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An Overview of Mycotoxin Contamination of Foods and Feeds

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with varying impacts on food processing. The major group of mycotoxins that contaminate foods and feeds include aflatoxins, fumonisins and patulin. Several studies conducted to reveal the metabolism of mycotoxins in the body are reviewed. Health implications of mycotoxins upon consumption of adequate doses are diverse. They include sub-acute mycotoxicosis, immune suppression, carcinogenicity, genotoxicity, morbidity and mortality in animals and humans as well as interaction with nutrient assimilation. Mycotoxicity of foods have tremendous effect on international trade, resulting in huge losses. There are regulations, though not in all countries, aimed at preventing and controlling Mycotoxins which operate only on industrially processed foods and those meant for exports but not locally processed ones. A number of strategies for preventing mycotoxins have been proposed but the awareness for implementation is very low. The use of media to create awareness is a viable option.

Keywords: Food; Feeds; Mycotoxins; Contamination; Mycotoxicity; Mycotoxin metabolism; Prevention

Introduction

Mycotoxins are secondary metabolites produced by moulds which contaminate foods and have toxic efects on the health of humans and animals. Mycotoxins are produced primarily by the fungi which belong to *Aspergillus, Penicillium* and *Fusarium* genera. Fungi proliferate to produce secondary metabolites under favorable environmental conditions, when temperature and moisture are suitable. Fungi are a normal part of the micro fora of standing crops and stored feeds, but t

the composition of the commodity and the

ting, handling and storage [1]. The amount ... physical factors (moisture, relative humidity, temperature and mechanical damage), chemical factors (carbon dioxide, oxygen, composition of substrate, pesticide and fungicides), and biological factors (plant variety, stress, insects, spore load).

Several fungal metabolites which are toxic in experimental systems abound, however, there are only fve that are of major agricultural importance: afatoxin, produced by *Aspergillus favus* and *A. parasiticus*; deoxynivalenol, produced by *Fusarium graminearum* and *F. culmorum*; *fumonisin*, produced by *Fusarium verticillioides* (ex-moniliforme); ochratoxin, produced by *Aspergillus ochraceus* and *Penicillium verrucosum*; and zearalenone, produced by various *Fusarium* species [2]. T ese toxins produced by fungal species remain stable throughout the processing periods and cooking of feeds and foods (afatoxin [3], ochratoxin [4], fumonisin [5], deoxynivalenol [6]. Fungal infection and subsequent production of Mycotoxin can occur at the feld during crop growth or harvesting, and may continue during storage. T e occurrence of this Mycotoxin at a considerably high level of concentration in foods can cause toxic efects ranging from acute to chronic (mutagenic, teratogenic, carcinogenic) manifestations in humans and animals [7]. Animals that have been fed with Mycotoxin-contaminated feeds release products which can be dietary sources of some Mycotoxin [8].

Human diseases arising from Mycotoxin cut across a large part of the globe without boundaries. T ere are thousands of fungal secondary metabolites currently known, but only a few groups are reported to be important from the safety and economic points of view; namely afatoxins (AFs), mainly produced by *Aspergillus* species; *ochratoxin A* (OTA), produced by *Aspergillus* and *Penicillium*0c a'A'A–

T e economic impact of Mycot

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d animal life to reduced livestock production, disposal of contaminated foods and feeds and investment in research [12]. As a result of deleterious efects of Mycotoxin on humans and farm animals, a good number of countries in the world have implemented several regulations which prescribe the limits of Mycotoxin in several food commodities intended for consumption. In 1993, the WHO-International Agency for Research on Cancer evaluated the carcinogenic potential of AFT, OTA, TCT, ZEA, and FUMs [13,14].

So many eforts have been made towards control and reduction of mycotoxin contamination of foods but the ubiquitous nature of toxigenic fungi enables their wide occurrence. It is also noted that in most rural areas of the world, no efort is made towards the control of toxigenic fungi in food contamination. T e aim of this work is have a general overview of Mycotoxins contamination of foods.

Foods implicated in mycotoxins contamination

Mycotoxins are reported to have occurred in many agricultural products ranging from raw to process, hence, becoming a worldwide issue [15]. T ey have the capacity to remain stable during processing of foods [16], indicating di f culty of getting rid of them. Reports that mycotoxin is naturally fairly distributed as contaminants of many cereals, (Table 1) as well as other food commodities [17] and feeds [18,19] along the food chain. While AFB1 and OTA are among the most frequently observed mycotoxin in foods [20], the other types are occasional contaminants depending on the factor prevailing on their occurrences where they are located. Several authors indicated the prevalence of a f atoxigenic and ochratoxigenic mould growth and toxin production [21,22].

