Mycotoxin problem in Africa: Current status, implications to food safety and health and possible management strategies

J.M. Wagacha [a,*], J.W. Muthomi [b]

[a] Institute for Crop Science and Resource Conservation, Section Phytomedicine, University of Bonn, 53115 Bonn, Germany
[b] Department of Plant Science and Crop Protection, University of Nairobi, P.O. Box 29053 Nairobi, Kenya

Abstract

Mycotoxins are toxic secondary metabolites of fungal origin and contaminate agricultural commodities before or under post-harvest conditions. They are mainly produced by fungi in the *Aspergillus*, *Penicillium* and *Fusarium* genera. When ingested, inhaled or absorbed through the skin, mycotoxins will cause lowered performance, sickness or death on humans and animals. Factors that contribute to mycotoxin contamination of food and feed in Africa 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 socio-economic status of majority of inhabitants of sub-Saharan Africa predisposes them to consumption of mycotoxin contaminated products either directly or at various points in the food chain. The resulting implications include immuno-suppression, impaired growth, various cancers and death depending on the type, period and amount of exposure. A synergistic effect between mycotoxin exposure and some important diseases in the continent such as malaria, kwashiorkor and HIV/AIDS have been suggested. Mycotoxin concerns have grown during the last few decades because of their implications to human and animal health, productivity, economics of their management and trade. This has led to development of maximum tolerated limits for mycotoxins in various countries. Even with the standards in place, the greatest recorded fatal mycotoxin-poisoning outbreak caused by contamination of maize with aflatoxins occurred in Africa in 2004. Pre-harvest practices; time of harvesting; handling of produce during harvesting; moisture levels at harvesting, transportation, marketing and processing; insect damage all contribute to mycotoxin contamination. Possible intervention strategies include good agricultural practices such as early harvesting, proper drying, sanitation, proper storage and insect management among others. Other possible interventions include biological control, chemical control, decontamination, breeding for resistance as well as surveillance and awareness creation. There is need for efficient, cost-effective sampling and analytical methods that can be used for detection analysis of mycotoxins in developing countries.
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* Corresponding author.
 E-mail address: wagacha2000@yahoo.com (J.M. Wagacha).

1. Introduction

Mycotoxins are toxic secondary metabolites produced by fungi and contaminate various agricultural commodities either before harvest or under post-harvest conditions (FAO, 1991). 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. The largest mycotoxin-poisoning epidemic in a decade was reported in Africa during the last 5 years (Lewis et al., 2005; CDC, 2004). Some mycotoxins such as aflatoxins are considered by the US Food and Drug Administration (FDA) to be unavoidable contaminant of food. The goal therefore has been to minimize contamination. However, mycotoxin management methods used in developed countries cannot realistically be used in developing countries because of the characteristics of the food systems and the technological infrastructure in those countries resulting in uncontrolled mycotoxin levels in these situations. The threat is made even more palpable by the fact that, staple diets in many African households are based on cereal crops such as maize, which are highly susceptible to mycotoxin contamination.

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). This review examines the extent of the mycotoxin problem in Africa, its implications to food/feed safety, health as well as economic implications of their contamination and poisoning. It also examines the scale and levels of human and animal exposure to different food crops in different parts of Africa; highlights the mycotoxins posing the greatest danger in Africa and explores possible management interventions.

2. 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 (WHO, 1999). 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; FDA, 1995).

From the African perspective, two classes of mycotoxins: — aflatoxins and fumonisins have been estimated to be widespread in major dietary staples (Table 1). 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

Table 1
Examples of food commodities and aflatoxin contamination levels in Africa reported in literature

