

A Review of Sudan's Major Food-Borne Viral and Fungal Diseases

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ABSTRACT

Objective: This review article was conducted to highlight both viral and fungal foodborne situations in Sudan and their health impacts aiming to help health policymakers in introducing strict intervention measures.

Method: The review uses up-to-date data via manual screening of the titles and abstracts of retrieved articles using string foodborne diseases in Sudan and foodborne illnesses as keywords to obtain publications from the electronic databases PubMed, Scopus, and Google Scholar from the year 2000-2020 using the publish or perish tool, the databases were reviewed from January to April 2022.

Results: Foodborne viruses and Mycotoxins produced by certain fungi; are considered among the top priorities and have become of great concern to the food industry over the last few years because their contamination can occur at any point in the nutritional supply chain besides their serious effects on human health both long and short term.

Conclusion: The review on major food borne viral and fungal diseases in Sudan is an important issue to guard against contamination with such micro-organisms and prevent their illnesses.

Keywords: Enteric viruses, Norovirus, Aflatoxins, Mold, Aspergillus flavus.

1. INTRODUCTION

Many serious foodborne outbreaks in the African region result from the consumption of contaminated ready-toeat foodstuffs informally sold as street foods. Thus, the health aspects and food safety pose a real challenge to the food safety regulators. But because of the shortage of food supplies, people care more about hunger satisfaction than food safety, which negatively affected the global situation. The main predisposing factors for foodborne illness include unhealthy practices, undercooking, and lack of refrigerators, besides environmental causes such as polluted water, unsafe waste disposal, and exposure of food to insects or dust (1). This review article aims to highlight both viral and fungal foodborne situations in Sudan and their health impacts.

1.1. Foodborne viruses in Sudan

Infectious diseases, heavy metals, chemical pollutants, and natural poisons like toxic mushrooms produced foodborne

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Content of this journal is licensed under a Creative Commons Attribution-NonCommercial 4.0 International License. illness pandemics. The earliest reported foodborne infection occurred about 323 B.C., when Alexander the Great died of typhoid (2). Foodborne illness was misdiagnosed as poisoning before current microbiology. Before the 1960s, epidemics were frequently poorly recorded and might not have been seen. The 1971 Bon Vivant Outbreak recorded such events. After the Bon Vivant case, many foodborne outbreaks from the 1960s or earlier were not completely written down and may not have been reported until the Jack-in-the-Box Outbreak (3). Foodborne illness outbreaks are most often caused by Norovirus. It was agreed that viral control measures are required all along the food processing chain. However, the effectiveness of such control procedures and how to validate properly their performance still needs to be understood (4). Because viruses do not reproduce in food, their infectivitey depends on virus stability and individual susceptibility. A load of foodborne enteric viral diseases is often difficult to assess since mild illnesses may pass unnoticed, however; such viruses can survive long periods by tolerating fluctuating environmental conditions. And so, testing for water contamination is important to guard against diarrheal disease caused by them. As a result of an enhancement in the sensitivity of detection methods and an increase in reporting, the number of outbreaks has recently increased (4).

1.2. Enteric Viruses

Enteric viruses are microorganisms that spread widely, producing human infections ranging from clinically asymptomatic most often, to gastroenteritis, hepatitis, and even meningitis (5). Such infections are communicated by the faeco-oral route. In the Western world, norovirus (NoV) and hepatitis A virus (HAV) are yet considered at the bleeding edge of human foodborne microbes considering the number of episodes and individuals influenced (6). Other viruses (rotavirus, hepatitis E viruses, and sapovirus) can also represent a risk to mankind (7).

1.3. Foodborne Viral Situation in Sudan

1.3.1. Rotavirus

Worldwide Rotavirus is the main causal agent of severe diarrhea among children under 5 years of age, causing 610,000–870,000 deaths yearly. Class A rotaviruses are the main causative agents of severe diarrhea in children. It is one of the Reoviridae family members that are non-enveloped and has double-stranded RNA. The Rotavirus genus has an 11-segment genome covered (1). In Sudan, viral genotyping was studied based on the viral proteins (VP4 and VP7) by nested PCR and gene sequencing results showed the VP7 predominant G class was G1 (8). Two epidemiological studies conducted on under-5-year-old children infected with rotavirus in Omdurman Pediatric Hospital, Sudan; The first was a setting-based study over one year, which concluded that rotavirus represents a public health problem among Sudanese children younger than five years and that effective

