International Livestock Research Institute

Aflatoxin literature synthesis and risk mapping: Special emphasis on sub-Saharan Africa

Christine Atherstone, Delia Grace, Farid Waliyar, Johanna Lindahl and Moses Osiru

August 2014

CGIAR

ILRI
INTERNATIONAL LIVESTOCK RESEARCH INSTITUTE

ICRISAT INTERNATIONAL CROPS RESEARCH INSTITUTE FOR THE SEMI-ARID TROPICS

Authors

Christine Atherstone, International Livestock Research Institute
P.O. Box 24384, Kampala, Uganda

Delia Grace, International Livestock Research Institute
P.O. Box 30709, GPO 00100, Nairobi, Kenya

Farid Waliyar, International Crops Research Institute for the Semi-Arid Tropics
BP 320 Bamako, Mali

Johanna Lindahl, International Livestock Research Institute
P.O. Box 30709, GPO 00100, Nairobi, Kenya

Moses Osiru, International Crops Research Institute for the Semi-Arid Tropics
BP 320 Bamako, Mali

Citation

Atherstone, C., Grace, D., Waliyar, F., Lindahl, J. and Osiru, M. 2014. *Aflatoxin literature synthesis and risk mapping: Special emphasis on sub-Saharan Africa*. ILRI project report. Nairobi, Kenya: ILRI.

Contents

List of tables

List of figures

Acronyms and abbreviations

AIDS	Acquired Immunodeficiency Syndrome
CAST	Council for Agricultural Science and Technology
CDC	Centers for Disease Control and Prevention
CI	confidence interval
CIMMYT	International Maize and Wheat Improvement Center
CPA	cyclopiazonic acid
DALYs	Disability-Adjusted Life Years
DNA	deoxyribonucleic acid
FAO	Food and Agriculture Organization of the United Nations
FDA	Food and Drug Administration
HIV	human immunodeficiency virus
IARC	International Agency for Research on Cancer
ICRISAT	International Crops Research Institute for the Semi-Arid Tropics
IFPRI	International Food Policy Research Institute
IITA	International Institute of Tropical Agriculture
ILRI	International Livestock Research Institute
KES	Kenya shilling(s)
MRC	South African Medical Research Council
OR	odds ratio
ppb	parts per billion
ppm	parts per million
UNICEF	United Nations Children's Fund
USAID	United States Agency for International Development
USD	United States dollar(s)
WHO	World Health Organization

Acknowledgements

This report was the work of many people. At the International Livestock Research Institute, Kenya, thanks to Pamela Ochungo for mapping of the aflatoxin prevalence studies and Isaiah Akuku for securing many of the journal articles summarized in this report. At the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Mali, thanks to Aoua Traoré for access to aflatoxin prevalence data from the ICRISAT laboratory. At the International Institute of Tropical Agriculture, Nigeria, thanks to Ranajit Bandyopadhyay for the detailed information on the biocontrol product Aflasafe™.

This report was supported by the CGIAR Research Program on Agriculture for Nutrition and Health, led by the International Food Policy Research Institute.

Executive summary

The availability of a safe and nutritious food supply comprises one of the cornerstones of food security across much of the developing world, particularly in Africa. Aflatoxins are naturally occurring mycotoxins produced by the fungus *Aspergillus* spp. These toxins contaminate an array of crops including maize (corn), groundnuts (peanuts), millet, wheat, rice, oats, barley, sorghum, teff (an African cereal), soybeans, beans and peas, edible oils, nuts (other than groundnuts), traditional plant remedies, spices, sesame seeds, dried fruit, dried vegetables, melons, eggs, milk (cow, goat, sheep, camel and buffalo), cheese, meat and fish in tropical and sub-tropical regions worldwide.

While almost a dozen different *Aspergillus* species can produce aflatoxins, *Aspergillus flavus* and *Aspergillus parasiticus* are the most important species for aflatoxin production in food crops. At least 14 different types of aflatoxins are produced in nature (Boutrif 1998). Aflatoxin B_1 is the most toxic aflatoxin and is produced by both *A. flavus* and *A. parasiticus*. Aflatoxin B_2 is also produced by both *A. flavus* and *A. parasiticus.* Aflatoxin G_1 and aflatoxin G_2 are produced exclusively by *A. parasiticus*. Aflatoxin M_1 and aflatoxin M_2 were originally discovered in the milk of cows fed on aflatoxin-contaminated grain. Aflatoxin M_1 and aflatoxin M_2 are the products of a conversion process of, respectively, aflatoxin B_1 and aflatoxin B_2 in the animal's liver. Aflatoxin M_1, a metabolite of aflatoxin B_1, and aflatoxin M_2, a metabolite of aflatoxin B_2, are found in human breast milk and in the milk of animal's fed on contaminated feeds.

Crops are particularly susceptible to infection in the field by *Aspergillus* spp. following prolonged exposure to high humidity, drought, insect damage and use of susceptible crop varieties. Favourable storage conditions that promote growth of *Aspergillus* spp. and aflatoxin contamination include high moisture content (at least 7%), high temperature and insect or rodent damage. Crops can become contaminated in the field, during harvesting and drying and under inadequate storage conditions. While the presence of *Aspergillus* spp. in food products does not always indicate harmful levels of aflatoxin are also present, it does imply a significant risk in consumption, particularly in food products with frequent and high consumption in developing world populations.

Humans are exposed to aflatoxins primarily through the consumption of contaminated agricultural or animal products. In recent years, hundreds of aflatoxicosis cases in Africa have been associated with consumption of contaminated home-grown maize. Chronic aflatoxin exposure leads to several health-related conditions, including acute and chronic aflatoxicosis, aflatoxin-related immune suppression, liver cancer, liver cirrhosis and nutritional-related problems. Chronic exposures are endemic in developing countries because aflatoxin monitoring is inadequate, populations tend to rely on just a few staple crops that are vulnerable to *Aspergillus* spp. infection and growing conditions favour mould growth.

In animals, chronic exposure through consumption of feed contaminated with low levels of aflatoxin can cause liver damage, gastrointestinal dysfunction, decreased appetite, decreased reproductive function, decreased growth and decreased production. In addition, immunosuppression results in greater susceptibility to other diseases. Trace levels of aflatoxins and their metabolites may also carry over into the edible tissue of meat-producing animals, eggs from poultry and milk from dairy animals.

Current technologies in improved field crop management, pre-harvest and post-harvest practices, clinical interventions and public information campaigns all have potential to reduce the risk of aflatoxin contamination in foods and feeds thereby reducing human and animal exposure in

developing countries. Many of these technologies and practices have proven to be effective during field trials but large-scale rollout still needs to be evaluated, particularly in their role in reducing aflatoxin contamination and exposure along the different commodity value chains.

At this time, aflatoxin contamination is often a problem of unknown dimensions on farms and in warehouses, processing facilities and food products. What is known, however, is that the pervasive and chronic consumption of aflatoxin-contaminated foods and feeds throughout Africa continues to pose a significant threat to both human and animal health.

1. Background

Worldwide, it is estimated that nearly a quarter of agricultural crops are contaminated with mycotoxins (Reddy et al. 2010). Mycotoxins are fungal toxins that contaminate staple foods consumed by many of the poorest and most vulnerable populations in the world. In livestock production, mycotoxins pose the greatest risk to animal feed safety (Bankole and Adebanjo 2004). The economic impact of these mycotoxins includes losses incurred by human and animal deaths, veterinary and physician costs, reduced productivity of animals, loss of livelihoods, costs of control measures, loss of trade, losses to farmers through disposal of contaminated foods or feeds and investment in aflatoxin research to come up with mitigation strategies. According to the World Development Report, diseases caused by mycotoxins lead to reduced life expectancy in developing countries (Bankole and Adebanjo 2004). The main mycotoxins of significance for human disease are aflatoxins, fumonisins, ochratoxin A, deoxynivalenol and zearalenone (Pitt et al. 2012).

Aflatoxins are highly carcinogenic, secondary metabolites of the fungi *Aspergillus flavus*, *Aspergillus parasiticus* and occasionally other *Aspergillus* species (Pitt et al. 2012). While almost a dozen different *Aspergillus* species can produce aflatoxins, *A. flavus* and *A. parasiticus* are the most important species for aflatoxin production in food crops. *A. flavus* is delineated into two morphotypes called the *S* and *L* strains. The *S* strain produces many small sclerotia (less than 400 µm in diameter), relatively few conidia and consistently high levels of aflatoxin. The *L* strain produces fewer, larger sclerotia (over 400 µm in diameter), more conidia and, on average, less aflatoxin than the *S* strain. A significant percentage of *L* strain isolates produce no aflatoxin which makes them good candidates for use as active agents in biocontrol products that manage aflatoxin contamination.

Aflatoxins are prevalent in food crops, particularly maize, groundnuts, oilseeds and tree nuts, in tropical and subtropical regions worldwide. Host crops are particularly susceptible to infection by *Aspergillus* following exposure to high humidity and temperature, or damage from stressful conditions such as drought and insects and the average aflatoxin-producing potential of the fungal community associated with the crops (Cotty and Jaime-Garcia 2007). Crops can become contaminated in the field, during harvesting and after harvest during food storage, transportation and processing (Probst et al. 2010; Wu et al. 2011a). While the presence of *Aspergillus* in food products does not always indicate harmful levels of aflatoxin are present, it does imply a significant risk in consumption, particularly in food products with frequent and high consumption in developing world populations. Maize and groundnuts are the major source of aflatoxin exposure in humans because of the frequent and high consumption rates of these foods worldwide and their susceptibility to *Aspergillus* infection (Strosnider et al. 2006). Seasonal variation in contamination levels is common (Cotty and Jaime-Garcia 2007; James et al. 2007) and has been linked anecdotally to rain at or near harvest and high daily temperatures during key stages of crop development.

Developing crops are frequently very resistant to infection by *A. flavus* and subsequent aflatoxin contamination unless environmental conditions favour both fungal growth and crop susceptibility. During the first phase of contamination, wounding of the developing crop by birds, mammals or insects or mechanically (e.g. by hail), or the stress of hot, dry conditions result in significant infections (Cotty and Jaime-Garcia 2007). For crops with the most severe contamination problems, the distribution and planting time are generally designed to avoid conditions conducive to *A. flavus*. However, because weather is not consistent, even well planned crops may become exposed to conditions favourable to contamination.

The second phase of contamination may occur at any time from crop maturation until consumption. During this phase, toxin increases in crops infected during the first phase and those infected after maturation. The second phase occurs when the mature crop is exposed to warm, moist conditions in the field or during transportation, storage or use. Under high humidity, initially dry seeds develop water content conducive to contamination. Substrate moisture content and temperature dictate the extent of contamination. Crop contamination with aflatoxins frequently involves both of these phases (Cotty and Jaime-Garcia 2007).

Favourable growth temperatures for *A. flavus* are a minimum of 10–12°C, a maximum of 43–48°C and an optimum of about 33°C (Pitt et al. 2012). Growth of *A. flavus* occurs over the pH range of 2.1–11.2 (the entire range examined) at 25°C, 30°C and 37°C, with optimal growth over a broad pH range of 3.4–10. *A. flavus* is also very heat resistant. At 45°C, the time required to kill at least 90% of the fungal population is 106 hours, and at 60°C, 1 minute (Pitt et al. 2012). The growth of *A. parasiticus* is very similar to that of *A. flavus*, with the exception that *A. parasiticus* grows at slightly lower temperatures, with a maximum temperature of up to 42°C (Pitt et al. 2012).

At least 14 different types of aflatoxins are produced in nature by the different species of *Aspergillus* (Boutrif 1998). Aflatoxin B_1 is considered the most toxic and is produced by both *A. flavus* and *A. parasiticus*. Aflatoxin B_1 is carcinogenic and teratogenic in both humans and animals. To date, aflatoxin B_1 is the only mycotoxin classified as a Group 1a human carcinogen by the International Agency for Research on Cancer (IARC 1982). Aflatoxin B_2 is also produced by both *A. flavus* and *A. parasiticus.* Aflatoxin G_1 and aflatoxin G_2 are produced exclusively by *A. parasiticus*. Aflatoxin M_1 and aflatoxin M_2 were originally discovered in the milk of cows fed on mouldy grain. These compounds are products of a conversion process in the animal's liver. Aflatoxin M_1 is a metabolite of aflatoxin B_1 in humans and animals and aflatoxin M_2, a metabolite of aflatoxin B_2 in milk of cattle fed on contaminated feeds.

Chronic human aflatoxin exposure causes acute liver damage and liver cirrhosis, as well as development of tumours (USAID 2003). For people chronically infected with hepatitis B virus, chronic aflatoxin consumption raises by up to thirty-fold the risk of liver cancer compared with either exposure alone (Groopman and Kensler 2005). Currently, aflatoxins have also been shown to have a related effect in inducing liver cancer in people with hepatitis C. The difference in aflatoxin-induced liver cancer between hepatitis B virus and hepatitis C virus infection is related to their epidemiology. From an infectious disease standpoint, humans are far more likely to acquire chronic hepatitis B virus infection early in life, usually in the first year of life. Infection early in life lengthens the time at which an individual is both chronically infected and also exposed to aflatoxin. The later in life hepatitis B virus is acquired, the less likely it is to become a chronic infection. The chance that an infection becomes chronic in adults is about 10%. Hepatitis C virus is typically acquired much later in life, mostly through contaminated needles/blood or sexual transmission. Hence, the period of time in which an individual is infected and also exposed to aflatoxin is relatively shorter (F. Wu, personal communication).

Acute aflatoxicosis is characterized by haemorrhage, acute liver damage, oedema and death and results from consumption of extremely high doses of aflatoxin. Chronic aflatoxin exposure is associated with immunosuppression (Turner et al. 2003; Williams et al. 2004; Jiang et al. 2005; Jiang et al. 2008), increased susceptibility to liver cancers in synergy with hepatitis B virus infection, liver cirrhosis and child growth impairment (Gong et al. 2002; Gong et al. 2004; Turner et al. 2007; Pitt et al. 2012).

While it has been stated that 40% of the productivity lost to diseases in developing countries is due to diseases exacerbated by aflatoxins (Bankole and Adebanjo 2004), this is an overly simplistic statement that disregards the complexity of the disease process. However, the statement tries to capture the severity of the problem of aflatoxin-contaminated foods, their consumption and health impacts on both people and livestock throughout much of the developing world.

In recent years, hundreds of aflatoxicosis cases in Africa have been associated with consumption of contaminated home-grown maize (Azziz-Baumgartner et al. 2005). In high concentrations, consumption of aflatoxins, particularly aflatoxin B_1, can cause rapid death (Beed 2013). Acute aflatoxicosis in East Africa has become an epidemic, particularly in arid and semi-arid areas. Dietary exposure in East Africa is highest from common foods made from maize, such as ugali in Kenya and posho in Uganda.

To limit aflatoxin exposure, over 100 nations worldwide have set maximum tolerated limits of aflatoxin in food (CAST 2003). These standards offer public health protection in industrialized nations but have little effect in less developed countries for several reasons. First, the food consumed from subsistence farms, which are widespread in less developed countries, rarely enters any sort of regulatory inspection for aflatoxin (Williams et al. 2004; Strosnider et al. 2006). Second, even if this food did meet the maximum tolerated limit of aflatoxin, many people in less developed countries consume such high levels of maize and groundnut products, putting them at risk of chronic aflatoxin exposure (Shephard 2008). Third, less developed countries that attempt to export maize and nuts abroad may find their export markets severely jeopardized by strict aflatoxin standards, resulting in potential countervailing risks of exporting the best foods and keeping the worst for domestic consumption (Wu 2004).

Therefore, it is estimated that about five billion people worldwide suffer from uncontrolled exposure to aflatoxins (Strosnider et al. 2006). Aflatoxin-associated health effects pervade sub-Saharan Africa and East Asia. These effects could be mitigated through effective use of current agricultural knowledge and public health practice (Khlangwiset and Wu 2010). The discussion of this problem and its remedies must include the underlying question of food insufficiency and more general economic challenges in developing countries (Strosnider et al. 2006). Moreover, developing countries face a range of public health risks and limited resources to manage them. Therefore, there is a pressing need for public health evidence and a risk-based approach to aflatoxin mitigation.

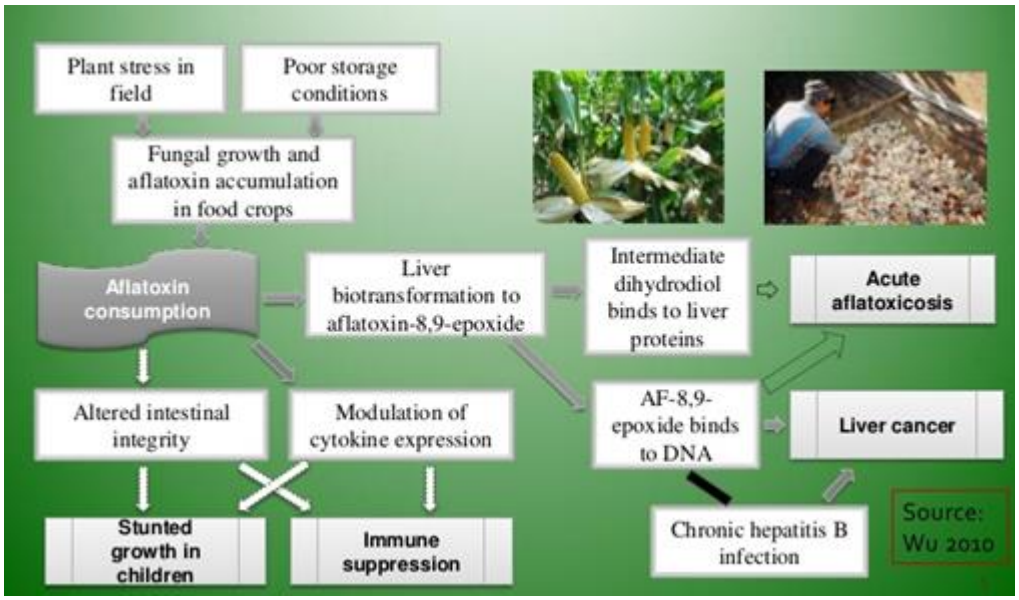
2. Methods

A systematic literature review was undertaken following 'Preferred Reporting Items for Systematic Reviews and Meta-Analyses' guidelines to capture information on aflatoxin prevalence, risk factors and control options and costs to support risk maps and evidence around costs and controls. Twenty-three databases were searched using a combination of the 'Medical Subject Headings' terms: mycotoxin, aflatoxin, Africa, sub-Saharan Africa, prevalence, maize, sorghum, groundnut, peanut, milk, fish, dairy, hepatitis, diet, climate, global warming, weather, drought, stunting, wasting, malnutrition, risk factors, exposure, proxies, outcomes, hepatocellular carcinoma, liver cancer, control, insect, pest, jaundice and cirrhosis. An initial 2700 papers were identified. After screening, 543 were retained for data extraction, included in this report and compiled into a prevalence database by region and commodity. The prevalence database was then converted into risk maps. Geographic Positioning System coordinates for the location of samples collected in each study included in the database were mapped and the maps included in this report.

3. Health

The major source of human and livestock exposure to aflatoxins is consumption of contaminated foods and feeds (Pitt et al. 2012). Aflatoxin contamination is widespread throughout Africa as well as several countries in Asia. Aflatoxins have been detected in maize (corn), groundnuts (peanuts), millet, wheat, rice, oats, barley, sorghum, teff (an African cereal), soybeans, beans and peas, edible oils, nuts (other than groundnuts), traditional plant remedies, spices, sesame seeds, dried fruit, dried vegetables, melons, eggs, milk (cow, goat, sheep, camel and buffalo), cheese, meat and fish in tropical and sub-tropical regions worldwide.

Exposure to aflatoxin leads to, or is associated with, several health-related conditions including acute and chronic aflatoxicosis, aflatoxin-related immunosuppression, liver cancer and liver cirrhosis, and nutritional-related problems such as stunted growth in children (Figure 1). Exposure to aflatoxin may also compound pre-existing health concerns.



Source: Wu et al. (2011b).

Figure 1: Influence diagram of aflatoxin and its health effects.

Aflatoxin exposure can be measured in two ways: through an analysis of prepared foods in combination with consumption data or through biological markers of exposure from blood or urine samples that are obtained and analysed for the presence of aflatoxin derivatives. Possibilities to minimize biological exposure include (i) chemoprotection through the use of drugs and dietary supplements that detoxify aflatoxin and (ii) enterosorptive food additives that bind to the toxin and render the aflatoxin biologically unavailable to the body.

3.1. Acute aflatoxicosis

Acute aflatoxicosis is associated with sporadic outbreaks caused by the consumption of highly contaminated foods. Early symptoms of acute aflatoxicosis include diminished appetite, malaise and low fever. Later symptoms, which include vomiting, abdominal pain and hepatitis, can signal potentially fatal liver failure (USAID 2003). Severe acute liver injury with high morbidity and mortality has been associated with high-dose exposures to aflatoxins (Chao et al. 1991). Ingestion of 2–6 mg of aflatoxin per day by adults for a month can cause acute hepatitis and death (Patten 1981).

Acute aflatoxicosis in animals was first documented in 1960 after more than 100,000 turkeys died following an outbreak in the United Kingdom. A survey during the outbreak showed an association

with feeds, namely, Brazilian peanut meal. It was discovered that this peanut meal was highly toxic to poultry and ducklings with symptoms typical of Turkey X disease. Speculations made during 1960 regarding the nature of the toxin suggested that it might be of fungal origin. In fact, the toxin-producing fungus was identified as *A. flavus* in 1961 and the toxin was given the name 'aflatoxin' by virtue of its origin (***A****spergillus* ***fla****vus* toxin) (Pitt et al. 2012).