Country	Commodity	Frequency of aflatoxin positive samples	Contamination rate/concentration	Reference
Botswana	Raw peanut	78% contained aflatoxins	Concentrations ranging 12–329 mg/kg	Barro et al. (2002)
Nigeria	Pre-harvest maize	*Aspergillus flavus* was isolated from 65% of the samples	Total aflatoxins ranged 3–138 mg/kg in positive samples	Maxwell et al., 2000
	Dried yam chips		Mean concentration of aflatoxin B1: 27.1 ppb.	Chauliac et al. (1998)
	Melon seeds		Aflatoxin B1 above 5 mg/kg in 32.2% of samples	Mensah et al. (2002)
Senegal	Peanut oil	Aflatoxin B1 found in over 85% of samples	Mean contents about 40 ppb	Muleta and Ashenafi (2001)
S. Africa	Traditionally brewed beers	33.3% of commercial beer samples contained aflatoxins	200 and 400 mg/l	Mensah et al. (1999)
Kenya	Maize	Samples from local markets	Up to 46,400 µg/kg	CDC (2004)
		Samples from government ware houses 350 maize products	Up to 1800 µg/kg 55% had levels>20 ppb; 35% had levels >100 ppb;	(CDC, 2004; Lewis et al., 2005)

Adapted from WHO, (2006), CDC, (2004), Lewis et al. (2005).

Kenya (Lewis et al., 2005; CDC, 2004; Ngindu et al., 1982). The 2004 aflatoxin-poisoning outbreak in Kenya was the largest and most severe documented worldwide. The outbreak covered more than seven districts and resulted in 317 case-patients and 125 deaths. Other smaller outbreaks were reported in 2005 and 2006, with about 30 and 9 deaths, respectively. The maize implicated in the 2004 aflatoxicosis outbreak was harvested in February during off-season, early rains. Maize obtained from local farms in the affected area was significantly more likely to have aflatoxin levels greater than the regulatory limit of 20 ppb compared to maize bought from other regions of the country. Contaminated homegrown maize bought from local farms entered the local market distribution system, resulting in widespread aflatoxin contamination. Therefore, efforts to successfully interrupt exposure to aflatoxin during an outbreak must consider the potential role of the market system in sustaining exposure.

3. Factors affecting occurrence of toxigenic fungi and toxins in African 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 (Cole et al., 1995). Humidity, temperature, and aeration during drying and storage are also important factors. 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 (Marsh and Payne, 1984). 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 (Hesseltine and Bothast, 1977). 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 socio-economic 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.

4. Food safety and health hazard implications associated with mycotoxins

In 1993, the International Agency for Research on Cancer (IARC) classified AFB1 and mixtures of aflatoxins as Group 1 carcinogens (IARC, 2002). Due to their various toxic effects and good thermal stability, the presence of mycotoxins on foods

and feeds is potentially hazardous to the health of both humans and animals (Fellinger, 2006; Barug et al., 2003). There is ample evidence that the inhabitants of sub-Saharan Africa are experiencing heavy dietary exposure to food-borne mycotoxins particularly aflatoxins and fumonisins. According to Miller (1996), 40% of the productivity lost to diseases in developing countries is due to diseases exacerbated by aflatoxins. Regrettably, many of the people in the region are not even aware of the effect of consuming mouldy products. Due to the low literacy levels and other socio-economic factors, even if steps were taken to make food products safe, the consumers might be unwilling to pay extra costs, and may still prefer to buy the cheap commodities.

Epidemiological studies of human populations exposed to diets naturally contaminated with aflatoxins revealed an association between the high incidence of liver cancer in Africa and elsewhere and dietary intake of aflatoxins (MERCK, 2006). For people who are infected with hepatitis B and C, which is common in sub-Saharan Africa, aflatoxin consumption raises the risk of liver cancer by more than ten-fold compared to either exposure alone (Turner et al., 2003). In addition, preliminary evidence suggests that there may be an interaction between chronic mycotoxin exposure and malnutrition, immuno-suppression, impaired growth, and diseases such as malaria and HIV/AIDS (Gong et al., 2003, 2004). In a recent study in Ghana, higher levels of aflatoxin B1-albumin adducts in plasma were associated with lower percentages of certain leukocyte immunophenotypes (Jiang et al., 2005) while a study in Gambian children found an association between serum aflatoxin albumin levels and reduced salivatory secretory IgA levels (Turner et al., 2003).