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control procedures are essential to reduce this problem (9). The other study found that rotavirus prevalence was 25% in children under the age of five, and that rotavirus vaccine, accurate and routine diagnosis of acute child diarrheal infection aids in accurate management, preventing antibiotic use, and reducing the prevalence in vulnerable children (10). Although fecal-oral contact is the known mode for transmission of rotavirus, contaminated hands and surfaces and the respiratory route are also reported (11). Another study targeted the spread of rotavirus and adenovirus, which are responsible for diarrhea among refugees in Khartoum, Sudan. All study population data was collected, and ICT and PCR were used as viral detection procedures. The study results showed that most diarrheal cases under five years of age were caused by rotavirus class A (VP6). It also indicated that most of the patients were using raw water obtained from donkey carts and those who lived in poor environmental conditions with no access to toilets (12). Considering the histological pattern of the upper gastrointestinal tract, rotavirus targeted epithelial cells of the proximal part of the small intestine (enterocytes) where they started to replicate. These cells undergo necrosis and shedding with the loss of their digestive enzymes, leading to primary malabsorption, especially for carbohydrates and proteins. Loss of enterocytes leads to villous atrophy with reactive inflammatory reactions and crypt cell hyperplasia. These changes will increase the severity of diarrhea. Recovery from villi takes 1-10 days. Infected children excrete billions of virus particles with each motion of their stools. These viruses can survive in the environment for several days or weeks (13). The malabsorptive component of diarrhea is due to the accumulation of unabsorbed carbohydrates, water, and other nutrients in the intestinal lumen. Since the revelation of the NSP4 enterotoxin, it has been suggested that there is an extra secretory part in the pathogenesis of diarrhea caused by rotavirus (14). Recovery from the first infectious episode may lower the severity of the following motions but does not induce long-lasting immunity (9). Rotavirus infection has a considerable economic impact on medical or non-medical resources, affecting all healthcare settings and resulting in considerable costs for national healthcare payers, patients' families, and employers (15). Detection methods for such viruses include electron microscopy and nucleic acid detection. Both are highly specific and sensitive. Rotavirus viral antigen detection is the most widely accepted diagnostic tool that depends on the revelation of protein antigens on virus molecules in stool samples (16). Molecular-based detection of Astro-virus and class A and B Rotavirus was carried out in children less than 5 years old with gastroenteritis in Aljazeera and Khartoum states, Sudan. All samples were tested by RT-PCR amplifications after RNA extraction to detect Astro-virus. According to this study, Astro-viruses are an important cause of gastroenteritis like rotavirus, and to avoid the spread of nosocomial viral gastroenteritis infections in pediatric units, the detection of rotavirus and Astro-virus assays in clinical diagnosis must be carried out (12).

1.3.2. Noroviruses (NOV)

With the universal improvements in the detection methods of viruses in foods, (NoV) is now considered the commonest virus that causes foodborne disease. The role of asymptomatic food handlers who contribute to the outbreak is becoming increasingly apparent, being responsible for up to a quarter of outbreaks (17). Despite their impacts and prevalence, little information about the life cycle and the pathological aspects related to the norovirus-induced disease is available. Intestinal infection is the basic side, but extra intestinal spread and correlating pathologies have also been detected. Preventive and control measures for Norovirus infections are very difficult because of their low infectious dosage, high shedding titer, and environmental stability. The virus can be transmitted by different routes; foodborne and person-toperson are the most significant (18). Hepatitis and Norovirus in Western countries, infections are among the most significant foodborne pathogens in terms of the number of episodes and the impact on individuals., the clinical result of NoV disease is generally gentle. Asymptomatic diseases are far-reaching and may take an interest in the spread of contamination (19). In any case, information is restricted about the rate and epidemiological information of such infections in Sudan. A study was led to examine the sub-atomic based on the study of disease transmission of NoVs in children under 5 years of age suffering from severe gastroenteritis in Al Gezira state, Sudan. It showed that acute gastroenteritis in children under 5 years of age is mainly caused by norovirus. These findings highlighted the importance of introducing routine norovirus laboratory investigation in hospitals admitting children with gastroenteritis (20). Another study was conducted targeting the molecular epidemiology of bacterial and viral causative agents of diarrhea among children in Khartoum State, Sudan. The study investigated the epidemiological and clinical factors of bacterial and viral etiology in children suffering from severe diarrhea in Khartoum State. The collected samples were tested by Multiplex RT-PCR. Results showed that rotavirus was the main causative agent (10.2%) of the isolated enteroviruses, followed by norovirus G2 (4.0%). Outbreak control is dependent on infection control procedures such as hand washing and disinfection, avoiding contact with infectious individuals, and sterilizing the environment (21).

1.3.3. Hepatitis A virus (HAV)

HAV is a small, non-enveloped, SS – RNA virus, it is resistant for acids and thermostable (22). The common route of infection in non-immunized people is the fecal-oral route. The disease is mainly associated with unsafe food or water, poor sanitation, inadequate personal hygiene, and analoral sex. HAV can live and persist in the environment, and it resists procedures used routinely in food production for inactivation and/or control of bacteria (23). Hepatitis A viruses multiply in the liver cells disturbing its function. It induces an immune reaction that causes inflammation of the liver. HAV infections aren't very well known in Africa, but the available data shows that the endemic is still high in most African countries, except for some parts of South Africa (18). Rate of HAV in asymptomatic food handlers in Khartoum State was targeted by investigating 70 food handlers using the ELISA method for detection and concluded that the high prevalence rates of HAV antibodies detected in their samples raised the possibility of being a source of HAV infection outbreaks in the community (24). Another study obtained the same conclusion after testing 90 food handlers working in Khartoum cafeterias, serologically using an enzyme-linked immuno-sorbent assay (ELISA) to detect IgG and IgM. The virus doesn't cause only chronic liver disease; but it can cause very bad acute liver symptoms that can lead to death. In the year 2016, WHO estimated that people who died due to hepatitis A globally were 134, representing 5% of the total death rate from hepatitis. A report of a very bad case of the same virus has been made by UN peacekeepers in South Sudan (23). The most widely accepted diagnostic tool for hepatitis A is the discovery of the emergence of antibodies, especially of the IgM type using an enzyme immunoassay. However, the detection of HAV RNA is thought to be a good way to tell if someone has the disease even if they don't have specific antibodies (25).

