Mycotoxin contamination of foods in Africa: Antinutritional factors

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Abstract

Mycotoxins are regulated in foods and feeds because of carcinogenic (aflatoxin), immunotoxic (deoxynivalenol), or environmental estrogenic (zearalenone) properties. In addition to having tumorigenic properties, many mycotoxins are antinutritional factors that cause unthrifty growth and immune suppression in young animals. In the developed world, human exposure, and particularly exposure of children, to dietary mycotoxins is virtually nonexistent because of regulatory standards. In developing countries, monitoring and enforcement of standards is rare, and mycotoxin-susceptible foods are often the primary staples in rather undiversified diets. In sub-Saharan Africa, people are exposed to unsafe levels of various mycotoxins, often in mixtures, and the consequences in terms of public health burden have been ignored. This paper presents information on the health effects that have been attributed to mycotoxin exposure from the medical research literature and data on existing mycotoxin levels in maize in West and Central Africa. The International Institute of Tropical Agriculture (IITA), in its Maize Integrated Pest Management Project, has recognized mycotoxins as one of the most important constraints to the goal of improving human health and well-being through agriculture. An overview of various research and development activities at the Institute is given.

Mycotoxin effects on human and animal health

Mycotoxins are chemical contaminants in foods produced by fungal infestation that adversely affect human or animal health. Although there are hundreds of fungal metabolites that are toxic in experimental systems, there are only five that are of major agricultural importance: aflatoxin, produced by *Aspergillus flavus* 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 [1].

Generally, these toxins are stable throughout the typical processing and cooking of feeds and foods (aflatoxin [2], ochratoxin [3], fumonisin [4], deoxynivalenol [5]). Products from animals that have been fed mycotoxin-contaminated feeds can be dietary sources of some mycotoxins [6].

A recent report by the US National Academy of Sciences [7] noted that even with the high-quality food system in the United States, carcinogenic mycotoxins in American diets may increase cancer rates. Mycotoxins are certainly a major public health problem for humans in developing countries [8]. In numerous geographic studies, significant correlations between areas with high risk of dietary exposure to mycotoxins and cancers have been found. The effects of mycotoxin exposure range from acute intoxication to chronic, subacute responses. The following are summary statements taken from a very large body of scientific literature, concerning specific effects of the various mycotoxins on human and animal health.

Subacute chronic toxicity and growth faltering

Subacute mycotoxicoses (toxic effects by mycotoxins) are manifested in a syndrome of symptoms, which commonly include moderate to severe liver damage, reproductive problems, appetite loss, digestive tract discomfort, diarrhoea, growth faltering, immune suppression, increased morbidity, and premature mortality [9]. Aflatoxin is also implicated in the degenerative diseases childhood hepatic cirrhosis and Reye's syndrome [10]. Aflatoxins have been shown to pass transplacentally, thus having the potential to affect prenatal

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infant development [11]. In Kenya, the mean birthweight of the offspring of women exposed to aflatoxins prenatally was lower than that of those who had not been similarly exposed [12, 13]. An important research question is: How much morbidity and growth faltering are occurring in infants who are at high risk of aflatoxin exposure relative to those at low risk?

Immune suppression: Increased morbidity and mortality in animals and humans

Aflatoxin B_1 is hepatotoxic in humans and animals and is nephrotoxic and immunosuppressive in animals [14]. Experimental exposure of animals to a chemical family of *Fusarium* toxins called trichothecenes causes severe damage to actively dividing cells in bone marrow, lymph nodes, spleen, thymus, and intestinal mucosa. At lower doses, these compounds can be immune suppressive [15].

As a result of their studies on animals, researchers have concluded that mycotoxins are likely to be immunotoxic to humans as well [9]. Pestka and Bondy [15] dismissed the problem for the developed world by stating that "These [high doses of mycotoxins] might be most likely encountered in animal feed that is not inspected for interregional or international commerce. In contrast, human food is regulated at the low parts per billion ranges in Canada, the United States, and most developed countries because of potent hepatocarcinogenicity of aflatoxins. Thus, vigilant monitoring should minimize the potential for aflatoxin-induced immune suppression in humans." Monitoring is effectively done in the developed world. In the developing world, except in cases of exports of vulnerable commodities such as groundnuts or coffee to the developed nations, monitoring of internal food supplies is rarely implemented.

