

Health Impacts of Foodborne Mycotoxins and Factors Affecting Occurrence of Toxigenic Fungi and Their Toxins in Stored Foods

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Abstract

Many cereal and other crops are susceptible to fungal attack either in the field or during storage. These fungi may produce as secondary metabolites a diverse group of chemical substances known as mycotoxins. Poor harvesting practices, improper storage and less than optimal conditions during transportation, marketing and processing can also contribute to fungal growth and increase the risk of mycotoxin production. However a large number of fungi are associated with groundnut kernel, maize, rice, and sorghum grain, chilli, and various spices, the most common mycotoxin-producing fungi are aspergillus spp(*Aspergillus flavus*, *Aspergillus parasiticus*, *Aspergillus ochraceus*), fusarium spp (*Fusarium verticillioides*, *Fusarium graminearum*, *F. proliferatum*) and *Penicillium* spp (*Penicillium verrucosum*). Among the mycotoxins fitting into this major group would be the aflatoxins, deoxynivalenol, fumonisins, zearalenone, T-2 toxin, ochratoxin and certain ergot alkaloids. The diseases (mycotoxicoses) caused by these mycotoxins are quite varied and involve a wide range of susceptible animal and humans. Most of these diseases occur after consumption of mycotoxin contaminated grain or products made from such grains but other routes of exposure exist. The resulting implications include immunosuppression, impaired growth, various cancers and death depending on the type, period and amount of exposure. Possible intervention strategies include good agricultural practices such as early harvesting, proper drying, and sanitation, storage, transportation, marketing and insect management among others. Other possible interventions include biological control, chemical control, and decontamination, breeding for resistance as well as surveillance and awareness creation.

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1. Introduction

Many cereal and other crops are susceptible to fungal attack either in the field or during storage. These fungi may produce as secondary metabolites a diverse group of chemical substances known as mycotoxins. There can be wide year to year fluctuations in the levels of mycotoxins in foods, depending on many factors, such as adverse conditions favoring fungal invasion and growth. Many mycotoxins were initially identified after they had caused a variety of subacute health problems in livestock as well as humans, with many target organs and systems affected. With modern farming, storage and processing practices, the aim is to reduce obvious contamination, and much of our concern now focuses on chronic effects at low levels of exposure (Magan and Olsen, 2004). The term mycotoxin was used for the first time in 1961 in the aftermath of a veterinary crisis in England, during which thousands of animals died. The disease was linked to a peanut meal, incorporated in the diet, contaminated with a toxin produced by the filamentous fungus *Aspergillus flavus* (Bennet & Klich, 2003; Richard, 2007). In general, mycotoxins are low-molecular-weight compounds that are synthesized during secondary metabolism by filamentous fungi; their chemical structure may range from simple C₄ compounds to complex substances (Paterson & Lima, 2010). Mycotoxins are natural contaminants in raw materials, food and feeds. Mould species that produce mycotoxins are extremely common, and they can grow on a wide range of substrates under a wide range of environmental conditions; they occur in agricultural products all around the world (Bennet & Klich, 2003). Many mycotoxins may be toxic to vertebrates and other animal groups and, in low concentrations, some of them can cause autoimmune illnesses, and have allergenic properties, while others are teratogenic, carcinogenic, and mutagenic (Bennet & Klich, 2003; CAST, 2003).

Worldwide, it is generally claimed that natural products are safe. However, contamination of human or animal food (or feed) via natural biotoxins produced by microbes might result in outbreaks of several diseases. Among the microbes, fungal toxins assume more importance due to their worldwide distribution. The colonizing fungi are capable of producing toxins, and can cause deleterious health effects in humans or in livestock consuming the contaminated products. Such cases of fungal poisoning may cause death in animals, but are rarely fatal to humans (Pfohl-Leszkowicz 2000).

Mycotoxins are toxic secondary metabolites produced by fungi and contaminate various agricultural commodities either before harvest or under post-harvest conditions. Tropical conditions such as high temperatures and moisture, monsoons, unseasonal rains during harvest and flash floods lead to fungal proliferation and production of mycotoxins (Bhat and Vasanthi, 2003). Poor harvesting practices, improper storage and less than optimal conditions during transportation, marketing and processing can also contribute to fungal growth and increase the risk of mycotoxin production. These climatic conditions as well as the food

production chains are characteristic in most parts of Africa. Consequently, the threat of mycotoxin contamination of foods and feeds resulting in human and livestock poisoning is real and of major concern (Lewis et al., 2005; CDC, 2004).

Poor post-harvest management can lead to rapid deterioration in nutritional quality of seeds. Microbial activity can cause undesirable effects in grains including discoloration, contribute to heating and losses in dry matter through the utilization of carbohydrates as energy sources, degrade lipids and proteins or alter their digestibility, produce volatile metabolites giving off-odours, cause loss of germination and baking and malting quality; affect use as animal feed or as seed. Filamentous fungal spoilage organisms may also produce mycotoxins that can be carcinogenic or cause feed refusal and emesis (Magan et al., 2004). The food-borne mycotoxins likely to be of greatest significance in Africa and other tropical developing countries are the fumonisins and aflatoxins (WHO, 2006). Mycotoxins attract worldwide attention because of the significant economic losses associated with their impact on human health, animal productivity and trade (WHO, 2006; Wu, 2006).

Cereal plants may be contaminated by mycotoxins in two ways: fungi growing as pathogens on plants or growing saprophytically on stored plants (Glenn, 2007). However, not all fungal growth results in mycotoxin formation and detection of fungi does not imply necessarily the presence of mycotoxins. Consumption of a mycotoxin-contaminated diet may induce acute and long-term chronic effects resulting in teratogenic, carcinogenic, and estrogenic or immune suppressive effects. Direct consequences of consumption of mycotoxin-contaminated animal feed include: reduced feed intake, feed refusal, poor feed conversion, diminished body weight gain, increased disease incidence (due to immune-suppression), and reduced reproductive capacities (Fink-Gremmels and Malekinejad, 2007; Morgavi and Riley, 2007; Pestka, 2007; Voss and Haschek, 2007) which leads to economic losses (Huwig et al., 2001; Wu, 2004; Wu, 2006).

Due to modern laboratory methods and a growing interest in this field of research, more than 300 different mycotoxins have been differentiated thus far. However for a practical consideration in the feed-manufacturing process only a small number of toxins are of relevance, with aflatoxins, trichothecenes, zearalenone, ochratoxins and fumonisins being of particular interest, although it has to be mentioned that the extent of harm each toxin (group) can cause is highly species-dependant (Erber and Binder, 2004). Approaches to prevent mycotoxicoses include pre- and post-harvest strategies; the latter are often categorized into physical, chemical and biological methods (Jouany, 2007). The best way would be the prevention of mycotoxin formation in the field in the first place, which is supported by proper crop rotation and fungicide administration at the right time. In case of toxin manifestation, measures are required that act specifically against certain types and groups of toxins. The most prevalent approach counteracting mycotoxins in the feed industry is to include sorbent materials into the feed, for more or less selective removal of toxins by means of adsorption within the route of the gastrointestinal tract, or to add enzymes or microbes capable of detoxifying certain mycotoxins or toxin groups (Leibetseder, 2005).