1.3.4. Hepatitis E virus

Hepatitis E virus is the main causative agent of hepatic toxicity globally (HEV), especially in young people, and it often causes fatal symptoms in pregnant women (10). Due to water pollution, the fecal-oral route is the mode of transmission, but there are also animal sources. The course of the HEV is like other acute hepatitis viruses, and the rate of infection is high in young people. The virus does not have a chronic clinical picture, but asymptomatic infections are common. The death rate among pregnant women reached 25% (26). In the periods between the epidemics of hepatitis E, some studies published individual cases in Africa and some research was conducted on the incidence of the disease in Sudan (in patients from the adjacent states of Kordofan and Darfur, 25% were diagnosed with a mortality rate of 10%, especially among pregnant women). In June 2004, a large epidemic of hepatitis E was reported among the displaced camps in the state of Darfur, western Sudan, and at the borders with Chad, which seem to be an attractive area for researchers. About 5,000 HEV infections were recorded over a 6-month duration. A group of work, sponsored by the non-governmental organization Epicenter in refugee camps in Darfur, Doctors Without Borders, WHO, and the Centers for Disease Control in refugee camps in Chad, conducted a field study of this large epidemic (26). Within the same study population mentioned above, another study was carried out to detect the genetic diversity in the composition of the hepatitis E virus in Darfur, western Sudan, and neighboring Chad. The sequence at the ORF2 site was compared between 23 Sudanese and 5 Chadians who were isolated from the virus, and by presenting the results, it was found that more than 99.7% of the strains belong to Genotype 1 with high similarity. While four Chadian isolates were close to Genotype 2, this sparked widespread debate about the possibility of multiple disease sources (27). A third study was carried out to review

hospital records of hospitalized patients to assess the effects of this virus on pregnant women in Mornay camps, Darfur, Sudan, during the epidemic period. The conclusion showed the drastic effect of this disease on pregnant women, with a particular death rate of 31.1% (26). Another study conducted among the same population aiming to impose the feasibility of comparing the amplification of HEV RNA obtained from dried blood spots or serum to explain the biological characteristics of asymptomatic individuals and patients concluded the first report using RT-nucleic acid amplification of the dry blood spot that was obtained in tropical conditions in the refugee camps at the time of the epidemic spread, and because this mechanism is the easiest for children as it only needs 50 ml of blood and it does not need sedimentation or cooling, it may be the most reliable and accurate way to use it in such research in developing countries (26). The role of water treatment methods was the topic of another study conducted among the same population in Darfur, Sudan. A case and a retrospective cohort study were conducted to detect risk factors for asymptomatic and clinical hepatitis E, respectively. Being a young adult and drinking chlorinated surface water were detected as risk factors for both asymptomatic and clinical HEV infection. Although this was not found to be statistically significant, two donkeys were positive for HEV RNA detected in serum samples, concluding that the current precautions to ensure a safe water supply may not be enough to deactivate HEV and control this epidemic. This study indicates the need to evaluate current water manipulation procedures and to identify other methods adapted to complex emergencies (28). A conclusive study conducted to investigate the serological epidemiology among mothers and neonates in Medani Hospital, Sudan, measured the level of HEV Immunoglobulin G (IgG) antibodies and showed a significant rise of these indicators in pregnant women in central Sudan without considering their age, gestational age, or even the number of their births. These results highlight that optimum preventive procedures should be used against HEV infection (29). The seroprevalence of HEV in South Sudan's homeless was detected in another study. The study indicated that infections with HEV are more prevalent than estimated previously, proposing the likelihood of a higher (yet not detected) burden than even reported (30). In a descriptive and cross-sectional study, using immunological tests (ELISA), the frequency of IgG and IgM antibodies in pregnant females at the Maternity Hospital of Dongola, Northern Sudan was determined in a descriptive and cross-sectional study. The study was conducted in November 2015. The results showed that HEV antibody (IgG) has a low frequency among pregnant women, and they recommended that HEV antibody testing should be performed on pregnant females in Sudan, and that prenatal screening for pregnant women will ensure that doctors can pay more attention to preventing the transmission of HEV in the perinatal period (31). A raised prevalence of HEV in pregnant women in Port Sudan city, Sudan in a cross-sectional study was documented in November and December 2015. It investigated the seroreactivity, and HEV RNA molecular positivity in pregnant women was determined (32). Another

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study in Port Sudan, Eastern Sudan, reported a Hepatitis E outbreak in the period of November 2010 to March 2011, where maternal mortality was as high as 28.2%. In this study, serological tests were done using ELISA to investigate IgG and IgM Anti-Hepatitis E virus in 39 pregnant women who came to Port Sudan hospital with different signs of viral hepatitis (33) The seroprevalence of hepatitis E in food handlers in Khartoum, Sudan was investigated among females and males who worked in different cafeterias in Khartoum state, using commercial tests with high specificity and sensitivity (ELISA) to detect the virus. The study concluded that HEV was found to be significantly high among food handlers (15.5%) in Khartoum state (34). The symptoms of foodborne illnesses are vomiting, diarrhea, which might lead to dehydration, fever, colic, headache, arthralgia, and myalgia. The final diagnosis can be done by stool culture or by advanced laboratory techniques. Anyhow, empiric treatment must be started before obtaining the laboratory results if a foodborne illness is suspected. Rehydration is a cornerstone if the patient is clinically dehydrated besides antibiotic therapy. Foodborne disease cases should be reported to health agencies (35).