Interaction with nutrient assimilation

Protein–energy malnutrition, kwashiorkor, and aflatoxin exposure appear to be seasonally linked in tropical regions where aflatoxins are present [16]. Research has shown that there is no specific cause-and-effect relationship between aflatoxin and kwashiorkor, but children with kwashiorkor who had tested positive for aflatoxin in blood and urine had statistically significantly longer hospital stays and suffered from more infections [17, 18]. Thus, aflatoxin acted in conjunction with kwashiorkor, possibly by immune suppression, to worsen the prognosis [19]. Vitamins are thought to ameliorate genotoxicity, and aflatoxin B_1 has been reported to interact with assimilation of vitamins A and E [20, 21].

Carcinogenicity and genotoxicity

Naturally occurring aflatoxins are carcinogenic in humans [14]. Intracountry correlation between estimates of the incidence of primary liver cancer and the intake of aflatoxins in the same population groups has been demonstrated in Swaziland [22, 23] and was corroborated by data from Mozambique [24]. In China, where maize was the major source of aflatoxin exposure, mortality from liver cancer was 372/100,000 in areas at high risk of food contamination versus 33/100,000 in areas at low risk [25]. A strong relationship exists between hepatitis B virus infection, aflatoxin, and liver cancer [19], and in some studies a multiplicative or synergistic effect has been clearly noted [14]. Aflatoxin B_1 is defined as genotoxic because it causes DNA damage, gene mutation, and chromosomal anomalies [14].

Ochratoxin is suspected as the cause of urinary tract cancers and kidney damage in areas of chronic exposure in parts of Eastern Europe [14, 26]. Human exposure to ochratoxin primarily occurs from wholegrain breads, although coffee and wine are also implicated when fungi infect the berries and grapes.

Fumonisins are suspected as primary causal factors in esophageal cancers in the Transkei, South Africa [24, 27]. Marasas [28] suggested that levels of 100–200 ppb would be safe for humans consuming large amounts of contaminated maize.

Acute toxicosis

Acute aflatoxicosis (severe aflatoxin poisoning) occurs in poultry, swine, and cattle consuming feeds contaminated with aflatoxins. The same can appear in humans, and cases of lethal toxic hepatitis attributed to consumption of aflatoxin-contaminated maize have occurred [10, 27, 29, 30]. Large-scale acute human toxicoses due to consuming wheat and rice contaminated with deoxynivalenol have occurred in modern times in India [31], China, and Korea, among other countries [32].

Prevalence of mycotoxins in West and Central Africa

Although mycotoxins are strictly regulated in most parts of the world, outside of South Africa there is very little information about the amount of exposure to these dietary antinutritional factors in sub-Saharan Africa. From 1993 to 1996, the International Institute of Tropical Agriculture (IITA) conducted toxin analyses on corn samples collected from five agroecological zones of Benin, Nigeria, and Cameroon. Mycotoxin amounts vary between seasons and years, depending on environmental conditions and crop and produce management practices. The hypothesis was that there

would be differences in the risk of mycotoxin contamination mediated by climatic conditions and crop management practices in the different agroecological zones [33, 34].

Risk of exposure to specific toxins from a maize-based diet

The Sahel, just south of the Sahara Desert, has high temperatures and the risk of drought stress, conditions that give *A. flavus* competitive advantage over other grain-infesting fungi. This zone also has a higher incidence of the much more toxic "S strain" of *A. flavus* than the other zones [35]. In the Sahel of both Benin and Nigeria, maize had a significantly higher risk of aflatoxin contamination after six months in storage than in the other zones (tables 1 and 2). In the dry savannah, ear-boring insects are known to increase aflatoxin contamination [37], and the dry savannah has a long monomodal rainy season during which farmers intercalate cotton, groundnuts, and maize in their fields. All of these crops are prone to *A. flavus* and aflatoxin buildup, and growing them together has the potential to increase contamination. In the dry savannah of both Benin and Nigeria, overall toxin levels were low, as a result of better crop husbandry and more advantageous climate [34, 38] (tables 1 and 2). The moist savannah has a bimodal rainy season, and farmers have difficulty in drying their first-season crop before storage, leading to insect infestation and rapid quality degradation. This zone had a significantly higher percentage of stores infected within the first three months of storage than the other zones in both countries. The high humidity and warm temperatures in coastal savannahs and humid forest regions increase the risk of fungal contamination of maize, but *A. flavus* and aflatoxin were relatively scarce, displaced by *Fusarium* and *Penicillium* species (tables 1 and 2). The cool climate found in the mid-altitude zones is more likely to promote the *Fusarium* species and the associated toxins such as fumonisin, deoxynivalenol, and zearalenone. These agroecological conditions exist across a large part of sub-Saharan Africa.