Most tactics aimed at mycotoxin prevention are essentially disease management practices whose goal is to reduce infection of the plants or grain by toxigenic fungi. Mitigation of mycotoxin problems can include tactics for reducing mycotoxin concentrations or simply diverting contaminated grain into uses with a greater tolerance for contamination (Leonard and Bushnell, 2003). Climate changes seem to be another important factor affecting mycotoxin contamination of foods and feedstuffs (Paterson and Lima 2010). Depending on the geographical and climate conditions, different fungal species can invade foods and feedstuffs. *Aspergillus*, *Penicillium*, and *Fusarium* species are the most important mycotoxin producers. *Penicillium* and *Aspergillus* species can grow at higher temperature and lower aw than *Fusarium*. *Fusarium* species grow well at higher aw and lower temperature (Bhat and others 2010). *Aspergillus* species can be found on nuts, cereals, palm kernels, cocoa, and coffee beans. Depending on the structure and biological origin, mycotoxins can be classified into 4 categories (polycetoacids, terpenes, cyclopeptides, and nitrogenous metabolites) (Bhat et al., 2010). Among all mycotoxins, aflatoxins (AFs), ochratoxin A (OTA), trichothecenes (deoxynivalenol (DON) and T-2 toxin), zearalenone (ZEN), and fumonisins (FMN) have received much attention due to high frequency and severe health effects in humans and animals (Bhat and others 2010). AFs, DON, and ergot alkaloids are usually produced at preharvest stages, while FMN and OTA are mainly produced during postharvest activities (Bhat et al., 2010). Naturally contaminated crops may contain multiple mycotoxins resulting in more severe effects. Multitoxin occurrence or co-occurrence of mycotoxins results in synergistic effects of mycotoxins, especially in acute toxicities in animals (such as AFs with DON, and T2 toxin, OTA with FMN, and FMN with DON) (Binder et al., 2007).

1.1 Objectives

- ✓ To review health impacts of foodborne mycotoxins in stored foods.
- ✓ To review factors affecting occurrence of mycotoxins in stored foods and their postharvest control and management.

2. Literature review

2.1 Occurrence and significance of food-borne mycotoxins in developing countries

The term mycotoxin literally means poison from fungi. Among the thousands of species of fungi, only about 100 belonging to genera *Aspergillus*, *Penicillium* and *Fusarium* are known to produce mycotoxins. Out of the 300–400 mycotoxins known, the most important are aflatoxins, ochratoxins, deoxynivalenol (DON or vomitoxin), zearalenone, fumonisin, T-2 toxin and T-2 like toxins (trichothecenes). Deoxynivalenol, zearalenone, T-2 toxin and fumonisin are all produced by fungi of the genera *Fusarium*. Crops in tropical and subtropical areas are more susceptible to contamination than those in temperate regions, since the high humidity and temperature in these areas provide optimal conditions for toxin formation (Thomson and Henke, 2000). The Food and Agricultural Organization of the United Nations (FAO) has estimated that up to 25% of the world's food crops are significantly contaminated with mycotoxins. However, the presence of mycotoxins in food is often overlooked in Africa due to public ignorance about their existence, lack of regulatory mechanisms, dumping of food products, and the introduction of contaminated commodities into the human food chain during chronic food shortage due to drought, wars, political and economic instability (MERCK, 2006). Ethical considerations also play a role during the manufacturing process of food products using heavily contaminated commodities and sometimes “diluting” contaminated agricultural products such as peanuts with good quality products to an “acceptable” level below the regulatory level (MERCK, 2006). While aflatoxins occur mostly in maize and groundnuts, the prevalence of fumonisins is 100% or close to it in all surveillance data that have been reported on maize from different parts of Africa (Bankole et al., 2006). Limited surveys have also established presence of ochratoxin A, trichothecenes and zearalenone in the continent (Bankole et al., 2006; Muthomi et al., 2002). Aflatoxin poisoning has been associated with eating home grown maize and storing it under damp conditions (Lewis et al., 2005). Acute aflatoxin poisoning has severally occurred in Eastern and Central provinces of Kenya (Lewis et al., 2005; CDC, 2004).

Table 1: Mycotoxins of public health concern, associated fungi, disease associated and food/feed crops at risk of contamination

Mycotoxin	Producing fungi	Associated food/feed crops	mycotoxicoses
Aflatoxins	<i>Aspergillus flavus</i> <i>A. parasiticus</i>	Maize, peanuts, tree nuts, copra, spices, cottonseed	carcinogenesis and inducement of hepato-cellular carcinoma (HCC)
Fumonisin	<i>Fusarium verticillioides</i> <i>F. proliferatum</i> <i>A. niger</i>	Maize	liver or pancreatic necroses
Ochratoxin A	<i>Penicillium verrucosum</i> <i>A. ochraceus</i> <i>A. carbonarius</i> <i>A. niger</i>	Maize, wheat, barley, oats, dried meats and fruits, coffee, wine	cause kidney and bladder dysfunction, increased water consumption, blood in urine and faecal samples, liver damage
Deoxynivalenol	<i>F. graminearum</i>	wheat, oats, and barle	associated with diarrhea, nausea, headache, dizziness, gastroenteritis, and ataxia
Zearalenone	<i>Fusarium graminearum</i> and <i>F. culmorum</i>	wheat, barley, sorghum and rye	reproductive disorders, ovarian dysfunction, infertility, increase in abnormal spermatozoa,
Ergot	<i>Claviceps</i>	several grass species; pearl millet	gangrene and loss of limbs
T-2 toxin	<i>F. sporotrichioides</i>	Corn, wheat, barley, oats, rice, rye	gastrointestinal tract, skin, lymphoid and erythroid cells

Source; (Wu et al., 2014)

2.2 Major Types of stored food Mycotoxins and Their Health Effects

2.2.1 Aflatoxins

Aflatoxins are polyketide secondary metabolites from toxigenic strains of *Aspergillus* and related species. Chemically they are difuro-coumarins which are freely soluble in chloroform and methanol. They are stable at high temperatures but unstable to UV light or polar solvents. These groups of mycotoxins are closely related in structure and there are 18 different relatives of aflatoxins, out of which four AFB1, AFB2, AFG1 and AFG2 are reported agriculturally common. However, AFB1 and AFB2 are the most important and AFB1 have been adjudged the most toxic (Bhat and Vasanthi, 2003; Greekmore, 2012; Da Costa et al. 2010). According to Razzaghi-Abyaney et al., 2010; *Aspergillus flavus*, *Emericella astellata*, *E. venezuelensis* and *A. pseudotamarii* produce AFB1 and AFB2 while *A. parasiticus*, *A. nominus*, *A. bombycus*, *A. miniscleotigenes*, and *A.*

arachidicola produce AFB1, AFB2, AFG1 and AFG2 toxins. The toxicity profile of aflatoxins is B1>M1>G1>B2>M2>G2 (Farang, 2008). AFB1 has been reported to contaminate 25% of the global food supply. Aflatoxigenic organisms are common fungi that inhabit the soil and a variety of decaying organic matter as saprophytes (Razzaghi-Abyanay *et al.*, 2013). A recent study documented that the distribution of aflatoxin contamination in different agro-ecological zones of Senegal established that the soil is a reservoir for field infections in much the same way as poor storage practices (Tiffany, 2013). *A. flavus* is widespread in both temperate and tropical countries (Greekmore, 2012). *Aspergillus spp.* can attack various commodities including groundnut, maize and spices, vegetables, cocoa, coffee, beer, eggs and meat amongst many others agricultural products. Affected grains may not appear overtly mouldy and producing fungus proliferates in improperly stored grains of moisture content greater than 14%, relative humidity greater than 70%, pH 4-6 and temperature 30-40°C (Greekmore, 2012; Whitlow and Hagler, 2013). Maize is prone to field infections by *Aspergillus* and the crop debris is reported as the primary source of inocula in the USA. The fungal sclerotia survive many years in the soil, germinate and produce numerous conidia during silking. Soil dwelling mites feed on the germinated sclerotia and thus spread the conidia. Contaminations generally are more frequent in tropical zones with inclement weather situations; in such zones replete with poor traditional storage facilities, rice is also prone to aflatoxin contamination. Insect damage exacerbates kernel infection in maize grown in the USA. *Aspergillus* ear rot and mouldiness are favoured by hail, drought stress and early frost. However, the amount of aflatoxin deposit in corn is influenced by high temperatures, high grain moisture content (15-18%), pH and nitrogen deficiency (John and Steve, 2010; Tiffany, 2013).