Exposure to large doses (>6000 mg) of aflatoxin may cause acute toxicity with lethal effect whereas exposure to small doses for prolonged periods is carcinogenic (Groopmann and Kensler, 1999). Symptoms of acute toxicity include reduced liver function, derangement of blood clotting mechanism, icterus (jaundice), and a decrease in essential serum proteins synthesized by the liver. Other general signs of aflatoxicosis are oedema of the lower extremities, abdominal pain, and vomiting. Severe acute liver injury with high morbidity and mortality has been associated with high dose exposures of aflatoxins (Chao et al., 1991). Ingestion of 2–6 mg/day of aflatoxin for a month can cause acute hepatitis and death (Patten, 1981). Early symptoms of hepatotoxicity from aflatoxicosis can manifest as anorexia, malaise and low-grade fever. Aflatoxicosis can progress to potentially lethal acute hepatitis with vomiting, abdominal pain, hepatitis and death (Etzel, 2002). No animal species is resistant to the acute toxic effects of aflatoxins. However, animal species respond differently in their susceptibility to the chronic and acute toxicity of aflatoxins. For most species, the LD50 value ranges from 0.5 to 10 mg/kg body weight. Toxicity is influenced by environmental factors, exposure level, and duration of exposure beside age, health, and nutritional status of diet (FAO, 2000).

Chronic exposure to aflatoxins is associated with impaired immunity, malnutrition and liver cancer which is the third most common cause of death from cancer in Africa (Williams et al., 2004). Chronic toxicity results from long-term exposure of moderate to low aflatoxin concentrations. Symptoms of chronic poisoning include decrease in growth rate, lowered milk or egg production, and immuno-suppression. The chronic incidence of aflatoxin in diets is evident from the presence of aflatoxin M1 in human breast milk in Ghana, Kenya, Nigeria, Sierra Leone, Sudan, Thailand, and the United Arab Emirates, and in umbilical cord blood samples in Ghana, Kenya, Nigeria, and Sierra Leone (Bhat and Vasanthi, 2003). There is some observed carcinogenecity, mainly related to aflatoxin B1. Liver damage is apparent due to the yellow colour that is characteristic of jaundice, and the gall bladder becomes swollen. Immuno-suppression is due to the reactivity of aflatoxins with T-cells, decrease in Vitamin K activities, and a decrease in phagocytic activity in macrophages. These immuno-suppressive effects of aflatoxins predispose the animals to many secondary infections due to other fungi, bacteria and viruses (McLean, 1995). A recent study by Azzi-Baumgartner et al. (2005) reported that males were more likely to die from aflatoxicosis, in spite of eating similar quantities of maize as females. The study and that of Ngindu et al. (1982) reported that aflatoxicosis patients reported dog deaths before developing aflatoxicosis. Therefore, in future, reports of deaths of dogs may warm public health officials of a potential aflatoxin contamination in the food supply.

Aflatoxin exposure has also been suggested as a causal or aggravating factor for kwashiorkor in African children (Ramjee et al., 1992). There is a striking association between aflatoxin and impaired growth in children (Egal et al., 2005; Gong et al., 2003). These adverse growth effects are strongly correlated with the change from breastfeeding to solid foods, including maize, which is widely used in ground form as the basis for porridge for weaning purposes. Whether the effects of weaning foods and associated reduced growth are a direct result of aflatoxin exposure has however, not been confirmed. Turner et al. (2003) detected AF-alb adducts in 93% of sampled children (6–9 years) in Gambia and provided evidence that igA in saliva may be reduced because of aflatoxin exposure. The study confirmed that children in rural areas of Gambia are frequently exposed to high levels of aflatoxin.

Fumonisins have been implicated in a number of animal diseases such as leucoencephalomalacia in equines, which involves a massive liquefaction of the cerebral hemisphere of the brain with neurological manifestations such as abnormal movement, aimless circling, lameness; porcine pulmonary oedema; rat liver cancer and haemorrhage in the brain of rabbits (Marasas, 1995). It can cause hepatotoxicity and nephrotoxicity in many animals (Howard et al., 2001). At high levels of exposure, fumonisin B1 has been shown to produce liver cancer, decreased the life span in female mice and also induced liver carcinoma in male rat (NTP, 1999). The prevalence of fumonisins has been reported to be 100% or close to it in all surveillance data that have been reported on maize from different parts of Africa (Bankole et al., 2006).