2. CONTROL OPTIONS OF VIRUSES IN FOOD PROCESSING

Control, prevention, and treatment of foodborne illnesses are very costly. Control measures are limited and include that the vomit and/or diarrhea must be cleaned up and disinfected, besides the avoidance of food handling by infected people. The development of diagnostic procedures increases the chances of the identification of enteric viruses, especially in developing countries where the data is still limited (36). Studies on the stability of foodborne viruses are also minimal because most of them don't grow in laboratory cultures. But such studies confirmed that viruses can survive a variety of preservation methods. Intervention procedures that induce the inactivation of microbes are necessary to obtain a 3-log degradation of the level of viruses. Purification of fresh bivalves, on the other hand, is insufficient to prevent viral outbreaks in any case, because the type of food and the virus tested affect the specific food preservation methods (37).

3. FOODBORNE FUNGI IN SUDAN

3.1. Impact of Mycotoxins on Human Health

Mycotoxins are harmful mixtures that a few kinds of mold (fungi) normally produce. Mold, which can excrete mycotoxins, develops in numerous food varieties. It can form either before or after collecting and during storage, on or in the actual food. This typically happens in hot, wet, and damp conditions. Most mycotoxins are synthetically stable and tolerate food preparation (38). In Sudan, the situation of the inability to address contamination with mycotoxins for basic foods makes all measures to reduce the formation of mycotoxins and pollution urgent and necessary for the Sudanese population (39). Mycotoxins of concern in sorghum in sub-Saharan African countries about prevalence, concentration, and potential exposure to health risks. The results of researchers who conducted a study on these toxins in four countries, including Sudan, indicate an urgent need to study, establish, and implement a coherent pre-and post-harvest management system to ensure safety at all levels in the sorghum production chain in those countries. These countries are encouraged to practice and implement programs to improve the application of good agricultural practices through transportation, storage, and final distribution (38).

3.2. Common Fungi Causing the Foodborne Diseases

Aflatoxins, ochratoxin A, patulin, fumonsens, zyaralenone, nevanol, and deoxy-nephallinol are the most widely recognized mycotoxins that are a source of concern for human and animal wellbeing (40). Mycotoxins show up in the food chain of life because of crop contamination with form both before and after the harvest. Intoxication can happen directly by ingesting contaminated food or indirectly by animals that have been fed with contaminated feed, especially the milk of these animals (41). Mycotoxins are generally found in food varieties, which is why they are of concern. Some foodborne mycotoxins have acute effects and are accompanied by severe clinical symptoms that appear quickly after consuming a contaminated food product, while other mycotoxins are connected to long-term health effects (cancer and immunodeficiency). Ahmed and Al-Bashir (42) examined samples of peanuts and their products that were collected from three Sudanese states: Khartoum in central Sudan, Kordofan in its west, and Gedaref in the east, which represent peanut producing states. The researchers examined the contents of samples of aflatoxin AFB1, AFB2, AFG1 and AFG2 using the method of high-performance liquid chromatography (HPLC) with fluorescence detection. They reported that a sample of peanuts or peanut products was not contaminated with AFG1 or AFG2. The highest numbers of specimens contaminated with mycotoxins are found in 38 analyzed 43 samples of peanut butter from Khartoum state and found 100% contamination.

3.3. Fungi That Producing Mycotoxin

Aspergillus flavus and Aspergillus parasiticus are responsible for the production of aflatoxins, the most toxic mycotoxins that grow in soil, deciduous plants, and grains. Crops frequently affected by the multiple Aspergillus species incorporate oilseeds (soybeans, peanuts, sunflower, and cottonseeds), grains (corn, sorghum, wheat, and rice), spices (chili pepper, black pepper, coriander, turmeric, and ginger) and nut fruits (pistachios, almonds, walnuts, and walnuts). M1 aflatoxin can be found in the milk of animals fed with contaminated feed (43).

Kabbashi et al. (2017) (44), collected and analyzed 25 samples of roasted peanuts in Khartoum for their content of aflatoxins. The results of analyzing aflatoxin content showed a high rate of contamination in all samples, and that there were significant differences between the samples collected from Khartoum, Khartoum North, and Omdurman, and that the contamination rate reached 84%, which is a worrying result that shows the risk of consuming roasted beans contaminated with aflatoxin for consumers, especially children. Large doses of aflatoxin can lead to acute toxicity (aflatoxin poisoning) and may be life-threatening, usually by damaging the liver. Aflatoxins have additionally been demonstrated to be genotoxic, implying that they are harmful to DNA and can cause cancer in animal species (45). A few types of Aspergillus and Mycobacterium produce ochratoxin A, which is produced by and is a common mycotoxin that contaminates food. Ochratoxin A is formed during crop storage and is known to cause several toxic effects on animal species. The most sensitive and prominent effect is kidney damage, but the toxin may also affect the development of the fetus and the immune system. On the contrary, while this link has not been proven in humans, the effect on the kidneys was demonstrated in 2016 (46).