Risk of multiple toxin exposure

Mycotoxin mixtures are likely to occur naturally, and these may alter immunity in an additive or synergistic manner. In Benin and Nigeria, mixtures of mycotoxigenic fungi were found in all samples (fig. 1). In Benin in 1993–94, 80 samples were analysed for fumonisin at the beginning of storage. Fumonisins were found in all samples, ranging from 0.1 to 8.3 ppm [39]. It was shown that grain could be simultaneously contaminated with both aflatoxins in fumonisin. The presence of various mycotoxigenic fungi in grain in the moist savannah of Benin suggests that people are being exposed to multiple toxins. For humans and animals, the potency of the contaminated material is related to the mixtures present [1].

TABLE 1. Percentage of samples in aflatoxin classes per agroecological zone in Benin after six months of storage in 1993–95 ($n = 301$)

Zone	< 5 ppb	5–10 ppb	10–20 ppb	20–50 ppb	50–100 ppb	> 100 ppb
Sahel savannah	67.8	1.6	4.8	1.6	—	24.2
Dry savannah	71.3	5.0	11.3	—	5.0	8.8
Moist savannah	68.4	1.3	7.6	7.6	7.6	7.6
Coastal savannah	85.5	3.8	2.5	1.3	—	7.5

Source: ref. 33.

TABLE 2. Aflatoxin B detected in maize samples in five agroecological zones of Nigeria in 1994 and 1995 ($n = 25$ per zone)

	1994				1995			
Survey	Mean aflatoxin (μg/kg)[a]	Range (μg/kg)	% stores positive	Positive > 20 ppb[b]	Mean aflatoxin (μg/kg)[a]	Range (μg/kg)	% stores positive	Positive > 20 ppb[b]
Sahel savannah	104.3	0–1,380	12.0	868.9	51.0	0–408.3	25.0	204.2
Dry savannah	0.0	0–0	0.0	0.0	3.8	0–16.7	21.4	0.0
Moist savannah	32.9	0–600	24.0	423.3	40.6	0–650.0	18.7	216.6
Mid-altitude	55.2	0–1,380	4.0	1,380.0	0.0	0–0	0	0.0
Humid forest	125.0	0–1,050	16.0	781.3	21.1	0–202.9	27.7	77.4

Source: refs. 34, 36.
a. Zone mean including all samples.
b. Mean of positive stores.

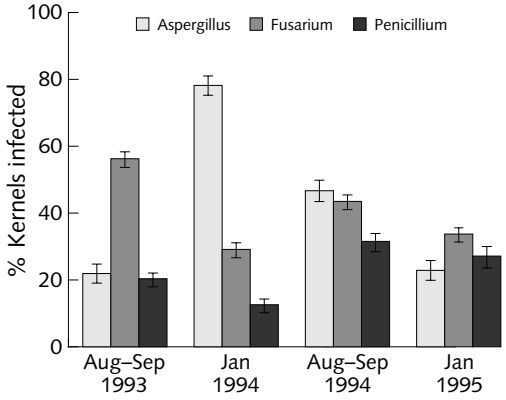

FIG. 1. Percent maize kernels from the southern Guinea Savannah of Benin that were infected with three genera of fungi during four sampling periods. Source: ref. 33

IITA strategy to ameliorate the problems of mycotoxin contamination in foods and feeds

Various projects are being coordinated by IITA, with multiple partners and donors, to try to reduce the amounts of mycotoxins that people are exposed to in West Africa. Inducing behavioural change—thus enabling families to improve their diets even without additional income—is often the most cost-effective way to improve nutritional status [40]. A three-year project, funded by Rotary International, is designed to conduct public information campaigns about the importance of consuming only good-quality grain. It is expected that an educated urban consumer will demand improved food quality from market sources. In partnership with governmental agencies and with market and consumer associations, a strategy for sustainable monitoring of markets will be put in place. A feasibility study will be conducted to look at methods for rendering poor-quality grain safe for animal feeds, in order to have an alternative market for the poorest-quality grain and so to help remove it from the human foods sector. Market demand should result in farmer demand for technologies to produce high-quality maize grain for human consumption.

To prepare a basket of technologies for on-farm reduction of contamination, research funded by the German BMZ/GTZ and the United States Department of Agriculture has three components. First, biological control of *Aspergillus flavus* is being developed to prevent aflatoxin contamination in all vulnerable crops. IITA is also working to introduce host plant resistance to *A. flavus* into tropically adapted maize. Second, farmer participatory research methods are being used to optimize field-to-store commodity management practices to improve grain quality and reduce aflatoxin contamination, and to assess the costs to the farmer of adopting improved management practices. Third, a medical epidemiology study is under way to detect the effect of exposure to aflatoxin in rural maize-based systems on the immune response to vaccination, and on the growth of children under the age of three. An assessment of the range of foods available in the households of the test children will be made to control for other nutritional deficiencies that can affect infant development.

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