Ochratoxin A is nephrotoxic, teratogenic, carcinogenic and immuno-suppressive in many animal species (Stoev, 1998; IARC, 1993a). The international Agency for Research on Cancer has classified OTA as possibly carcinogenic in humans (group 2B carcinogen) (IARC, 1993a). Sedmikova et al. (2001) reported the possibility of OTA increasing the mutagenic ability

of aflatoxin B1 in cases of simultaneous occurrence of the two mycotoxins in the same crop.

Bearing these facts in mind, the really important questions are: how contaminated are the diets in Africa, how much of the ingested dose is significant to human health and nutrition and what are the thresholds for effects on human immunology and nutritional health? These questions gain more weight in the consideration that food security concerns of many of the people at risk mean that even knowing that food is contaminated would not help, because the people have no alternative sources of food. This was recognized in the report of the Third Joint FAO/WHO/UNEP International Conference on mycotoxins (Van Egmond, 1999). It would therefore be logical to argue that the amounts of mycotoxins allowed in foods by *Codex Alimentarius* have no relevance to most of the people in Africa; their consumption of traded food items is small, and laboratories to test their foods are not available or are inaccessible. Where trade does occur, the least contaminated foods and feeds are exported, which may lead to enhanced exposure of the producers because the more highly contaminated products are retained at home for consumption by a population that is already at the greatest risk of aflatoxin exposure (Lewis et al., 2005).

5. Magnitude of mycotoxin problem in Africa and its implications

Most of the mycotoxin-poisoning problems occur in the sub-Saharan Africa where maize and groundnuts are a dietary staple. Whereas maize is a dietary staple in Africa, groundnut production has been on the increase reaching 8,520,221 metric tons in 2004 (Afla-guard, 2005). About 250,000-hepatocellular carcinoma related deaths occur annually in parts of sub-Saharan Africa due to aflatoxin ingestion alone. About 5.2 million cancer deaths occur each year, 55% of which occur in developing countries. 80% of cases and deaths of liver cancer occur in these countries particularly in Western and Central Africa while 78% of cervical cancers occur in developing countries (Dow, 1994). An expert group meeting on aflatoxins and health in Brazzaville, Republic of Congo concluded that mycotoxins impacted negatively on livelihoods, particularly of poor people. Besides the direct health risk and causing premature deaths in Africa, economic losses arising from mycotoxicoses are enormous in the region (Fellinger, 2006; MERCK, 2006; Wu, 2004).

6. Aflatoxins

Up-until the mid 1990s, reports of acute aflatoxin poisonings, approximately 25% of which result in death were found in scientific journals, but subsequent reports are usually in the daily press. The largest reported outbreak of aflatoxicosis to date occurred in Kenya in 2004 where 317 cases and 215 recognized deaths were reported (Azzi-Baumgartner et al., 2005; CDC, 2004; Lewis et al., 2005; MMWR, 2004). Other documented fatal aflatoxicosis outbreaks have been reported in Western India in 1974: 397 cases and 106 reported deaths (Krishnamachari, Nagaarajan, Bhat and Tilak, 1975); Nigeria in 2005: more than

100 deaths (Afla-guard, 2005); Kenya in 1981: 20 cases (Ngindu et al., 1982); Kenya in 2005: 80 cases and 30 reported deaths and 9 deaths in 2006 (Anonymous, 2006). In the 2004 outbreak in Kenya, concentrations of aflatoxin B1 in maize was found to be as high as 4400 ppb, which is 220 times greater than the 20 ppb, limit for food suggested by Kenya authorities (Azzi-Baumgartner et al., 2005; CDC, 2004). Of great concern is what seems to be an annual outbreak of aflatoxicosis in Eastern province of Kenya (Azzi-Baumgartner et al., 2005).

