Food Additives and Contaminants, October 2007; 24(10): 1109–1114

Taylor & Francis
Taylor & Francis Group

Relative severity of aflatoxin contamination of cereal crops in West Africa

RANAJIT BANDYOPADHYAY[1], MANJULA KUMAR[1], & JOHN F. LESLIE[2]

[1]*International Institute of Tropical Agriculture, Oyo Road, Ibadan, Nigeria, and* [2]*Department of Plant Pathology, Kansas State University, Manhattan, KS 66506, USA*

(*Received 19 April 2007; accepted 4 July 2007*)

Abstract
Aflatoxins are a common contaminant of cereals that can cause cancer, liver disease, immune suppression, retarded growth and development, and death, depending on the level and duration of exposure. Maize is an introduced crop to Africa and there have been efforts over the last 20 years or so to replace traditional cereal crops, such as sorghum (*Sorghum bicolor*) and pearl millet (*Pennisetum glaucum*), with maize. We found that maize was significantly more heavily colonized by aflatoxin-producing *Aspergillus* spp. than either sorghum or millet, with overall aflatoxin levels being correspondingly higher. On average, Nigerians consume 138 kg cereals annually. If the primary cereal is sorghum instead of maize, then the risk of aflatoxin-related problems is reduced 4-fold; if it is pearl millet, then the risks are reduced 8-fold. Development programs and other ventures to increase maize production in marginal cropping areas of Africa should be reconsidered and, instead, efforts to improve/maintain traditional crops encouraged.

Keywords: Aspergillus, Fusarium, *maize, mycotoxins, pearl millet, sorghum*

Introduction

Mycotoxins in general (CAST 2003) and aflatoxins (Williams et al. 2004) in particular pose major but unappreciated risks to humans residing in the less-developed countries of tropical regions. Aflatoxins are produced naturally by several species of *Aspergillus* (mainly *A. flavus*, *A. parasiticus*) and are usually associated with peanuts and cereals, although they may be recovered from commodities as diverse as chili, rice, sorghum, cottonseed, copra, nut crops, soybean, cheese, wheat, cassava, yam and melon seeds, eggs, milk and milk products (CAST 2003; Williams et al. 2004).

In the European Union, regulations limit the amount of total aflatoxins to $4\,ng\,g^{-1}$, whereas guidelines in a few developing countries and the US limit total aflatoxins to no more than $20\,ng\,g^{-1}$ in foodstuffs intended for human consumption (FAO 2004). In Nigeria, the National Agency for Food, Drug Administration and Control has set $20\,ng\,g^{-1}$ as the maximum permissible limit for total aflatoxin in foodstuff (FAO 2004). These regulations are usually enforced and problems associated with aflatoxin contamination are generally rare in developed countries. In most less-developed countries, regulations on aflatoxin contamination are, usually, either nonexistent or unenforceable since much of the agricultural produce never enters any official channel, but moves instead through local markets where ''caveat emptor'' remains the basic rule. While discoloured grain is often more difficult to sell than its normal counterpart, the relative scarcity of food in many regions often forces consumers into distressing decisions, such as to eat contaminated grain today and worry about the consequences tomorrow (or some other time in the future) or starve today and perhaps not even have a tomorrow. Thus, food containing high levels of aflatoxin is often consumed in less-developed countries.

Aflatoxins usually come to public prominence when the deaths of domesticated animals, e.g. contaminated dog food produced by Diamond Pet Foods in the US in 2005 (http://www.fda.gov/oc/po/firmrecalls/diamond12_05.html) or human mortalities, such as occurred in Kenya in both

Correspondence: Ranajit Bandyopadhyay. E-mail: r.bandyopadhyay@cgiar.org

ISSN 0265–203X print/ISSN 1464–5122 online
DOI: 10.1080/02652030701553251

2004 and 2005 (Lewis et al. 2005), are associated with aflatoxin contamination. Yet long-term exposure to sub-lethal levels of aflatoxin are probably more widespread and more insidious than acute aflatoxicosis (Williams et al. 2004). Aflatoxins are classified as Class I carcinogens by IARC (IARC 1993) and have long been associated with liver cancer in both humans and domesticated animals (Gorelick et al. 1993). The immune suppression associated with aflatoxin consumption means that people are more susceptible to infections in general and that vaccination programs to reduce communicable diseases are less likely to be successful (Jiang et al. 2005). Retardation of early childhood growth and development (Gong et al. 2002, 2004) means that human resources within a country will not be realized and subsequent development of the country is hindered.