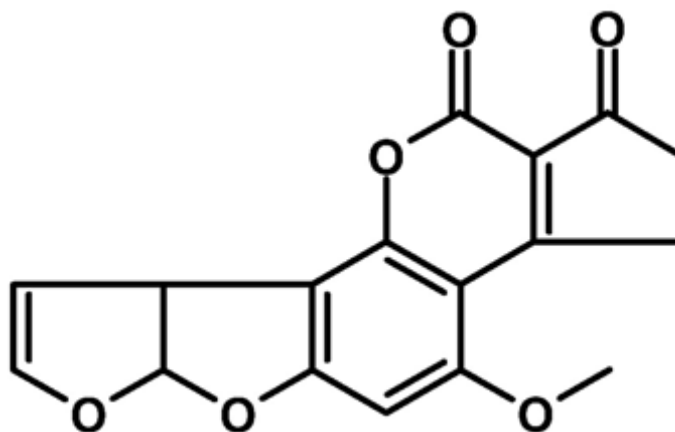


Fig 1 Structure of aflatoxin B1 as a representative of the aflatoxins.

Aflatoxins are heat stable, not eliminated completely by normal cooking procedure (Kishore *et al.*, 2002). They are efficiently absorbed in the small intestine perhaps at the duodenum. The liver is their main target organ but may affect other organs based on their reactivity with DNA, RNA, enzymes and proteins. Mycotoxins undergo metabolism via cytochrome p450 enzymes in the liver. WHO in 2006 reported the presence of AFM1 in human breast milk in Nigeria and affirmed that children feeding on such breast milk will be stunted and may suffer neurological impairment. Aflatoxin epoxide (8, 9 epoxide) is their major toxic metabolite which binds to DNA or RNA to form adducts. Adducts especially at N7-guanine implicates the compound in carcinogenesis and inducement of hepato-cellular carcinoma (HCC). One such mutation is suspected to occur at human P53 tumor suppression gene at codon 249, leading to the conversion of guanine (G) to thymine (T) at the third nucleotide of the codon (Murphy *et al.*, 2006).

Besides these, aflatoxin contamination causes inferior crop quality and barriers international marketability of produce; especially oil seeds such as groundnut which require stricter lower limits of aflatoxins for exports into the EU and USA (Razzaghi-Abyanay *et al.*, 2010). According to Tiffany (2013), 85% of samples of peanut oil from Senegal contain on the average 40 ppb of AFB1. Maize samples from Nororo district of the same country showed up to 852.5 ppb while locally produced snack peanuts and feed cakes showed aflatoxin levels of between 25-236 ppb. These toxins jeopardize food safety exceeding standards set for food for human consumption and impairing exportability of these commodities into the USA or UAE and EU with maximum allowable total aflatoxin limits of 20 ppb and 4 ppb respectively (Farang, 2008; Allameh *et al.*, 2011; Tiffany, 2013). Consequently, Africa loses 670-750 million USD annually to EU restrictive standard for aflatoxins on cereals, dried fruits and nuts; and billions on the same issue the world over (Okello *et al.*, 2010). Members of the African Groundnut Council put the annual costs of programmes aimed at preventing or reducing aflatoxin contamination at US\$ 7.5 million (Bhat and Vasanthi, 2003). Economic losses however, may reach 100% when the entire product is rejected by the markets (Okello *et al.*, 2010).

2.2.2 Fumonisin

Fumonisin were first isolated from culture studies in South Africa in 1988 (Viljoen, 2013) and are widely

reported throughout the continent of Africa today (Okello *et al.*, 2010). As a matter of fact fumonisins have assumed as much toxicological importance as aflatoxins (Bhat, 2008). Chemically they are similar in structure to sphingosine a component of sphingolipids which are concentrated in myelin and certain nerve tissues of living organisms. Fumonisin B1, B2 and B3 are produced by *Fusarium moniliforme* (*F. verticilloides*), *F. proliferatum*, *F. nygamai* and *Alternaria alternata* (Withlow and Hagler, 2013; Reddy *et al.*, 2010a; Viljoen, 2013). These *Fusarium* pathogens have been implicated in causing fusarium kernel rot of maize and other cereals, a disease associated with dryness and insect damage. Other fumonisin organisms are *F. chlamydosporium*, *F. sambucinum*, *F. semitectum* and *F. equiseti* (Goswami *et al.*, 2008). *Fusarium verticilloides* has been underscored as cause of seedling blight, stalk rot and ear rot of some cereals (Reddy *et al.*, 2010a). *Fusarium spp.* are widely distributed plant pathogens and known contaminants of stored agricultural products or waste grains (Greekmore, 2010). Hot humid conditions with temperature ranges of 40-65°C is reported to favour fusariotoxin production on grains (Reddy *et al.*, 2010a). According to Bryden (2007), fumonisins are not only associated with maize but also commonly found in maize-based products. In like manner studies showed that *F. equiseti* isolated from ginseng fields attacked a wide range of legumes and cereals such as common bean, bush bean, broad bean, alfalfa, canola and wheat (Goswami *et al.*, 2008). This organism in an evaluation in South Africa produced fumonisin B1 ranging from 0.12-0.61µg/g on cowpea seeds; with total fumonisins peaking at 25.30µg/g in the study (Kritzinger *et al.*, 2003). Fumonisin organisms attack stressed and senescent crops in the field. *Fusaria* and their related species occur systemically in roots, stems, leaves and kernels of susceptible crops and can be recovered from all maize kernels. Studies according to this source suggested that the fungi seem to have a mutualistic association with maize crop producing metabolites of value such as fusaric acid and gibberellins for the plant. Organisms exposed to fumonisins show pathological changes such as liver or pancreatic necroses, kidneys and liver weight increases, liver damage, increased concentration of haemoglobin, diarrhoea and neurotic effects (Mycotoxin Information, 2013). At low doses fumonisins caused liver and kidney damage in pigs. They affect swines, donkeys and mules; and the fatal disease of horses' equine leukoencephalomalacia (ELEM) has been attributed to fumonisins. Fusarial metabolites are hepatotoxic and their cancer promoting activity in rats has been reported. Fumonisin B1 is also possible human carcinogen (Withlow and Hagler, 2013). Oesophageal cancer cases in China, Northeast Italy and Transkei Region of South Africa have been reported to be linked to fumonisins (Bhat and Vasanthi, 2003; Reddy *et al.*, 2010a; Withlow and Hagler, 2013). According to John and Steve (2010), up to 10 fumonisins have been identified; however Fumonisin B1, B2 and B3 are commonly of agricultural importance with B1 reputed as the most toxic. Reddy *et al.* (2010) noted that intact fumonisins have low bioavailability. Heating such as roasting during food processing noted these authors, converts fumonisins side chains to chemically reactive forms that couple the toxins covalently to amino groups on proteins. Studies have also shown that frying may increase the inherent toxicity of hydrolysed fumonisins by converting them to N-fatty acetyl-hydrolysed fumonisins which have been found 10 times more toxic in culture trials. These toxic metabolites affect sphingolipid synthesis and the biosynthesis of the polyketides. Fumonisin toxicity is thought to result from causing electrolyte loss in affected tissues, blockage of sphingolipid synthesis; or disruption of sphingolipid metabolism through inhibition of ceramide synthase activity (Withlow and Hagler, 2013).