Muriuki and Siboe (1995) reported 100% contamination incidence of three packed maize brands in Kenya with aflatoxins B1 and B2 (0.4–2.0 µg/kg). Aflatoxin levels of about 30 times higher than the legal limits (10 ppb) have been reported in peanut butter given to school children in Eastern Cape, South Africa. (MERCK, 2006). A study by Gong et al. (2003) reported high contamination levels of staple foods by aflatoxins in West Africa. In Benin and Togo, aflatoxin levels in maize have been reported to average five times the safe limit in up to 30% of household grain stores. In the two countries, studies have shown that 99% of fully weaned children had nearly two-fold higher aflatoxin albumin adduct levels compared to those breast-fed (Egal et al., 2005; Hell et al., 2005). Udoh et al. (2000) reported that 33% of maize samples from different agro-ecological zones of Nigeria were contaminated with aflatoxins. Hell et al. (2000) found that the percentage of maize samples with more than 5 µg/kg aflatoxin levels was between 9.9% and 32.2% in the different agro-ecological zones of Benin before storage, but that this increased to 15.0% and 32.2% after 6 months storage. All the maize samples collected from silos and warehouses in Ghana contained aflatoxins at levels ranging from 20 to 355 µg/kg, while fermented maize dough collected from major processing sites contained aflatoxin levels of 0.7 to 313 µg/kg (Kpodo, 1996). Yameogo and Kassamba (1999) reported that seeds of groundnuts from Burkina Faso inoculated with *A. flavus* excreted all the four major aflatoxins, which peaked at 170 ppb after 6 days. Aflatoxin was detected in 98% of samples of dried yam chips surveyed in Benin with levels ranging from 2.2 to 220 µg/kg and a mean of 14 µg/kg (Bassa et al., 2001). Aflatoxin B1 was detected in 54.2% of dried yam chips in Nigeria (Bankole and Adebanjo, 2003a) while Bankole and Eseigbe (1996) detected aflatoxins in 35% of tiger nut (*Cyperus esculentus*) with concentrations ranging from 10–120 µg/kg in Nigeria.

Aflatoxin albumin (AF-alb) adducts were detected in 99% of children in Benin and Togo (Gong et al., 2003). Over 90% of West African sera were reported to contain detectable level AF-alb adducts, with exposure occurring throughout life, including *in utero* and via breast milk (Turner et al., 2000; Wild et al., 2000). In Togo and Benin, the level of aflatoxin in stored maize has been shown to exceed 100 ppb in 50% of tested samples (Hell et al., 2000). In fact some of the highest levels of AF-alb ever measured have been reported in children between 9 months and 5 years in Benin and Togo where 5.4% had levels greater than 200 pg/mg with a maximum level in one child of 1064 pg/mg (Gong et al., 2003). A study in Nigeria by Uriah et al. (2001) found that blood and semen aflatoxin levels ranged from 700 to 1393 ng/ml and 60 to 148 ng/ml, respectively in infertile men and were significantly higher than that in fertile men.