The objective of this study was to compare *Aspergillus* contamination and aflatoxin levels in maize, sorghum and pearl millet grown side-by-side by subsistence farmers in Africa.

Materials and methods

IITA routinely runs multi-location ''on-farm'' trials of traditional and improved maize, sorghum and pearl millet lines to determine yields and the effects of agronomic and other parameters not always observable in fields at research stations. The cooperating farmers are provided with seed and other inputs, e.g. fertilizers and pesticides, and allow the collection of data on yield, maturity and pest and disease incidence. For this study, we used samples grown by farmers at 14 locations in two of the agricultural/ecological zones in western Africa – the Northern Guinea savanna and the Southern Guinea savanna. Cereal/root crop mixed farming system is a common feature of these two zones that run from east to west across West Africa from Liberia to the Central African Republic (Dixon et al. 2002). Maize, sorghum and pearl millet are the primary cereal components in this cropping system (Dixon et al. 2002). Crop varieties suitable for use in one agricultural/ecological zone may not be appropriate for another. All three crops were grown at two locations, maize and sorghum only at 11 locations and maize and pearl millet only at one location.

Crops were planted in late June 2005 (actual dates varied according to occurrence of adequate rainfall for sowing) and grown in Nigeria according to standard practices, which varied somewhat by region. Farmers in the Northern Guinea Savanna agro-ecozone grew the trials at Bichi, Kaya, Dunki, Danbrinin, Kachia, Kufana, Idon, Gwaram, Gar, Badara, Grazing reserve and Kasuwamagani, and in the Sudan Savanna agro-ecozone in Wudil and Danbua. At least two, but often three or four cultivars of sorghum (out of Local, ICSV 400, KSV 8, Local Kaura and Local SK) and maize (Local, HE 97TZE and Across 97) were sampled in the locations where these two crops were grown. The number of cultivars sampled depended on the number of cultivars grown by the farmers. Pearl millet was grown in only three locations since farmers in other locations do not traditionally grow this crop. All cultivars (Local, Sossatc88 and LCIC 9702) of pearl millet in the trials by farmers were sampled. All three crops were grown in Bichi and Wudil, only pearl millet and sorghum in Danbua, and only sorghum and maize in the remaining 11 locations.

Grain was harvested during the first half of October at plant maturity (~12% grain moisture) and ~1 kg of each sample (five sub-samples of 200 g) shipped immediately to the IITA laboratory in Ibadan, where it was received in 2 days, and stored in paper bags at 4°C until analyzed. The samples were processed within 3 weeks for mycological and aflatoxin analysis. One hundred kernels of each grain sample were surface sterilized in 1% NaOCl for 3 min, washed in three/four changes of sterile water, placed on moist filter paper in a Petri dish and incubated for 6 days on a laboratory bench at 25–28°C. The proportion of kernels infested with one or more *Aspergillus* spp., one or more *Fusarium* spp., other fungi, or free of any fungal contamination was determined. This method underestimates the frequency of *Fusarium* spp. in millet and sorghum as they are often outperformed by other faster growing fungi already present.