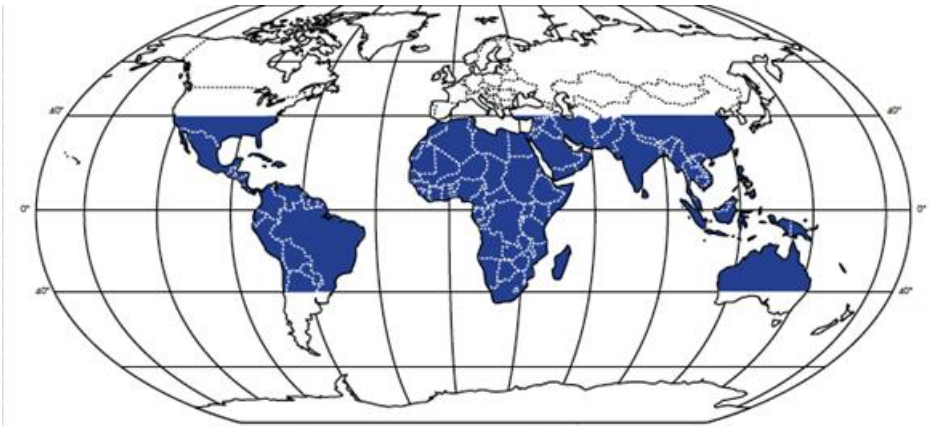
Kenya has experienced several recurrences of acute aflatoxicosis in humans and has recorded hundreds of deaths in the last four decades. The largest reported outbreak of aflatoxicosis to date occurred in Kenya in 2004 where 317 cases and 125 deaths were reported with significant mortality among domesticated livestock and widespread socio-economic impact (Azziz-Baumgartner et al. 2005; Lewis et al. 2005; Wagacha and Muthomi 2008). Other documented fatal aflatoxicosis outbreaks have been reported in Western India in 1974: 397 cases and 106 reported deaths (Krishnamachari et al. 1975); Nigeria in 2005: more than 100 deaths (Afla-guard 2005); Kenya in 1981: 20 cases (Ngindu et al. 1982) and Kenya in 2005: 80 cases and 30 reported deaths, and nine deaths in 2006 (Wagacha and Muthomi 2008). Probst et al. (2010) found that in the eastern region of Kenya, where the aflatoxicosis outbreaks happen, *A. flavus* found in maize samples was primarily the *S* strain, which produces more aflatoxins.

In the 2004 outbreak in Kenya, concentrations of aflatoxin B_1 in maize were found to be as high as 4400 parts per billion (ppb), which is 220 times greater than the 20 ppb limit for food suggested by Kenyan authorities (Azziz-Baumgartner et al. 2005). During this outbreak, children younger than 14 years (51% of the child population) had a greater predisposition to aflatoxicosis risk (Obura 2013).

A study by Azziz-Baumgartner et al. (2005) reported that males were more likely to die from aflatoxicosis, in spite of eating similar quantities of maize as females. One hypothesis for males being at higher risk of dying from aflatoxicosis is alcohol consumption. This study and that of Ngindu et al. (1982) reported that aflatoxicosis patients reported the death of their dogs before developing aflatoxicosis themselves. Therefore, in future, reports of deaths of dogs may warn public health officials of a potential aflatoxin contamination in the food supply.

3.2. Chronic aflatoxicosis

Chronic aflatoxicosis is caused by long-term exposure to low to moderate levels of aflatoxins in the food supply. It is estimated that more than five billion people in developing countries worldwide are at risk of chronic aflatoxin exposure through contaminated foods (Figure 2).



Source: Williams et al. (2004).

Figure 2: Areas and populations at risk of chronic exposure to uncontrolled aflatoxin contamination.

Chronic exposure to moderate or even low levels of aflatoxin has been linked to development of liver cancer. In a study of Gambian liver cirrhosis patients, those that had eaten groundnuts at least once daily over the previous two months were classified in the high aflatoxin intake group (Kuniholm et al. 2008). The moderate aflatoxin intake group had eaten groundnuts 2–6 times a week and the low aflatoxin intake group had eaten groundnuts once or less a week. The occurrence of groundnut consumption was self-reported in this study. As to what constitutes moderate and low levels of aflatoxin intake is yet to be standardized and it is left to the publishing authors to create their own criteria.

In addition to the links to liver cancer, chronic aflatoxin exposure has been associated with impaired growth and immunosuppression in young West African children (Turner et al. 2003; Gong et al. 2004). Immunosuppression predisposes humans and animals to many secondary infections by fungi, bacteria and viruses (McLean 1995). Thus, chronic aflatoxin exposure could exacerbate the burden of disease in already vulnerable populations.

Chronic aflatoxin exposure is evident from the presence of aflatoxin M_1 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).

However, data regarding other potential health effects of chronic aflatoxin exposure are scarce, resulting in a significant limitation of current research (Strosnider et al. 2006). Primary limitations for conducting this research include difficulties in defining clinical outcomes in often remote or resource-constrained environments and difficulty in accurately assessing aflatoxin exposure.

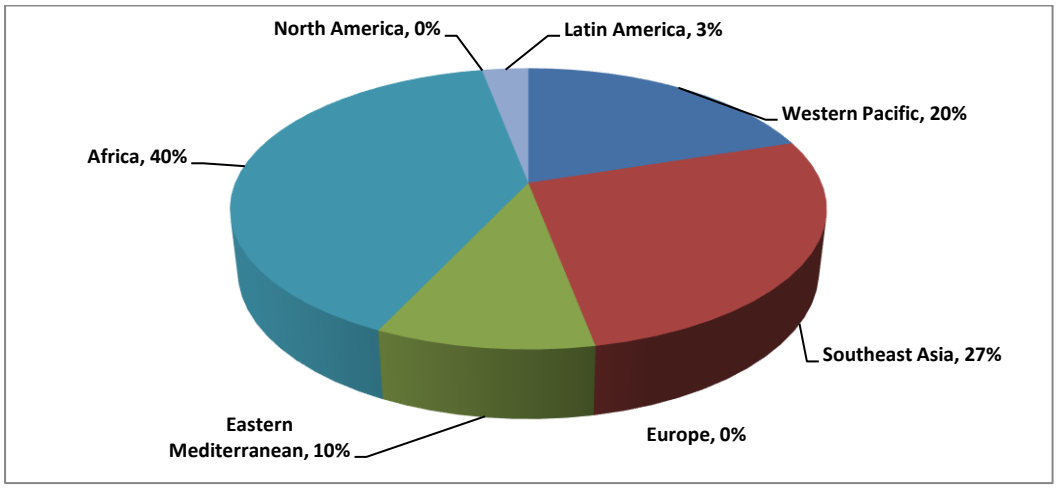
When discussing disease processes in the body, correlation between two variables does not necessarily imply that one causes the other. This is particularly important in regards to aflatoxin exposure and health outcomes because research is still ongoing and there are many variables to tease apart. Much of the research linking aflatoxin consumption and health outcomes relies on the patients' knowledge of what they consumed, sometimes much later. Additionally, given the burden of disease, compromised immune status and widespread malnutrition of many people living in developing countries, elucidating associations between aflatoxin exposure and health consequences is a high priority. Additional factors such as consumption patterns, age, gender and environmental factors will also contribute to different outcomes. Developing countries face a range of public health risks and limited resources to manage them. In order to determine the best intervention measures, a better understanding of the risk of varying levels of aflatoxin exposure and disease outcome is warranted.

3.3. Liver cancer

A large body of experimental, clinical and epidemiological evidence has defined aflatoxin as one of the most potent naturally occurring liver cancer-causing agents. Globally, it is estimated that aflatoxin exposure contributes to 4.6–28.2% of all liver cancer cases, most of which occur in sub-Saharan Africa, southwest Asia and China (Figure 3), the regions with the highest aflatoxin exposure. Each year 550,000–600,000 new cases of liver cancer are recorded worldwide, and of these, approximately 25,200–155,000 are attributable to aflatoxin exposure (Liu and Wu 2010). According to the World Health Organization (WHO), liver cancer is the third leading cause of cancer deaths globally. Approximately 83% of liver-related deaths in East Asia and sub-Saharan African are due to liver cancer (USAID 2003).

Epidemiological studies of human populations exposed to diets naturally contaminated with aflatoxins reveal an association between the high incidence of liver cancer in Africa and elsewhere

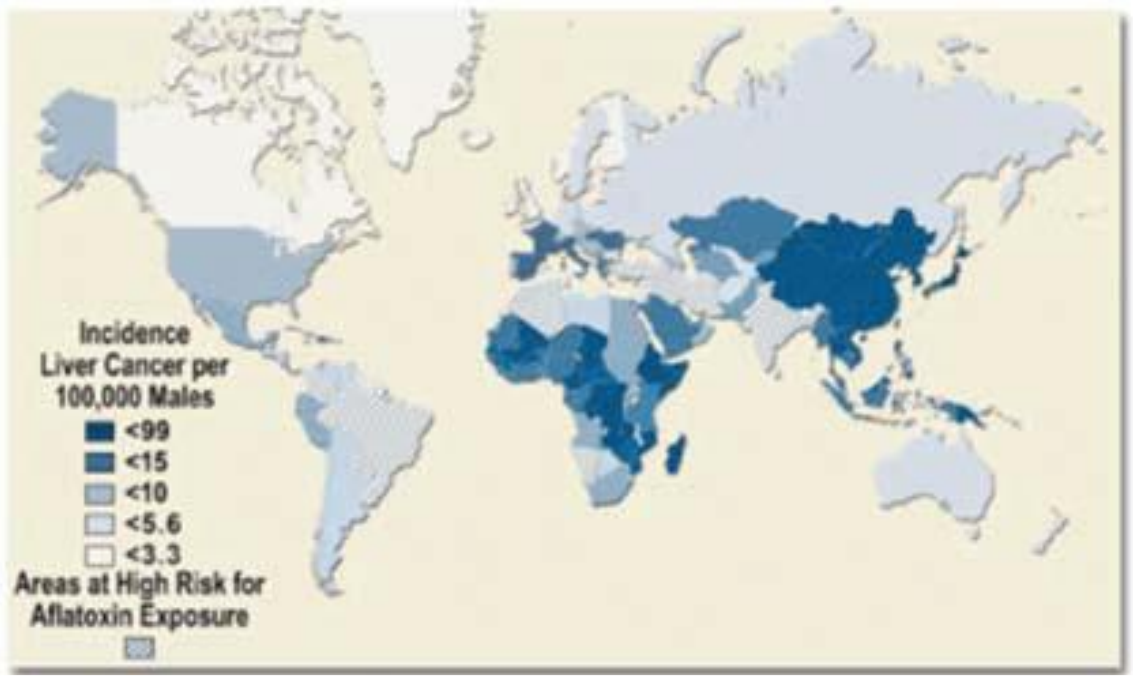
and dietary intake of aflatoxins (MRC 2006). Hepatitis B virus infection and chronic aflatoxin exposure places a person at a risk 30 times greater of developing liver cancer than people who are hepatitis B virus negative. Sub-Saharan African and Asian populations that have endemically high rates of infection of hepatitis B virus and hepatitis C virus are, therefore, likely to have a significantly increased disease burden from liver cancer (USAID 2003).



Source: USAID (2003).

Figure 3: Distribution of liver cancer attributable to aflatoxin.

The global disease burden of aflatoxin is influenced greatly by the geographic and temporal incidence patterns of liver cancer. Figure 4 depicts the correlation between high liver cancer rates and high risk of chronic exposure to aflatoxin.



Source: Williams et al. (2004).

Figure 4: Correlation between high liver cancer rates and high risk of chronic exposure to aflatoxin.

The International Food Policy Research Institute (IFPRI)-led Aflacontrol project quantified the impact of aflatoxin-induced liver cancer into disability-adjusted life years (DALYs).The results are presented in Table 1.

Table 1: Disability-adjusted life years from aflatoxin-induced liver cancer, by world region

World region	Disability-adjusted life years annually attributable to aflatoxin-induced liver cancer
Africa	147,940–778,700
North America	143–182
Latin America (including Central America)	8749–65,520
Eastern Mediterranean	10,231–219,960
Southeast Asia	41,600–583,700
Western Pacific	117,260–360,230
Europe	2093–7228

Source: Wu et al. (2011b).

Given the high burden of disease of aflatoxin induced liver cancer, public health interventions to reduce aflatoxin exposure and hepatitis B virus infection are critical. Reducing aflatoxin exposure to non-detectable levels could reduce liver cancer cases in high-risk areas by about 23% (Liu et al. 2012).

3.4. Liver cirrhosis

Worldwide, cirrhosis of the liver is the sixteenth leading cause of death, responsible for hundreds of thousands of deaths each year. People with cirrhosis of the liver are at high risk of developing liver cancer. A study on aflatoxin exposure and the cause of liver cirrhosis in the Gambia found that chronic hepatitis B virus infection and aflatoxin exposure—either separately or in synergy—were the agents most likely responsible for most cirrhosis cases in that West African population (Kuniholm et al. 2008). However, the association between aflatoxin and liver cirrhosis is not as well documented as with liver cancer.

3.5. Reproductive health

It has been hypothesized that aflatoxins have an impact on reproductive health. Globally, one useful indicator of reproductive health is infant birth weight. It is estimated that more than 20 million infants worldwide, representing 15.5% of all births, are born with low birth weight (birth weight less than 2500 g), with 95.6% occurring in developing countries. Additionally, almost a decade after the declaration of the Millennium Development Goals, there has largely been no change in maternal mortality rates and child mortality rates barely decreased by 27% (Shuaib et al. 2010b).

There was no consensus on findings regarding the relationship between aflatoxins and birth weight. While four studies (Abulu et al. 1998; Abdulrazzaq et al. 2002; Abdulrazzaq et al. 2004; Turner et al. 2007) reported a negative correlation between birth weight and aflatoxin levels (with p values ranging from < 0.001 to < 0.05), two studies found this relationship only in female infants ($p < 0.5$) (Vries et al. 1989; Jonsyn et al. 1995a). In a study in the United Arab Emirates, 100% (43 of 43) of neonates born with low birth weight had detectable aflatoxin M_1 in their cord blood but only 55% (68 of 123) of neonates with normal birth weight had detectable aflatoxin M_1 in their cord blood (Abdulrazzaq et al. 2004). One study conducted in Ibadan, Nigeria did not find any correlation between the presence of aflatoxins and birth weight (Maxwell et al. 1994). Similarly, Vries et al. (1989) did not find any correlation between aflatoxins in maternal blood and cord blood and birth weight. Two studies reported the occurrence of stillbirths among women who had significantly higher levels of maternal serum aflatoxins (Lamplugh et al. 1988) or both maternal and neonatal serum aflatoxin (Vries et al. 1989). One study by Sadeghi et al. (2009) in Iran found an association between aflatoxin M_1 concentration in breast milk and length of the infant at birth ($p < 0.01$). Abdulrazzaq et al. (2003) did not find any significant correlation between aflatoxin M_1 and gestational age, postnatal age, gender or clinical condition.

Four studies (Ahmed et al. 1995; Sodeinde et al. 1995; Abulu et al. 1998; Abdulrazzaq et al. 2004) reported findings relating aflatoxin biomarkers and jaundice among newborns. Only one study found that aflatoxin serum levels of infants were a risk factor for neonatal jaundice (odds ratio [OR], 2.68; confidence interval [CI], 1.18–6.10) (Sodeinde et al. 1995). Of the two studies that did not find any statistically significant correlation between aflatoxins and jaundice, one used serum from the neonate (Ahmed et al. 1995) while the other used cord blood (Abdulrazzaq et al. 2004). The fourth study reported that aflatoxins were associated with jaundice in low birth weight babies but did not state whether any association exists between aflatoxins and jaundice in babies of normal weight (Abulu et al. 1998). It is noteworthy that the aflatoxin levels in body fluids vary by season, as was demonstrated by three studies that noted that the frequency of detection of aflatoxins was higher during the wet than the dry season (Lamplugh et al. 1988; Vries et al. 1989; Abulu et al. 1998). This further complicates drawing conclusions on the association between aflatoxin serum levels and disease outcomes.

Due to the high frequency of anaemia in pregnant woman, a study in Ghana investigated associations between anaemia and aflatoxin B_1. The mean aflatoxin-albumin level was 10.9 pg/mg (range = 0.44–268.73 pg/mg); 30.3% of participants were anaemic. The odds of being anaemic increased 21% (OR, 1.21; $p = 0.01$) with each quartile of aflatoxin-albumin reaching an 85% increased odds in the 'very high' compared to the 'low' category (OR, 1.85; CI, 1.16–2.95). This association was stronger among women with malaria and findings were robust when women with evidence of iron deficiency anaemia were excluded. This study found a strong consistent association between anaemia in pregnancy and aflatoxin biomarkers (Shuaib et al. 2010a).

In another study by the same author of socio-demographic determinants of aflatoxin levels in pregnant women, aflatoxin-albumin as well as the percentage of women having high aflatoxin-albumin levels (≥ 11.34 pg/mg; upper quartile) were inversely associated with indices of higher socio-economic status. Higher income, being employed, having one child (versus no children) and having a flush toilet (versus no toilet facilities) were each independently associated with a 30–40% reduced odds of high aflatoxin-albumin levels (Shuaib et al. 2012). Having a flush toilet has no bearing on aflatoxin exposure, but this study points out the impact of socio-economic status and aflatoxin exposure. Studies continue to show that the most vulnerable populations that consume large quantities of staple crops susceptible to aflatoxin contamination are also among the poorest in the world.

One study examined the possible association between aflatoxins and male fertility. Semen from 40% of infertile men had aflatoxins compared to semen from 8% of fertile men. The concentrations of aflatoxins detected in the semen were consistently higher among infertile men compared to the fertile men. Fifty percent of infertile men with high aflatoxin semen levels also showed abnormalities (sperm count, morphology and motility) of their spermatozoa on semen analysis. In comparison, only 10–15% of the fertile men showed comparative abnormalities of spermatozoa (Ibeh et al. 1994).

These studies show the challenges of drawing conclusions about cause and outcome from cross-sectional data on aflatoxin exposure. It is clear that aflatoxin consumption does not directly cause having a flush toilet. On the other hand, it is not clear if aflatoxins cause anaemia. Ingestion of aflatoxin at very high levels (above 6000 mg) results in liver failure and death within 1–2 weeks of exposure (Obura 2013). An increasing body of evidence suggests that aflatoxins modulate the immune system and may lead to stunted growth in children. While aflatoxin exposure is associated with changes in markers of human immune systems, how these changes actually correlate to

disease outcomes is less clear and was beyond the scope of the studies. Furthermore, at the moment, because of the relatively small number of epidemiological studies undertaken and the limited nature of dose-response relationships, it is not possible to definitively link an aflatoxin dose with a particular risk of stunting in a population. However, while causality has not yet been confirmed, the body of evidence consistently shows an association between aflatoxin exposure and growth impairment in children (Wu 2013).

3.6. Childhood growth performance

Childhood growth performance is usually measured by one or more of three indicators: height-for-age, weight-for-age and weight-for-height. Based on WHO definitions, children whose height-for-age, weight-for-age and weight-for-height Z-scores are two standard deviations or more below the WHO growth standards are considered to be stunted, underweight and wasted, respectively. Wasting is an indicator of deficits in tissue and fat mass, which may be caused by acute malnutrition, whereas stunting is regarded as an indicator of chronic malnutrition. The prevalence of severe wasting decreases by 24 months of age, whereas stunting prevalence increases by age and reaches a plateau at 24–36 months (Black et al. 2008). Once established, stunting and its effects usually last for years. Children who are stunted often develop long-term development and cognitive problems and are more vulnerable to infectious diseases (Ricci et al. 2006). Globally, 26% of children under five years of age were stunted in 2011, roughly 165 million children worldwide. But this burden is not evenly distributed around the world. Sub-Saharan Africa and South Asia are home to 75% of the world's stunted children (UNICEF 2013). Globally, 21% of deaths and DALYs in children aged five years and under are estimated to be attributed to stunting, severe wasting and intra-uterine growth restriction (Black et al. 2008). It has been estimated that children with a weight-for-age Z-score of -1 to -2 are twice as likely to die from diarrhoeal diseases compared to children with normal weight, whereas children with weight-for-age Z-scores ranging from -2 to -3 are five times as likely to die. Additionally, 52% of pneumonia deaths in children aged five years and under are associated with low body weight (Caulfield et al. 2004).

There is a growing body of literature trying to link aflatoxin exposure with impaired growth in children (Gong et al. 2003; Egal et al. 2005). This impaired growth is strongly correlated with the change from breastfeeding to solid foods. Maize is widely used 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.

3.6.1. Stunted and underweight children

A study in Benin and Togo found that stunted and underweight children had, on average, 30–40% higher levels of aflatoxin-albumin in their blood than children with normal body weight (Gong et al. 2002). Aflatoxin-albumin levels increased with age until three years of age. This trend reflected the transitioning of children from breastfeeding to weaning and post-weaning foods. Children who were completely weaned had higher levels of aflatoxin-albumin than breastfed or partially breastfed children (Gong et al. 2003; Gong et al. 2004). Clear dose-response relationships were found between mean aflatoxin-albumin levels and lower height-for-age and weight-for-age Z-scores. Children who were stunted had 30–40% higher mean aflatoxin-albumin levels compared to non-stunted children.

Another study in Benin and Togo investigated aflatoxin exposure in children around the time of weaning and correlated the data with food consumption, socio-economic status, agro-ecological zone of residence and anthropometric measures (Gong et al. 2003). Blood samples from 479 children (aged 9 months to 5 years) from 16 villages in four agro-ecological zones were assayed for aflatoxin-albumin as a measure of recent past (2–3 months) exposure. Aflatoxin-albumin adducts

were detected in 99% (475/479) of children (geometric mean, 32.8 pg/mg; 95% CI, 25.3–42.5). Adduct levels varied markedly across agro-ecological zones with mean levels being approximately four times higher in the central than in the northern region. The central region has two maize growing seasons, compared to one season in the north, and higher rainfall and humidity than the northern region. The aflatoxin-albumin level increased with age up to three years and, within the 1–3 year age group, was significantly (p = 0.0001) related to weaning status. Weaned children had approximately two-fold higher mean aflatoxin-albumin adduct levels (38 pg/mg) than children receiving a mixture of breast milk and solid foods, after adjustment for age, sex, agro-ecological zone and socio-economic status. A higher frequency of maize consumption, but not groundnut consumption, by the child in the preceding week was correlated with higher aflatoxin-albumin adduct level. In this study, aflatoxin exposure among these children was widespread (99%) and growth faltering associated with high blood aflatoxin-albumin adducts. Children in these two categories had 30–40% higher mean aflatoxin-albumin levels than the remainder of the children and strong dose–response relationships were observed between aflatoxin-albumin levels and the extent of stunting and being underweight.

However, another study done in Benin and Togo found high aflatoxin-albumin adduct levels were correlated with high prevalence of *A. flavus* and aflatoxin in groundnut, but significance was weak after adjustment for weaning status, agro-ecological zone and maternal socio-economic status (p = 0.091 and p = 0.083, respectively). Ingestion of *A. flavus* and aflatoxin was high in certain agro-ecological zones and among the higher socio-economic strata due to higher frequencies of groundnut consumption. Contamination of groundnuts was similar across socio-economic and agro-ecological boundaries. In conclusion, dietary exposure to aflatoxin from groundnut was less than from maize in young children from Benin and Togo (Egal et al. 2005).

A study carried out in the Gambia by Turner et al. (2003) found that elevated aflatoxin-albumin levels were associated with stunting and underweight among children aged 6 to 9 years. The study detected aflatoxin-albumin adducts in 93% of sampled children and provided evidence that immunoglobulin A in saliva may be reduced because of aflatoxin exposure (Turner et al. 2003). The study confirmed that children in rural areas of the Gambia are frequently exposed to high levels of aflatoxin.