Patulin is a fungal toxin produced by a range of molds, notably Aspergillus, Penicillium, and Byssochlamys. Patulin, which is normally found in apples and spoiled apple items, may also be found in natural products, grains, and other rotten food varieties. The primary wellsprings of patulin in the human eating routine are apples and apple syrup, both produced using defiled organic products. In animals, severe manifestations include liver, spleen, and kidney damage, as well as immune system inebriation. With respect to the individual, queasiness, gastrointestinal unsettling influences, and spewing have been accounted for. Patulin is genotoxic, but the potential for causing cancer has not yet been established (47). Fusarium fungi are common in soil and produce several different toxins, including trichococcins such as deoxy-nephallinol, nephallinol, T2 and HT2 toxins, zearalenone, and phaeomonsenate (48). Mold and toxins occur on a range of different cereal crops. Various fusarium toxins are associated with certain types of grains. For example, Zyaralenone is commonly associated with wheat, the T2 and HT2 toxins are associated with oats, and phyomonsenat is associated with corn. Trichococcins can be highly toxic to humans, causing rapid skin or gastric mucosal irritation and leading to diarrhea. Chronic effects reported to occur in animals include suppression of the immune system. At high intake levels, particularly in pigs, zyaralenone has been shown to have effects on hormones and estrogen and may cause sterility at high intake levels. As for the phyomonsens, it is associated with esophageal cancer in humans and liver and kidney toxicity in animals (49). Patulin is genotoxic, but the potential for causing malignant growth has not yet been established (50). Fusarium is a large genus of soil fungi that produces trichococcins such as deoxynephallinol, nephallinol, T2 and HT2 poisons, zearalenone, and phaeomonsenate (51). Shape and poisons occur in a variety of grain crops. Different fusarium poisons are related to kinds of grains. For instance, Zyaralenone is ordinarily connected with wheat, the T2 and HT2 poisons are related to oats, and phyomonsenat is related to corn. Trichococcins can be profoundly harmful to people, causing fast skin or gastric mucosal disturbances that lead to the runs. Persistent impacts happen in animals that incorporate concealment of

the immune system. Zyaralenone has been demonstrated to have consequences for chemicals and estrogen and may cause sterility at high admission levels, especially in pigs. The phyomonsens is linked to esophageal cancer in humans and liver and kidney toxicity in animals (49).

3.4. How to Reduce the Health Risks from Mycotoxins

Note that the mold that produces mycotoxins can develop on various crops and food sources and get into food sources deeply, not simply on a superficial level. Normally, mold does not grow on foods that are well dried and preserved, so effective drying of goods and keeping them dry, or storing them appropriately, is an effective measure to control mold growth and the production of mycotoxins (52). Ferreira, et al. (53) reported that the UV-C radiation therapy is efficient in fungal decontamination, photodegradation of mycotoxins, and release of bound phenolics in black and red rice grains after exposure for one hour, without affecting changes in cooking and color qualities.

3.5. Aflatoxins

The effect of heat treatment on the level of aflatoxin BI on some Sudanese peanut (Arachies hypogaea L.) samples and peanut products was investigated. In this study, samples of peanuts from Al-Managel, Sudan (irrigated sector), and Kordofan (rainfed sector) regions were obtained and examined. The fungal count was higher in samples collected from the rainfed sector compared to the irrigated sector. The mean level (ppb) of aflatoxin BI (AFBI) was 9.33, 4.67, and 3.33 in the Kordfan 1, Kordofan 2, and Al-Managel samples, respectively (Figure 1). However, AFBI was absent in the Gezira samples. Results also showed that roasting lowered the levels of AFBI in most of the samples that were tested. The greatest reduction was achieved when samples were roasted at 175 °C for 20 minutes of Kordofan samples led to a reduction of 19% in the levels of AFBI, while the reduction was 78% by the roasting-blanching method. The traditional confectionery products made from the most contaminated peanut samples displayed low levels of AFBI in Mundaco and Fulia, while Lucom was devoid of any AFBI. This was because the alkaline conditions in the latter were different from those in the first (54).

Table 1. Permissible limits for some mycotoxins in food

Type of mycotoxin	Permissible limit (μg / kg)	Type of crop
Aflatoxin B + G	0-50	Peanuts – corn – other foods
	0-1000	Fodder
Aflatoxin M1	1.0-0.05	Milk
Ochratoxin A	300	Corn – rice – barley – legumes
Zeralone	30 - 1000	All foods
Patulin	20-30	Apple juice

It has been demonstrated that aflatoxin contamination was found to be high in seeds from insect-damaged pods

compared to mechanically harvested damaged pods. On the other hand, healthy, intact pods are free from aflatoxin contamination. As regards the contamination of the various peanut products (paste, grey and red roasted nuts), both grey and roasted nuts were highly contaminated when compared to the healthy groundnut seeds (55).

In another study, it was found that chips that were sold in the local supermarkets in Wad-Medani city contained high levels of aflatoxins. These snack foods, which are preferred by children, are more likely to be infected with this type of mycotoxin.

In a study conducted (54), they investigated the levels of aflatoxin contamination with A. flavus and the aflatoxin contamination of groundnut samples collected from various locations in Gezira State (Figure 2). Aspergillus flavus development and aflatoxins creation was explored were at their minimum in samples collected from the villages (producing areas) in comparison to elevated levels in samples collected from the supermarkets in Wad Medani city. However, samples collected from local markets in the producing areas were less contaminated. The inhibitory impact of some fundamental oils on Aspergillus flavus development and the creation of aflatoxins was explored (54). The outcomes showed that clove oil was the best inhibitor oil in the diminishing mycelial growth of the organism, concerning both mycelial spiral growth and mycelial dry weight. Also, even though cumin oil was not hindering mycelial development totally, it was second best to clove oil. It gave significantly preferable inhibition over control at all its tried concentrations.

Table 2. Aflatoxin contamination of some peanut products (Packed or non-packed) from various Local markets in the Gezira State, Sudan.