A total of 500 g of grain from each sample was ground with a Romer Series II mill (Romer Labs, Union, MO, USA) and a fine powder (particle size <841 μm) obtained after sifting through a 20-Mesh sieve. Then, 1 g of the powder was suspended in sterile H_2O to make 10 ml total volume. Ten μl of the suspension was spread on plates of a medium selective for *Aspergillus* (Cotty 1997). The number of colony forming units (cfu) of all *Aspergillus* species was recorded. To determine the nature of *Aspergillus* species, 20–36 isolates from two/three different crop samples from each location were further characterized as ''S'' (more toxigenic) and ''L'' (less toxigenic) types of *A. parasiticus* and *A. tamari* based on their morphology and toxin production potential (Cotty 1997). The amount of toxin production was not quantified but the type of aflatoxin (B or G or both) produced was recorded from TLC plates visualized under long-wave UV light (365 nm) to distinguish the toxin profiles of ''S'' and ''L'' types. ''L'' type isolates of

Aspergillus that produced only B aflatoxin and had smooth conidial surface were considered as ''L'' type *A. flavu*s. ''S'' type isolates that produced both B and G aflatoxins and had smooth conidial surface were considered as the unnamed *Aspergillus* taxon S_{BG} (Cotty and Cardwell 1999). Isolates with dark green colonies that produced both B and G aflatoxins and had rough conidial surface were considered as *A. parasiticus*.

A total of 20 g of powdered grain was extracted by blending at high speed with 100 ml of 70% methanol for 3 min. The slurry was allowed to settle and the supernatant containing the toxin was filtered through Whatman No. 1 filter paper (Whatman International Ltd., Maidstone, UK) and 15 ml of the filtrate collected for further evaluation. The sample was diluted by adding 100 µl of extract to a microtiter well containing 100 µl of enzyme conjugate and processed according to the instructions for use of the CD-ELISA kit (EZ-Quant^{TM} Aflatoxin Plate Kit, Diagnostix Limited, Mississauga, Canada). Solutions containing known amounts of aflatoxin served as the controls. OD_{450} was measured with a Dynatech MR 250 microwell reader (Dynatech Laboratories Inc., Chantilly, VA, USA) and plotted against a standard curve generated with each microtiter plate from aflatoxin solutions of known concentration to determine the amount of aflatoxin present. Samples with more aflatoxin than in the most concentrated standard were diluted appropriately with 70% methanol and reassayed. The minimum detection limit was $1\,ng\,g^{-1}$. The degree of variation between subsamples was <15%. Based on spiked recovery controls using 5, 10, 15, 20, and $25\,ng\,g^{-1}$ levels in maize, sorghum and pearl millet grains, ≥80% of the aflatoxin present was recovered by this method.

Results

Kernels of all grains could be contaminated with *Aspergillus*, *Fusarium* and other species of filamentous fungi, but these species were not present equally on all of grains (Table I). Kernels of maize were four- and nine-fold more likely to be contaminated with *Aspergillus* than comparable samples of sorghum and pearl millet, respectively, and 1.8-fold more likely to be contaminated with *Fusarium* than sorghum and pearl millet. Sorghum, however, was more likely to be contaminated with other filamentous fungi than were either maize or millet. Within the 623 strains of *Aspergillus* species characterized from maize (313) and sorghum (310), *A. flavus* ''L'' type was always dominant (>80%) with one or two of the other types occasionally isolated but never at a frequency >17% of the total (Table II). In the three pearl millet samples analyzed, all 66 characterized strains were ''L'' type with a mean contamination level of 1367 cfu g^{-1}.

Table I. Percentage of maize, sorghum and pearl millet kernels with no visible fungal infection or infected with *Aspergillus* spp. or *Fusarium* spp. (blotter test).

Crop	Sample size (*n*)	Fungal Genus present			
		Aspergillus	*Fusarium*	Others	None detected
Maize	23	17.6 a*	47.3 a	3.9 a	31.2 a
Sorghum	40	4.2 b	26.3 b	43.2 c	26.3 a
Pearl millet	7	1.9 b	26.1 b	55.6 b	16.4 a

*Within each column, mean values followed by similar letters are not significantly different ($p \leq 0.05$).