A study of Gambian infants found a strong correlation between maternal aflatoxin exposure during pregnancy and growth in the first year of life. Aflatoxin-albumin in maternal blood was a strong predictor of both weight (p = 0.012) and height (p = 0.044) gain, with lower gain in those with higher aflatoxin exposure (Turner et al. 2007). While the correlation between aflatoxin consumption and stunting and underweight children is yet to be elucidated, the proportion of childhood growth stunting is directly correlated with the proportion of the population living below the national poverty line and inversely correlated with gross domestic product per capita (Khlangwiset and Wu 2010).

3.6.2. Wasting and weaning foods

A cross-sectional study in Kenya found significant association (p = 0.002) between aflatoxin exposure and wasting. Aflatoxins were also more frequently detected in the flour fed to stunted and underweight children compared to that fed to normal children (Okoth and Ohingo 2004). The weaning process in West African countries starts in many cases at early ages, when the children are about 3–6 months old. Up to 50% of children in Makurdi, Nigeria consume pap, a porridge made from maize, as their main weaning food, followed by Cerelac, a commercial infant formula (26.5%) and pap mixed with other food (11%). Weaning foods in West Africa are usually made of maize, groundnuts, sorghum, millet and guinea corn. Likewise, maize is a major weaning food in countries

in East Africa. In Uganda, 89% of children are fed maize porridge regularly. About 24.5% of children aged 3–28 months consume maize porridge seven days a week. Gruels prepared from maize are used as weaning foods in Ethiopia, Kenya and Tanzania. Other staple crops are also used to prepare weaning foods in these East African countries. Some of them include barley and wheat in Ethiopia, sorghum and millet in Kenya and sorghum in Tanzania. Sorghum porridge (nasha) is a traditional weaning food in Sudan (Khlangwiset et al. 2011). Research is still needed to strengthen the evidence for an association between aflatoxin consumption in weaning food and the impact on childhood growth.

Because of multiple routes of exposure beginning in the foetal environment, high percentages of children in various countries have been exposed to aflatoxins, as detected in multiple studies. About 85–100% of children in African countries, such as the Gambia, Guinea, Kenya, Benin, Togo and Senegal, have either detectable levels of serum aflatoxin-albumin or urinary aflatoxins (Wild et al. 1990; Wild et al. 1993; Turner et al. 2000; Gong et al. 2002; Gong et al. 2003; Turner et al. 2003; Gong et al. 2004; Turner et al. 2007; Polychronaki et al. 2008). None of these studies suggested non-negligible aflatoxin exposure among the populations of children studied. However, many of the studies were done among populations of children in which one would expect to find aflatoxin exposure such as rural areas where consumption of susceptible staple crops is high and poverty limits the food quality, quantity and variety for the children.

Among the risk factors associated with growth impairment, aflatoxin emerges as playing a potentially important contributory role. The weight of evidence linking aflatoxins with growth impairment has been increasing over the last five decades of research. One critical piece of information that is currently unavailable is a mechanism by which aflatoxin causes growth impairment in humans and animals. If such a mechanism could be elucidated, then the weight of evidence linking aflatoxin with growth impairment would become even stronger (Khlangwiset et al. 2011).

3.6.3. Breast milk

Several studies have also found aflatoxin B_1 and aflatoxin M_1 in excreted breast milk samples (Jonsyn et al. 1995b; Polychronaki et al. 2006; Polychronaki et al. 2007; Sadeghi et al. 2009; Gürbay et al. 2010; Tchana et al. 2010; El-Tras et al. 2011; Afshar et al. 2013), further compounding aflatoxin exposure and its health risks in breastfeeding infants and children. On the whole, there were significant differences in aflatoxin contamination of breast milk between studies that were conducted in developing countries and those in developed countries. While breast milk samples from three studies conducted in developed countries had contamination rates ranging from 0% in France to 5% in Italy (mean concentration 55.35 ng per litre), 34–95% of breast milk samples from the studies in developing countries were contaminated with aflatoxins (Lamplugh et al. 1988; Saad et al. 1995). About 30–60% of breast milk samples from Sudanese (Coulter et al. 1984), Kenyan (Maxwell et al. 1989), Ghanaian (Lamplugh et al. 1988; Maxwell et al. 1989) and Egyptian (Polychronaki et al. 2006; Polychronaki et al. 2007) mothers contained detectable levels of aflatoxins. In Sierra Leone, 88% (99/113) of breast milk samples from mothers contained detectable levels of aflatoxins (Jonsyn et al. 1995b). However, only 11% of breast milk samples from Zimbabwean mothers (Wild et al. 1987) and 5% of breast milk samples from mothers in Cameroon (Tchana et al. 2010) were aflatoxin M_1 positive. Levels of aflatoxin M_1 in breast milk samples taken in Ankara, Turkey ranged from 60.0–299.99 ng/l and aflatoxin B_1 from 94.5–4123.8 ng/l (Gürbay et al. 2010).

Levels of aflatoxin M_1 in breast milk were significantly associated with cereal consumption (Sadeghi et al. 2009), consumption of raw milk (El-Tras et al. 2011), high corn oil consumption,

obesity, number of children and early lactation stage (Polychronaki et al. 2006). A study done in Egypt found that the average daily exposure of newborns to aflatoxin M_1 via consumption of maternal breast milk was 52.7 ng (El-Tras et al. 2011).

There was a significant difference ($p < 0.0001$) in aflatoxin M_1 exposure between infants fed on maternal breast milk and those fed on formula milk, with breastfeeding being considered a risk factor for aflatoxin M_1 exposure in early infancy (Table 2). It is worthwhile to note that the detectable levels are inconsistent across countries, which implies that the marker may be unreliable with linking to growth impairment. However, these studies imply that lactating mothers are exposed to aflatoxin and can transfer it to their babies through breastfeeding.

Table 2: Comparison of aflatoxin levels in breast milk, raw cow's milk and weaning foods, by country

Country	Population	Aflatoxin M_1 (µg/kg)		Aflatoxin B_1(µg/kg)		Source
		Range	Mean	Range	Mean	
Brazil	42 raw milk samples	0.3–1.97				Sassahara et al. (2005)
Brazil	50 lactating mothers	0.024	0.024			Navas et al. (2005)
Egypt	50 raw milk samples	0.01–0.25				Motawee et al. (2009)
Egypt	388 lactating mothers	0.00056–0.5131	0.000135			Polychronaki et al. (2006)
Ghana	264 lactating mothers	0.02–1.816				Lamplugh et al. (1988)
Iran	111 raw milk samples	0.15–0.28				Kamkar (2005)
Iran	80 samples of milk-based cereal weaning foods			0.003–0.035	0.0168	Oveisi et al. (2007)
Iran	160 lactating mothers	0.0003–0.0267	0.00082			Sadeghi et al. (2009)
Iran	98 raw bulk tank samples	0.0003–0.392	0.039			Tajkarimi et al. (2007)
Iran	186 raw milk samples	0.010–0.410	0.0434			Ghiasian et al. (2007)
Iran	88 raw milk samples	0.013–0.394	0.052			Fallah et al. (2011)
Iran	319 raw bulk tank samples		0.057			Tajkarimi et al. (2008)
Iran	75 raw milk samples	0.005–0.05	0.0601			Rahimi et al. (2010)
Italy	341 raw milk samples	0.05				Decastelli et al. (2007)
Italy	231 lactating mothers	0.194	0.194			Turconi et al. (2004)
Italy	82 lactating mothers		0.05535			Galvano et al. (2008)
Nigeria	22 raw milk samples	2.04–4.0				Atanda et al. (2007)
Nigeria	7 weaning foods	4.6–530		181.6–4806[a]		Oluwafemi and Ibeh (2011)
Nigeria	48 maize-based weaning gruels			0.142–6.516[b]		Oyelami et al. (1996)
Sierra Leone	113 lactating mothers	0.2–99	0.8			Jonsyn et al. (1995b)
Sudan	99 lactating mothers		19			Coulter et al. (1984)
Sudan	44 bulk milk samples	0.22–6.9	2.07			Elzupir and Elhussein (2010)
Thailand	11 lactating mothers	0.039–1.736	0.664			El-Nezami et al. (1995)
Thailand	240 raw and bulk tank samples	0.05–0.101	Winter: 0.084 Rainy season: 0.073 Summer: 0.053			Ruangwises and Ruangwises (2010)
Uganda	5 baby food products			1–20		Ismail et al. (2008)
UAE	445 lactating mothers	0.2–0.3				Saad et al. (1995)
Zimbabwe	42 lactating mothers	0–50				Wild et al. (1987)
Zimbabwe	54 lactating mothers		3.6			Nyathi et al. (1989)

Note: UAE = United Arab Emirates.

[a]Aflatoxin B_2: 103–8290 µg/kg

[b]Total aflatoxin

Aflatoxin levels vary widely between countries and commodities. It should be noted that very high levels of aflatoxins were found in two of the three studies that analysed customary weaning foods in their countries. In Egypt, Italy, Sudan and Thailand, breast milk from lactating mothers had higher levels of aflatoxins compared to sampled raw cow's milk. In Brazil and Iran, raw cow's milk had higher levels of aflatoxins than breast milk from lactating mothers. Given the small number of

studies done in each of the countries and the seasonal variation in aflatoxin levels, it is hard to draw any concrete conclusions on whether cow's milk or human breast milk poses a higher risk of aflatoxin exposure to infants and weaned children. However, given the high contamination levels of cereal-based weaning foods in Nigeria and Uganda (countries with warm, humid climates that favour *Aspergillus* growth), the risk of aflatoxin consumption in cereal-based weaning foods seems particularly high. Additional studies to determine the extent of contamination in popular weaning foods is definitely warranted.

3.7. Kwashiorkor

Aflatoxin exposure has also been suggested as a causal or aggravating factor for kwashiorkor, a form of protein-energy malnutrition (Ramjee et al. 1992; Tchana et al. 2010) and chronic hepatomegaly (Gong et al. 2012) in African children. Because kwashiorkor reduces the capacity of the liver to detoxify aflatoxins, much higher aflatoxin levels have been found in the blood, urine and livers of children with the disease than in similar age-matched children (Hendrickse et al. 1983; Hendrickse 1984).

The number of children suffering from kwashiorkor at hospitals in Durban, South Africa has risen since 1992. These cases of kwashiorkor, marasmus (severe underweight) and underweight that were reported during this period correlated with findings of impaired liver function (USAID 2003). Researchers have suggested that aflatoxins may play a role in the pathogenesis of kwashiorkor (Fapohunda 2011). However, children prone to kwashiorkor might also be prone to eating the type of foods which are likely to have higher levels of aflatoxins. The association between aflatoxin exposure and kwashiorkor, if any, has yet to be proven.

3.8. Immunosuppression

Research into links between aflatoxin exposure and immunosuppression is still ongoing. In a recent study in Ghana, higher levels of aflatoxin B_1-albumin adducts in plasma were associated with lower percentages of certain leukocyte immunophenotypes (Jiang et al. 2005). A study of Gambian children found an association between serum aflatoxin-albumin levels and reduced salivary secretory immunoglobulin A levels because of dietary levels of aflatoxin exposure. In a multivariable analysis, secretory immunoglobulin A was markedly lower in children with detectable levels of aflatoxin-albumin compared to those with non-detectable levels (50.4 µg/mg protein [95% CI, 48.0–52.8] and 70.2 µg/mg protein [95% CI, 61.1–79.2], respectively; $p < 0.0001$) (Turner et al. 2003).Given the high burden of infection-related mortality throughout the developing world, further investigation of the immune effects of aflatoxin exposure in children is merited.

3.9. Links to HIV and tuberculosis

New research has linked high aflatoxin levels to an increased risk of developing tuberculosis in human immunodeficiency virus (HIV)-positive individuals (Keenan et al. 2011). Hypotheses of the link between HIV infection and aflatoxin exposure suggest two possible routes: (1) HIV infection decreases the levels of antioxidant nutrients that promote the detoxification of aflatoxin or (2) the high degree of co-infection of HIV-infected people with hepatitis B also increases the biological exposure to aflatoxin (Williams et al. 2004). However, this research is still in its early stages and more work is needed to determine whether aflatoxin consumption is correlated with tuberculosis infection. It is possible that poverty is associated with both tuberculosis and eating foods contaminated with aflatoxins and there is no link between aflatoxins and tuberculosis.

In a study done on HIV-positive Ghanaians, hazard ratios for developing symptomatic tuberculosis were significantly higher for those in the highest aflatoxin-albumin quartile (hazard ratio 3.30; 95% CI 1.34–8.11) compared to those in the lowest quartile. Those with the highest levels of aflatoxin-

albumin from dietary intake have an increased hazard of symptomatic tuberculosis but not malaria, hepatitis B virus or pneumonia (Keenan et al. 2011). However, these findings were based on analysis of physician findings and there is no mention of whether tuberculosis, malaria, hepatitis B virus or pneumonia were laboratory confirmed. Additionally, the sample size of the study was small (n = 141) and the patients in the study were on a variety of medications, including anti-retroviral drugs. It is possible that these drugs may have interfered with the metabolism of aflatoxins, influencing aflatoxin-albumin levels in these patients. As stated previously, there may be confounding factors rather than correlation between aflatoxin exposure and symptomatic tuberculosis.

Another recent study concluded that the frequency of HIV transmission is positively associated with maize consumption in Africa. While the article suggested that improvements in the quality of maize may avoid up to 1 million transmissions of HIV annually (Williams et al. 2010), there was no clear evidence to support anything more than a connection between maize consumption and the frequency of HIV transmission. In 2010, HIV ranked as the top cause of DALYs in sub-Saharan Africa with 15,782,000 DALYs recorded (Ortblad et al. 2013). Further research is still needed into the correlation between consumption of aflatoxin- or multiple mycotoxin-contaminated maize and the frequency of HIV transmission. Once again, poverty may be a confounding factor in the association between maize consumption and HIV transmission.

In Ghana, a study to investigate the possible interaction of aflatoxin and HIV on immunosuppression found that among both HIV-positive and HIV-negative participants, high aflatoxin-albumin was associated with lower perforin expression on CD8+ T-cells ($p = 0.012$). HIV-positive participants with high aflatoxin-albumin had significantly lower percentages of CD4+ T regulatory cells (Tregs; $p = 0.009$) and naive CD4+ T-cells ($p = 0.029$) compared to HIV-positive participants with low aflatoxin-albumin. In addition, HIV-positive participants with high aflatoxin-albumin had a significantly reduced percentage of B-cells ($p = 0.03$) compared to those with low aflatoxin-albumin (Jiang et al. 2008).

These results suggest that high aflatoxin-albumin accentuates some HIV-associated changes and may facilitate HIV-associated immune hyper-activation and lead to more severe disease. However, there are many possible confounding issues such as poverty, burden of other diseases and nutritional status.

In another study in Ghana, HIV-infected participants had significantly higher aflatoxin-albumin levels (median for HIV-positive and HIV-negative participants was 0.93 and 0.80 pmol/mg albumin, respectively; $p < 0.01$) and significantly lower levels of vitamin A (-16.94 µg/dL; $p < 0.0001$) and vitamin E (-0.22 mg/dL; $p < 0.001$) (Obuseh et al. 2011). For the total study group, higher aflatoxin-albumin was associated with significantly lower vitamin A (-4.83 µg/dL for every 0.1 pmol/mg increase in aflatoxin-albumin). People infected with hepatitis B virus had significantly lower vitamin A (-5.66 µg/dL; $p = 0.01$). Levels of vitamin A and vitamin E were inversely associated with HIV viral load ($p = 0.02$ for both) and low levels of vitamin E were associated with lower CD4 counts ($p = 0.004$).

The finding of the significant decrease in vitamin A associated with aflatoxin-albumin suggests that aflatoxin exposure significantly compromises the micronutrient status of people who are already facing overwhelming health problems, including HIV infection. However, the study did not control for the consumption of foods rich in vitamin A, such as confounding factors where people prone to eating aflatoxin-contaminated foods are also prone to not eating foods rich in vitamin A because of poverty.

4. Diagnostics

Aflatoxins are difficult to detect because they are dangerous at very low levels and they are not distributed evenly in foods or feeds. Since aflatoxins cannot be completely prevented in crops, regulations are needed to prevent highly contaminated crops from entering the food chain. However, regulations are not enough. In Kenya, for example, where the vast majority of crops are sold in the informal market, regulations regarding aflatoxins are enforced only in the formal market, leaving most of the population, especially the poorest portion, unprotected. The same situation occurs for animal feeds, where only the formal market chain has tests imposed on them. Generally, the difficulty of obtaining a representative sample is recognized as the major cause of insecurity in aflatoxin testing. Tests seek to find very small amounts of aflatoxins. Most standards are expressed in ppb. Finding one ppb of aflatoxin is the equivalent of detecting one second in 32 years or of finding seven people in the population of the world.

Another problem is that aflatoxins are often not distributed evenly throughout the material being sampled. As a result, repeated tests on the same cereals or nuts often give different results. Moulds do not grow uniformly in crops and therefore toxins are unevenly distributed (Turner et al. 2009). In the case of maize and groundnuts, individual kernels and nuts, respectively, can contain very high levels of aflatoxins. Considering that one maize kernel can have 50,000 ppb, just 30 of these kernels would be enough to put a 50 kg bag of maize above the limits of 10 ppb. This means the samples have to find just one in 5000 kernels.

The variability of aflatoxins in crops and the dependence on a large sample size was demonstrated early (Whitaker et al. 1976; Whitaker et al. 1979). In animal feeds, where the crops are milled, there is increased homogeneity but variability will still depend on how sampling, mixing and subsampling are done (Coker et al. 2000).

Sampling errors can lead to different types of problems. False positives occur when samples are rejected when they are actually safe for consumption. This leads to losses for producers and decreases the amount of feed available for animals and food for people. The other type of problem is a false negative (that is, accepting a sample as safe even though its aflatoxin level exceeds the standards); this error exposes people and animals to contaminated food or feed. A number of protocols for sampling of commodities for mycotoxins have been developed, with different risks for consumers (accepting food or feed that should have been rejected) and producers (rejecting food or feed that should have been accepted).

For smallholders, it may be unfeasible to attain the desired amount of animal feed for sampling and less may need to be taken (Pitt et al. 2012). Since milk is more homogenous, it is assumed that there is less variability in testing for aflatoxin M_1 but this has not been proven. However, there is also variability between different laboratories and different laboratory methods. Most methods require a correct extraction and clean-up of samples and the way these are done may have effects on the outcome (Turner et al. 2009).

Highly reliable methods are liquid chromatography–mass spectrometry and high (or ultra) performance liquid chromatography, and these often serve as references for other methods. Total aflatoxins can also be measured by direct fluorescence of purified extracts. Different immunoassays have also been developed, such as enzyme-linked immunosorbent assays, which are easy and cost-effective (Turner et al. 2009; Pitt et al. 2012). There are a number of rapid tests

providing a result over or under a certain limit (agristrips and dipsticks). Table 3 summarizes the characteristics of different types of tests available for detecting aflatoxins.

Table 3: Characteristics of different tests available for detecting aflatoxins

Test	Cost	Complexity	Portable	Detects < 10 ppb	Detects other mycotoxins
LC-MS	$$$	+	No	Yes	Yes
TLC	$	++	No	Yes	No
UPLC	$$	+++	No	Yes	No
DF	$	+++	No	Yes	No
ELISA	$	+++	No	Yes	No
NIRS	$$	+	Yes	No	Potentially
Agristrips and dipsticks	$	++	Yes	Yes	No

Source: Modified from Harvey et al. (2013).

Notes: ppb = parts per billion; LC-MS = liquid chromatography–mass spectrometry; TLC = thin-layer chromatography; UPLC = ultra performance liquid chromatography; DF = direct fluorescence; ELISA = enzyme-linked immunosorbent assay; NIRS = near infrared spectroscopy; $ = low cost; $$ = medium cost; $$$ = high cost; + = low complexity; ++ = medium complexity; +++ = high complexity

While these current technologies can provide an accurate measurement of aflatoxin levels, they are generally expensive, have low throughput and are not readily portable. A promising technology is near infrared spectroscopy, an instrumental technique used to identify substances by measuring their absorption of infrared radiation.

Because of the non-uniform distribution of aflatoxins in crops, it is possible that subsequent tests on the same batch of cereals or oilseeds will give very different results, and there have been several studies to identify robust sampling protocols. Unlike analytical methods, sampling schemes cannot be collaboratively tested; usually a particular sampling plan is proposed, based on statistical consideration of the measured toxin distribution, and thereafter adopted as an official procedure. Due to the difficulties in assessing mycotoxin levels, it is important to have a reference system where local laboratories can be accredited and ring tests performed, both within a country and in a region. This way, the reliability of laboratory results can be established.

5. Consumption and exposure data

The Centers for Disease Control and Prevention (CDC) estimates that 4.5 billion people in the developing world may be chronically exposed to aflatoxins in their diets (CDC 2012). In Kenya, aflatoxin levels were analysed in serum samples previously collected for the 2007 nationwide Acquired Immunodeficiency Syndrome (AIDS) survey.

Seventy-eight percent of the serum samples had detectable levels of aflatoxin B_1-lysine (range = < limit of detection–211 pg/mg albumin; median = 1.78 pg/mg albumin). Aflatoxin exposure did not vary by sex, age group, marital status, religion or socio-economic characteristics. Aflatoxin exposure varied by province ($p < 0.05$); it was highest in Eastern (median = 7.87 pg/mg albumin) and Coast (median = 3.70 pg/mg albumin) provinces and lowest in Nyanza (median = < limit of detection) and Rift Valley (median = 0.70 pg/mg albumin) provinces (Yard et al. 2013).

In addition to province, aflatoxin exposure was also closely related to occupation. Those engaged in crafts and trades (e.g. miners, machine mechanics and food preparers) and elementary occupations (e.g. street vendors, farm hands and construction/manufacturing labourers) had significantly higher aflatoxin adduct levels ($p < 0.05$). Aflatoxin levels were higher in urban (median = 2.23 pg/mg albumin) than in rural participants (median = 1.49 pg/mg albumin; $p < 0.05$).

Participants who reported that they were sick in the past week had higher aflatoxin adduct levels (median = 2.29 pg/mg albumin) than did those who reported not being sick (median = 1.67 pg/mg albumin; $p < 0.01$). Participants seeking health care outside the home in the past three months had higher aflatoxin adduct levels (median = 2.67 pg/mg albumin) than did those not seeking health care outside the home (median =1.59 pg/mg albumin; $p < 0.01$). Participants living in a household in which someone (including themselves) sought outpatient care during the past four weeks had higher aflatoxin adduct levels (median=3.06 pg/mg albumin) than did households with no one seeking outpatient care (median=1.61 pg/mg albumin; $p < 0.01$). While the causal association between aflatoxin exposure and illness is still be elucidated, the findings from this study suggest that aflatoxin exposure is widespread throughout Kenya and poses a public health problem through the country.

