried rice at two child day care centers--Virginia, 1993. *MMWR Morb Mortal Wkly Rep.* 1994;43(10):177-178.
16. Sanon V, Sainvil F, Tulp OL, Awan AA, Einstein GP. Haiti's Cholera Epidemic: When the Unexpected Must Be Expected. *FASEB J.* 2021;35(S1).
17. Severe K, Alcenat N, Rouzier V. Resurgence of Cholera in Haiti amid humanitarian Crises. *N Engl J Med.* 2022; 387(25):2389-2391.
18. Taylor SL, Stratton JE, Nordlee JA. Histamine poisoning (scombroid fish poisoning): an allergy-like intoxication. *J. Toxicol Clin Toxicol.* 1989; 27(4-5): 225-240.
19. Lehane L, Lewis RJ. Ciguatera: recent advances but the risk remains. *Int J Food Microbiol.* 2000; 61(2-3): 91-125.
20. Magnussen A, Parsi MA. Aflatoxins, hepatocellular carcinoma, and public health. *World J Gastroenterol.* 2013;19(10):1508-1512.
21. Zhang W, He H, Zang M, Wu Q, Zhao H, Lu L, et al. Genetic Features of Aflatoxin-Associated Hepatocellular Carcinoma. *Gastroenterology.* 2017; 153(1): 249.e2-262.e2.
22. Stoll A. (October 1932). "Ergot and ergotism". *The Science of Nature.* 20(41): 752-757. Bibcode:1932NW...20.752S. doi:10.1007/BF01493390.S2CID 29050640.
23. Bianco MI, Luquez C, de Jong LI, Fernandez RA. Presence of *Clostridium botulinum* spores in *Matricaria chamomilla* (chamomile) and its relationship with infant botulism. *Int J Food Microbiol.* 2008;121(3): 357-360.
24. Chamomile. *Drugs and Lactation Database (LactMed)* [Internet]. Bethesda, MD: U.S. National Library of Medicine. 2021. Bookshelf ID: NBK501808.
25. Schardl CL, Panaccione DG, Tudzynski P. Ergot alkaloids--biology and molecular biology. *Alkaloids Chem Biol.* 2006;63:45-86.
26. Nichols D (May 24, 2003). "Hypothesis on Albert Hofmann's Famous 1943 "Bicycle Day"". *Hofmann Foundation*.
27. Tulp OL, Mensah CP, Kakwere RT. Efficacy of Botulinum Toxin Stereotype A and Its Relevant Mechanisms of Action in Both Cosmetic and Therapeutic Uses. *Curr Trends ToxiPharmaRes.* 2021;1(1): 1-6.
28. Tetanus vaccines: WHO position paper--February 2017. *Wkly Epidemiol Rec.* 2017; 92(6): 53-76.
29. Widdowson MA, Sulka A, Bulens SN, Beard RS, Chaves SS, Hammond R, et al. Norovirus and foodborne disease, United States, 1991-2000. *Emerg Infect Dis.* 2005; 11(1): 95-102.
30. Bernstein DI. Rotavirus overview. *Pediatr Infect Dis J.* 2009; 28(3 Suppl):S50-S53.
31. Lehane L, Lewis RJ. Ciguatera: recent advances but the risk remains. *Int J Food Microbiol.* 2000; 61(2-3): 91-125.
32. Virus Taxonomy: 2021 Release. *International Committee on Taxonomy of Viruses (ICTV)*.
33. WHO. Oral Rehydration Salts (ORS). 1 January 2006. Finkelstein RA. (1996). Cholera, *Vibrio cholerae* O1 and O139, and Other Pathogenic Vibrios. Ch24, In: *Medical Microbiology*, 4th edition. UTMB Galveston.