Average aflatoxin contamination (Table III) was much higher in maize ($36\,ng\,g^{-1}$) than in either sorghum ($8.8\,ng\,g^{-1}$) or pearl millet ($4.6\,ng\,g^{-1}$). The median amount of aflatoxin in a sample was similar for all three grains (4.2, 5.0 and $4.4\,ng\,g^{-1}$, respectively), suggesting that the major problem was samples that were heavily contaminated. Of the 23 maize samples, four (17%) exceeded the $20\,ng\,g^{-1}$ Nigerian guideline as did two (5%) of the 40 sorghum samples but none of the pearl millet samples. In addition to having a higher proportion of samples that did not meet the guidelines, maize samples also contained higher levels of aflatoxin than did non-conforming sorghum samples. Maize samples had up to 24-fold the recommended maximum aflatoxin level, while sorghum samples (4.5-fold) were less heavily contaminated with aflatoxin, even in the worst case. The likelihood of aflatoxin exposure to humans from maize is particularly high in zones where the frequency of maize consumption, the presence of aflatoxin in maize or the presence of *A. flavus* on maize is relatively high (Egal et al. 2005).

Discussion

Our data clearly show that maize is more likely to be contaminated with aflatoxins and to be contaminated at a higher level than either sorghum or millet. A comparative study in the US has shown that pearl millet, commonly used in poultry diets, harbours less aflatoxin-producing fungi and is less prone to aflatoxin contamination compared to maize (Wilson et al. 2006). Aflatoxin contamination usually increases during storage and, as samples were taken immediately after harvest, the values obtained are likely to be the lowest occurring on

these grains during the year. Food safety issues, especially in rural areas, are such that all of grain produced is consumed locally and usually by humans.

Aflatoxin exposure in humans is high in West Africa; >99% of the population in many areas are positive for long term exposure (Gong et al. 2002). Subsistence farmers in the savannas of Africa on average consume locally grown maize virtually every day of the week (Cardwell and Henry 2004). Given a typical diet of Nigerians, which includes 138 kg cereal person^{-1} year^{-1} (FAO 2005), aflatoxin exposure from maize consumption would be ~5.0 mg person^{-1} year^{-1} (calculated by multiplying mean aflatoxin level in maize, i.e. 36 ng g^{-1}, by annual cereal consumption per person, i.e. 138 kg) and the corresponding value for sorghum would be ~1.2 mg person^{-1} year^{-1}. Thus, the average difference between consuming maize and sorghum would be almost 3.8 mg year^{-1}. The average daily aflatoxin exposure per person would be 13.6 μg day^{-1} for maize (which exceeds the Nigerian recommended maximum of 7.6 μg day^{-1} for foodstuff), 3.3 μg day^{-1} for sorghum (less than half the Nigerian recommended maximum) and 1.7 μg day^{-1} for pearl millet (less than one quarter of the Nigerian recommended maximum) (Table IV). Sorting grain, usually by discolouration, can significantly reduce exposure to Fusarium toxins (Afolabi et al. 2006) and could also reduce aflatoxin contamination levels in non-discoloured grain. However, poor quality, discoloured grain, enriched with aflatoxin, is likely to be consumed by the poorest consumers who are also the most susceptible to the subclinical

Table II. Number of colony forming units (cfu) of four *Aspergillus* spp. per gram of milled grain of maize, sorghum and pearl millet.

			Aspergillus spp. (% of total, range)			
Crop	Sample size (*n*)	cfu g^{-1}grain	*A. flavus* "L"	Unnamed taxon "S_{BG}"	*A. parasiticus*	*A. tamarii*
Maize	13	9900**	83–100	0–17	0–15	0–5
Sorghum	13	2400	83–100	0–12	0–4	0–10

**Significant at $p = 0.001$ ($n = 39$; three subsamples each from 13 locations).

Table III. Relative levels of aflatoxin in freshly harvested maize, sorghum and pearl millet in 2005.