During aflatoxicosis outbreaks in Kenya in 2004, 2005 and 2010, geometric mean aflatoxin levels among patients with potential liver dysfunction ranged from 120 to 1200 pg/mg albumin (Azziz-Baumgartner et al. 2005). Although most of the participants in the above mentioned study in Kenya had much lower albumin levels, six participants (1%) had aflatoxin adduct levels above 120 pg/mg albumin. This is notable for three reasons. First, the survey was conducted in 2007, the first year since 2004 that no aflatoxicosis outbreaks were reported in Kenya. Second, maize samples collected from Kenya's Eastern Province had lower aflatoxin levels in 2007 (84% of maize samples contained less than 20 ppb aflatoxin) than in 2006 (only 48% of maize samples contained less than 20 ppb aflatoxin). Third, this study sampled patients regardless of symptoms. The fact that extensive aflatoxin exposure was found during a relatively low-risk year suggests that even during optimal times, aflatoxin remains a persistent health threat in Kenya (Yard et al. 2013). Research suggests that chronic aflatoxin exposure at the levels seen in this study could stunt growth (Gong et al. 2004; Khlangwiset et al. 2011) and impair immunity (Jiang et al. 2005).

An estimate of the health impacts of aflatoxin in Kenya determined that the approximate daily consumption of maize and peanuts is 357 and 44 grams per person, respectively. Therefore, the lifetime average daily dose of aflatoxin in Kenyan adults is 5.2–200 ng/kg body weight per day (Wu et al. 2011b). The wide variance in aflatoxin exposure is probably due to the varying levels of consumption of commonly contaminated crops and varying aflatoxin levels throughout the different seasons and years. The hepatitis B virus prevalence in Kenya is 11–15%. Therefore, the estimated number of aflatoxin-induced liver cancer cases are between 82 and 4080 per year. The estimated DALYs associated with aflatoxin-induced liver cancer in Kenya are 1066–53,040 per year (Wu et al. 2011b). These numbers do not take into account occasional exposure to very high aflatoxin levels. Another interesting finding from this estimate is that average daily maize consumption in rural areas (624 g per person) is 1.77 times higher than that in urban areas (352 g per person), putting rural populations at a higher risk of aflatoxin exposure (Wu et al. 2011b). However, this finding is in contrast to the results from a study of a similar population in Kenya that found urban participants had higher aflatoxin-albumin levels than rural participants (Yard et al. 2013).

In Korea, it was estimated that the probable daily intake of aflatoxin B_1 fell in the range of 1.19–5.79 ng/kg body weight (Park et al. 2004). This range exceeded the estimated provisional tolerable daily intakes in Korea. Rice is the major contributor to the dietary intake of aflatoxin B_1 in Korea.

In China, the mean estimated daily aflatoxin B_1 intakes were 0.218–0.222 ng/kg body weight for children and 0.106–1.08 ng/kg body weight for adults (Ding et al. 2012). The risk of liver cancer was

estimated at 0.003–0.17 cancer cases per year per 100,000 people and 24.7–1273 margins of exposure values.

The traditional diet in most countries of sub-Saharan Africa is predominantly cereals (rice, maize, millet and sorghum) and tubers (cassava, yam and cocoyam). A recent report found acutely toxic aflatoxin levels of 30,000 ppb in boiled groundnuts and 24,000 ppb in roasted groundnuts in Lagos, Nigeria (Thomas et al. 2003). During the 2004 aflatoxicosis outbreak in Kenya, aflatoxin levels in maize products in markets of affected areas varied from 1–46,400 ppb, with over 20 ppb in 55% of maize products, over 100 ppb in 35% and over 1000 ppb in 7% (Lewis et al. 2005). The mean aflatoxin levels in maize from the households of those affected by aflatoxicosis was 354.5 ppb compared to 44.1 ppb in control households (Azziz-Baumgartner et al. 2005). Multiple studies in sub-Saharan Africa have shown widespread exposure (Gong et al. 2002; Gong et al. 2003; Cardwell and Hendy 2004; Gong et al. 2004; Egal et al. 2005; Jolly et al. 2006; Jolly et al. 2007; Polychronaki et al. 2008; Khlangwiset et al. 2011) in addition to high levels of contamination in staple crops and milk (Lewis et al. 2005; Muture and Ogana 2005; Urio et al. 2006; Atanda et al. 2007; Atehnkeng et al. 2008a; Mwihia et al. 2008; Kang'ethe and Lang'a 2009; Mutegi et al. 2009; Elzupir and Elhussein 2010; Daniel et al. 2011; Kamika and Takoy 2011; Ezekiel et al. 2012; Ndung'u et al. 2013; Wagacha et al. 2013). Table 4 lists the studies in published literature of aflatoxin consumption and exposure.

Table 4: Literature review of aflatoxin consumption and exposure data

Country or region	Commodity	Amount	Population	Aflatoxin M_1 intake	Aflatoxin B_1 levels (urine)	Aflatoxin exposure	Aflatoxin-albumin levels (serum)	Source
Africa	Milk			0.1 ng/person per day				WHO (2002)
Africa	Milk	0.04 kg/day						Prandini et al. (2009)
Benin and Togo			Children (9 months to 5 years)				32.8 pg/mg	Gong et al. (2003)
Benin, February			Children (16–37 months)				37.4 pg/mg	Gong et al. (2004)
Benin, June			Children (16–37 months)				38.7 pg/mg	Gong et al. (2004)
Benin, October			Children (16–37 months)				86.8 pg/mg	Gong et al. (2004)
Brazil	Beans		Adults			1.58 ng/kg b.w. per day		Jager et al. (2013)
Brazil	Milk		Adults	0.1 ng/kg b.w. per day				Jager et al. (2013)
Brazil	Milk		Children	1 ng/kg b.w. per day				Shundo et al. (2009)
Brazil	Milk		Adults	0.188 ng/kg b.w. per day				Shundo et al. (2009)
Brazil	Peanuts		Adults			1.56 ng/kg b.w. per day		Jager et al. (2013)
Cameroon			Children, partially weaned (1.5–4.5 years)		1.43 ng/ml			Ediage et al. (2013)
Cameroon			Children, fully weaned (1.5–4.5 years)		2.82 ng/ml			Ediage et al. (2013)
China (southern Guangxi)	Market samples		Adults			6.5–53 ng/kg b.w. per day (aflatoxin B_1)		Shank et al. (1972)
Egypt			Children, 1–2.5 years		13.2 pg/ml			Polychronaki et al. (2008)
Egypt	Maternal breast milk		Infants	52.684 ng/day				El-Tras et al. (2011)
Europe	Milk			6.8 ng/person per day				WHO (2002)
Europe	Milk	0.29 kg/day		0.11 kg b.w./day				Prandini et al. (2009)
Far East	Milk			12 ng/person per day				WHO (2002)
Far East	Milk	0.03 kg/day		0.2 kg b.w./day				Prandini et al. (2009)
France			Children			0.323–0.89 ng/kg b.w. per day		
France	Core foods		Children			0.001–0.01 ng/kg b.w. per day		Sirot et al. (2013)
France	Total diet study		Children			0.323–0.89 ng/kg b.w. per day		Leblanc et al. (2005)
Gambia			Children (6–9 years)				22.3 pg/mg	Turner et al. (2003)
Gambia			Infants (> 1 year)				8.7 pg/mg	Turner et al. (2007)

Gambia			Adults			1.4 µg/day		Wild et al. (1992)
Gambia			Adults (18–70 years)				19.3 mg/pg	Miele et al. (1996)
Ghana			Adults				0.89 mg/pg	Jolly et al. (2006)
Ghana			Adults, HIV-negative				0.9 pmol/mg	Obuseh et al. (2011)
Ghana			Adults, HIV-positive				1.1 pmol/mg	Obuseh et al. (2011)
Ghana			Pregnant women				5 pg/mg	Shuaib et al. (2012)
Ghana			Pregnant women				10.9 pg/mg	Shuaib et al. (2010a)
Ghana	Groundnuts	0.15 kg/week	Millers					Jolly et al. (2008)
Ghana	Groundnuts	0.42 kg/week	Consumers					Jolly et al. (2008)
Ghana	Groundnuts	0.50 kg/week	Processors					Jolly et al. (2008)
Ghana	Groundnuts	0.63 kg/week	Retailers					Jolly et al. (2008)
Ghana	Groundnuts	0.93 kg/week	Farmers					Jolly et al. (2008)
Ghana	Groundnuts	0.94 kg/week	Poultry					Jolly et al. (2008)
Ghana	Groundnuts	35 kg/year	Per capita					Jolly et al. (2008)
Greece	Breakfast cereals		Children			0.07–10.75 ng/kg b.w. per day		Villa and Markaki (2009)
Guinea			Children, 2–4 years		26.6 pg/ml			Polychronaki et al. (2008)
Japan	Total diet study		Young children			0.006–0.007 ng/kg b.w. per week		Kumagai et al. (2008)
Japan	Total diet study		Older children			0.005–0.006 ng/kg b.w. per week		Kumagai et al. (2008)
Japan	Total diet study		Young children	0.013–0.014 ng/kg b.w. per week				Sugita-Konishi et al. (2010)
Japan	Total diet study		Older children	0.011–0.12 ng/kg b.w. per week				Sugita-Konishi et al. (2010)
Kenya							8.4 pg/mg	Jones et al. (2001)
Kenya	Maize	400 g/day	Per capita					Muriuki and Siboe (1995)
Kenya	Overall (range of < limit of detection to 211 pg/mg)						7.87 pg/mg	Yard et al. (2013)
Kenya	Uncooked food samples from the home		Adults			3.5–14.8 ng/kg per day		Peers and Linsell (1973)
Korea	Rice					1.19–5.79 ng/kg b.w. per day (aflatoxin B_1)		Park et al. (2004)
Latin America	Milk			3.5 ng/person per day				WHO (2002)
Latin America	Milk	0.16 kg/day		0.06 kg b.w. per day				Prandini et al. (2009)
Middle East	Milk			0.7 ng/person per day				WHO (2002)
Middle East	Milk	0.12 kg/day		0.1 kg b.w. per day				Prandini et al. (2009)
Mozambique	Samples cooked at home		Adults			38.6–183.7 ng/kg per day (aflatoxin B_1)		Van Rensburg et al. (1985)

Netherlands			Children	0.02–0.44 ng/kg b.w. per day		Bakker et al. (2009)
Nigeria	Cereals	138 kg/year	Per capita			Bandyopadhyay et al. (2007)
Spain	Infant formula		Infants	0.08–37.47 ng/kg b.w. per day		Hernández-Martínez and Navarro-Blasco (2010)
Spain (Catalonia)	Total diet study		Adolescents	0.19–1.31 ng/kg b.w. per week		Cano-Sancho et al. (2013)
Spain (Catalonia)	Total diet study		Children	0.03–0.34 ng/kg b.w. per week		Cano-Sancho et al. (2013)
Swaziland	Uncooked food samples from the home		Adults	5.1–43.1 ng/kg per day (aflatoxin B_1)		Linsell and Peers (1977)
Swaziland	Uncooked food samples from the home		Adults	11.4–159 ng/kg per day (aflatoxin B_1)		Peers et al. (1987)
Taiwan			Adults, liver cancer positive		56.5 pmol/mg	Wu et al. (2009)
Taiwan			Adults, liver cancer negative		59.8 pmol/mg	Wu et al. (2009)
Thailand	Samples of cooked food		Adults	6.5–53 ng/kg per day (aflatoxin B_1)		Shank et al. (1972)
Transkei	Samples cooked at home		Adults	16.5 ng/kg per day (aflatoxin B_1)		Van Rensburg et al. (1985)

Note: b.w. = body weight

6. Prevalence maps

To better visualize the prevalence of aflatoxins globally and to identify gaps in prevalence data, a literature review of 23 databases of aflatoxin prevalence studies was undertaken. The data from these prevalence studies were compiled by region and commodity. The resulting spreadsheets were converted into risk maps.

6.1. Maize

A total of 89 studies from 2000 to 2014 in maize and maize products were mapped. Maize samples in East Africa contain aflatoxin at levels that are consistently well above limits, as evidenced by the number of studies with maize samples containing aflatoxins above 10,000 ppb (Figure 5). To get a better picture of the situation in sub-Saharan Africa, the studies in this region were mapped separately (Figure 6). As would be expected, there were a number of surveys in Kenya, particularly in the eastern part of the country, where recorded acute aflatoxicosis cases have occurred. Several studies also highlighted highly contaminated maize in West Africa. Perhaps most telling for this mapping exercise is the lack of prevalence studies throughout much of sub-Saharan Africa. Given the favourable environmental conditions and the public health implications, it seems that many countries in the region are unaware of the magnitude of the aflatoxin problem.

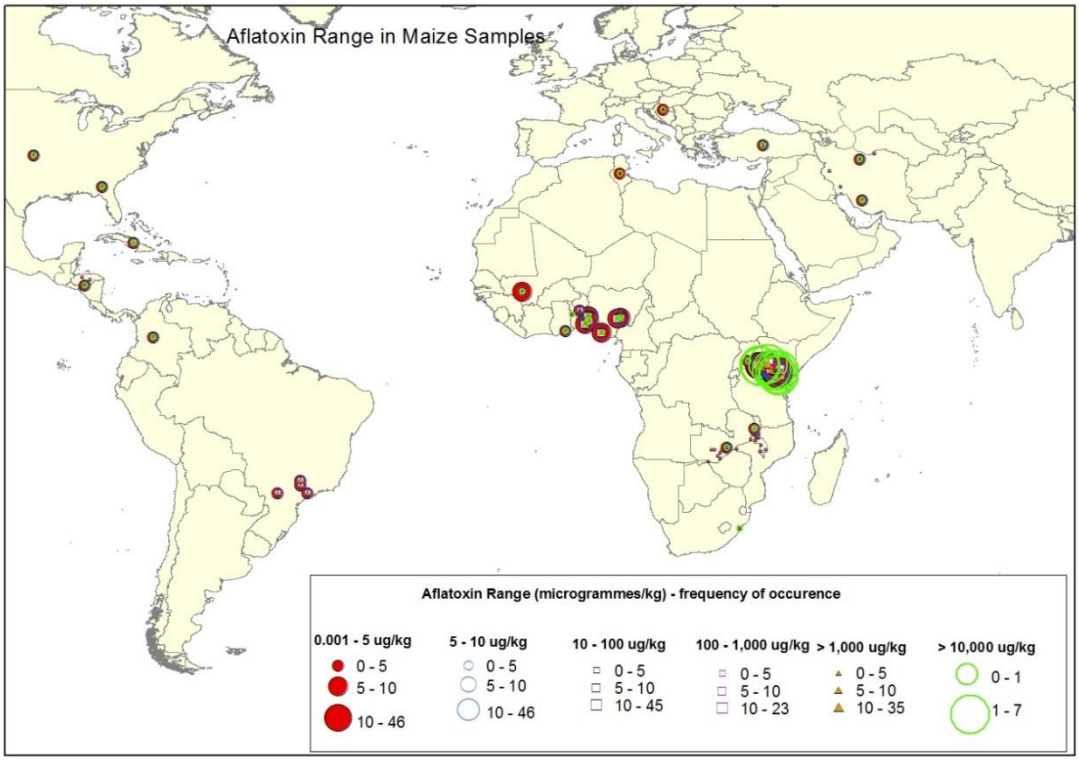


Figure 5: Aflatoxin range in maize samples.

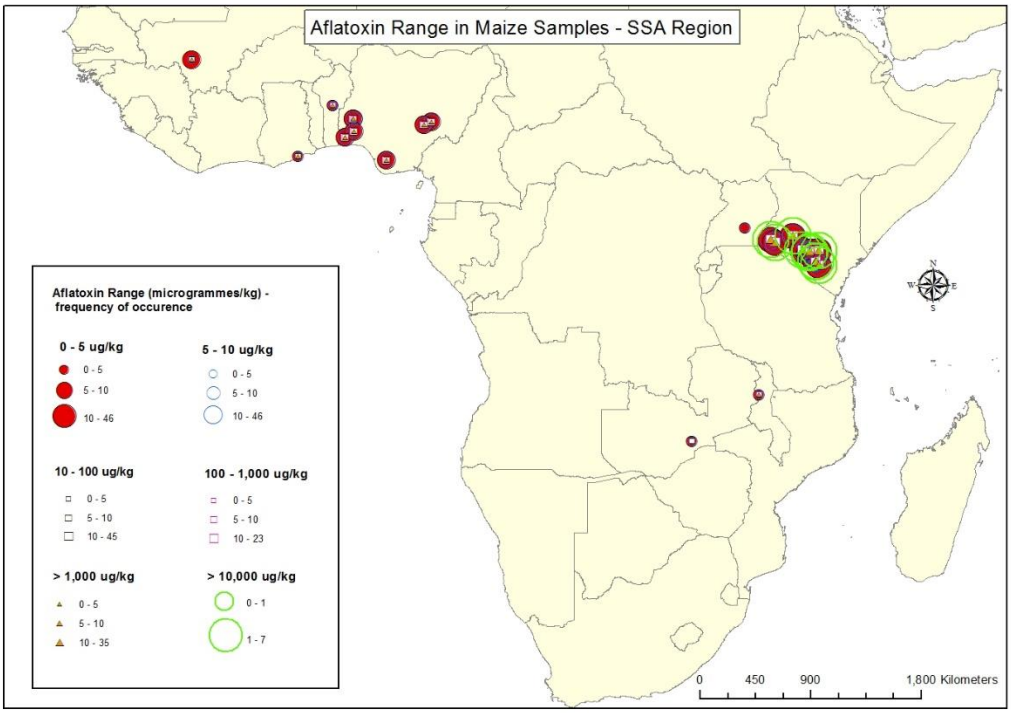


Figure 6: Aflatoxin range in maize samples from sub-Saharan Africa.

6.2. Groundnuts

In groundnuts and groundnut-based snacks, 66 surveys from 2000 to 20014 were mapped. While there were fewer groundnut surveys, the surveys are more geographically dispersed (Figure 7). Because groundnuts are a significant source of protein throughout sub-Saharan Africa, this area was mapped separately (Figure 8).

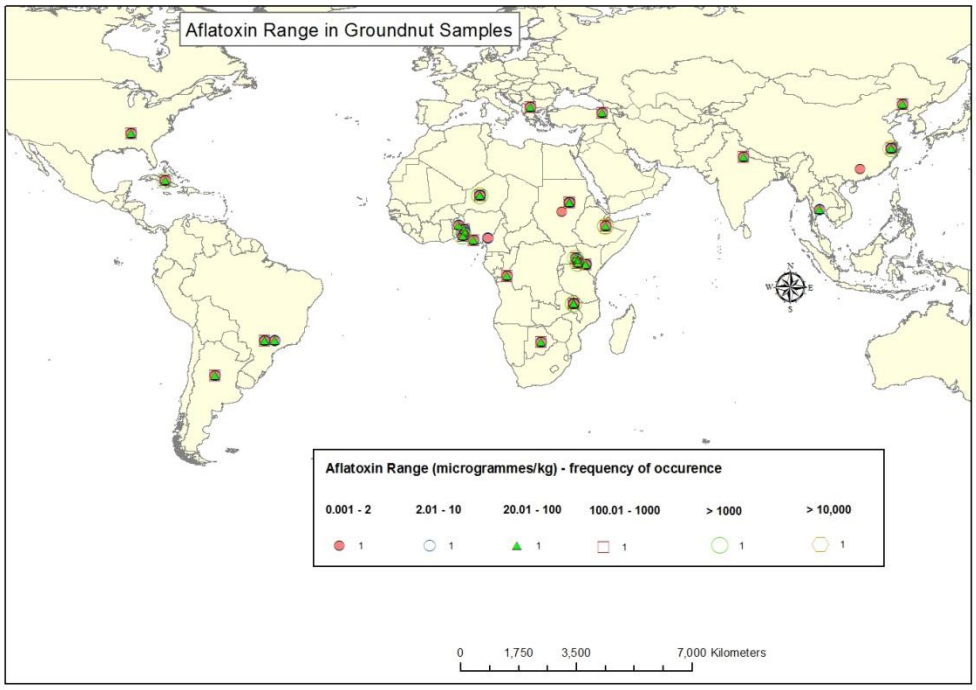


Figure 7: Aflatoxin range in groundnut samples.

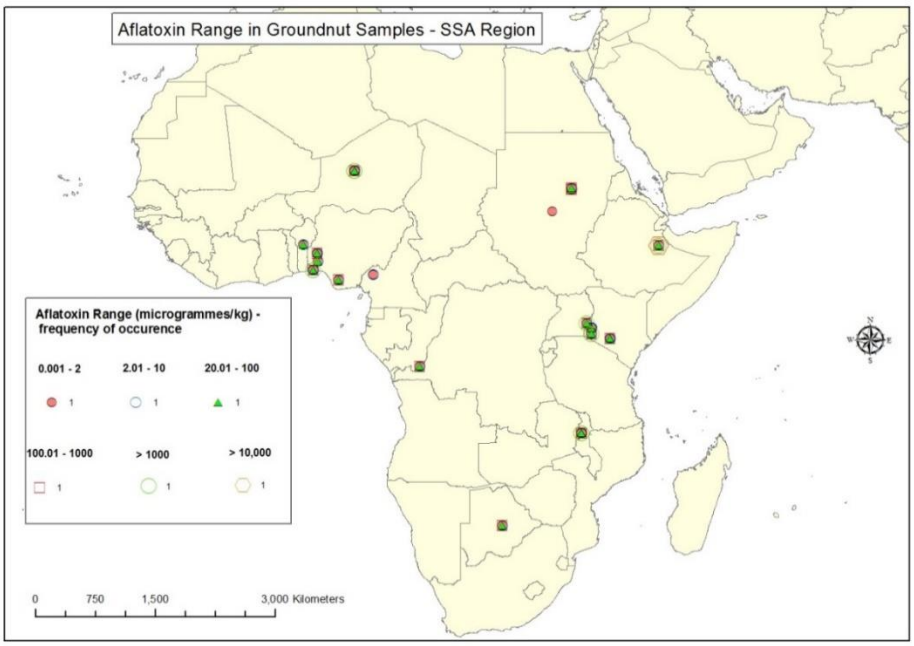


Figure 8: Aflatoxin range in groundnut samples surveyed in sub-Saharan Africa.

While fewer groundnut studies than maize have been done in East and West Africa, the maps highlight that surveys have found high levels of aflatoxin contamination in groundnuts sampled. There are definite gaps in prevalence throughout the region, emphasizing a need for further research.