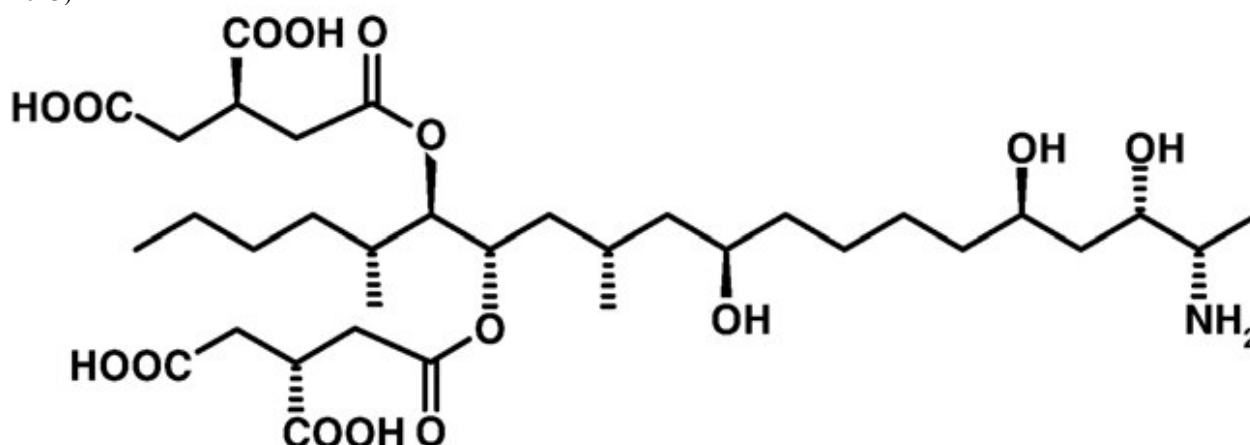


Fig. 2 Structure of fumonisin B1 as a representative of the fumonisins

2.2.3 Ochratoxins

Ochratoxins are naturally occurring mycotoxins soluble in organic solvents, aqueous solution of sodium bicarbonate and slightly soluble in water (Sorrenti *et al.*, 2013). Chemically they consist of isocoumarin moiety linked by a peptide bond to phenylalanine (Mycotoxin Information, 2013). They are produced by *Aspergillus ochraceus*, *A. auricomus*, *A. glaucus*, *A. carbonarius*, *A. alliaceus*, *A. melleus*, *A. niger*, *Penicillium verucosum*

and related fungi. *A. niger* plays roles in industrial manufacture of enzymes and citric acid; as such care should be taken that only a toxigenic strains of the fungus is employed in such processes. *Penicillium verucosum* is common to temperate Northern Europe while, *A. ochraceus* is pan-tropical in distribution. These groups of mycotoxins are entirely actively produced in storage though there are some reports of *P. verucosum* infecting produce between anthesis and harvest (Reddy *et al.*, 2010a). Ochratoxins are mainly found associated with cereals but have also been isolated from wines, coffee, spices and dried fruits (Bryden, 2007). Its occurrence on barley is high on a world scale (Reddy *et al.*, 2010a), but common on cocoa in SSA (Fapohunda, 2013). Four ochratoxins have been identified as ochratoxins A, B, C, and D with ochratoxin A (OTA) a non-ribosomal peptide synthase being the most potent poison. They have been adjudged nephrotoxic and carcinogenic (Fapohunda, 2013). Mycotoxin Information (2013) reported that ochratoxins cause kidney and bladder dysfunction, increased water consumption, blood in urine and faecal samples, liver damage, diarrhoea and suppressed immunity to environmental and microbial stressors as some symptoms presented by organisms affected by OTA.

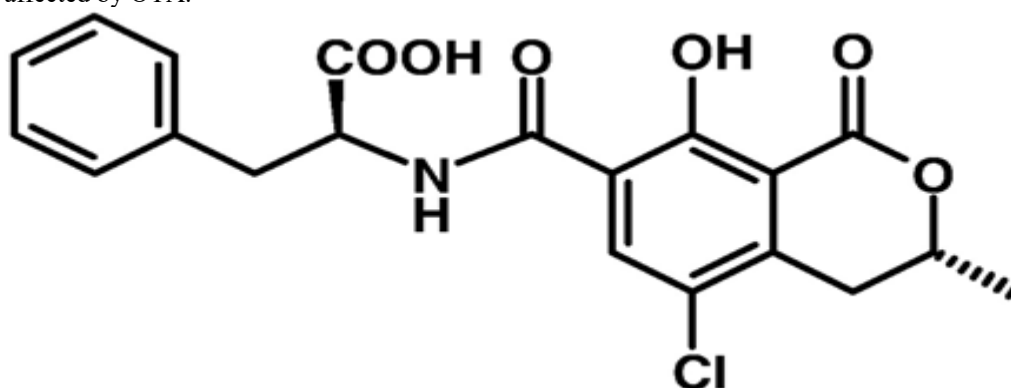


Fig.3 Structure of ochratoxin A

2.2.4 Deoxynivalenol

DON, also called food-refusal factor or vomitoxin, is a member of the trichothecene mycotoxins class. The major DON producing mold is *F. graminearum* (Hallen-Adams *et al.*, 2011; Ortega *et al.*, 2013). Optimum conditions for DON production was at 0.995 aw and 30 °C after 6 wk. DON is very heat stable; however, evidence suggests that concentration of DON may be decreased by processing primarily by boiling in water as DON levels were reduced in noodles and spaghetti by cooking in a large volume of water (Visconti *et al.*, 2004; Kushiro *et al.*, 2012). DON exhibits toxicity in a variety of ways. It inhibits synthesis of RNA, DNA, and protein (Eriksen and Pettersson 2004). Furthermore, studies on experimental animals have revealed that DON causes genotoxicity, cytotoxicity (Awad *et al.*, 2014), teratogenicity, and induction of fetal skeletal deformities (Zhao *et al.*, 2012). DON is associated with diarrhea, nausea, headache, dizziness, gastroenteritis, and ataxia in animals and humans. Consumption of DON-contaminated food stimulates vomiting and at exceedingly higher concentrations it is lethal (Pestka 2007; Sobrova *et al.*, 2010).

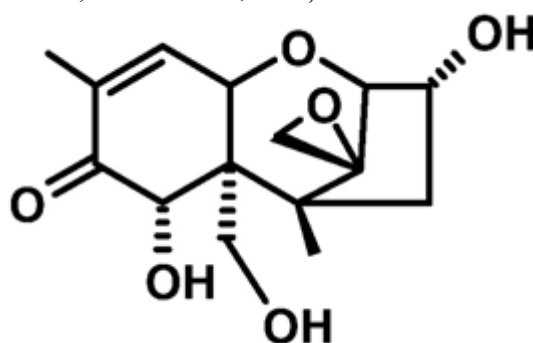


Fig 4 Structure of deoxynivalenol

Corn and small grains such as wheat, oats, and barley are the major crops affected but DON can be found in corn as well (CAST, 2003). The organisms survive on residue left on the field from the previous season's crop, providing an inoculum source for the new crop. The organisms do well in cool, moist conditions with contamination of the crop occurring when conidia of the organism are windblown to the corn silks or in small grains to the anthers which emerge outside the floret during anthesis. The fungus penetrates the host ear or floret and produces the disease which may be ear rot in corn or head blight in small grains. Certain environmental conditions may allow for late growing season development of DON in crops. Apparently, DON production is necessary for the organism to produce disease in some crops. Storage under good conditions (<14% moisture),

including control of insect pests, will minimize further elaboration of the toxin by these toxigenic fungi. Again, if grains have matured and are stored under appropriate conditions, DON does not further accumulate in storage. Swine are the animals most usually affected by this toxin and exhibit reduced intake of contaminated grain; if they do eat it they may vomit. Levels above 1 µg/g are considered potentially harmful to these animals. Pet foods prepared with wheat contaminated with this toxin have been involved in acute toxicities. DON is immunosuppressive and may cause kidney problems in animals. Humans are thought to exhibit a similar vomition syndrome when consuming DON-contaminated grain (Bhat *et al.*, 2003). DON does not appear to be significantly carried over into tissues or fluids of animals consuming toxic levels. Baking and malting using contaminated wheat and barley are adversely affected (Richard, 2000).