		Aflatoxin (ng g^{-1})			
Crop	Sample size (*n*)	Mean ± SD	Median	Range	% Samples with > 20 ng g^{-1} aflatoxin
Maize	23	36.0 ± 100	4.2	1.1 – 480	17
Sorghum	40	8.8 ± 14	5.0	1.6 – 90	5
Pearl millet	7	4.6 ± 1.8	4.4	2.6 – 8.1	0

Table IV. Calculated aflatoxin exposure of Nigerians consuming maize, sorghum or pearl millet in their diet compared with aflatoxin-safe levels in maize as per Nigerian food safety guidelines.

		Aflatoxin exposure		
Commodity	Aflatoxin level (ng g^{-1})*	μg person^{-1} year^{-1}†	ng person^{-1} day^{-1}	ng kg^{-1} body weight day^{-1}‡
Maize	36.0	4968	13611	226.8
Sorghum	8.8	1214	3327	55.5
Pearl millet	4.6	634	1739	29.0
Aflatoxin-safe foodstuff	20.0	2760	7562	126.0

*Values for maize, sorghum and pearl millet are means based on analysis of field samples, whereas the value for aflatoxin-safe maize is the maximum limit recommended by the National Agency for Food, Drug Administration and Control of Nigeria.
†Calculated by multiplying the average cereal consumption per year (i.e. 138 kg person^{-1} year^{-1}) by the corresponding aflatoxin level in grain.
‡For a person with average body weight of 60 kg.

problems associated with aflatoxin poisoning. Other relatively simple processing steps can reduce toxin levels in other commodities (Desjardins et al. 2000; Turner et al. 2005) and could be applied either directly or in principle to help reduce or prevent contamination. Detoxification is not economically feasible for most people in this region, although some calcium montorillonite clays, which can bind aflatoxin to prevent its absorption, have been used in animal feeds in developed countries and might be economically viable (Phillips et al. 1995; Phillips 1999).

In other studies on aflatoxins in cereals from Africa, Shephard (2005) reviewed aflatoxin contamination and food safety issues in grain available in local markets. Sangare-Tigore et al. (2006) suggested that some west African maize has only minor problems with contamination with either aflatoxin B_1 or other commonly encountered maize contaminants, e.g. fumonisins, ochratoxins and zearalenone. Such relatively low levels might be found routinely in grain destined for export, but are probably rare in grain consumed locally, especially in rural areas, given the relatively high aflatoxin B_1 albumin adducts known to occur in this region (Jolly et al. 2006).

Our study differs from those mentioned above in that we evaluated field data rather than market data. Grain in the markets is often the cleanest grain available, as the poorer quality grain is often unmarketable and consumed on the farm of origin. Data on field levels of aflatoxins and on commodities other than maize and peanut are much less common than data on market samples (Shephard 2005). In Ethiopia, Ayalew et al. (2006) reported that a small proportion (6%) of sorghum field samples were contaminated with up to $26\,ng\,g^{-1}$ aflatoxin B_1 (mean $0.9\,ng\,g^{-1}$, average $10\,ng\,g^{-1}$) – levels considerably lower than those observed in this study. Sorghum and millet from Sudan (Abdel-Rahim et al. 1989), millet from Gambia (Hudson et al. 1992) and sorghum from Botswana (Siame et al. 1998) have also been reported to be occasionally contaminated with low levels of aflatoxin.

Maize has become a favoured cereal in parts of West Africa due to its taste and cooking properties. The area devoted to its production has increased nearly 10-fold (from 465 000 ha in 1980 to 4 466 000 ha in 2004) in Nigeria alone in the last 25 years (FAOSTAT data 2006). Certainly, maize production will not cease due solely to the problem of aflatoxin contamination, but additional management practices need to be introduced to reduce aflatoxin levels throughout the region. The best way to reduce aflatoxin contamination is to prevent it from ever forming on foodstuffs. Maize planted in marginal regions usually encounters both heat and drought stress – two factors that increase the risk for aflatoxin contamination in grain (Diener et al. 1987; Bruns 2003). Replacing maize in these marginal environments with indigenous sorghum or pearl millet would disproportionately reduce aflatoxin exposure since maize with extreme levels of contamination would be encountered less frequently and the crops replacing it would have a much lower risk of contamination.