6.3. Milk

A total of 135 surveys from 2000 to 2014 were mapped for milk (raw, pasteurized and ultra-heat treated). Only surveys done on cow's milk were included in this map (Figure 9). A number of studies have been done on milk in eastern Europe. Some of this is a result of trade with the European Union and the need to comply with their aflatoxin standards. Additionally, the formal trade in milk in this area better lends itself to surveys for consumer safety.

A few studies have been done in sub-Saharan Africa where the vast majority of milk is informally traded. As such, testing is harder and awareness of the risk of aflatoxins in milk very low. However, several surveys have found samples containing aflatoxins at levels well above national and regional standards. This is particularly troubling for infants and children who consume more milk and hence are more susceptible to the adverse health effects associated with chronic aflatoxin exposure.

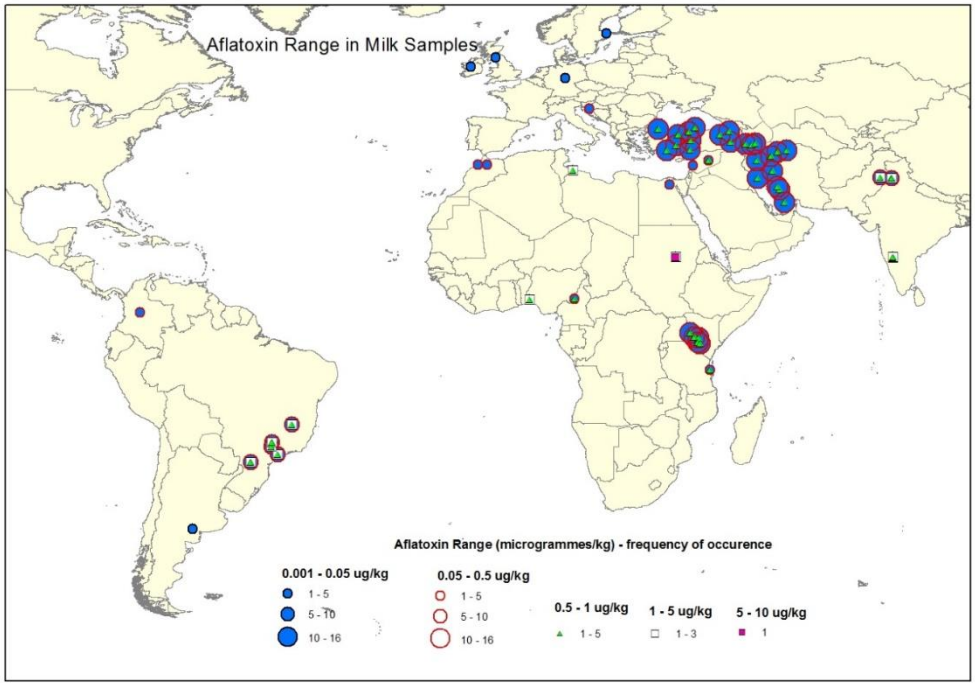


Figure 9: Aflatoxin range in milk samples.

6.4. Other commodities

In addition to maize, groundnuts and milk, there are many other commodities in sub-Saharan Africa that have been analysed for aflatoxin contamination. The results of these studies are summarized in Table 5.

Table 5: Literature review of aflatoxin prevalence in other commodities

Commodity	Aflatoxin B_1 (ppb)		Total aflatoxin (ppb)		% of samples with over 20 ppb aflatoxin	Country/region	Study
	Range	Median	Range	Median			
Barley			0.6–0.8			Tunisia	Ghali et al. (2010)
Barley	1.6–5	18.4	3.5–11.5	7.0		Tunisia	Ghali et al. (2008)
Barley	Trace–11.7	3.8			1.7	Ethiopia	Ayalew et al. (2006)
Beans			0.2–6.2	2.4		Cameroon	Njobeh et al. (2010)
Breadfruit	40.06–48.59	45.37				Nigeria	Odoemelam and Osu (2009)
Cassava-based street-vended snack	0					Nigeria	Rubert et al. (2013)
Cassava chips	0					Benin	Gnonlonfin et al. (2008)
Cassava chips	5.2–14.5					Cameroon	Essono et al. (2009)
Coconut-based street-vended snack	0–23	23				Nigeria	Rubert et al. (2013)
Cottonseed oil (refined)	0.2–0.8					Sudan	Idris et al. (2010)
Cowpea		3.58				Benin	Houssou et al. (2009)
Dried baobab leaves	0					Benin, Mali, Togo	Hell et al. (2009)
Dried fruit	0.7–50	6.4	1.5–45	9.7		Tunisia	Ghali et al. (2008)
Dried fruit			0.13–40.6			Tunisia	Ghali et al. (2010)
Dried hot chilies	0–6					Benin, Mali, Togo	Hell et al. (2009)
Dried okra	0–3.2					Benin, Mali, Togo	Hell et al. (2009)
Dried onion leaves	0					Benin, Mali, Togo	Hell et al. (2009)
Dried tomatoes	0					Benin, Mali, Togo	Hell et al. (2009)
Garri (cassava-based food)			0.12–5.71			Nigeria	Ogiehor et al. (2007)
Ground red pepper	250–525					Ethiopia	Fufa and Urga (1996)
Guinea corn	27.22–36.13	30.53				Nigeria	Odoemelam and Osu (2009)
Hazelnut			25–175			Egypt	Abdel-Hafez and Saber (1993)
Hazelnut			15–25			Egypt	Abdel-Hafez and Saber (1993)
Melon seeds (shelled)	0					Benin, Mali, Togo	Hell et al. (2009)
Millet			2.6–8.1	4.4		West Africa	Bandyopadhyay et al. (2007)
Millet	0–260					Kenya, Malawi	Kenji et al. (2000)
Millet	34-40.3	37.52				Nigeria	Odoemelam and Osu (2009)
Rice			0			Tunisia	Ghali et al. (2010)
Rice	0		2–7.5	4.7		Tunisia	Ghali et al. (2008)
Rice	0–1642	200.19				Nigeria	Makun et al. (2007)
Rice	4.1–309	37.2	27.7–371.9	82.5		Nigeria	Makun et al. (2011)
Sesame oil (unrefined)	0.2–0.8					Sudan	Idris et al. (2010)
Sesame seeds	0–25					Nigeria	Mbah and Akueshi (2009)
Sorghum			1.8–90	5.0	5	West Africa	Bandyopadhyay et al. (2007)
Soybeans			0.2–3.9	2.1		Cameroon	Njobeh et al. (2010)
Spices			0.92–17.1			Tunisia	Ghali et al. (2010)
Spices	0					Ghana	Ahene et al. (2011)
Spices	1.7–38.9	11.3	3.6–87.4	28.5		Tunisia	Ghali et al. (2008)
Teff	Trace–15.6	5.1			5.7	Ethiopia	Ayalew et al. (2006)
Traditional herbal remedies			0			South Africa	Katerere et al. (2008)
Wheat			0.15–18.6			Tunisia	Ghali et al. (2010)
Wheat	1.1–3.4	2.2	4.0–12.9	6.7		Tunisia	Ghali et al. (2008)
Wheat	17.01–36.13	19				Nigeria	Odoemelam and Osu (2009)
Wheat	Trace–12.3	8.7			1.7	Ethiopia	Ayalew et al. (2006)
Yam chips			5–27			Nigeria	Jimoh and Kolapo (2008)
Yam chips	0					Benin	Gnonlonfin et al. (2008)
Yam flour	0–3.2					Nigeria	Somorin et al. (2012)

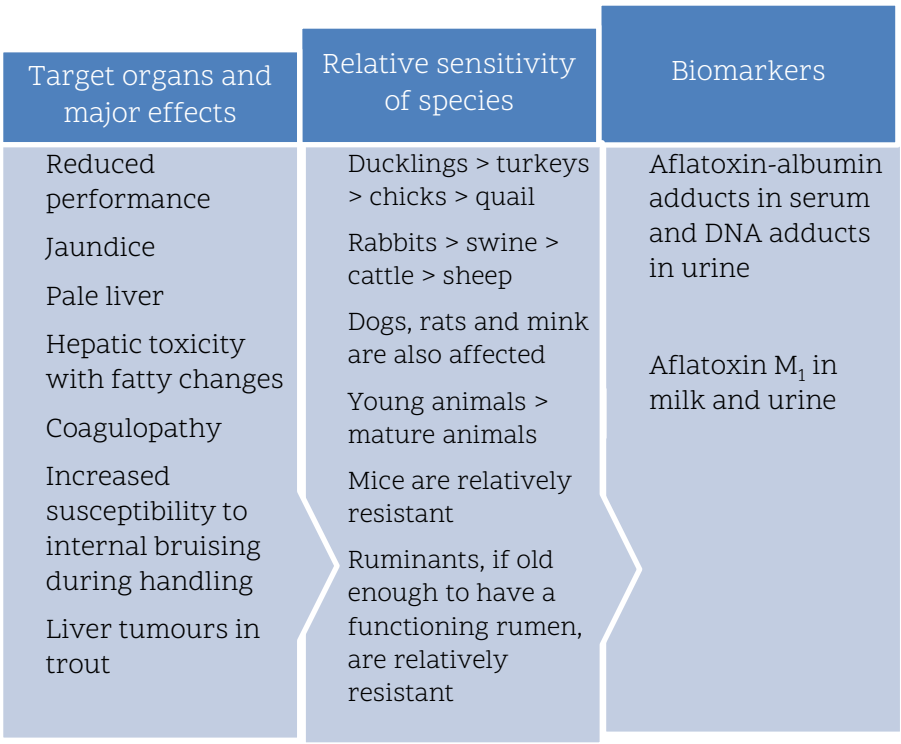
7. Impact of aflatoxins

A recent assessment concluded that 4.5 billion people in the developing world are chronically exposed to uncontrolled amounts of aflatoxins (CIMMYT 2004). There is ample evidence that inhabitants of sub-Saharan Africa are experiencing heavy dietary exposure to food-borne mycotoxins, particularly aflatoxins and fumonisins. According to Miller (1995), 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 (Wagacha and Muthomi 2008). Due to low literacy levels and other socio-

economic factors, even if steps were taken to make food products safe, consumers might be unable or unwilling to pay extra costs and may still prefer to buy the cheap commodities (Wagacha and Muthomi 2008). Besides the direct health risks and premature deaths in Africa, aflatoxin contamination has wide ranging impacts on trade, food safety and food security throughout Africa. (Wu 2004; Fellinger 2006; MRC 2006).

7.1. Aflatoxins in animals and animal-source foods

No animal species is completely resistant to the acute toxic effects of aflatoxins. However, animal species respond differently in their susceptibility to chronic and acute toxicity of aflatoxins (Figure 10). For most species, the aflatoxin median lethal dose (the dose required to kill 50% of a population of test animals) ranges from 500 to 10,000 µg/kg body weight. Toxicity is influenced by environmental factors, exposure level and duration of exposure, besides age, health and nutritional status of diet (Wagacha and Muthomi 2008). Foetuses are very susceptible to even low levels of aflatoxins, and young and fast-growing animals are more affected than adults. Some studies report that males are more susceptible than females (Cassel et al. 2012; Grace 2013). Aflatoxin residues can be found in eggs, milk and meat of animals following consumption of contaminated feeds (Reddy et al. 2010).



Source: Pitt et al. (2012); Grace (2013).
Note: DNA = deoxyribonucleic acid.
Figure 10: Expected toxic effects, species sensitivity and potentially useful biomarkers of aflatoxins in farm animals.

The first recorded case of aflatoxicosis in animals was responsible for the deaths of over 100,000 turkeys in the United Kingdom in 1960. This new disease was called 'Turkey X' until the aetiological cause was discovered and linked to consumption of aflatoxins in the feed. Mortality is also documented in ducks, chickens, pheasant, calves and pigs. In the United States of America and elsewhere, field outbreaks causing mortality have been well documented in turkeys, laying hens,

pigs, cattle, rainbow trout and dogs. In the case of poultry, pigs and farm-raised trout, large numbers of animals were involved.

Acute toxicity is easily recognized, but the more subtle effects are probably of greater concern to farmers. Chronic consumption of aflatoxins at lower levels can cause liver damage, gastrointestinal dysfunction, decreased appetite, decreased reproductive function, decreased growth and decreased production. In addition, immunosuppression results in greater susceptibility to other diseases. Adverse impacts are more severe where there is co-contamination with other mycotoxins (Pitt et al. 2012; Grace 2013).

In turkeys, a sensitive species, reduced weight gain is seen at a dose of 125 µg/kg diet, impaired immune response and increased mortality at 250 µg/kg and acute mortality at 500µg/kg. A similar relative dose–response occurs in pigs but at higher levels of exposure because they are less affected by aflatoxins. In cattle and chickens, much higher levels are required to induce a decrease in performance and in chickens, impaired immune response can occur at levels that have no effect on the growth rate (Pitt et al. 2012).

7.1.1. Aflatoxins in animal feeds

Aflatoxins occur in many animal feed concentrates including cereal grains, soybean products, oil cakes (from groundnuts, cottonseed, sunflower, palm and copra) and fishmeal. Brewers' grains, a by-product of the manufacture of cereal-based alcoholic drinks, can also be contaminated with aflatoxins (Odhav and Naicker 2002). Pasture, hay, straw and silage are more prone to contamination with other types of mycotoxins besides aflatoxins (Grace 2013). Poorly stored homemade dairy concentrates are suspected as the main source of aflatoxin exposure to livestock on smallholder farms in Kenya (Lanyasunya et al. 2005). Little is known about aflatoxin levels in animal feeds in sub-Saharan Africa. Surveys of animal feeds from 2000 to 2014 were mapped (Figure 11). There are issues of aflatoxin contamination in animal feed due to the use of ingredients prone to contamination. However, given the small number of surveys, it is hard to quantify the scale and impact. Further work undertaken by the International Livestock Research Institute on behalf of the East African Community to determine the current situation of aflatoxin regulations in animal feed in East Africa is to be published in late 2014.

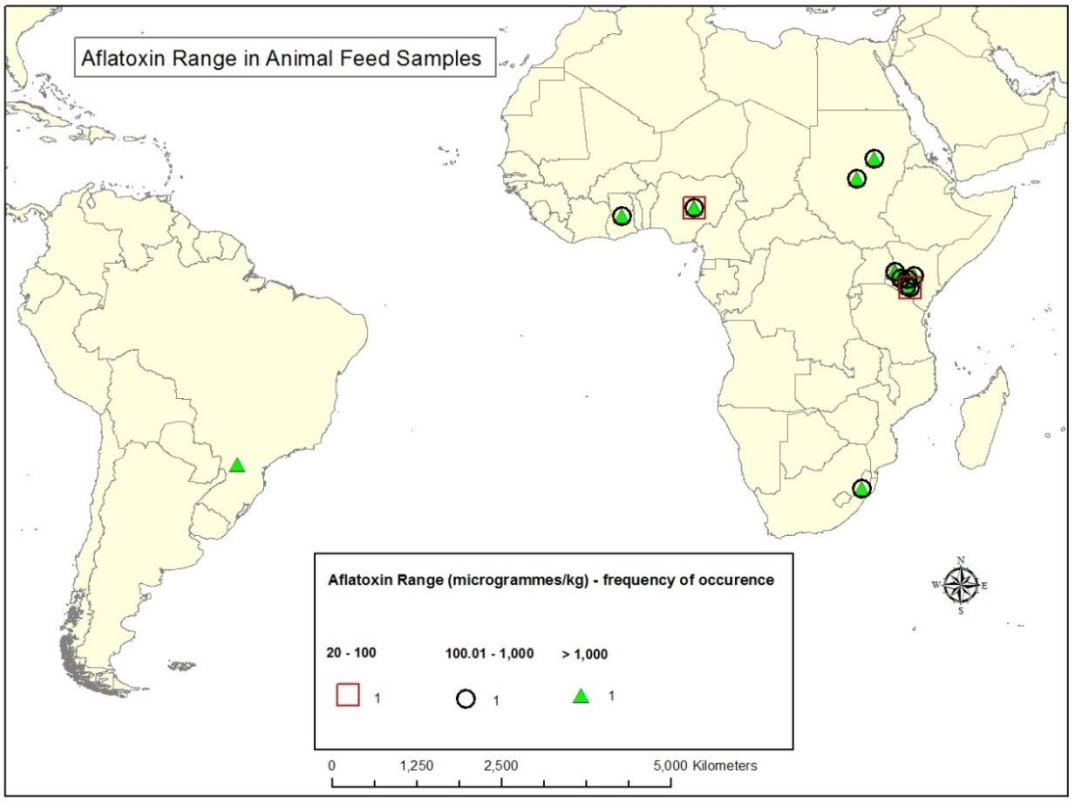


Figure 11: Aflatoxin range in animal feed samples.

In general, livestock in intensive systems are at higher risk of dietary exposure to aflatoxins than animals in extensive systems due to the greater use of concentrates containing products susceptible to aflatoxin contamination in intensive systems. Worldwide, a high and increasing proportion of dairy cattle, poultry and swine are kept in intensive systems; aflatoxins are thus likely to be an increasing problem (Grace 2013).

Chronic aflatoxicosis probably has greater economic impacts than acute disease. Numerous studies show a worsening in feed conversion rations, a decrease in average daily gain and a decrease in body weight for animals experimentally fed aflatoxins (Table 6). Additional losses occur to the livestock sector if grain and feed do not meet the standards for animal feed. Moreover, the nutritive value of grains and cereals is reduced by contamination with the mould that produces aflatoxins. Economic loss also occurs if livestock and fish products do not comply with the standards for aflatoxins in human foods (Grace 2013).

Table 6: Animal studies on the effect of aflatoxin exposure on animal growth

Animal	Aflatoxin dose and duration of experiment	Results	Study
Pigs (n = 50)	0 (A), 0.2 (B), 0.7 (C), 1.1. (D) mg/kg feed for 16 weeks	No significant difference in body weight between groups. Increase in FCR (4.53 [A], 4.55 [B], 4.67 [C], 4.76 [D]) ($p < 0.05$)	Armbrecht et al. (1971)
Pigs (n = 60)	0 (A), 1.0 (B), 2.0 (C), 4.0 (D) mg/kg feed for 13 weeks	Increase in FCR (3.14 [A], 3.82 [B], 4.13 [C], NA [D]) ($p < 0.001$)	Armbrecht et al. (1971)
Pigs, weanlings (n = 110)	< 2 (A), < 8 (B), 51 (C), 105 (D), 233 (E) µg/kg feed for 120 days	No significant effect on weight gain or feed conversion	Keyl and Booth (1971)
Pigs, weanlings (n = 110)	< 6 (A), 450 (B), 615 (C), 810 (D) µg/kg feed for 120 days	Decrease in ADG at 615 and 810 µg/kg feed (0.71 kg [A], 0.60 kg [C], 0.47 kg [D]) ($p < 0.05$)	Keyl and Booth (1971)
Pigs (n = 32; 8 for each of 4 groups of pigs)	20 (A), 385 (B), 750 (C), 1480 (D) µg/kg (control: 20 µg/kg group)	Decrease in ADG (dose-related) (0.77 kg [A], 0.67 kg [B], 0.57 kg [C], 0.41 kg [D]) and ADFI (2.87 kg [A], 2.53 kg [B], 2.15 kg [C], 1.61 kg [D]) ($p < 0.05$). Increase in FCR in the 1480 µg/kg group (3.74 [A], 3.97 [D]) ($p < 0.05$)	Southern and Clawson (1979)
Pigs, 5–6 weeks old (n = 30; 10 each in control, 300 and 500 µg/kg groups)	0, 300 and 500 µg/kg feed for 10 weeks	Decrease in weight gain in both aflatoxin-treated groups up to 2 kg in 10-week period and feed consumption in high-dose group compared with controls ($p < 0.01$)	Panangala et al. (1986)
Pigs, weanlings (n = 90)	0 (A), 420 (B), 840 (C) µg/kg for 49 days	Decrease in ADG (0.52 kg [A], 0.46 kg [B], 0.28 kg [C]) and ADFI (1.13 kg [A], 0.95 kg [B], 0.67 kg [C]). Increase in FCR (1.72 [A], 1.92 [B], 2.70 [C]) (linear $p < 0.01$ and quadratic $p < 0.05$)	Lindemann et al. (1993)
Pigs, weanlings (n = 63)	0 (A), 800 (B) µg/kg feed for 42 days	Decrease in ADG (0.64 kg [A],0 .41 kg [B]) and ADFI (1.32 kg [A], 0.82 kg [B])	Lindemann et al. (1993)
Pigs, weanlings (n = 96)	0 (A), 992 (B) µg/kg feed for 6 weeks	Decrease in ADG (0.505 kg [A], 0.392 kg [B]) and ADFI (1.1 kg [A]), 0.88 kg [B]) ($p < 0.01$)	Schell et al. (1993a)
Pigs, weaned (n = 54)	0 (A), 880 (B) µg/kg feed for 4 weeks	Decrease in ADG (0.64 kg [A], 0.48 kg [B]) ($p < 0.05$) and ADFI (1.32 kg [A], 1.0 kg [B]) (p < 0.05). Increase in FCR (2.08 [A], 2.43 [B]) ($p < 0.05$)	Schell et al. (1993b)
Pigs, weaned (n = 81)	0 (A), 500 (B) µg/kg feed for 5 weeks	Decrease in ADG (0.66 kg [A], 0.46 kg [B]) and AFDI (1.41 kg [A], 0.97 kg [B]) ($p < 0.05$)	Schell et al. (1993b)
Pigs, weaned (n = 63)	0 (A), 800 (B) µg/kg feed for 4 weeks	Decrease in ADG (0.63 kg [A]), 0.52 kg [B]) ($p < 0.05$) and ADFI (1.29 kg [A], < 1.02 kg [B]) ($p < 0.01$)	Schell et al. (1993b)
Pigs, growing barrow (n = 40)	0 (A), 3 (B) mg/kg feed for 28 days	Decrease in weight gain (19.1 ± 0.73 kg [A], 10.7 ± 1.06 kg [B]) ($p < 0.05$)	Harvey et al. (1994)
Pigs (n = 27)	0 (A), 2.5 (B) mg aflatoxin/kg feed, 2.5 mg aflatoxin/kg feed + 2400 IU tocopherol (C) for 32 days	Decrease in body weight (38.4 ± 3.9 kg [A], 22.0 ± 2.0 kg [B], 23.5 ± 3.0 kg [C]) and feed consumption (138 ± 20 kg [A], 41 ± 4.5 kg [B], 45 ± 2.0 kg [C]) ($p < 0.05$)	Harvey et al. (1995a)
Pigs (n = 18)	0 (A), 2.5 (B) mg aflatoxin/kg, 2.5 mg aflatoxin + 100 mg fumonisin B_1/kg feed (C) for 35 days	Decrease in body weight (49.2 kg [A], 33.2 kg [B], 23.9 kg [C]), weight gain (31.6 kg [A], 15.8 kg [B], 6.3 kg [C]) and feed consumption per pen (153.7 kg [A], 89.0 kg [B], 42.7 kg [C])	Harvey et al. (1995b)
Pigs, 4-week-old weaned (n = 36)	0 (A), 240 (B), 480 (C) µg/kg feed for 30 days	Decrease in ADG (489 ± 18 g [A], 453 ± 12 g [B], 326 ± 17 g [C]) ($p < 0.05$)	Marin et al. (2002)
Piglets, 7 weeks old (n = 15)	0 (A), 2.0 mg aflatoxin (B), 2.0 mg ochratoxin (C), 2.0 mg aflatoxin + 2.0 mg ochratoxin (D) per kg feed for 28 days	Decrease in body weight gain in all aflatoxin-treated groups (18.2 ± 0.9 kg [A], 13.5 ± 0.8 kg [B], 13.8 ± 1.0 kg [C], 8.8 ± 0.9 kg [D]) ($p < 0.05$)	Harvey et al. (1989)
2- to 3-year-old sows and their piglets (n = 24)	0 (A), 800 µg/kg aflatoxin G_1 (B), 800 µg/kg aflatoxin B_1 (C), 800 µg/kg aflatoxin G_1 + 800 µg/kg aflatoxin B_1 (D) for 60 days of pregnancy to 28 days lactation	Decrease in piglets' body weight in aflatoxin B_1-treated group but not in aflatoxin G_1-treated group: 6.51 ± 0.42 g (A), 5.66 ± 0.39 g (B), 5.32 ± 0.63 g (C), 5.25 ± 0.44 g (D); $p < 0.05$ and $p < 0.005$ for C and D, respectively	Mocchegiani et al. (1998)
Steers, young cross-bred (n = 50)	0 (A), 100 (B), 300 (C), 700 (D), 1000 µg/kg feed for 133 days	Decrease in ADG at 700 and 1000 µg/kg ($p < 0.01$) (1.14 kg [A], 0.86 kg [D], 0.79 kg [E]). Increase in FCR at 700 and 1000 µg/kg ($p < 0.01$) (5.7 [A], 6.4 [D], 6.6 [E])	Keyl and Booth (1971)
Chickens (n = 900)	0 (A), 0.3 (B), 1.25 (C), 2.0 (D) mg/kg for 28 days	Decrease in body weight and food intake. Increase in FCR ($p < 0.001$)	Bryden et al. (1979)
Broiler chicks (n = 40–48)	0 (A), 5 (B) mg/kg feed, exercise (C), 5 mg/kg feed + exercise (D) for 24 days	Decrease in body weight in aflatoxin-treated group which can be partially improved by exercise (557.6 ± 9.3 g [A], 542.7 ± 9.0 g [B], 366.8 ± 7.4 g [C], 412.5 ± 7.4 g [D]). Increase in FCR in aflatoxin-treated group (1.54 [A], 1.89 [C])	Randall and Bird (1979)
Layer chicks (n = 40–48)	0 (A), 5 (B) mg/kg feed, exercise (C), 5 mg/kg feed + exercise (D) for 33 days	Decrease in body weight in aflatoxin-treated group which can be partially improved by exercise (469.5 ± 9.9 g [B], 370.8 ± 20.2 g [C], 384.1 ± 14.4 g [D]). Increase in FCR in aflatoxin-treated group (1.59 [A], 1.75 [C])	Randall and Bird (1979)