7. Fumonisins

Consumption of fumonisin has been associated with elevated human oesophageal cancer incidence in various parts of Africa, Central America, and Asia (Marasas et al., 2004) and among the black population in Charleston, South Carolina, USA (Sydenham et al., 1990). Because fumonisin B1 reduces uptake of folate in different cell lines, fumonisin consumption has been implicated in neural tube defects in human babies (Marasas et al., 2004). Some correlation studies have suggested a link between the consumption of maize with high incidence of *F. verticillioides* and fumonisins and the high incidence of human oesophageal carcinoma in certain parts of S. Africa (IPCS, 2000; Yoshizawa et al., 1994). Fumonisin has been demonstrated to induce apoptosis in cultured human cells and in rat kidneys (Tollenson et al., 1996) and to be possible human carcinogens (IARC, 1993a,b). Hell et al. (1996) reported a significant positive correlation between fumonisin and aflatoxin levels in maize samples in Benin. In West Africa, fumonisin levels of 640 µg/kg in maize from Benin (Doko et al., 1995) and 65–1830 µg/kg (mean 390 µg/kg) from Nigeria have been detected (Bankole and Adebanjo, 2003b). Fumonisin B1 was detected in 55 out of 108 maize samples in Nigeria (Bankole and Adebanjo, 2003b). All samples of home-brewed Xhosa maize beer in S. Africa were positive for fumonisin B1 (range 38 to 1066 ng/ml and mean 281 ng/ml) and total fumonisins (B1, B2 and B3) ranged from 43 to 1329 ng/ml, with a mean of 369 ng/ml (Shephard et al., 2005). These levels are well above the provisional maximum tolerable daily intake of 2 µg/kg of body weight/day set by the joint FAO/WHO Expert Committee on Food Additives. In the U.S., the Food and Drug Administration (FDA) has set industry guidelines for levels of fumonisin acceptable in human food and animal feed. The most stringent of these standards applies to degermed dry-milled corn products for human food, with a recommended total fumonisin maximum level of 2 mg/kg. Few other nations currently have fumonisin standards for food.

8. Ochratoxins

Ochratoxin A (OTA) is produced by fungi of the genera *Aspergillus* and *Penicillium*. The major species implicated in OTA production includes *A. alutaceus* (Syn. *A. ochraceus*), *A. carbonarius*, *A. melleus*, *A. sclerotiorum A. sulphureus, P. verrucossum. Aspergillus niger* and *P. purpurescens* are less important OTA producers (Benford et al., 2001). OTA is a frequent natural contaminant of many foodstuffs such as cocoa beans, coffee beans, cassava flour, cereals, fish, peanuts, dried fruits, wine, poultry eggs and milk (Weidenbörner, 2001). In Africa, OTA has been reported as a contaminant of cocoa and coffee beans, tiger nuts, wines and maize. OTA has been detected in cocoa powder in Ivory Coast, Guinea, Nigeria and Cameroon up to 4 mg/kg higher than the EU regulatory level (Bonvehi, 2004). Aroyeun and Adegoke (2007) recently reported over 50% occurrence of *A. ochraceus* and *A. niger* in cocoa beans in Nigeria with a corresponding 40–60 ppb OTA concentration. About 22% of the cocoa powder sold in Italian shops contained OTA (Tafuri et al., 2004) while 1800 ng/kg OTA was reported in German cocoa powder from 1996–1999 (Petzinger and Weidenbach, 2002).

There have also been concerns regarding contamination of coffee with OTA. Romani et al. (2000) detected OTA in 76/84 green coffee samples from 8 African countries at levels ranging from 0.5 to 48 µg/kg (mean 4 µg/kg). Shephard et al. (2003) found that all 24 tested S. African wines contained detectable levels (>0.01 µg/l) of OTA with a mean of 0.16 µg/l and 0.24 µg/l in white and red wines respectively. The mycotoxin was reported in 35% in "under-five clinics" of breast milks in Southern province of Sierra Leone with up to 22% co-occurrence with aflatoxins (Jonsyn et al., 1995). However, the scientists observed that whenever OTA was detected in high levels, AFB1 was absent or present at very low levels and vice versa which suggests some sort of competition between these toxins either at the production level in foodstuffs or in their rate of absorption in the gastrointestinal tract. OTA has also been reported as a contaminant of tiger nuts (Adebajo, 1993) and fermented maize dough (Kpodo, 1996) in West Africa.

9. Possible intervention strategies in mycotoxin management

Other than the direct health risk, economic losses and implications arising from mycotoxicoses are enormous. Many developing countries have realised that reducing mycotoxins levels in foods will not only reduce financial burden on health care but also confer international trade advantages such as exports to the attractive European markets. Factors fundamental to country's ability to protect its population from mycotoxins include the political will to address mycotoxins exposure and capability to test food for contamination, which determines whether requirements can be enforced. Management of mycotoxins contamination in Africa could be addressed in three fronts:

a) Prevention of exposure to mycotoxins
b) Decontamination
c) Constant surveillance and monitoring of moulds in contaminated food/feed stuff.