Acknowledgements

This research was supported in part by the Kansas Agricultural Experiment Station and by the Sorghum and Millet Collaborative Research Support Program (INTSORMIL) AID/DAN-1254-G-00-0021-00 from the US Agency for International Development. Contribution no. 08-17-J from the Kansas Agricultural Experiment Station, Manhattan.

References

Abdel-Rahim AM, Osman NA, Idris MO. 1989. Survey of some cereal grains and legume seeds for aflatoxin in the Sudan. Zentralblatt für Mikrobiologie 144:115–121.

Afolabi CG, Bandyopadhyay R, Leslie JF, Ekpo EJA. 2006. Effect of sorting on incidence and occurrence of fumonisin and fumonisin-producing *Fusarium* species on maize (*Zea mays* L.) in Nigeria. Journal of Food Protection 69:2019–2023.

Ayalew A, Fehrmann H, Lepschy J, Beck R, Abate D. 2006. Natural occurrence of mycotoxins in staple cereals from Ethiopia. Mycopathologia 162:57–63.

Bruns HA. 2003, Controlling aflatoxin and fumonisin in maize by crop management. Journal of Toxicology: Toxin Reviews 22:153–173.

Cardwell KF, Henry SH. 2004, Risk of exposure to and mitigation of effect of aflatoxin on human health: A west African example. Journal of Toxicology: Toxin Reviews 23:217–247.

Cotty PJ. 1997. Aflatoxin-producing potential of communities of *Aspergillus* section *Flavi* from cotton producing areas in the United States. Mycological Research 101:698–704.

Cotty PJ, Cardwell KF. 1999. Divergence of West African and North American Communities of *Aspergillus* section *Flavi*. Applied and Environmental Microbiology 65:2264–2266.

Council for Agricultural Science and Technology. 2003. Mycotoxins: Risks in plant, animal, and human systems. CAST Task Force Report Number 139. Ames, IA: CAST.

Desjardins AE, Manandhar G, Plattner RD, Maragos CM, Shrestha K, McCormick SP. 2000. Occurrence of *Fusarium* species and mycotoxins in Nepalese maize and wheat and the effect of traditional processing methods on mycotoxin levels. Journal of Agricultural Food Chemistry 48:1377–1383.

Diener UL, Cole RJ, Sanders TH, Payne GA, Lee LS, Klich MA. 1987. Epidemiology of aflatoxin formation by *Aspergillus flavus*. Annual Review of Phytopathology 25:249–270.

Dixon J, Gulliver A, Gibbon D. (2002). Farming Systems and Poverty 2001: Improving Farmers' Livelihoods in a Changing World. FAO/World Bank: Rome/Washington, DC.

Egal S, Hounsa A, Gong YY, Turner PC, Wild CP, Hall AJ, Hell K, Cardwell KF. 2005. Dietary exposure to aflatoxin from maize and groundnut in young children from Benin and Togo, West Africa. International Journal of Food Microbiology 104:215–224.

FAO. 2004. Worldwide regulations for mycotoxins in food and feed in 2003. FAO Food and Nutrition Papers 81. Food and Agriculture Organization of the United Nations: Rome, Italy. pp 1–180.

FAO. 2005. Africa Report 3. Food Supply Situation and Crop Prospects in Sub-Saharan Africa (GIEWS). Food and Agriculture Organization of the United Nations: Rome, Italy. pp 1–69.

FAOSTAT data [internet]. Available: http://faostat.fao.org/. Accessed: 25 November 2006.

Gong YY, Cardwell KF, Housna A, Egal S, Turner PC, Hall AJ, Wild CP. 2002. Dietary aflatoxin exposure and impaired growth in young children from Benin and Togo: Cross-sectional study. British Medical Journal 325:20–21.