Location	Peanut paste		Roasted Peanut		Fried Peanut	
	Packed	Unpacked	Packed	Unpacked	Packed	Unpacked
Al – Hoosh Al-Medina		0.1	7.0	0.2	9.2	0.01
Arab	8.0	2.0	9.1	0.5	10.1	0.20
Al_Fao	8.5	3.0	10.2	0.6	9.3	0.33
Soug Merkazi	25.3	5.0	29.5	6.6	25.3	6.1
Malaga	28.5	3.0	29.8	7.0	20.0	8.0

4. CONCLUSION

Foodborne diseases are considered one of the main reasons for illness and death, particularly in countries that suffer from poor economic conditions, as in Sudan. The review reported that many types of research revealed that viral and fungal foodborne infections are critical, life-threatening health problems in Sudan; Hoping that national food safety systems

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and protective measures such as awareness and training of employers should be followed and applied in Sudan.

Table 3. Levels of aflatoxin contamination (ppm) of seeds of certain crops other collected from the two markets of Wad medani, Sudan and stored differently for one month

Crops	Packed	Unpacked	Packed	Unpacked
Wheat	3.26	0.21	2.91	0.91
Faba bean	5.16	1.16	3.67	1.68
Phaseolus	9.60	3.25	3.67	2.50
Chickpea	6.33	2.33	5.33	2.10
Sorghum	3.50	0.91	1.90	0.80

Source: Abdelrahim, et al., (2012).

REFERENCES

- WHO, Manual of rotavirus detection and characterization methods', Biologicals, 2009, p. 143.
- [2] Derber CF, Gadkowski LB. Foodborne Infectious Diseases— Historical Perspective and Overview. Food Microbiology: In Human Health and Disease. 2016; Jan 5:1.
- [3] Seeger, Matthew W, and Julie MN. Modeling the recall and warning process in the foodborne contamination event: Perspectives from disaster warnings and crisis communication. Inter J Mass Emerg & Dis. 2010;28, (1): 115-144. DOI: 10.1177/028.072.701002800105
- [4] Bosch A, Gkogka E, Le Guyader F, Loisy-Hamon F, Lee A, van Lieshout L, Marthi B, Myrmel M, Sansom A, Schultz A C, Winkler A, Zuber S, Phister T. Foodborne viruses: Detection, risk assessment, and control options in food processing, Inter J Food Microb. 2018; 285, 110–128. DOI:10.1016/j. ijfoodmicro.2018.06.001.
- [5] Theng-Theng F, and Lipp E, K. Enteric Viruses of Humans and Animals in Aquatic Environments: Health Risks, Detection, and Potential Water Quality Assessment Tools', Microb. and Mol. Bio. Rev. 2005; 69(2): 357–371. DOI: 10.1128/ MMBR.69.2.357.371.2005.
- [6] Sattar SA, Jason T, Bidawid S, Farber J. Foodborne spread of hepatitis A: Recent studies on virus survival, transfer, and inactivation. Can. Infect. Dis. Med. Microbiol. 2000; 11(3): 159–163. DOI:10.1155/2000/805156
- Harrison L and DiCaprio E. Hepatitis E Virus: An Emerging Foodborne Pathogen, Front. Sustain. Food Syst. 2018; 08, (5): 2018 DOI:10.3389/fsufs.2018.00014
- [8] Magzoub MA, Bilal NE, Bilal JA, Alzohairy MA, Elamin B K, Gasim G I. Detection, and sequencing of rotavirus among Sudanese children. The Pan Afric. Med. J. 2017; 28, 87. DOI:11604/pamj.2017.28.87.11008
- [9] Elawad M, and Masaad T. Epidemiology of Rotavirus Infection in Children Less than Five Years in Omdurman Paediatric Hospital, Khartoum, Sudan. Inter J Biomed Res. 2015; 6, (3):219-222. DOI:10.7439/ijbr.v6i3.1804
- [10] Abdalla AE, Nagi, AM, Elawa, EH. Frequency of Rotavirus Infection among Children with Diarrhea in Omdurman Pediatric Hospital, Sudan. Sudan J. Med. Sci. 2013; 8(4):163– 168.
- [11] Dennehy PH. Transmission of rotavirus and other enteric pathogens in the home. Pedia. Infect. Dis. J. 2000; Oct;19, (10Suppl):S103-5. DOI:10.1097/00006.454.200010001-00003. PMID: 11052397.