2.2.5 Zearalenone

ZEA, also called mycoestrogen, is synthesized as a secondary metabolite by *Fusarium* species, primarily *Fusarium graminearum* and *F. culmorum*. ZEA is a worldwide contaminant of various cereals and animal feed. Due to its binding affinity for estrogen receptors, ZEA has significant effects on the reproductive system. ZEA may cause severe reproductive disorders, ovarian dysfunction, infertility, increase in abnormal spermatozoa, and other problems. ZEA is also linked to early onset of puberty in young children. It is a potential stimulator of human breast tumorigenesis and, moreover, it is hepatotoxic, hematotoxic, genotoxic, and immunotoxic (Hueza *et al.*, 2014). Zearalenone (previously known as F-2 toxin) was produced by some *Fusarium* species *Fusarium graminearum* (*Gibberella zeae*), *Fusarium culmorum*, *Fusarium cerealis*, *Fusarium equiseti*, *Fusarium crookwellense* and *Fusarium semitectum*. These fungi infected contaminants of cereal crops worldwide (Bennett and Klich, 2003). The concentration of accumulated Zearalenone (ZEA) in cereals depended on several factors such as the substrate, temperature, duration of *Fusarium* growth and strain of fungal species. Moreover, the humid tropical climate promoted microbial proliferation on food and feedstuffs and finally mycotoxin biosynthesis (Nuryono *et al.*, 2005). Toxicity of ZEA and its metabolites was related to the chemical structure of the mycotoxins, similar to naturally occurring estrogens (Gromadzka *et al.*, 2009). ZEA was heat-stable, which made it difficult to remove and/or decomposed from food. Additionally, it was observed that during food and feed processing (e.g. milling, extrusion, storage and heating) ZEA was not decomposed (Yumbe-Guevara *et al.*, 2003). Zearalenone imitates the effect of female hormone oestrogen and at low doses, increases the size or early maturity of mammary glands and reproductive organs. At higher doses Zearalenone interferes with conception, ovulation, implantation, fetal development and the viability of new born animals (Zinedine *et al.*, 2007). Zearalenone causes estrogenic responses in dairy cattle and large doses of this toxin are associated with abortions. Other responses of dairy animals to zearalenone are reduced in feed intake, decreased milk production, vaginitis, increase vaginal secretions, poor reproductive performance and mammary gland enlargement in heifers. It is recommended that zearalenone should not exceed 250 ppb in the total diet (Zinedine *et al.*, 2007).

Grains infected with the above organism may exhibit the pink color associated with the production of a pink pigment simultaneously produced with the zearalenone. Most often this mycotoxin is found in corn. However, it is found also in other important crops such as wheat, barley, sorghum and rye throughout various countries of the world (CAST, 2003).

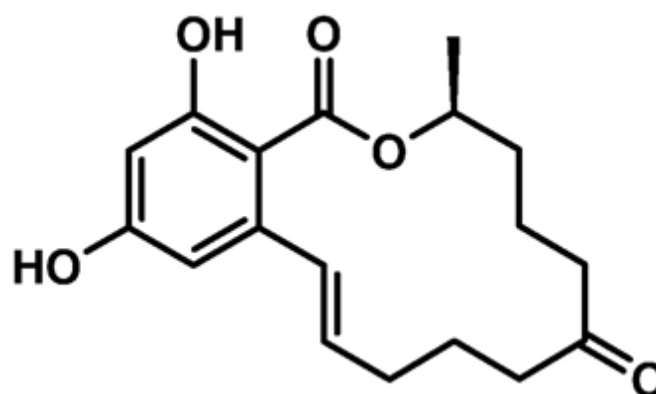


Fig. 5. Structure of zearalenone.

2.2.6 Ergot

Ergot is often used as a name for any condition which causes for the production or accumulation of ergot alkaloids either in hardened masses of fungal tissue (sclerotia), the most typical association, or liberated into host plant tissue by endophytic fungi (Richard, 2007). Ergot alkaloids are a large group of compounds produced by fungi that attack a wide variety of grass species, including small grains, during the growing season. They are divided into the clavine alkaloids, lysergic acids, simple lysergic acid amides and peptide alkaloids (Fig. 6). The major ergot fungus is *Claviceps* which produces sclerotia in several grass species with *C. purpurea* being the most commonly found species, however, *C. fusiformis* has produced ergot in pearl millet and *C. paspali* has been

associated with problems in dallis grass poisonings (CAST, 2003; Richard, 2007). The endophytic fungi, *Neotyphodium* or *Epichloe* produce ergot peptines such as ergovaline in tissues of selected grasses such as fescue. In the United States, *Neotyphodium coenophialum* inhabits tall fescue and produces ergovaline which is implicated in an ergot-like toxicosis in animals grazing on this grass (CAST, 2003; Richard, 2007). The entire life cycle of the organism *Claviceps* is quite complex, but for simplicity, species of this genus replace the developing ovaries of the seed with masses of fungal tissue which harden into sclerotia (sometimes called "ergots"). The sclerotia are brown to purple-black in color and contain the ergot alkaloids. The fungus gains entry into the host plant from sclerotia that have been in the soil. The infecting fungal elements (ascospores) are ejected forcibly but also are assisted by wind and splashing rain in gaining access to the host plant where the florets are invaded with subsequent sclerotial development (Richard, 2007). The sclerotia are harvested with the grain and if not eliminated by screening or some other process they can end up in feed or food made from the contaminated grain (Richard, 2007).

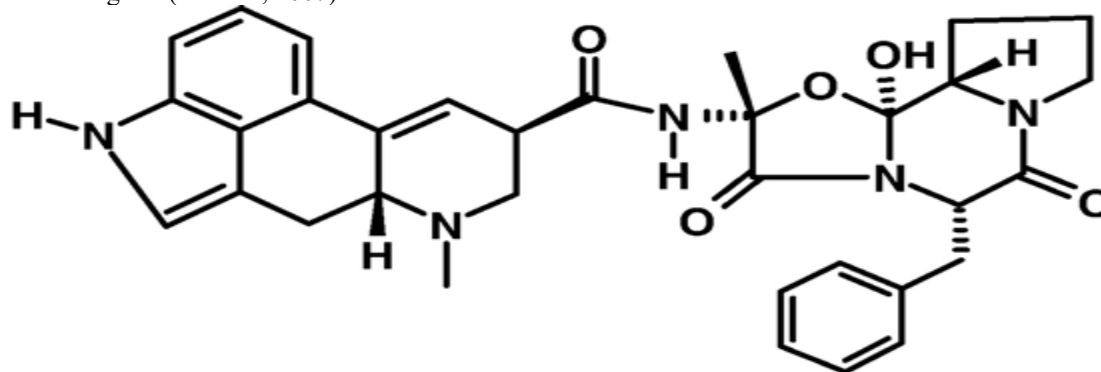


Fig. 6. Structure of ergotamine H as a representative of the ergot alkaloids.

2.2.7 T-2 toxin

This mycotoxin is a representative of a large group of mycotoxins called trichothecenes (Fig. 7). It belongs to the Type A chemical class of non-macrocylic trichothecenes while DON, as mentioned earlier, belongs to the Type B nonmacrocylic trichothecenes. The principal fungus responsible for the production of T-2 toxin is *F. sporotrichioides* (CAST, 2003; Richard, 2007). Some strains of this fungus also produce some closely related mycotoxins (HT-2 toxin and diacetoxyscirpenol) belonging to the same chemical class. Corn, wheat, barley, oats, rice, rye and other crops have been reported to contain T-2 toxin (CAST, 2003; Richard, 2007). Natural occurrence has been reported in Asia, Africa, South America, Europe and North America. Natural levels range from near zero to 10 µg/g with few exceptions up to levels of 40 µg/g. Toxin production is greatest with moisture and temperatures ranging from 6 to 24°C. Visible evidence of the producing fungus may appear on corn as white mold growth which, in some instances, may appear pink to reddish, often beginning at the tip of the ear. Adequate storage with low moisture and insect control will minimize further fungal growth and T-2 toxin production. In the United States, T-2 toxin is infrequently found and if found, likely results from inadequate storage of products (Richard, 2007).