Broiler chicks (n = 40–48)	0 (A), 5 (B) mg/kg feed, exercise (C), 5 mg/kg feed + exercise (D)	Decrease in body weight in aflatoxin-treated group which can be partially improved by exercise (510.5 ± 12.5 g [A], 502.0 ± 12.0 g [B], 414.9 ± 19.8 g [C], 434.0 ± 8.1 g [D]). No change in FCR	Randall and Bird (1979)
Broiler chickens (n = 75)	0 (A), 0.075 (B), 0.225 (C), 0.675 (D) mg/kg feed for 7 weeks	Decrease in body weight in all aflatoxin-treated groups (2256 ± 21 g [A], 2098 ± 26 g [B], 1989 ± 20 g [C], 2047 ± 24 g [D])($p < 0.05$)	Doerr et al. (1983)
Broiler chickens (n = 75)	0 (A), 0.3 (B), 0.9 (C), 2.7 (D) mg/kg in feed for 7 weeks	Decrease in body weight in group D only (2024 ± 30 g [A], 1671 ± 36 g [D]) ($p < 0.05$)	Doerr et al. (1983)
1-day-old broilers (n = 70)	0 (A), 0.625 (B), 1.25 (C), 2.5 (D), 5.0 (E), 10.0 (F) mg/kg in feed for 3 weeks	Aflatoxin dose-related decrease in body weight in groups D, E and F (511 ± 32 g [A], 463 ± 16 g [D], 386 ± 25 g [E], 286 ± 13 g [F]) and feed consumption (851 ± 52 g [A], 773 ± 50 g [D], 703 ± 55 g [E], 734 ± 14 g [F]) ($p < 0.05$)	Huff (1980)
1-day-old broiler chicks (n = 48)	0 (A), 5 (B) mg/kg of feed aflatoxin B_1 in feed for 3 weeks	Decrease in weight gain (866 ± 12.7 g [A], 699 ± 38.5 g [B]) ($p < 0.05$) and feed intake (1369 ± 45.7 g [A], 957 ± 183.5 g [B]) ($p < 0.05$). No change in FCR.	Pimpukdee et al. (2004)
14-day-old broiler chicks (n = 200)	0 (A), 100 (B), 200 (C), 400 (D), 800 (E) µg/kg aflatoxin B_1 for 35 days	No significant difference in weight gain ($p < 0.05$). Increase in FCR at dose E (2.02 [A], 2.11 [E])	Giambrone et al. (1985)
Male broiler chicks (n = 180)	0 (A), 2.5 (B) mg/kg aflatoxin, 2.5 mg/kg aflatoxin + 16 mg/kg of deoxynivalenol (C) for 3 weeks	Decrease in body weight (626 ± 11 g [A], 521 ± 12 g [B], 488 ± 9 g [C]), weight gain (490 ± 10 g [A], 397 ± 10 g [B], 365 ± 8 g [C]) and protein serum (2.9 ± 0.1 g/100 ml [A], 2.0 ± 0.1 g/100 ml [B], 2.1 ± 0.1 g/100 ml [C]) ($p < 0.05$)	Huff et al. (1986)
105-day-old cockerels (n=120)	0 (A), 2.5 (B), 5.0 (C),10.0 (D) mg/kg in feed for 4 weeks	Aflatoxin dose-related decrease in body weight ($p > 0.05$) (1.85 ± 0.03 g [A], 1.57 ± 0.05 g [B], 1.51 ± 0.04 g [C], 1.47 ± 0.03 g [D])	Shukla and Pachauri (1985)
1-day-old broilers and layer chicks (n = 40 each)	0 (A),1 (B), 4(C) mg/kg in feed for 4 weeks	Aflatoxin dose-dependent decrease in body weight ($p < 0.05$). Broiler chicks: 332 ± 17.81 g (A), 254 ± 14.35 g (B), 239 ± 13.5 g (C). Layer chicks: 158 ± 3.6 g (A), 139 ± 4.41 g (B), 126 ± 5.82 g (C)	Ram et al. (1988)
1-day-old broiler chicks (n = 40)	0 (A), 0.5 (B) mg/kg in feed for 32 days	Decrease in body weight (246.32 ± 2.14 g [A], 140.79 ± 1.31 g [B]), percentage weight gain (100% [A], 57% [B]) and total feed consumption (691.0 g [A], 590.0 g [B]) ($p < 0.01$)	Prabaharan et al. (1999)
14-day-old turkeys (n = 200)	0 (A), 100 (B), 200 (C), 400 (D), 800 (E) µg/kg aflatoxin B_1 for 35 days	Decrease in percentage weight gain at dose D and higher (averaged 5-week percentage weight gain: 48.2% [A], 33.2% [D], 19.7% [E]). Increase in FCR at the two highest doses (FCR averaged in 5 weeks: 1.81 [A], 1.89 [D], 2.28 [E]) ($p < 0.05$)	Giambrone et al. (1985)
Channel catfish (n = 450)	0, 100, 404, 2154 or 10,000 µg/kg for 10 weeks	Decrease in weight gain in the 10,000 µg/kg group by 24% compared to the control ($p < 0.05$). Weight gain per fish in the highest dosed group = 60 g compared to 80 g/fish in the control.	Jantrarotai and Lovell (1990)
Nile tilapia (n = 160)	0 (A), 0.94 (B), 1.88 (C), 0.375 (D), 0.752 (E), 1.50 (F), 3.0 (G) mg/kg diet for 25 days following with basal diet for 50 days	Decrease in ADG and ADFI but not FCR in group C and higher ADG: 10.87–11.30 g (A), 7.28 g (C), 7.1 g (D), 4.78 g (E), 3.25 g (F), 3.66 g (G) ($p < 0.01$). ADFI: 0.143–0.160 g (A), 0.115 g (C), 0.116 g (D), 0.711 g (E), 0.052 g (F), 0.048 g (G) ($p < 0.01$)	Chavez-Sanchez et al. (1994)
Lambs (n = 44)	0 mg aflatoxin in soybean meal (A), 0 mg aflatoxin in fish meal (B), 2.5 mg/kg diet soybean meal (C) or 2.5 mg/kg diet fish meal (D) for 35 days followed by 32-day wash out period	Decrease in feed intake and daily gain in aflatoxin-fed lambs ($p < 0.05$) during treatment and wash out periods. ADG: 0.53 kg (A), 0.24 kg (C), 0.50 kg (B), 0.05 kg (D). ADFI: 4.19 kg (A), 2.74 kg (C), 4.05 kg (B), 1.7 kg (D). Increase in FCR in aflatoxin-fed lambs ($p < 0.05$); FCR: 7.6 (A), 11.2 (C), 7.6 (B), 45.5 (D)	Edrington et al. (1994)
Lambs (n = 46)	23 lambs fed 2500 ppb aflatoxins for 21 days (A), 13 lambs control (B)	Reduction in body weight 19.2 (A), 17 (B)	Fernández et al. (1997)
Kids (n = 20)	0 (A), 50 ppb (B), 100 ppb (C), 150 ppb (D) for 12 weeks	Final weight 11.5 kg (A), 9.9 kg (B), 9.48 kg (C), 9.1 kg (D)	Ewuola et al. (2013)

Source: Khlangwiset et al. (2011).

Notes: ADG = average daily gain; ADFI = average daily feed intake; FCR = feed conversion ratio; NA = data not available.

Dietary levels of aflatoxin generally tolerated in the above studies are ≤ 50 ppb in young poultry, ≤ 100 ppb in adult poultry, ≤ 50 ppb in weaner pigs, ≤ 200 ppb in finishing pigs, < 100 ppb in calves, < 300 ppb in cattle and < 100 ppb in Nile tilapia.

7.1.2. Aflatoxins in dairy cattle and dairy products

In ruminants, aflatoxin B_1 is metabolized to aflatoxin M_1 in the liver and excreted in the milk of dairy cows. Aflatoxin intoxication in dairy cattle is characterized by liver cell injury, fatty liver syndrome, poor feed conversion and a significant reduction in milk yield. High-yielding dairy cows are considered to be more sensitive to aflatoxins than fattening cattle. Even low levels of aflatoxins

are able to affect the cellular and humoral immune system, resulting in increased susceptibility to infectious diseases in exposed animals (Fink-Gremmels 2008). Aflatoxicosis is usually considered a herd rather than an individual cow problem (Feddern et al. 2013).

Because aflatoxins are degraded by flora in the cow's rumen, the amount of aflatoxin M_1 excreted in milk is only around 1–7% of the total amount of aflatoxin B_1 ingested (Fink-Gremmels 2008). Cows in early lactation can excrete 3.8–6.2% of dietary aflatoxin B_1 as aflatoxin M_1 and cows in late lactation can excrete 1.8–2.5% of dietary aflatoxin B_1 as aflatoxin M_1 (Coppock et al. 2012). Higher-yielding animals consuming large amounts of concentrates typically have higher levels of aflatoxin in their milk. The dietary threshold for aflatoxin excretion in cows' milk appears to be 15 ppb or 230 µg aflatoxin B_1 per cow per day (Coppock et al. 2012). The presence of mastitis may increase the secretion of aflatoxins.

While levels of mycotoxins in cereals may reach thousands of ppb, levels in milk are generally less than 100 ppb. However, aflatoxins in milk are of concern because milk consumption is often higher among infants and children, who are likely to be more vulnerable. Accordingly, many countries set a lower threshold for aflatoxins in milk. According to European Union and Codex Alimentarius Commission standards, the maximum level of aflatoxin M_1 in liquid milk and dried or processed milk products should not exceed 0.05 µg/kg and in the United States of America, the maximum level permitted is 0.5 µg/kg.

Aflatoxin levels are around three times higher in soft cheese and five times higher in hard cheese than in the milk of origin. Since cheese is more concentrated, using aflatoxin-contaminated milk for cheese production is risk mitigating (for example, if 10 litres of milk makes 1 kg of cheese and aflatoxins are five times higher in hard cheese than in milk, then the exposure to aflatoxin by consuming 1 kg of cheese is half as much as that from consuming 10 litres of milk). Aflatoxins may also be present in yoghurt and other dairy products. Recent studies have suggested that a related toxin called aflatoxicol may also be excreted in significant amounts in milk, a subject that requires further research (Grace 2013).

7.1.3. Aflatoxins in meat-producing animals

Trace levels of aflatoxins and their metabolites may also carry over into the edible tissue of meat-producing animals. Aflatoxins are generally found in the liver, kidney and edible parts of the gastrointestinal tract. Aflatoxins are not known to accumulate in body fat. Studies have shown that frequency of processed meat contamination with aflatoxin B_1 was low and the toxin level within meat was usually less than 10 ppb (Miller et al. 1982; Trucksess et al. 1982; Trucksess et al. 1983; Richard et al. 1986; Beaver et al. 1990; Madden and Stahr 1992; Qureshi et al. 1998; Bailly and Guerre 2009; Feddern et al. 2013). Table 7 summarises a review of the published literature on aflatoxin levels in animal-source foods.

Table 7: Literature review of published aflatoxin levels in animal-source food products

Product	Aflatoxin M_1 (ppb)		Aflatoxin B_1 (ppb)		Total aflatoxin (ppb)		Country	Study
	Range	Mean	Range	Mean	Range	Mean		
Beef heart, dried				0.0143			Nigeria	Olufunmilayo and Oyefolu (2010)
Beef heart, fresh				0.0285			Nigeria	Olufunmilayo and Oyefolu (2010)
Beef kidney, dried				0.0348			Nigeria	Olufunmilayo and Oyefolu (2010)
Beef kidney, fresh				0.0435			Nigeria	Olufunmilayo and Oyefolu (2010)
Beef liver, dried				0.0021			Nigeria	Olufunmilayo and Oyefolu (2010)
Beef liver, fresh				0.0714			Nigeria	Olufunmilayo and Oyefolu (2010)
Beef, dried				0.0013			Nigeria	Olufunmilayo and Oyefolu (2010)
Beef, dried (kilishi)				113.10			Cameroon	Jones et al. (2001)
Beef, fresh				0.01			Nigeria	Olufunmilayo and Oyefolu (2010)
Buffalo milk	10–250						Egypt	Motawee et al. (2009)
Camel milk	10–250						Egypt	Motawee et al. (2009)
Cheese	0.16–0.35	0.21					Libya	Elgerbi et al. (2004)
Eggs						0.82	Cameroon	Tchana et al. (2010)
Fish, fresh					22–70.5		Egypt	Hassan et al. (2011)
Fish, salted					18.5–50		Egypt	Hassan et al. (2011)
Fish, smoke-dried			1.5–8.1				Nigeria	Adebayo-Tayo et al. (2008)
Fish, smoked					32–96		Egypt	Hassan et al. (2011)
Goat milk	10–250						Egypt	Motawee et al. (2009)

Aflatoxins in poultry

While chickens are relatively resistant to aflatoxins, turkeys and ducks are highly susceptible. Aflatoxin ingestion by chickens results in many different symptoms, such as reduced growth and increased susceptibility to infectious agents. The liver is considered the aflatoxins' target organ due to the protein production inhibition pathway of aflatoxin elicited in the hepatocytes (Dhanasekaran et al. 2009). Long-term consumption of feed contaminated with relatively low aflatoxin content causes immunosuppression in broilers by impairment of humoral and cellular immune response. Heavy loss due to the interaction of infectious bursal disease and aflatoxicosis has been reported (Otim et al. 2005). The symptoms observed in aflatoxicosis were anorexia and lack of thriftiness and the mortality rate was 0.03%. The interaction of infectious bursal disease and aflatoxicosis led to an increased mortality rate of 35.6% as compared to 3–21% in infectious bursal disease and 0.03% in aflatoxicosis (Otim et al. 2005).

As low as 0.2 parts per million (ppm) of aflatoxin B_1 have been documented to reduce egg production and egg mass in laying hens from 22 to 40 weeks of age (Azzam and Gabal 1998). An experiment was conducted on 1-week-old White Leghorn female chicks to study the effect of aflatoxin B_1 on weight gain, feed intake, feed gain ratio, age at sexual maturity, production and quality of eggs and retention of nutrients, and of aflatoxin B_1 residues in eggs and muscles of hens. The chicks were assigned to four dietary treatments: D1 (without aflatoxin B_1), D2 (2.50 mg/kg aflatoxin B_1), D3 (3.13 mg/kg aflatoxin B_1) and D4 (3.91 mg/kg aflatoxin B_1) up to the age of 40 weeks. At the end of the experiment, the mean body weight gain and feed intake were significantly lower in all aflatoxin-fed groups compared to the control. The feed gain ratios were noted as 13.41, 13.59, 13.82 and 14.71, with the group fed the highest concentration of aflatoxin B_1 showing a significantly poorer ratio than the other groups. Age at sexual maturity was also affected adversely by dietary aflatoxin B_1: 193 days for D4 compared to as early as 148 days for D1. Retentions of dry matter, crude protein, ether extract, calcium and metabolizable energy were adversely affected at various levels of aflatoxin B_1 compared to the control. Patho-anatomical and histopathological studies showed various adverse changes in the liver, kidneys, heart, ovaries and bursa of Fabricius in

aflatoxin B_1-fed groups. Aflatoxin residues were detected in eggs and breast muscles of hens in all aflatoxin B_1-fed groups (Pandey and Chauhan 2007).

At two weeks of age, groups of chickens were placed on diets containing minimum levels of 0, 2.5, 3.13 and 3.91 ppm aflatoxin B_1. These diets were fed for 40 weeks. Tissues and eggs were collected on day 40 for aflatoxin analyses. Aflatoxin B_1 in eggs increased with feed levels; the mean levels were 1.43, 1.39 and 1.63 ppb, respectively, for the different treatment groups. Levels in breast meat were 18, 26 and 26 ppb, respectively, for the chickens in the different treatment groups. Young birds have been shown to have higher levels of aflatoxins and require longer to clear aflatoxins (Hussain et al. 2010).

The transfer of aflatoxin B_1 from diet to eggs was studied in 12-week-old hens given diets containing 0, 100, 300 or 500 ppb aflatoxin B_1 (Oliveira et al. 2000). Aflatoxin B_1 was only detected at levels from 0.05 to 0.16 ppb (mean 10 ppb) in the eggs from hens on the 500 ppb diet. In this study, the transfer rate was 5000:1 diet to egg ratio.

In a feeding trial of 2-week-old turkey poults, at 50 ppb feeding level for 11 weeks, aflatoxin B_1 was found in the liver (0.02–0.09 ng/g), kidney (0.01–0.02 ng/g) and gizzard (0.043–0.162 ng/g) whereas aflatoxin M_1 could not be detected in the same organs (Richard et al. 1986). Feeding 50 ppb aflatoxins for 13 weeks increased the residues of aflatoxin B_1 and aflatoxin M_1. For aflatoxin B_1, liver contained 0.02–0.13 ng/g, kidney contained 0.01–0.34 ng/g and gizzard contained trace levels to 0.113 ng/g whereas aflatoxin M_1 in liver was 0.11–0.14 ng/g and kidney contained 0.01–0.07 ng/g. At the 150 ppb feeding level, fed for 11 weeks, aflatoxin B_1 in liver was 0.08–0.13 ng/g, kidney was 0.025–0.08 ng/g and gizzard contained trace levels to 0.22 ng/g whereas aflatoxin M_1 levels were 0.03–0.10 ng/g in liver and 0.09–0.13 ng/g in kidney. Aflatoxin M_1 was not shown to be present in the gizzard. Breast and thigh muscles did not contain aflatoxins (Richard et al. 1986).

A meta-analysis of studies on the effect of aflatoxins on growth performance found that for every mg/kg increase of aflatoxin in the diet, the growth rate in broilers would be reduced by 5% (Dersjant-Li et al. 2003). Additionally, dietary concentrations that would cause a 5% reduction in growth rate were estimated at 1 mg/kg from broilers. Due to the rapid metabolism of aflatoxins in the body of a chicken (Hussain et al. 2010), exposure to aflatoxins through consumption of chicken liver and meat is probably not a significant public health risk.

Aflatoxins in pigs

Pigs are highly susceptible to aflatoxins. The most susceptible feed components and those used in commercially available pig feedstuffs are groundnuts, maize and cottonseed. Aflatoxin B_1, aflatoxin G_1 and aflatoxin M_1 can be present in the sow's milk and different levels are possible depending on the initial contamination of the feed (Kanora and Maes 2009). Experimental intoxications have shown damaged lymphocytes and macrophages in piglets, indicating a loss of immune-competence due to exposure of sows to aflatoxins. Clinical signs of acute aflatoxicosis include anorexia, nervous signs and sudden death (Kanora and Maes 2009). In sows and gilts, aflatoxin consumption at levels above 2000 ppb produced acute hepatosis and death in 3–10 days. At levels of 500–750 ppb, there were no observed effects on conception in sows and gilts, piglets were normal but had slower growth due to aflatoxins in sow's milk (Osweiler 2006).

The transfer of aflatoxins into edible tissues of pigs has been studied. The half-life of aflatoxin residues is very short. In feed concentrations of 355–551 µg/kg, the average half-life was 24 hours. After 48 hours, only minute quantities of residues were found (less than 0.05 µg/kg) and after four days there were no residues (Kanora and Maes 2009).

Jacobson et al. (1978) fed feeder pigs (54–72 kg body mass) diets containing pure aflatoxin B_1 at 2000, 400 and 100 ppb for four weeks. In decreasing dietary level, aflatoxin B_1 levels in liver were 1.5, 0.5 and 0.2 ppb, respectively, levels in skeletal muscle were 1, 0.5 and 0.2 ppm, respectively and levels in kidney were 4.4, 0.7 and 0.2 ppb, respectively.