Cereals	Corn (grains, gluten); Rice; Wheat; Barley; Oats; Rye; Sorghum; Millet
Cereal products for human consumption	Cracked grains; Cereal cleanings; Wheat bran
cereal feed products	

	A. carbonarius, A. sclerotiorum, A. sulphureus	
Citrinin	*Penicillium citrinum, P. verrucosum, P. viridicatum, Monascus purpureus*	Oats, rice, corn, beans, fruits, fruit and vegetable juices, herbs and spices
Zearalenone	*Fusarium graminearum, F. sporotrichoides, F. culmorum, F. cerealis, F. equiseti, F. incarnatum*	Maize, soybean, cereals
Deoksynivalenol	*Fusarium graminearum, F. culmorum, F. crokwellense*	Maize, soybean, cereals
Alternariol, alternariol monomethyl ether	*Alternaria alternata, A. brassicae, A. capsici-anui, A. citri, A. cucumerina, A. dauci, A. kikuchiana, A. solani, A. tenuissima, A. tomato, A. longipes, A. infectoria, A. oregonensis*	Vegetables, fruit, cereals, soybean
Tenuazonic acid	*Alternaria alternata, A. capsici-anui, A. citri, A. japonica, A. kikuchiana, A. mali, A. solani, A. oryzae, A. porri, A. radicina, A. tenuissima, A. tomato, A. longipes*	Vegetables, fruit, cereals, soybean
Fumonisins	*Fusarium proliferatum, F. verticillioides*	Maize, soybean, cereals

Table 2: Mycotoxigenic fungi and mycotoxins [64-72,80].

Aflatoxin	**Zearalenone**	**Deoxynivalenol**		**Fumonisin**	**Ochratoxin A**
Tested samples	11,967	15,533	17,732	11,439	7,495
Positive samples	3,142	5,797	9,960	6,204	1,902
Percentage of positives	26%	37%	56%	54%	25%
Average positives (µg/kg)	57	286	1,009	1,647	14
Median positives (µg/kg)	11	85	453	750	2.6
1st quartile positives (µg/kg)	3	43	234	332	1.1
3rd quartile positives (µg/kg)	40	225	972	1,780	6.2
Maximum (µg/kg)	6,323	26,728	50,289	77,502	1,589
Sample origin	Myanmar	Australia	Central Europe	China	China
Sample type (analysis year)	other feed (2012)	silage (2007)	wheat (2007)	finished feed (2011)	finished feed (2011)

Table 3: Summary of the global survey of mycotoxins [73].

Trichothecenes pathway begins with an enzyme trichodiene synthase which cyclize farnesyl pyrophosphate (FPP) to trichodiene. T e enzyme possesses sub-units molecular mass of 45 kDa and usually isolated from *Fusarium sporotrichioides* [73]. T e subsequent pathway involves esteri f cation and oxygenation of trichodiene diacetoxyscirpenol, T-2 toxin and 3-cetyldeoxynivalenol [74]. T e genes involved are: tri 5; Tri 4 and Tri 3 [75].

Fungal species	**Mycotoxins**
Aspergillus flavus; Aspergillus parasiticus	Aflatoxins
Aspergillus flavus	Cyclopiazonic acid
A. ochraceus; Penicillium viridicatum; P. cyclopium	Ochatoxin A
P. expansum	Patulin
Fusarium culmorum; F. graminearum; F. sporotrichioides	Deoxynivalenol
F. sporotrichioides; F. poae	T-2 toxin
F. sporotrichioides; F. graminearum; F. poae	Diacetoxyscirpenol
F. culmorum; F. graminearum; F. sporotrichioides	Zearalenone

F. moniliforme	Fumonisins
Acremonium coenophialum	Ergopeptine alkaloids

Table 4: T e major toxigenic species of fungi and their principal mycotoxins [81].

Fumonisins are synthesized by the condensation of the amino acid alanine to an acetate-derived precursor. Structurally, they possess C–20 diester of propane–1, 2, 3 tricarboxylic acid and peritahydroxylcosane with primary amino group. T e enzyme adenosyl methionine transferase is attached to the C-12 and C-16 of the branded chain methyl group [76], though, the isolation of this enzyme has not been documented [77].