The major effect of T-2 toxin and other trichothecenes is that they inhibit protein synthesis which is followed by a secondary disruption of DNA and RNA synthesis. It affects the actively dividing cells such as those lining the gastrointestinal tract, skin, lymphoid and erythroid cells. It can decrease antibody levels, immunoglobulins and certain other humoral factors such as cytokines. The manifestations of disease include signs of weight loss or poor weight gains, bloody diarrhea, dermal necrosis or beak and mouth lesions, hemorrhage and decreased production of milk and eggs. The Type A trichothecenes are more toxic to poultry species than the Type B trichothecenes. Yellow caseous plaques, occurring at the margin of the beak, mucosa of the hard palate, angle of the mouth and tongue, characterize typical oral lesions in poultry (Richard, 2007).

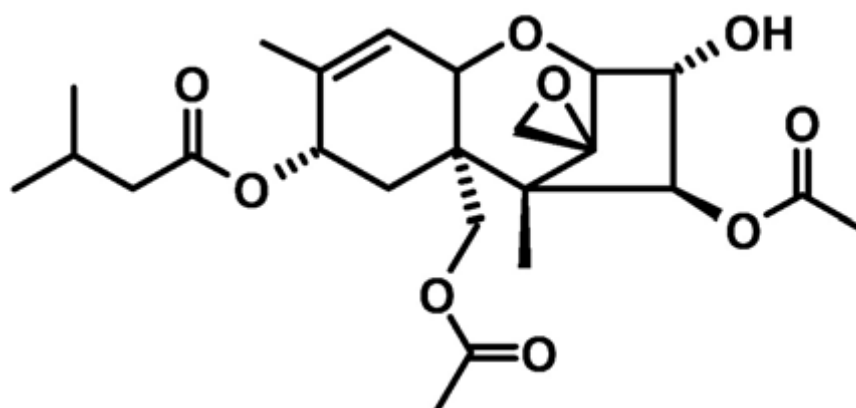


Fig 7 Structure of T-2 toxin

2.3 Factors affecting occurrence of toxigenic fungi and toxins in stored produce

Mycotoxins are produced by fungal action during production, harvest, storage and food processing. Once the crop becomes infected under field conditions, the fungal growth continues with increasing vigour at post-harvest and storage conditions. Genotypes, drought, soil types, and insect activity are important in determining the likelihood of pre-harvest contamination. Humidity, temperature, and aeration during drying and storage are also important factors (Wagacha and Muthomi, 2008). Maize is one of the richest substrates for aflatoxin elaboration and even standing crop get high degrees of infestation. Other cereal crops like wheat, barley, oat and sorghum are not very susceptible to extensive pre-harvest aflatoxin contamination. Conidia of *Aspergillus flavus* are the major source of primary inoculum in maize fields (Scheidegger and Payne, 2003). The fungus appears to form many sclerotia in insect-damaged kernels before harvest. These sclerotia are dispersed into the soil during harvest. The sclerotia survive in soil and produce conidiophores and conidia during the following season. Invasion of maize by *A. flavus* occurs via silks (Wagacha and Muthomi, 2008). Once *A. flavus* is present in plant tissue, it can continue to grow (Scheidegger and Payne, 2003). Senescencing silk is a suitable media for microbial growth and provide entry for fungi into the ear. The fungal mycelium spreads superficially among the kernels and penetrates the kernels mainly through the pericarp. Insects that feed on maize ears in the field and stored maize predispose kernels to fungal infection through physical damage while storage insect pests open the kernels to fungal invasion (Avantaggio *et al.*, 2002). Therefore, insect damage of maize is a good predictor of mycotoxin contamination, and can serve as an early warning. Insects carry the spores from plant surfaces to the interior of the stalk or kernels or create infection wounds due to the feeding of the larvae on stalks or kernels (Munkvold and Hellminch, 2000). Insect-damaged kernels provide an opportunity for the fungus to circumvent the natural protection of the integument and establish infection sites in vulnerable interior (St. Leger *et al.*, 2000). Wounding by insects may provide infection courts and allow kernels to dry down to moisture content more favourable for growth of *A. flavus* and aflatoxin production. In addition, there seems to be a correlation between socioeconomic status of majority of sub-Saharan countries and exposure to mycotoxins. A case in point is where maize is traditionally stored in granaries but storage of improperly dried maize inside homes occurs during periods of food shortage, which may facilitate contamination of maize with mycotoxins (Azziz- Baumgartener *et al.*, 2005). Poor aeration in the houses and dirty floors may promote fungal growth on wet maize kernels. Drought conditions stress plants and render them susceptible to contamination by *Aspergillus spp* (Robertson, 2005; Holbrook *et al.*, 2004). Other factors favouring mycotoxin contamination are stress factors during plant growth, late harvesting of crops, high ambient humidity preventing thorough drying, unscientific storage practices and lack of awareness (Wagacha and Muthomi, 2008).

2.3.1 Availability of nutrients and conditions for mould growth

The fact that a strain of mould has the genetic potential to produce a particular mycotoxin is not enough for it to do so. There must be enough nutrients to encourage mould growth and the level of mycotoxin production would in part be influenced by the nutrients available to the mould. Typically, moulds require a source of energy in the form of carbohydrates or vegetable oils in addition to a source of nitrogen either organic or inorganic, trace elements and available moisture for growth and toxin production (Makun, 2013). Substrate may also play a role in selecting for or against toxin producing strains of a given species, e.g., there are a high proportion of toxin-producing strains of *A. flavus* isolated from peanuts and cottonseed than from rice or sorghum. It has also been found that strains of ochratoxin and citrinin producing *P. viridicatum* isolated from meat were more unstable than those isolated from grain and rapidly lost toxin-producing ability (Makun, 2013). Field fungi like *Fusarium* and *Alternaria* contaminate grains before or during harvest. The storage fungi (e.g. *Penicillium* and *Aspergillus*) are capable of growing at lower water content than the field fungi and they tend to contaminate the grains in silos

and other storage places. It is known that aflatoxin production is favoured by prolonged end of season drought and associated elevated temperatures (Rachaputi *et al.*, 2002). Moulds can grow and produce mycotoxins under a wide temperature range with optimum generally between 20 to 30°C. However, temperatures optimum for toxin production need not correspond to that optimum for growth: *Fusarium tricinctum* grows well at 25°C but produces T-2 toxin best near freezing temperatures. *Penicillium martensii* produces penicillic acid rapidly at 20-30°C, but considerably more toxin eventually accumulates between 4 to 10°C (Makun, 2013).

2.3.2 Farming systems and agricultural techniques

A number of farming techniques have been shown in various reports as stimulating mould growth in agricultural produce. For example produce harvested from land on which groundnut has been planted the previous year were infested more by *Aspergillus flavus* and contained more aflatoxin than crops grown on land previously planted with rye, oats, melon or potatoes indicating that crop rotation influences mycotoxigenic mould growth. Likewise, previously fungicide-treated soil has been shown to reduce incidence of *A. flavus* in groundnuts to very low levels (Makun, 2013).

2.3.3 Soil types and soil conditions

Soil is a natural factor that exerts a powerful influence on the incidence of fungi. Crops grown in different soil types may have significantly different levels of mycotoxin contamination. For example, peanuts grown in light sandy soils support rapid growth of the fungi, particularly under dry conditions, while heavier soils result in less contamination of peanuts due to their high water holding capacity which helps the plant to prevent drought stress (Codex Alimentarius Commission, 2004).

2.3.4 Pre-harvest conditions

Genotypes, drought, soil type, plant density, fertilization level, and insect activities are important components in determining the likelihood of pre-harvest contamination. However, the most important factor appears to be high night time temperatures, which favour fungal growth and toxin production at a time when the plant is deprived of its usual energy source and thus least able to resist fungal attack (Abbas *et al.*, 2002, 2007).