- [12] Elhussein AM, Mustafa MO, Enan K. Molecular Detection of Rotavirus (A and B) and Astrovirus in Children Less than 5 Years with Gastroenteritis in Khartoum and Aljazeera States, Sudan, (January). Int. J. S. Res. Sci. Engg. Technol. September-October-2018; 4(10): 07-13.
- Rzeżutka A and Cook, N. Survival of human enteric viruses in the environment and food', FEMS Microb, Rev. 2004; 28(4): 441–453. DOI: 10.1016/j.femsre.2004.02.001.
- [14] Carter MJ. Enterically infecting viruses: Pathogenicity, transmission, and significance for food and waterborne infection. J App Microb. 2005; 98(6):1354–1380. DOI: 10.1111/ j.1365-2672.2005.02635. x.
- [15] Riewpaiboon A, Shin S, Le TP, Vu DT, Nguyen TH, Alexander N, Dang DA. Rotavirus Economic Study Group. Cost of rotavirus diarrhea for programmatic evaluation of vaccination in Vietnam. BMC Pub Health. 2016; Aug 11,16(1):777. DOI: 10.1186/s12889.016.3458-2
- [16] Cook SM, Glass RI, Lebaron CW, Ho, Mei-shang. Global seasonality of rotavirus infection. Bull. WHO. 1990; 68(2) 171-177. ISSN: 00439686.
- [17] Iturriza-Gomara M. and O'Brien SJ. Foodborne viral infections. Curr Opin Infect Dis. 2016 Oct; 29(5):495-501. DOI: 10.1097/ QCO.000.000.000000299
- Barclay L, Park GW, Vega E, Hall A, Parashar U, Vinjé J, Lopman B. Infection control for norovirus. Clin Microb Infec. 2014; 20(8): 731–740. DOI:10.1111/1469-0691.12674.
- [19] Koopmans M. and Duizer E. Foodborne viruses: An emerging problem. Inter. J. Food Microb. 2004; Jan 1, 90(1):23-41. DOI:10.1016/s0168-1605(03)00169-7.
- [20] Tatay, EM, El Hussein, AM, Mustafa, MO, Elkhidir, IM, Enan, KA. Molecular Detection of Norovirus 1, 2 in Children Less than 5 Years with Gastroenteritis in Al Jazeera State, Sudan. Inter J Sci Res Sci Eng Tech. 2018; 4, (9): 458–462.
- [21] Ada, MA, Wang J, Enan, KA, Shen, H, Wang, H, El Hussein, AR, Musa, A, Khidi, IM, Ma, X. Molecular survey of viral and bacterial causes of childhood diarrhea in Khartoum State, Sudan. Front Microb. 2018; 9:112. DOI: 10.3389/fmicb.2018.00112.
- [22] Franco E, Meleleo C, Serino L, Sorbara D, Zaratti L. Hepatitis A: Epidemiology and prevention in developing countries. World J Hep. 2012;4(3):68-73. DOI:10.4254/wjh. v4.i3.68.
- [23] https://www.who.int
- [24] Nor MAM, and Ali MA. Prevalence Rate of Hepatitis A Virus among Asymptomatic Food Handlers in Khartoum State (Sudan). Afri J Med. 2019; 4 (7):1-5.
- [25] de Paula VS. Laboratory Diagnosis of Hepatitis A. Fut Viro. 2012;7(5):461-472.
- [26] Gue PJ, Guthmann J, Nicand E. Outbreak of Hepatitis E Virus Infection in Darfur, Sudan: Effectiveness of Real-Time Reverse Transcription-PCR Analysis of Dried Blood Spots. J Clin Microbiol. 2009; Jun;47(6):1931-3. DOI:10.1128/JCM.02245-08.
- [27] Nicand E, Armstrong GL, Enouf V, Guthmann JP, Guerin J-P, Caron M, Andraghetti, R. Genetic heterogeneity of hepatitis E virus in Darfur, Sudan, and neighboring. Chad J Med Viro. 2005; 77: 519–521. DOI:10.1002/jmv.20487.
- [28] Boccia D, Guthmann JP, Klovstad H, Hamid N, Tatay M, Ciglenecki I, Nizou JY, Nicand E, Guerin PJ. High mortality associated with an outbreak of hepatitis E among displaced persons in Darfur, Sudan. Clin Infect Dis. 2006; Jun 15;42(12):1679-84. DOI: 10.1086/504322.

- [29] Eltayeb R, Gasim GI, Elhassan EM, Rayis, D, Abdullahi H. Maternal and newborn seroprevalence of hepatitis E virus at Medani Hospital, Sudan. F1000Res 2015; 4:823. DOI: 10.12688/f1000research.7041.
- [30] Azman AS, Bouhenia M, Iyer AS, Rumunu J, Laku RL, Wamala JF, Rodriguez-Barraquer I, Lessler J, Gignoux E, Luquero FJ, Leung DT, Gurley ES, Ciglenecki I. High Hepatitis E Seroprevalence Among Displaced Persons in South Sudan. Amer J Trop Med Hyg. 2017; 96(6):1296-1301. DOI:10.4269/ajtmh.16-0620.
- [31] Younes HA. Seroprevalence of Hepatitis E virus (HEV) antibodies (IgG, IgM) among Sudanese Pregnant women in Dongola Maternity Hospital, Northern Sudan. MSc. Thesis, Faculty of Medical Laboratory, Sudan University of Science & Technology, Khartoum, Sudan. http://repository.sustech.edu/ handle/123456789/12802. 2015
- [32] Mustafa E, Mustafa MO, Khair OM, Hussein AM, Elkhidir IM, Enan KA. Seroprevalence and Molecular Detection of Hepatitis E Virus (HEV) Among Pregnant Women in Port Sudan State. Inter J Sci Res Sci Eng Tech. 2017;3, (2):2394-4099.
- [33] Rayis DA, Jumaa, AM, Gasim, GI, Karsany, MS., Adam, I. An outbreak of hepatitis E and high maternal mortality at Port Sudan, Eastern Sudan. Pathog Glob Health. 2013; Mar; 107(2): 66–68. DOI: 10.1179/204.777.3213Y.000.000.0076.
- [34] Yusuf SU, Garbi MI, Saad FMS. Seroprevalence of hepatitis e among food handlers in central Khartoum Sudan. Inter J Multidis Res Develop. 2015; 2, (11): 351-354.
- [35] Switaj TL, Winter KJ, Christensen SR. Diagnosis and Management of Foodborne Illness. Amer Fam Phys. 2015; Sep 1;92(5):358-365. DOI:10.1037/e548332006-001.
- [36] Yeargin T, Gibson KE. Key characteristics of foods with an elevated risk for viral enteropathogen contamination. J. Appl. Microbiol. 2019; Apr;126(4):996-1010. DOI:10.1111/ jam.14113.
- [37] Baert L, Debevere J, Uyttendaele M. The efficacy of preservation methods to inactivate foodborne viruses. Inter J Food Microb. 2009; 31, (131):2-3):83-94. DOI: 10.1016/j. ijfoodmicro.2009.03.007.
- [38] Ssepuuya G, Van Poucke C, Ediage EN, Mulholland C, Tritscher A, Verger P, Kenny M, Bessy C, De Saeger S. Mycotoxin contamination of sorghum and its contribution to human dietary exposure in four sub-Saharan countries. Food Addit Contam Part A Chem Anal Control Expo Risk Assess. 2018 Jul; 35(7):1384-1393. DOI:10.1080/19440.049.2018.1461253.
- [39] Motarjemi Y. and Lelieveld H. Food safety management: a practical guide for the food industry. 2013; Academic Press.
- [40] Elzupir AO, Suliman MA, Ibrahim IA, Fadul MH, Elhussein, AM. Aflatoxins levels in vegetable oils in Khartoum State, Sudan. Mycot Res. 2010; 26(2):69-73. DOI:10.1007/ s12550.010.0041-z.
- [41] 41. Bennett J.W. and Klich, M. Mycotoxins. Clin Microb Rev. 2003;16, 497-516. DOI:10.1128/CMR.16.3.497.516.2003
- [42] 42. Ahmed SAA. and Elbashir A A. Determination of Aflatoxins in Groundnut and Groundnut products in Sudan using AflaTest[®] and HPLC. Memorias del Instituto de Investigaciones