10. Prevention of exposure to mycotoxins

This approach of preventing exposure to mycotoxins aims at ensuring that foods have the lowest practical mycotoxins concentration. To achieve this, certain practices in the production (field), harvesting, storage, transportation, marketing, processing and legislation need to be observed.

11. Good agricultural practices

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

i. Early harvesting — 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 (Amyot, 1983). 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.

ii. Proper drying — 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 *Syzigum 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.

iii. Physical treatment — 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) and Lopez-Garcia and Park (1998). 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 (Davidson et al., 1982) and mouldy and stained peanut (Fandohan et al. 2005; Turner et al., 2005).

iv. Sanitation — 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).

v. Proper storage — 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).

vi. Insect management — 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.

vii. Other methods — Cultural practices including crop rotation, tillage, planting date, and management of irrigation and fertilization, have limited effects on infection and subsequent mycotoxins accumulation (Champeil et al., 2004; Munkvold, 2003).

12. Biological control

Significant inroads have been made in establishing various bio-control strategies such as development of atoxigenic bio-control fungi that can out-compete their closely related, toxigenic strains in field environments, thus reducing the levels of mycotoxins in the crops (Cleveland et al., 2003; Dorner et al., 1999). The International Institute for Tropical Agriculture (IITA) has for the last couple of years been researching on biological control of mycotoxins-producing fungi through competitive exclusion strategy (IITA, 2003b). The organization's researchers have found a less toxigenic strain of *A. flavus* that grows on grain stored under warm humid conditions, which can displace harmful strains that produce large amounts of toxins (IITA, 2003a). Potential bio-control agents include atoxigenic strains of *A. flavus* and A. *parasiticus* which upon introduction to soil of developing crops have resulted in 74.3 to 99.9% reduction in aflatoxin contamination in peanuts in the USA (Dorner et al., 1998). Dorner and Cole (2002) reported a field application of non-toxigenic strains of *A. flavus* and *A. parasiticus* that reduced post-harvest aflatoxin contamination by 95.9%. Use of biological agents to suppress growth of fumonisin-producing fungi has been reported. Desjardins et al. (1998) observed inhibition of fumonisin formation by atoxigenic *F. verticillioides* strains although these caused higher disease incidence when applied through the silk channel. The observation implied that the ability to produce fumonisins is not required to produce ear rot and that effective colonization of plant with atoxigenic strains could competitively exclude fumonisin-producing strains or prevent them from producing fumonisins. Luongo et al. (2005) also reported suppression of saprophytic colonization and sporulation of toxigenic *F. verticillioides* and *F. proliferatum* in maize residues by non-pathogenic *Fusarium* species. Control of fumonisin-producing fungi by endophytic bacteria has also been reported (Bacon et al., 2001). Competitive exclusion, whereby the

bacteria grow intercellularly precluding or reducing growth of intercellular hyphae was thought to be the mechanism involved. Masoud and Kaltoft (2006) reported *in vitro* inhibition of OTA production by *A. ochraceus* by three yeasts (*Pichia anomala*, *P. kluyveri* and *Hanseniaspora uvarum*). Fungal strains of *Trichoderma* have also been demonstrated to control pathogenic fungi through mechanisms such as competition for nutrients and space, fungistasis, antibiosis, rhizosphere modification, mycoparasitism, biofertilization and the stimulation of plant-defense mechanisms (Benítez et al., 2004). The ability of fungal antagonists to control toxigenic types is, however, dependent on the differential effect of macro and micro-climatic conditions on the antagonist–pathogen interaction (Luongo et al., 2005).

Important criteria for evaluating the effectiveness of mycotoxin bio-control agent include ability to colonize the target substrate or plant part, ability to be active under various environmental conditions in the field or during storage so that its growth and that of the pathogen coincide and compatibility with other control procedures without inducing effects that compromise end use quality of the commodity (Bacon et al., 2001). In this regard, atoxigenic strains of *F. verticillioides* and *F. proliferatum* would be superior bio-control agents for toxigenic strains since they occupy the same ecological niche as the toxigenic strains in the host plant and share similar growth conditions.