2.3.5 Time of harvesting

Harvest is the first stage in the production chain where moisture content becomes the most important parameter in terms of the management and protection of the crop. It also marks a shift from problems caused by plant pathogenic fungi, like *Fusarium*, to problems caused by storage fungi. Ideally, grains will always be harvested after a spell of dry weather when it is at safe moisture content, so that immediate drying is not necessary. Another important control measure at harvest will be visual examination of the grain for symptoms of disease, and the segregation of diseased batches from healthy grain. Early harvesting reduces fungal infection of crops in the field and consequent contamination of harvested produce. Even though majority of farmers in Africa are well aware of the need for early harvesting, lack of storage space, unpredictable weather, labour constraint, need for cash, threat of thieves, rodents and other animals compel farmers to harvest at inappropriate time (Bankole and Adebajo, 2003; Kaaya *et al.*, 2006). aflatoxin levels increased by about 4 times by the third week and more than 7 times when maize harvest was delayed for 4 weeks. However, if products are harvested early, they have to be dried to safe levels to stop fungal growth. Lower aflatoxin levels and higher gross returns of 27% resulting from early harvesting and threshing of groundnuts (Rachaputi *et al.* 2002; Makun, 2013).

2.3.6 Pest infestation

Insects are the chief causes of deterioration and loss of grains and seeds. Their invasion of cereals decreases the quality, grade and market value of these agricultural products which in most instances are rendered unsafe for human and animal consumption. Pest infestation is largely due to improper post-harvest and storage conditions and the level of insect damage influences the extent of mycotoxin contamination (Makun, 2013). Avantaggio *et al* (2002) found that insect damage of maize is good predictor of *Fusarium* mycotoxin contamination. Insects carry spores of mycotoxin-producing fungi from plant surfaces to the interior of the stalk or kernels or create infectious wounds through their feeding habits (Munkvold, 2003).

2.3.7 Post-harvest handling

The post-harvest stages are those stages following harvest and leading up to primary processing such as milling. This will typically involve drying (if required), storage and transportation steps. Post-harvest movement of food/feed commodities can be complex, passing as it may between a number of intermediaries such as traders and intermediate processors, who may be situated at different geographical locations. In the simplest case, produce may remain on-farm in store or buffer storage for short periods of time before being passed directly onto the processor (Makun, 2013). In more complex cases it may pass through the hands of merchants or third party drying facilities (if harvested wet e.g. grains) and held in storage for periods of time before finally arriving at the processors. At all times the produce can become susceptible to fungal contamination and mycotoxin production if the storage conditions are not strictly controlled (Makun, 2013).

2.3.8 Drying conditions and duration

Rapid drying of agricultural products to low moisture level is critical as it creates less favourable conditions for fungal growth, proliferation, and insect infestation. It helps keep products longer (Lanyasunya *et al.*, 2005).

Ayodele and Edema (2010) evaluated the Critical Control Points (CCP) in the production of dried yam chips with a view to reducing mycotoxin contamination and identified the drying stage as a CCP. Aflatoxin contamination can increase 10 fold in a 3-d period, when field harvested maize is stored with high moisture content (Hell *et al.*, 2008). The general recommendation is that harvested commodities should be dried as quickly as possible to safe moisture levels of 10 – 13 %. Even, when drying is done in the dry season, it is not completed before loading grains into stores as observed by Mestre *et al* (2004) and products can be easily contaminated with aflatoxins. During storage, transportation and marketing, low moisture levels should be maintained by avoiding leaking roofs and condensation arising from inadequate ventilation (Makun, 2013).

2.3.9 Storage factors

Mycotoxin contamination of foods or feeds may result from inadequate storage and/or handling of harvested products. To preserve quality in storage, it is necessary to prevent biological activity through adequate drying to less than 10% moisture, elimination of insect activity that can increase moisture content through condensation of moisture resulting from respiration, low temperatures, and inert atmospheres (Lanyasunya *et al.*, 2005; Turner *et al.*, 2005). And the post-harvest contamination is normally characterized by the activities of the ‘storage’ fungi, typically *Aspergillus* and *Penicillium* species that are able to grow in relatively dry conditions (Makun, 2013).

2.4 Post-Harvest Control and Management of mycotoxins

Significant levels of grain mould infection begin from the field being encouraged by damp conditions at the time of harvesting, delayed harvesting and continue in storage due to improper or insufficient drying, insect infestation and poor storage conditions of stored produce (Reddy *et al.*, 2013; Tiffany *et al.*, 2013). Proper harvesting and handling of agro-produce are good starting points of post-harvest control of mycotoxins in produce. This is because such practices minimize abrasions and breakages of produce which provide portals of entry for mycotoxigenic fungi. According to the WHO (2006) good postharvest practices are directed to achieving minimization of mycotoxin contamination through proper drying and storage techniques as well as elimination of undue moisture migration into produce during storage or marketing. In many traditional storage systems of Senegal Tiffany (2013) reported that storage is done in bins, rooms and roomlets. In such systems aflatoxin contamination has been reported. In many third world economies, WHO (2006) noted that the combination of insufficient drying and humid atmospheric conditions warranted unacceptable levels of mycotoxins on harvested maize, groundnut etc. The parameters of moisture and temperature are known to influence to a large degree the activities of fungi (Bryden, 2007), with moisture regarded as the single most important determinant of how rapidly mycotoxigenic moulds grow in storage (Whitlow and Hagler, 2013). Therefore, ensuring that harvester combines are set properly to reduce breakage of kernels; and that produce are sorted and winnowed properly to remove bruised and broken tubers or kernels which could become easily infected in the store will go a long way to reduce or remove contamination in produce (WHO, 2006; Tiffany, 2013). To this end therefore, we should dry grains always to safe moisture content (13-14%) before storing. Usually drying should be done on covered surfaces not on bare grounds as this will increase the propensity of mycotoxin contamination. And store only clean grains in insecticide-treated bags on racks in a properly maintained, fumigated and ventilated store-house; and ensure that produce are dried to safe moisture levels before storing. It is important that old crops are evacuated before introducing new ones and that stored produce are stored only for minimal possible time periods (John and Steve, 2010). These simple postharvest technologies in subsistence farm levels of west Africa have been found to reduce disease burdens due to aflatoxins as reported by WHO (2006). The essence of good store structure, condition and hygiene is not only to reduce fungal load on grains but to minimize insect and moisture migration into the bin which could stimulate and ultimately quicken the growth and sporulation of the storage fungi (Enyiukwu *et al.*, 2014a).

2.4.1 Good agricultural practices

Agronomic practices have been shown to have profound effect on mycotoxins contamination of crops in the field.

Early harvesting reduces fungal infection of crops in the field before harvest and consequent contamination of harvested produce. Even though majority of farmers in Africa are well aware of the need for early harvesting, unpredictable weather, labour constraint, need for cash, threat of thieves, rodents and other animals compel farmers to harvest at inappropriate time (Makun, 2013). Rachaputi *et al.* (2002) reported that early harvesting and threshing of groundnuts resulted in lower aflatoxin levels and higher gross returns of 27% than in delayed harvesting. Rapid drying of agricultural products to low moisture level is critical as it creates less favourable conditions for fungal growth and proliferation, insect infestation and helps keep longer (Lanyasunya *et al.*, 2005). Hamiton (2000) reported that drying harvested maize to 15.5% moisture content or lower within 24–48 h would reduce the risk of fungal growth and consequent aflatoxin production. Awuah and Ellis (2002) demonstrated that when groundnuts were dried to 6.6% moisture level, they were free of fungi regardless of the local storage protectant used for 6 months, whereas at 12% moisture, only jute bags with the plant *Syzigium aromaticum* effectively suppressed the cross infection of healthy kernels. However, when the moisture content was increased to 18.5%, the latter treatment was not as effective. A community-based intervention trial in Guinea, West Africa

focused on thorough drying and proper storage of groundnuts in subsistence farm villages and achieved a 60% reduction in mean aflatoxin levels in intervention villages (Turner *et al.*, 2005). During storage, transportation and marketing, maintenance of low moisture levels should be maintained by avoiding leaking roofs and condensation arising from inadequate ventilation (Makun, 2013).