Gong YY, Hounsa A, Egal S, Turner PC, Sutcliffe AE, Hall AJ, Cardwell KF, Wild CP. 2004. Postweaning exposure to aflatoxin results in impaired child growth: A longitudinal study in Benin, West Africa. Environmental Health Perspectives 112:1334–1338.

Gorelick NJ, Bruce RD, Hoseyni MS. 1993. Human risk assessment based on animal data: Inconsistencies and alternatives. In: Eaton D, Groopman JD, editors. The toxicology of aflatoxins: Human health, veterinary, and agricultural significance. London: Academic Press. pp 508–511.

Hudson GJ, Wild CP, Zarba A, Groopman JD. 1992. Aflatoxins isolated by immunoaffinity chromatography from foods consumed in the Gambia. Natural Toxins 1:100–105.

IARC. 1993. Some naturally occurring substances: Food items and constituents, heterocyclic aromatic amines and mycotoxins. IARC Monographs on the evaluation of carcinogenic risks of chemicals to humans, Vol. 56. Lyon, France: International Agency for Research on Cancer. pp 1–599.

Jiang Y, Jolly PE, Ellis WO, Wang J-S, Phillips TD, Williams JH. 2005. Aflatoxin B_1 albumin adduct levels and cellular immune status in Ghanaians. International Immunology 17:807–814.

Jolly P, Jiang Y, Ellis W, Awuah R, Nnedu O, Phillips T, Wang JS, Afriye-Gyawu E, Tang L, Person S, et al. 2006. Determinants of aflatoxin levels in Ghanaians: Sociodemographic factors, knowledge of aflatoxin and food handling and consumption practices. International Journal of Hygiene and Environmental Health 209:345–358.

Lewis L, Onsongo M, Njapau H, Schurz-Rogers H, Luber G, Kieszak S, Nyamongo J, Backer L, Dahiye AM, Misore A, et al. 2005. Aflatoxin contamination of commercial maize products during an outbreak of acute aflatoxicosis in eastern and central Kenya. Environmental Health Perspectives 113:1763–1767.

Phillips TD. 1999. Dietary clay in the chemoprevention of aflatoxin-induced disease. Toxin Science 52:118–126.

Phillips TD, Sarr AB, Grant PG. 1995. Selective chemisorption and detoxification of aflatoxins by phyllosilicate clay. Natural Toxins 3:204–213.

Sangare-Tigori B, Moukha S, Kouadio HJ, Betbeder A-M, Dano DS, Creppy EE. 2006. Co-occurrence of aflatoxin B_1, fumonisin B_1, ochratoxin A and zearalenone in cereals and peanuts from Côte d'Ivoire. Food Additives and Contaminants 23:1000–1007.

Shephard GS. 2005. Aflatoxin and food safety: Recent African perspectives. In: Abbas HK, editor. Aflatoxin and food safety. London: Taylor and Francis.

Siame BA, Mpuchane SF, Gashe BA, Allotey J, Teffera G. 1998. Occurrence of aflatoxins, fumonisin B_1 and zearalenone in foods and feeds in Botswana. Journal of Food Protection 61:1670–1673.

Turner PC, Sylla A, Gong YY, Diallo MS, Sutcliffe AE, Hall AJ, Wild CP. 2005. Reduction in exposure to carcinogenic aflatoxins by postharvest intervention measures in west Africa: A community-based intervention study. Lancet 365: 1950–1956.

Williams JH, Phillips TD, Jolly PE, Stiles JK, Jolly CM, Aggarwal D. 2004. Human aflatoxicosis in developing countries: A review of toxicology, exposure, potential health consequences, and interventions. American Journal of Clinical Nutrition 80:1106–1122.

Wilson JP, Jurjevic Z, Hanna WW, Wilson DM, Potter TL, Coy AE. 2006. Host-specific variation in infection by toxigenic fungi and contamination by mycotoxins in pearl millet and corn. Mycopathologia 161:101–107.