en Ciencias de la Salud, 2016;14(2):35-39. DOI: 10.18004/ mem.iics/1812-9528/2016.014(02)35-039.

- [43] 43. Kang'ethe EK and Lang'a KA. Aflatoxin B1 and M1 contamination of animal feeds and milk from urban centers in Kenya. Afri. Health Sci. 2009; 9(4): 218–226.
- [44] 44. Mokhtari A, and Van Doren JM. An agent-based model for pathogen persistence and cross-contamination dynamics in a food facility. Risk Anal. 2019; 39: 992–1021. DOI: 10.1111/ risa.13215.
- [45] 45. Verma R J. Aflatoxin Cause DNA Damage. Inter. J. Human Gen. 2004; 4, (4): 231-236. DOI:10.1080/09723.757.2004.11 885899.
- [46] 46. Kőszegi T, and Poór M. Ochratoxin A: Molecular Interactions, Mechanisms of Toxicity and Prevention at the Molecular Level. Toxins (Basel). 2016; Apr 15;8(4):111. DOI:10.3390/ toxins8040111.
- [47] 47. Patial V, Asrani RK. Thakur M. Food-borne mycotoxicoses: Pathologies and public health impact, in Foodborne Diseases. Handbook of Food Bioengineering, Elsevier, 2018; pp. 239– 274.
- [48] 48. Perincherry, L, Lalak-Kańczugowska J, Stępi Ł. Fusariumproduced mycotoxins in plant-pathogen interactions. Toxins (Basel), 2019; Nov 14;11(11):664. DOI: 10.3390/ toxins11110664.
- [49] 49. Reddy KE, Lee W, Jeong JY, Lee Y, Lee HJ, Kim MS, Kim DW, Yu D, Cho A, Oh YK, Lee SD. Effects of deoxynivalenol – and zearalenone-contaminated feed on the gene expression profiles in the kidneys of piglets. Asian-Austr J Anim Sci. 2018; 31(1): 138–148. DOI:10.5713/ajas.17.0454.
- [50] 50. Pal, Singh N, Ansari, KM. Toxicological effects of patulin mycotoxin on the mammalian system: an overview. Toxic Res. 2017; 6, (6):764–771. DOI:10.1039/c7tx00138j.
- [51] 51. Jimenez-Garcia SN, Garcia-Mier L, Garcia-Trejo JF, Ramirez-Gomez XS, Guevara-Gonzalez RGA, Feregrino-Perez, A. Fusarium Mycotoxins and Metabolites that Modulate Their Production. In (Ed.), Fusarium – Plant Diseases, Pathogen Diversity, Genetic Diversity, Resistance and Molecular Markers. IntechOpen. 2018; DOI:10.5772/intechopen.72874
- [52] 52. Bhat R, Rai, RV, Karim, AA. Mycotoxins in food and feed: present status and future concerns. Comp Rev Food Sci Food Saf. 2010; 9(1): 57–81.
- [53] 53. Ferreira, CD, Lang, GH, Lindemann, IS, Timm, NS, Hoffmann, JF, Ziegler, V, de Oliveira, M. Postharvest UV-C irradiation for fungal control and reduction of mycotoxins in brown, black, and red rice during long-term storage. Food Chem. 2021; 339, 127810. DOI:10.1016/j.foodchem.2020.127810.
- [54] 54. Sulieman, AE, Ali, MA, Abdelrahim, AM. Effect of heat treatment on the level of aflatoxin BI (AFBI) on some Sudanese peanut (*Arachis hypogaea* L.) samples and peanut products. Sudan J Agri Res. 2007; 11, (9): 16-21.
- [55] 55. Abdel-Rahim, AM, Alsheikh, SM, Suleiman, AME. Aflatoxin Contamination of Some Crop Seed Types and their Products in the Gezira State, Sudan. Gezira J Eng Appl Sci. 2010; 5(2); 1-16.

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