T e biosynthetic pathway of A f atoxin has been well documented [78-80]. A polyketide synthase converted to norsolorinic acid by a fatty acid synthase. Conversion of enzymes occurs up to 12-17 ways with series of intermediates in polyketide. T en, AFB1 and AFG1 are produced a f er the formation of versicolorin B. Several enzymes that occur in a f atoxin and sterigmatocystin biosynthetic pathway are: O–methyl tranferase; fatty acid synthase; polyketide synthase; desaturase; versicolorin B synthase; verisconal hemiacetal acetate reductase; and norsolorinic acid reductase. T e genes involved in a f atoxin are: fas1A, toxins called trichothecenes causes severe damage to actively dividing cells in bone marrow, lymph nodes, spleen, thymus, and intestinal mucosa [109]. T ese trichothecenes can be immune suppressive at lower doses [101]. Miller and Trenholm [106] concluded that mycotoxins are likely to be immunotoxic to humans as well following their studies on animals. Pestka and Bondy [101] dismissed the problem for the developed world with the reason that the high doses of mycotoxins might be most likely encountered in animal feed that is not inspected for interregional or international commerce. However, human food is regulated at the low parts per billion ranges in Canada, the United States, and most developed countries because of potent hepato carcinogenicity of a f atoxins. T us, vigilant monitoring should minimize the potential for a f atoxin-induced immune suppression in humans." Monitoring is e f ectively done in the developed world. In the developing world, except in cases of exports of vulnerable commodities such as groundnuts or co f ee to the developed nations, monitoring of internal food supplies is rarely implemented [109].

Interaction with nutrient assimilation: Hendrickse [110] reported that protein–energy malnutrition, kwashiorkor, and a f atoxin exposure appear to be seasonally linked in tropical regions where a f atoxins are present. However, research has shown that there is no speci f c cause-and-e f ect relationship between a f atoxin and kwashiorkor, but children with kwashiorkor who had tested positive for a f atoxin in blood and urine had statistically signi f cantly longer hospital stays and su f ered from more infections [111,112]. T us, a f atoxin acted in conjunction with kwashiorkor, possibly by immune suppression, to worsen the prognosis [113]. Vitamins are thought to ameliorate genotoxicity, and a f atoxin B1 interacts with assimilation of vitamins A and E.

Carcinogenicity and genotoxicity: To underscore the correlation between cancer and a f atoxin, the incidence of primary live0e demonstrated in Swazils d [114] and corroborated by data f rom Mozambiqu] ʿ In China, maize was the major source of a f atoxin exposure hence a correlation was established in mortality rate from live0` s cer ea 372/100,000 as against low risk

Human exposure to ochratoxin primarily occurs from whole grain breads, although co f ee and wine are also implicated when fungi infect berries and grapes. Marasas [117] suggested that levels of 100–200
ppb would be safe for humans consuming large amounts of
contaminated maize.

Acute a f atoxicosis (severe a f atoxin poisoning) occurs in poultry, swine, and cattle consuming feeds contaminated with a f atoxins. T e same can appear in humans, and cases of lethal toxic hepatitis attributed to consumption of a f atoxin-contaminated maize have occurred [107,117,118]. Large-scale acute human toxicoses due to consuming wheat and rice contaminated with deoxynivalenol have occurred in modern times in India [119] China, and Korea, among other

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to sorting, or other physical treatment, before human consumption as well as spices, dried fgs, almonds, pistachios, apricot kernels, hazelnuts, and Brazil nuts intended for direct human consumption; and 15 g/kg for peanuts and other oilseeds, almonds, pistachios, apricot kernels, hazelnuts, and Brazil nuts subjected to sorting, or other physical treatment, before human consumption [124]. T e FDA action level is 20 g /kg for total AFs in peanuts, Brazil nuts, pistachios, and other foods for direct human consumption [127]. T ough all the limits set by the regulatory bodies followed the results of intensive research over the years, there is no doubt that constant review of these mycotoxins maximum limits in foods is important because of the development of mutant strains of fungi likely to produce more potent toxins.

Toxin	**Product**	**Maximum limit (μg/kg)**
Aflatoxin	Peanuts, oilseeds, cereals, processed products	4
	Tree nuts, dried fruits, maize, rice, spices, almonds, pistachios, hazel nuts	10
Fumonisins	Processed cereal-based foods	

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