A study conducted in Benin by Fandohan *et al.*, (2005) to determine the fate of aflatoxins and fumonisins through traditional processing of naturally contaminated maize and maize based foods, demonstrated that sorting, winnowing, washing, crushing combined with de-hulling of maize grains were effective in achieving significant mycotoxins removal. Similar results have been reported by Park (2002). This approach is based on separation of contaminated grain from the bulk and depends on the heavy contamination of only a small fraction of the seeds, so that removing those leaves a much lower overall contamination. Study of the distribution of aflatoxin in peanuts shows that a major portion (80%) of the toxin is often associated with the small and shrivelled seed and mouldy and stained peanut (Fandohan *et al.*, 2005; Turner *et al.*, 2005). Basic sanitation measures such as removal and destruction of debris from previous harvest would help in minimizing infection and infestation of produce in the field. Cleaning stores before loading new produce has been shown to be correlated with reduced aflatoxin levels (Hell *et al.*, 2000). To preserve quality in storage, it is necessary to prevent biological activity through adequate drying to less than 10% moisture, elimination of insect activity that can increase moisture content through condensation of moisture resulting from respiration, low temperatures, and inert atmospheres (Lanyasunya *et al.*, 2005; Turner *et al.*, 2005).

The level of insect damage influences the extent of mycotoxins contamination. Avantaggio *et al* (2002) found that insect damage of maize is good predictor of *Fusarium* mycotoxins contamination. Insects carry spores of mycotoxins producing fungi from plant surfaces to the interior of the stalk or kernels or create infection wounds through their feeding habits (Munkvold, 2003). Therefore, proper management of insect pests through any appropriate control strategy would reduce mycotoxins contamination problem. Cultural practices including crop rotation, tillage, planting date, and management of irrigation and fertilization, has limited effects on infection and subsequent mycotoxins accumulation (Champeil *et al.*, 2004; Munkvold, 2003).

2.4.2 Chemical control

Appropriate use of pesticides during the production process could help in minimizing the fungal infection or insect infestation of crops and consequently mycotoxin contamination. Fumonisins contamination could be reduced by application of fungicides that have been used in control of *Fusarium* head blight such as prochloraz, propiconazole, epoxyconazole, tebuconazole cyproconazole and azoxystrobin (Haidukowski *et al.*, 2004; Matthies and Buchenauer, 2000). On the other hand, fungicides such as itraconazole and amphotericin B have been shown to effectively control the aflatoxin producing *Aspergillus* species (Ni and Streett, 2005). However, use of fungicides is being discouraged due to economic reasons and growing concern for environment and food safety issues.

2.4.3 Decontamination

Decontamination of food/feed contaminated with mycotoxins could be achieved through either chemoprotection or enterosorption. Chemoprotection of aflatoxins has been demonstrated with the use of a number of chemical compounds like Oltipraz and Chlorophyllin or dietary intervention like broccoli sprouts and green tea that either increase an animal's detoxification processes (Kensler *et al.*, 2004) or prevent the production of the epoxide that leads to chromosomal damage. This intervention might not however be sustainable in the long-term in most African countries since it involves drug therapies, which are expensive besides the possible side effects. Enterosorption is based on the discovery of certain clay minerals, such as Novasil, which can selectively adsorb mycotoxins tightly enough to prevent their absorption from the gastrointestinal tract (Wang *et al.*, 2005; Phillips, Lemke and Grant, 2002). There are different adsorption agents but their efficacy in preventing mycotoxicosis varies. Selected calcium montorillonites have proven to be the most highly selective and effective of enterosorbents. However, with enterosorption, there is a risk that non-specific adsorption agents may prevent uptake of micronutrients from the food. Essential oils and aqueous extracts of *Aframomum danielli* were recently reported to reduce OTA in spiked cocoa powder by between 64 and 95% (Aroyeun and Adegoke, 2007). Although ochratoxin molecule is stable, it is acknowledged that around 40 to 90% of OTA is destroyed during roasting of coffee beans (Makun, 2013).

2.4.4 Legislation

Mycotoxin regulations have been established in about 100 countries, out of which 15 are African, to protect the consumer from the harmful effects of these mycotoxins (Fellinger, 2006; Barug *et al.*, 2003; Van Egmond, 2002). Human foods are allowed 4–30 ppb aflatoxin, depending on the country involved (FDA, 2004). In the US 20 µg/kg is the maximum aflatoxin residue limit allowed in food for human consumption, except for milk (Wu, 2006) while 4 µg/kg total aflatoxin in food for human consumption are the maximum acceptable limits in the EU, the strictest in standard worldwide (EC, 2006; Wu, 2006). OTA has been evaluated at the 37th, 44th and 56th meetings of the Joint FAO/WHO Expert Committee on Food Additives (JECFA), and a provisional tolerable weekly intake (PTWI) of 100 ng/kg body weight has been established (Benford *et al.*, 2001).

3. Summary and Conclusion

3.1 summary

Mycotoxins are toxic secondary metabolites of fungal origin and contaminate agricultural commodities before or under post-harvest conditions. However a large number of fungi are associated with groundnut kernel, maize, rice, and sorghum grain, the most common mycotoxin-producing fungi are *Aspergillus flavus*, *Aspergillus parasiticus*, *Aspergillus ochraceus*, *Fusarium verticillioides*, *Fusarium graminearum* and *Penicillium* spp. While maize and groundnut have been identified as very high-risk commodities for mycotoxin contamination, many other commodities have also been identified as vulnerable to this menace. Among the mycotoxins fitting into this major group would be the aflatoxins, deoxynivalenol, fumonisins, zearalenone, T-2 toxin, ochratoxin and certain ergot alkaloids. Factors that contribute to mycotoxin contamination of food and feed include environmental, socio-economic and food production. Environmental conditions especially high humidity and temperatures favour fungal proliferation resulting in contamination of food and feed. The diseases (mycotoxicoses) caused by these mycotoxins are quite varied and involve a wide range of susceptible animal and humans. Most of these diseases occur after consumption of mycotoxin contaminated grain or products made from such grains but other routes of exposure exist. The resulting implications include immuno-suppression, impaired growth, various cancers and death depending on the type, period and amount of exposure. Possible intervention strategies include good agricultural practices such as early harvesting, proper drying, and sanitation, storage, transportation, marketing and insect management among others. Other possible interventions include biological control, chemical control, and decontamination.

3.2 Conclusion

The presence of mycotoxins in food products has serious implications for human and animal health. Many countries have enacted regulations stipulating maximum amounts of mycotoxins permissible in food and feedstuffs. Most of the developed countries will not permit the importation of commodities containing amounts of mycotoxins above specified limits. Mycotoxins therefore have implications for trade between nations. Most of the outbreaks of mycotoxicoses described are a consequence of the ingestion of food that is contaminated with mycotoxins. Prevention of fungal invasion of commodities is far the most effective method of avoiding mycotoxins problems. Mycotoxins consideration should be an integral commodity management program focusing on the maintenance of commodity quality from the field to the consumer. Several effective ways for prevention and control of hazardous fungi and their dangerous mycotoxins have been presented. The methods include biological control, physical and chemical treatments. Following the good agricultural practices described in this paper during both pre- and post-harvest periods, would minimize the risk of feed contamination by mycotoxins. These include cultural practices of cereals in fields as well as harvest, transport and storage conditions. Physical, chemical or biological treatments of contaminated feed have poor efficacies and are not economically viable.

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