13. 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.

14. 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 Chlorophylin 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 (Hayes et al., 1998). 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. Entersorption 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 (Phillips et al. 1993). 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 (Mayura et al., 1998). Essential oils and acqueous 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.

15. Breeding for resistance

This is one of the most promising long-term strategies in mycotoxins contamination menace in Africa. Sources of resistance to *A. flavus* and *Fusarium* spp., particularly *F. verticillioides* have been identified and have been incorporated into public and private breeding programs (Munkvold, 2003). Potential biochemical and genetic resistance markers have been identified in crops, particularly maize in different parts of the world which are being utilized as selectable markers in breeding for resistance to aflatoxin contamination. Prototypes of genetically engineered crops have been developed which a) contain genes for resistance to the phytotoxic effects of certain trichothecenes, thereby helping reduce fungal virulence or b) contain genes encoding fungal growth inhibitors for reducing fungal infection in the USA. Gene clusters housing the genes that govern formation of trichothecenes, fumonisins and aflatoxins have been elucidated and are being targeted in strategies to interrupt the biosynthesis of these mycotoxins (Cleveland et al., 2003) Scientists at United States Department of Agriculture have identified two maize lines that are resistant to *A. flavus* and *F. moniliforme* (Hamiton, 2000). However, few if any, commercial cultivars have adequate levels of resistance to mycotoxins-producing fungi (Munkvold, 2003). Many organizations such as IITA are continuously working on resistance breeding programs in Africa (Hell et al., 2005). To devise effective strategies to control fungal infection and minimize mycotoxin production in host plants, a better knowledge of genetic variability and population structure at the intra-specific level and ability to detect cryptic populations or lineages which might arise that possess significant features in terms of toxin profile or host preferences is necessary (Mulé et al., 2005).

16. 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; Henry et al., 1999). In the US, 20 µg/kg is the maximum aflatoxin residue limit allowed in food for human

consumption, except for milk (Wu, 2006; FAO, 1996) 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). The European Union (EU) has recently issued a proposal to lower maximum tolerated limits for several mycotoxins in food and feed which became effective from 1st October 2006 (EC, 2006).

17. Surveillance and awareness creation

This could be a long-term intervention strategy as has been advocated by WHO (2006) and James (2005). It is imperative for African countries to strengthen nationwide surveillance, increase food and feed inspections to ensure food safety, and local education and assistance to ensure that food grains and animal feeds are harvested correctly, dried completely, and stored properly. Awareness of what mycotoxins are and the dangers that they pose to human and animal health could be done through government bodies, private organizations, non-governmental organizations, national media networks such as radios and television programs as well as features in newspapers and magazines. Seminars and workshops could be used as avenues and bridges of information exchange and dissemination between researchers and the populace respectively. Such events also serve as forums to assess past and present work and define and streamline areas of future studies. WHO (2006), has put plans in place to focus on field projects, strengthening surveillance and awareness raising and educating consumers on matters related to mycotoxins in Africa among others.

It is imperative that critical evaluation of the intervention strategies is done to put into consideration the sustainability, cultural acceptability, economic feasibility, ethical implication, and overall effectiveness of potential interventions.

18. Way forward: challenges and current needs

A regional experts meeting in 2005 on aflatoxins problem with particular reference to Africa made certain recommendations that could be instrumental in addressing or reducing mycotoxins contamination in the continent. The consultation noted that the achievement of mycotoxins reduction and control is dependent on the concerted efforts of all actors along the food production chain. Multidisciplinary approaches are therefore critical. The meeting recommended continued mycotoxins awareness as a public health issue, strengthened laboratory and surveillance capacities as well as establishing early warning systems. The participants also identified several research needs including cost-benefit analysis of interventions and research on the occurrence of mycotoxins in foods (WHO, 2006). In addition, there remains a need for efficient, cost-effective sampling and analytical methods that can be used for the detection of mycotoxins in developing countries (WHO, 2006; CTA, 1997).

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