A meta-analysis reviewed 85 articles published between 1968 and 2010, totalling 1012 treatments and 13,196 animals. Mycotoxins resulted in a 15% reduction in weight gain in females and 19% in males. The effects were greater in younger animals and at higher doses. For each additional 1000 ppb (1 mg/kg) of aflatoxins in the feed, there was a 3.9% reduction in pig weight gain. Methionine and protein were protective (Andretta et al. 2011).

Another meta-analysis of studies on the effect of aflatoxins on growth performance found that for every milligram per kilogram increase of aflatoxin in the diet, the growth rate in pigs would be reduced by 16% (Dersjant-Li et al. 2003). Additionally, dietary concentrations of aflatoxins that would cause a 5% reduction in growth rate were estimated at 0.3 mg/kg for pigs.

Aflatoxins in beef cattle

Beef cattle are relatively resistant to aflatoxins. Steers given ad libitum feed with aflatoxins at 700 ppb showed reduced weight gain and at 1000 ppb, death resulted after 59 days. However, even at 100 ppb, increases in liver weight have been noted (Whitlow and Hagler 1997). Steers fed a diet containing 800 ppb aflatoxins for 15 weeks and then placed on an aflatoxin-free diet for 2.5 weeks did not have residues of aflatoxin in the heart, skeletal muscle, liver and kidney (Table 8).

Table 8: Aflatoxin levels in tissues after steers were fed a diet containing 800 ppb aflatoxins for 15 weeks

Toxin	Level of aflatoxin (ng/g or ng/ml)					
	Liver	Kidney	Muscle	Heart	Lung	Rumen contents
Aflatoxin B_1	0.37	0.09	0.002	0.004	0.014	13.05
Aflatoxin M_1	1.07	4.82	0.115	0.14	0.29	0.14

Source: Richard et al. (1983).

Poultry feed contaminated at the level of 3000 ppb may result in levels of 3 ppb in poultry meat. Aflatoxins may be carried over from feed to eggs at ratios ranging from 5000–125,000 to 1 (Zaghini et al. 2005). These transfer rates are much lower than for milk and surveys in developing countries typically find trace levels in meat and offal. Given the relatively low quantities of animal-source food consumed, this is not likely to present a major contribution to overall consumption of aflatoxins in the diet. Consumption of aflatoxin-contaminated milk by infants and children poses the greatest animal-source food risk, based on present information.

Another source of aflatoxin exposure is processed fish which has been found to be significantly contaminated with aflatoxins (Adebayo-Tayo et al. 2008) (Figure 12). However, given the small number of surveys of aflatoxin contamination in fish and varying consumption levels across sub-Saharan Africa, the risk and impact of aflatoxin consumption from fish needs further research.

Finally, mould-fermented foods such as fermented meat may also contain aflatoxins but there is very little information regarding the level of aflatoxins in traditionally processed foods (Grace 2013).

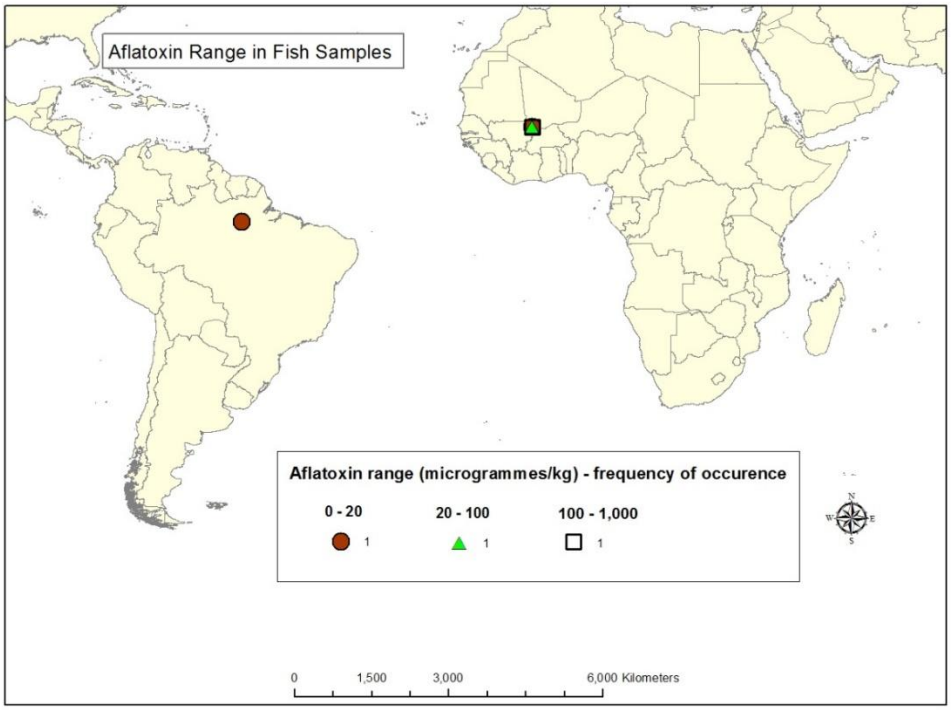


Figure 12: Aflatoxin range in fish samples.

Figure 13 shows the number of surveys on aflatoxins in animal-source food products and animal feed to identify countries with research gaps in aflatoxin contamination in animal-source foods and feeds. Overall, there have been far more studies of aflatoxin levels in milk and dairy products. As livestock intensification increases to meet food demands, the magnitude and impact of aflatoxin contamination on livestock health and productivity, animal-source foods and food safety will continue to be monitored, risk mitigating strategies employed along the value chains and alternative uses found for highly contaminated products.

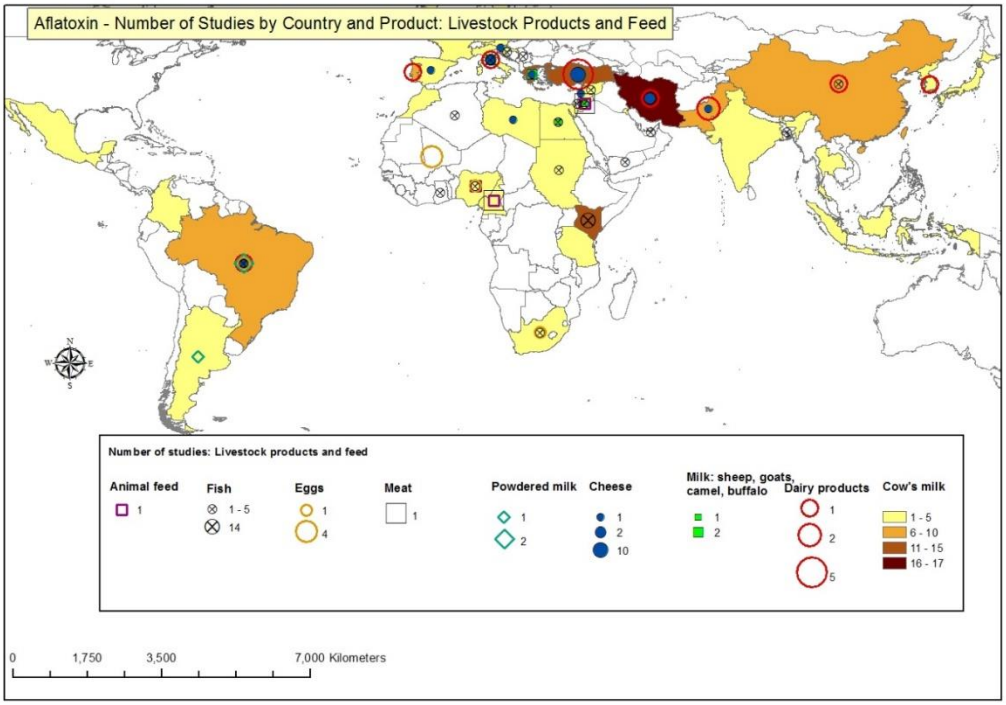


Figure 13: Number of surveys on aflatoxins in animal-source foods, 2000 to 2014.

7.2. Impact in East Africa

The recurrent cases of aflatoxin poisoning in East Africa have become a revolving epidemic, particularly in the arid and semi-arid areas of the region. This epidemic has been attributable primarily to crop planting and post-harvest practices. Retail stores and open markets have been shown to pose highest risk of crop contamination. Rates of aflatoxin exposure and hepatitis B prevalence in rural populations are higher than those in urban populations, even within the high-burden developing countries. This disparity may be explained by differences between an urban diet that is more diverse and the rural population's staple-based diet consisting of maize, peanuts and other foods prone to aflatoxin contamination (Liu and Wu 2010).

Kenya has experienced several aflatoxicosis outbreaks during the last 25 years, most of which have occurred in Makueni and Kitui districts in Eastern Province. Both districts are prone to food shortages due to poor and unreliable rainfall and high temperatures. In Kenya, more than 40% of rural and urban diets consist of maize and maize products (Mwihia et al. 2008). The 2004 Kenyan aflatoxicosis outbreak followed a poor maize harvest that had been damaged and consequently made susceptible to mould by drought. To guard against theft of the meagre harvest, people stored the maize in their houses, which were warmer and more moist than the granaries where the crop was usually stored. Maize samples taken during the outbreak showed aflatoxin B_1 concentrations as high as 4400 ppb, 220 times higher than the Kenyan limit for aflatoxin in food (USAID 2003). Until appropriate post-harvest handling and storage methods for maize are adopted by the local population, aflatoxin poisoning will continue to be a public health concern.

In Uganda, research in the 1960s and early 1970s indicated that a significant portion of the population was regularly exposed to aflatoxin-contaminated foods (Kaaya and Warren 2005). Because Sudan is the leading world producer of groundnuts (Younis and Malik 2003), the high standards for the export market have resulted in thorough sorting of peanut products to eliminate contaminated kernels. However, contaminated kernels may still find their way into the local market, particularly for oil processing factories (Idris et al. 2010).

The East African Community, the regional trade body, is developing aflatoxin standards for food and feed. However, the majority of the people in East Africa are subsistence farmers who produce and consume their own foods which do not pass through the government regulatory systems (Wild 2007). Grains sold at local markets are also not commonly screened for mycotoxins, thus consumers of foods from such markets are also at a risk of exposure. Poverty and food insecurity in the region has led to further increased mycotoxin exposure as the people are more concerned with having food to eat without regard of the quality or risks (Shephard 2008). Additionally, alternative uses for highly contaminated crops need to be developed.

7.3. Impact in Southern Africa

Several aflatoxin surveys have been conducted in Botswana, Malawi and South Africa. In Botswana, half of the maize meal samples contained aflatoxins at concentrations greater than 20 ppb (Mphande et al. 2004). In Malawian brewing grains, aflatoxin levels of up to 1020 ppb were reported. Malted maize and millet are used to make local brews that are widely consumed in Kenya and Malawi. Previous studies determined that toxins present in grains are not affected by normal cooking temperatures, indicating that beer may be contaminated with aflatoxins due to the use of contaminated grains in production (Kenji et al. 2000). Aflatoxin levels 30 times higher than the South African legal limit (10 ppb) have been reported in peanut butter given to school children in Eastern Cape, South Africa (MRC 2006). Researchers in South Africa have also noted a high incidence of mycotoxin contamination in maize porridge (Shephard et al. 2002).

7.4. Impact in West and central Africa

Exposure to aflatoxin is widespread in West Africa, probably starting in utero, and blood tests have shown that a very high percentage of West Africans is exposed to aflatoxins (Bankole and Adebanjo 2004). Aflatoxin-albumin adducts were detected in 99% of children in Benin and Togo (Gong et al. 2003). Over 90% of West Africans were reported to contain detectable levels of aflatoxin-albumin adducts, with exposure occurring throughout life, including in utero and via breast milk (Turner et al. 2000; Wild et al. 2000). In Benin and Togo, 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 aflatoxin-albumin ever measured have been reported in children aged 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 700–1393 ng/ml and 60–148 ng/ml, respectively, in infertile men and were significantly higher than those in fertile men.

Burkina Faso, Ghana, Nigeria, Senegal and Togo and have recorded aflatoxin contamination at varying levels in sorghum, maize, cotton seeds, groundnuts and groundnut products, yams and cassava. Studies in Ghana that collected samples from major processing sites in Accra reported aflatoxin levels that ranged from 2–662 ppb (Shuaib et al. 2010a), levels that far exceeded both Codex Alimentarius Commission and United States of America regulations. 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 (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 was 9.9–32.2% in the different agro ecological zones of Benin before storage, but this increased to 15.0–32.2% after six months of storage. All the maize samples collected from silos and warehouses in Ghana contained aflatoxins at levels ranging from 20–355 µg/kg, while fermented maize dough collected from major processing sites contained aflatoxin levels of 0.7–313 µg/kg (Kpodo 1995). Aflatoxins were detected in 98% of samples of dried yam chips surveyed in Benin with levels ranging from 2.2–220 µg/kg and a mean of 14 µg/kg (Bassa et al. 2001). Aflatoxin B_1 was detected in 54.2% of dried yam chips in Nigeria (Bankole and Mabekoje 2004) while Bankole and Eseigbe (1996) detected aflatoxins in 35% of tiger nut (*Cyperus esculentus*) in Nigeria, with concentrations ranging from 10–120 µg/kg.

As early as 1961, scientists at the National Stored Products Research Institute and the Institute of Agricultural Research, with the assistance of the Tropical Products Research Institute of London, demonstrated the susceptibility of groundnuts to aflatoxin contamination in Nigeria. The prevalence of the toxic *Aspergillus* strains on maize kernels from three agro-ecological zones in the northern part of Nigeria has been well established (Atehnkeng et al. 2008a).

In West Africa, many people are not only malnourished but also chronically exposed to high levels of mycotoxins. A study to determine the level of aflatoxin exposure among young children from Benin and Togo suggests a link with food consumption, socio-economic status, agro-ecological zones of resilience and culture-specific measures. Elevated aflatoxin levels were associated with child stunting, child mortality, immunosuppression and child neurological impairment (Gong et al. 2003). In a study carried out in the Gambia, Guinea, Nigeria and Senegal, over 98% of the subjects tested positive to aflatoxin markers (Bankole and Adebanjo 2004).

In Cameroon, researchers determined that cassava chips consumed by locals contained elevated aflatoxin levels, which may have occurred as a result of processing practices, conditions in storage facilities and long duration of storage (Essono et al. 2009).

In the Gambia, aflatoxins have been detected sera, maternal intravenous blood, breast milk and umbilical cords of patients in the maternity wards (Zarba et al. 1992). Peanuts are a staple food and the primary cash crop in the country and their common consumption results in high and prolonged exposure to aflatoxin. Furthermore, extensive research efforts have documented high liver cancer incidence resulting from childhood hepatitis B infections, lifetime dietary aflatoxin exposure and chronic hepatitis C infections (Kirk et al. 2006). The Gambia Intervention Study showed that hepatitis B vaccination can be implemented in the national immunization programs of developing countries and that immunization is highly effective in preventing chronic hepatitis B infection and the likely onset of liver cancer (Kirk et al. 2006).

7.5. Trade

With global trade of commodities susceptible to aflatoxin contamination and the impact of these food products on public health, aflatoxin regulations to facilitate trade and safeguard human and animal health have been developed. These standards vary by region and country.

7.5.1. Aflatoxin regulations

Appropriate levels of sanitary and phytosanitary standards are needed to mitigate food-borne health risks. However, import restrictions have been imposed without sufficient support in international science. In 1997, the European Commission proposed a uniform standard for total aflatoxins, setting the acceptable level of the contaminant in food products. It set a standard of 4 ppb total aflatoxin in cereals, edible nuts, dried and preserved fruits and groundnuts intended for direct human consumption, and 10 ppb in groundnuts subject to further processing. It also established the maximum allowable level for aflatoxin M_1 in milk at 0.05 ppb. For eight European Union members (Belgium, Greece, Ireland, Italy, Luxembourg, the Netherlands, Spain and Sweden), the new regulation resulted in a reduction in the level of acceptable food imports by more than 50%.

In part as a result of the objections raised by European trading partners, the commission relaxed the aflatoxin standard for cereals, dried fruits and nuts. A July 1998 commission regulation established the total aflatoxin standard in groundnuts subject to further processing at 15 ppb (8 ppb for aflatoxin B_1) and in other nuts and dried fruit subject to further processing at 10 ppb (5 ppb for aflatoxin B_1). A more stringent standard on cereals and dried fruits, and nuts intended for direct human consumption was set at 4 ppb (2 ppb for aflatoxin B_1). According to the revised regulation in March 2001, European Union members were to implement the necessary laws to comply with the new standards no later than April 2002.

The World Trade Organization Agreement on Sanitary and Phytosanitary Standards encourages member countries to harmonize national standards with international standards and recommendations developed by other World Trade Organization member governments in international organizations, such as the joint Food and Agriculture Organization of the United Nations (FAO)/WHO Codex Alimentarius Commission for food safety. The Codex Alimentarius Commission permits importing countries to impose more stringent measures than the international standards. Real world experience indicates that the costs of regulatory intervention can be high. The loss arising from rejection is not limited to the value of the product. It also includes transportation and other export costs. How regulatory costs for exporters compare with possible gains in the higher sanitary and phytosanitary levels in importing countries is a key part of today's trade policy. In general, a tighter standard on aflatoxin B_1 is applied to food products intended for direct human consumption compared to those subject to further processing.

Table 9 outlines the more common aflatoxin standards for peanuts, maize and milk. A full list of aflatoxin standards by country is provided in Appendix 1. These stringent aflatoxin regulations have raised questions and concerns, particularly on the impact the standards have on trade.

Table 9: Aflatoxin standards for peanuts, maize and milk

Standard	Product	Total aflatoxin (ppb)	Aflatoxin B_1 (ppb)	Aflatoxin M_1 (ppb)
Codex Alimentarius Commission	Peanuts, for further processing	15		
	Milk			0.5
European Union	Peanuts, to be sorted	15	8	
	Peanuts, processed	4	2	
	Maize	4	2	
	Maize, to be sorted	10	5	
	Milk			0.05
United States of America	Maize, processed peanuts	20		
	Milk			0.5

7.5.2. Impact of aflatoxin regulations on trade

In 2001, a study estimated that African food exporters lost 670 million United States dollars (USD) per year by not meeting European Union safety standards alone (Otsuki et al. 2001). According to FAO, only 15 African countries had regulatory limits for aflatoxins as of 2003 (Van Egmond and Jonker 2004), but even in countries with regulations, food that does not move through formal market channels, e.g. almost all food sold in local markets in Africa, is effectively unregulated. Contamination is proving to be a major obstacle in linking African farmers to export markets as aflatoxin contamination prevents commodities from meeting international, regional and local regulations and standards governing agricultural trade and food safety. Up to 25% of world food crops are affected and countries that are situated between 40°N and 40°S are most at risk (Van Egmond and Jonker 2004). The cost of compliance with these aflatoxin regulations can exceed total government development budgets for all expenditures in the least developed countries (Otsuki et al. 2001).

The joint FAO/WHO Expert Committee on Food Additives estimated that implementing a 10 ppb total aflatoxin standard leads to a risk of 39 cancer deaths per year per billion people, with an uncertainty range between 7 and 164 people. In comparison, a 20 ppb standard yields a risk of 41 cancer deaths per year per billion people with an uncertainty range between 8 and 173 people. It is therefore estimated that lowering the aflatoxin threshold from 20 to 10 ppb in countries where the percentage of carriers of hepatitis B1 is around 1% (e.g. countries in the European Union) would result in a drop in the population risk of approximately two cancer deaths a year per billion people. Approximately 0.2 cancer deaths will be prevented each year by tightening total aflatoxin standards by 1 ppb (Otsuki et al. 2001).

If both market and heath economic impacts of mycotoxins can be estimated, the cost-effectiveness of different interventions to reduce mycotoxin risk can then be assessed. Various studies have attempted to quantify the potential market losses associated with mycotoxins in crops. In the United States of America, Vardon et al. (2003) estimated the total annual losses due to three mycotoxins (aflatoxin, fumonisin and deoxynivalenol) to reach as high as USD 1 billion. Almost all of this loss was borne by maize, groundnut and wheat growers. However, a small portion of this loss was estimated to be suffered by livestock producers due to adverse animal health effects.

In three Asian countries (Indonesia, the Philippines and Thailand), the total estimated annual loss due to aflatoxin was about half a billion Australian dollars (Lubulwa and Davis 1994). The loss was

a combination of market impacts, through rejected lots with excessively high mycotoxin levels, and adverse health effects, specifically, the impacts of liver cancer in these populations (Table 10).

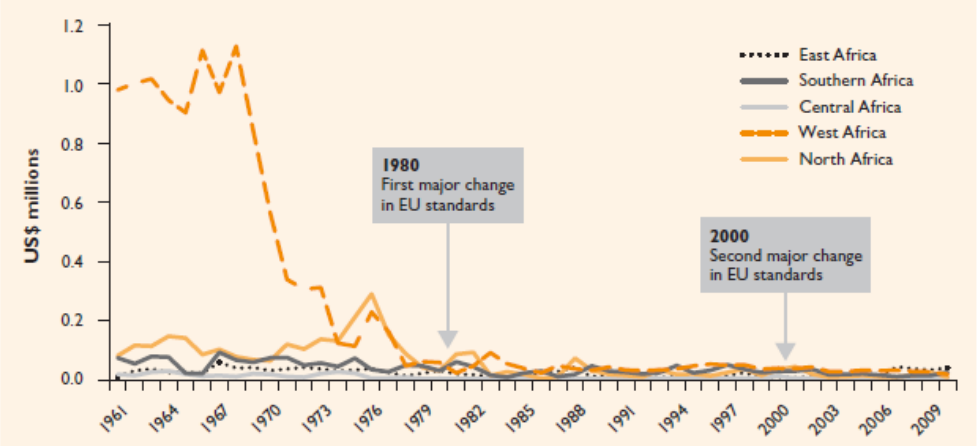
Table 10: Estimates of the 1991 annual social costs of aflatoxins in Indonesia, the Philippines and Thailand

Sector	Impact of aflatoxin considered	Parameter used in social cost estimation	Total cost for three countries (million Australian dollars)		
			Maize	Peanuts	Maize and peanuts
Grains sector households	Product spoilage effects	Change in wastage rates and post-harvest costs	70.9	36.8	107.7
	Human health effects	Cost of premature death due to aflatoxin-related primary liver cancer	112.7	73.2	185.9
	Human health effects	Cost of disability due to aflatoxin-related primary liver cancer	63.8	41.5	105.3
Poultry	Increased mortality rates and reduced feed-to-weight conversion	Reduction in the unit cost of production when the aflatoxin content of feed is reduced	28.9	2.5	31.4
Hen eggs	Increased mortality rates and reduced feed-to-weight conversion	Reduction in the unit cost of production when the aflatoxin content of feed is reduced	6.6	0.6	7.2
Pig meat	Increased mortality rates and reduced feed-to-weight conversion	Reduction in the unit cost of production when the aflatoxin content of feed is reduced	36.2	3.1	39.3
Total			319.1	157.7	476.9

Source: Lubulwa and Davis (1994).

Wu (2004) estimated the market impacts on the world's top maize and groundnut exporting countries of conforming to hypothetical harmonized standards for fumonisin in maize and aflatoxin in groundnuts. If the current United States of America standard for total aflatoxin level (20 µg/kg) were adopted worldwide, total annual groundnut losses in exporting countries would be USD 92 million, whereas if the European Union standard (4 µg/kg) were adopted worldwide, the total annual losses would increase to USD 450 million.

However, Xiong and Beghin (2011) showed that the standards set by the European Union had no significant trade impact on groundnut exports from Africa across various methods of estimation. Their findings concluded that the trade potential of African groundnut exporters is more constrained by domestic supply issues rather than by limited market access. African exports were already declining and African exporters would likely not have met the earlier less restrictive standards either (Figure 14).



Source: Roy (2013).

Figure 14: Value of exports of shelled groundnuts from African regions.

However, standards are meant to protect human health and testing alone is an inefficient and ineffective approach to the control of food contaminants (Clarke and Fattori 2013). Adopting good practices at all stages of the food chain to minimize infection by toxigenic moulds and the accumulation of mycotoxin contamination is the best way to reduce levels of these fungal toxins in the food supply.

7.5.3. Mapping of surveys with aflatoxin levels above European Union standards

Maps were developed from the literature review of aflatoxin surveys. From the maps, it is apparent that many countries in sub-Saharan Africa have aflatoxin contamination levels well above the European Union aflatoxin standards for groundnuts. Many parts of the world, including sub-Saharan Africa, also have contamination levels above the European Union standards for milk. These maps highlight that many countries have both high levels of contamination and a need for aflatoxin risk mitigating strategies, particularly in groundnuts and milk. These maps may under-estimate contamination levels, as only published surveys were mapped and those countries without surveys would appear to lack aflatoxin contamination at levels above European Union standards.

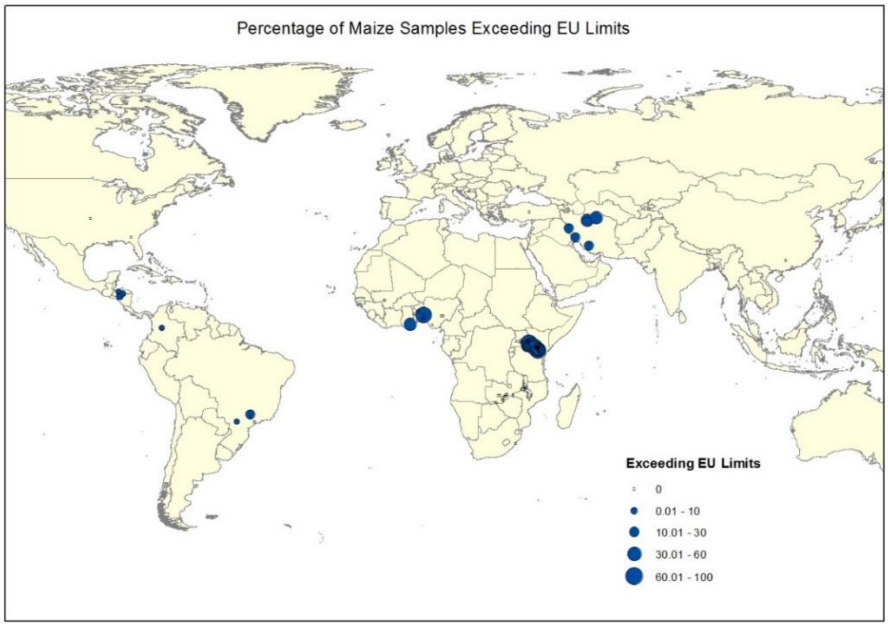


Figure 15: Percentage of maize samples with aflatoxin levels above European Union limits.

From Figure 15, it appears that the number of surveys with maize samples containing aflatoxins at levels above European Union standards is quite low. However, many surveys did not specifically state whether they had samples containing aflatoxins at levels above European Union limits. However, despite the small number of surveys mapped, there was a high percentage of samples with aflatoxin levels above European Union standards, indicating high frequency of contamination. From the surveys of aflatoxin-contaminated groundnuts, a high percentage of samples had aflatoxin levels above European Union limits, especially in parts of sub-Saharan Africa (Figure 16).

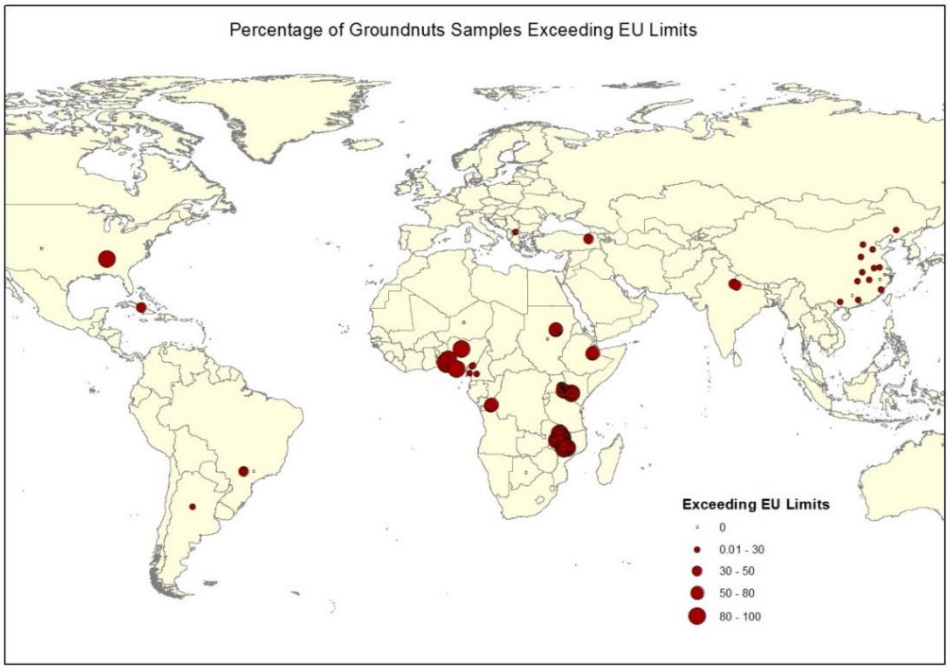


Figure 16: Percentage of groundnut samples with aflatoxin levels above European Union limits.

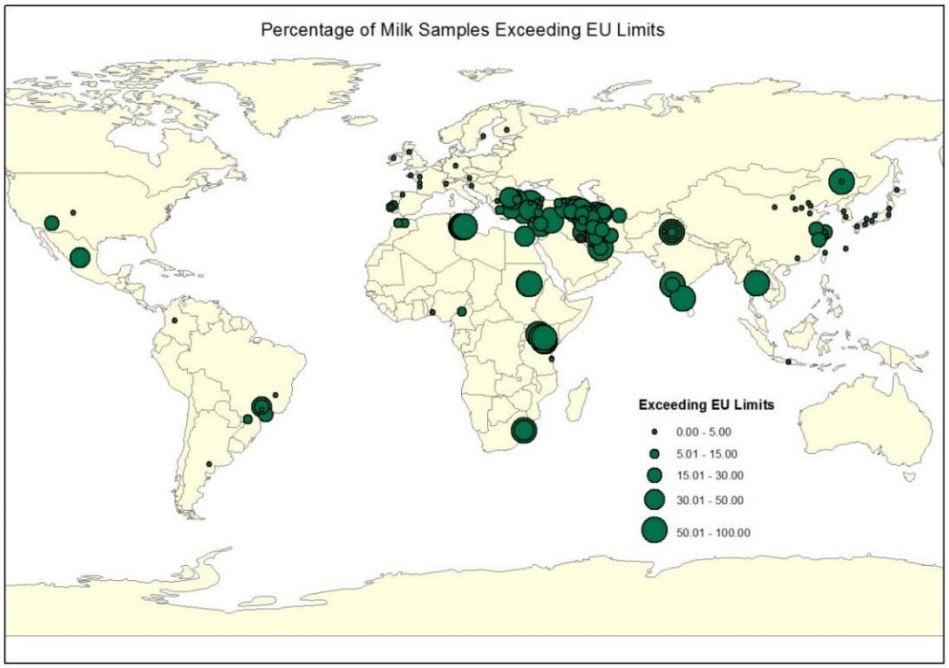


Figure 17: Percentage of cow milk samples with aflatoxin levels above European Union limits.

A large number of countries have reported aflatoxin levels in milk above European Union limits (Figure 17). The 0.05 µg/kg standard is quite strict, especially for countries with favourable environmental conditions that make it hard to control aflatoxin levels in grains and other ingredients that comprise animal feeds. Given the large number of aflatoxin-positive milk samples and the fact that only about 7% of aflatoxins ingested by cows ends up in their milk, it appears from the maps that there is a high degree of contamination in grains typically fed to dairy cattle.

7.5.4. Mapping of surveys with aflatoxin levels above United States of America standards

The United States of America has less strict aflatoxin regulations than the European Union. However, as the maps show, even meeting less stringent aflatoxin standards, particularly in milk, would still be difficult for countries in sub-Saharan Africa. These maps may underestimate contamination levels, as only published surveys were mapped and those countries without surveys would appear to lack aflatoxin contamination at levels above United States of America standards.

It appears that the number of surveys with maize samples containing aflatoxins at levels exceeding the United States of America standards is quite low (Figure 18). However, many surveys did not specifically state whether they had samples containing aflatoxins above these standards. The few studies mapped had a high percentage of samples with aflatoxin levels above the United States of America standards, indicating high frequency of contamination.

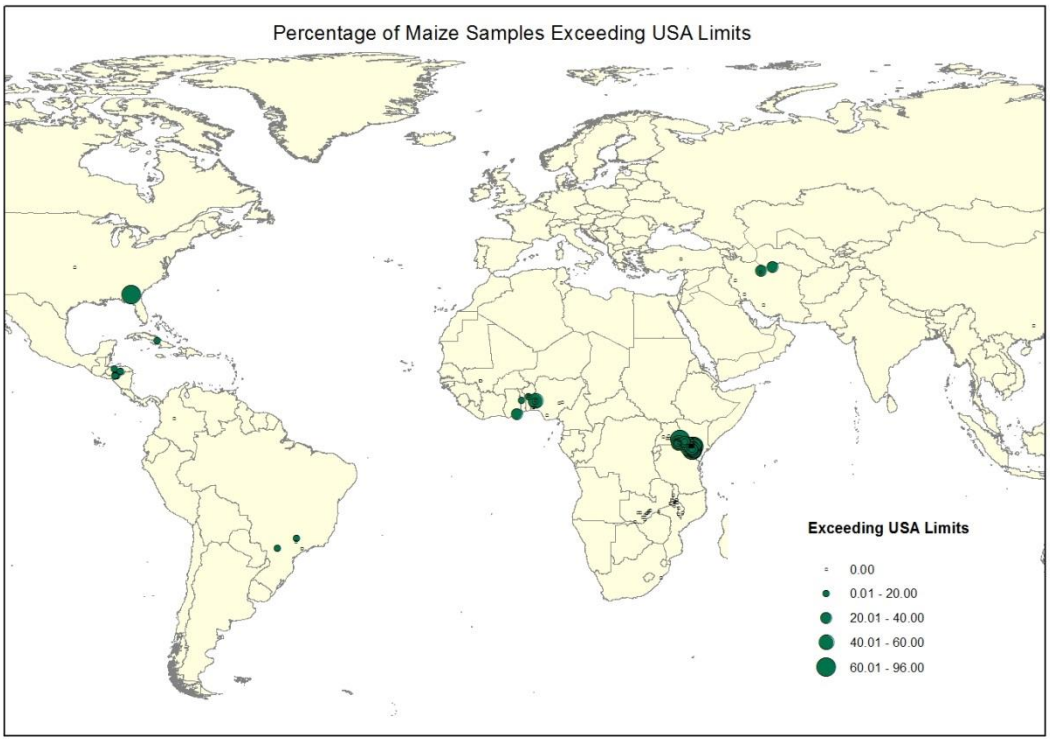


Figure 18: Percentage of maize samples with aflatoxin levels exceeding United States of America limits.

Similarly, mapping of surveys of groundnut samples contaminated with aflatoxins found that a high percentage of samples contained aflatoxins at levels above United States of America limits, especially in parts of sub-Saharan Africa (Figure 19).

Despite the less stringent standards for aflatoxin in milk in the United States of America (0.5 µg/kg), many countries still had a high percentage of samples containing aflatoxins at levels above the country's standards (Figure 20). Appendix 2 contains maps detailing the number of surveys carried out, by commodity and country.

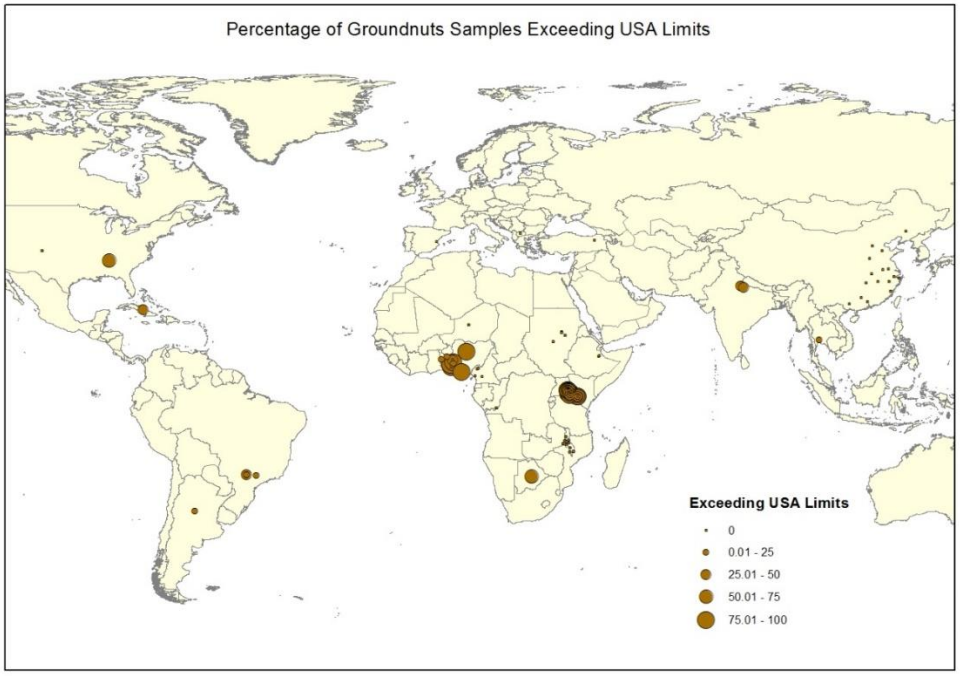


Figure 19: Percentage of groundnut samples with aflatoxin levels exceeding United States of America limits.

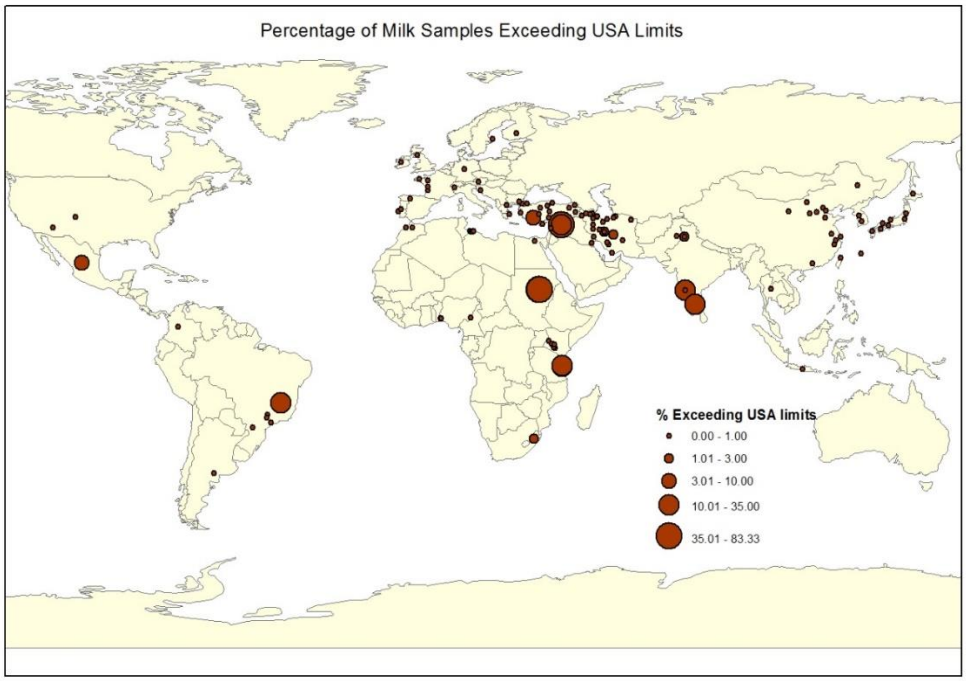


Figure 20: Percentage of cow milk samples with aflatoxin levels above United States of America limits.

8. Prevention and control

Aflatoxin contamination is a serious health concern present throughout the entire food chain, necessitating a multidisciplinary approach to analysis, action and solution. Current technologies in improved field management, pre-harvest and post-harvest practices and public information campaigns can all potentially reduce the risk of aflatoxin contamination in foods and feeds and reduce human and animal exposure (James et al. 2007; Mutegi et al. 2007).

8.1. Field management and pre-harvest practices

Most aflatoxin problems begin and develop in the field (Khlangwiset and Wu 2010). Strategies are needed to prevent infection of growing plants by toxigenic moulds. Pre-harvest management interventions seek to reduce or eliminate fungal infection in the field. Damage by insects, birds and stress, such as drought, predispose crops to *A. flavus* infection.

8.1.1. Good agricultural practices

Field management practices that increase crop yields can also reduce the risk of aflatoxin development. Listed below are pre-harvest good agricultural practices in groundnut production (Waliyar et al. 2013).

- Use of *A. flavus* resistant or tolerant varieties
- Selection of healthy seeds
- Early planting
- Avoidance of mono-cropping
- Application of *Trichoderma* at 1 kg/hectare
- Ploughing before sowing
- Appropriate weeding
- Application of farmyard manure at 2.5 tonnes/hectare before planting
- Treatment of foliar diseases using 1–2 sprayings of Kavach
- Application of lime or gypsum at 400 kg/hectare at flowering
- Mulching with crop residues at 40 days after planting
- Maintenance of optimal density of plants in the field
- Avoidance of end-of-season drought through irrigation (if possible)
- Removal of dead plants from the field before harvest

Resistant or tolerant varieties

Developing genetic resistance to *Aspergillus* spp. in maize and groundnuts is a high priority. Worldwide, the advantages of using resistant plant genotypes include direct health and economic benefits, the lack of impact on crops or the environment and the ability to use these genotypes in combination with other aflatoxin control strategies (Menkir et al. 2006). For instance, groundnut genotypes with drought resistance have also shown aflatoxin reduction (Holbrook et al. 2006; Guo et al. 2008).

Despite global efforts, progress in breeding for aflatoxin resistance in groundnuts has been limited due to the low level of resistance to different components of resistance, variable performance due to high genetic and environmental interaction, lack of reliable screening protocols and limited understanding of genetics of resistance (Nigam et al. 2009). Studies indicate that the three components of resistance (seed infection, seed colonization and aflatoxin production) are inherited independently (Waliyar et al. 2007a). Efficacy in reducing aflatoxin has been shown to be as high as 90–98% in resistant maize varieties developed and tested in the United States of America (Guo et al. 1996). Groundnuts bred for aflatoxin resistance in the United States of America achieve at least 70% reduction in pre-harvest aflatoxin contamination in multiple environments (Holbrook et al. 2006). Similarly, naturally aflatoxin-resistant lines in India had significantly lower aflatoxin levels compared to susceptible lines and produced higher pod yields, reducing aflatoxin contamination to less than 4 ppb (ICRISAT 2006).

Other plant traits may also help to some extent to mitigate the problem of aflatoxin contamination. These traits include short growth duration to match the period of soil moisture availability to avert

moisture stress, uniform pod maturity and longer root systems to extract moisture from the deeper soil layers to maintain plant–water status (Nigam et al. 2009).

Pre-harvest aflatoxin contamination is most frequent in groundnuts cultivated under rain-fed conditions. Drought and heat stress at maturity exacerbate fungal invasion, and high temperature and humidity during harvesting and storage lead to further fungal invasion and higher production of the toxin in the kernels (Waliyar et al. 2007a). Under the present circumstances, genetic resistance alone cannot eliminate the problem of aflatoxin contamination in groundnuts unless it is accompanied by other good management practices such as soil amendments, biocontrol, soil–water management, soil pest control and proper drying, curing and storage.

Biocontrol

Biocontrol of aflatoxin refers to the use of living organisms to reduce the incidence, growth and/or pathogenicity of *Aspergillus* spp. in susceptible crops in order to reduce aflatoxin contamination. The most widely used biocontrol method employs atoxigenic strains of *Aspergillus* that can competitively exclude toxigenic strains from colonizing crops. There is a strong correlation between the presence of atoxigenic strains and aflatoxin reduction (Dorner and Horn 2007). Toxigenic strains of *A. flavus* are more aggressive colonizers than atoxigenic strains. Therefore, achieving long-term aflatoxin reduction would depend on maintaining a high atoxigenic to toxigenic strain ratio in soil through repeated applications of the atoxigenic strain in years when susceptible crops are grown (Dorner and Horn 2007).

The International Institute of Tropical Agriculture (IITA) has developed Aflasafe™, a biocontrol technology that introduces native local atoxigenic strains of *A. flavus*. The introduced atoxigenic strain competitively displaces toxigenic *A. flavus*, drastically reducing its population. Aflasafe™ is broadcast by hand 2–3 weeks before the flowering stage of maize to prevent the aflatoxin-producing fungus from colonizing and contaminating the crop while it remains in the field and subsequently in storage. Aflasafe™ can also be used in fields of groundnuts and chilies. Field testing of Aflasafe™ in Nigeria between 2009 and 2012 consistently showed a decrease in contamination in maize and groundnuts by 80–90% or more (Cotty and Bandyopadhyay 2013). Per hectare, Aflasafe™ costs USD 18 and provides crop protection in the field and during storage. In Senegal, a major groundnut exporter, use of Aflasafe™ would annually add USD 281 million in value to groundnut exports (Bandyopadhyay et al. 2013a).

Other biocontrol methods by competitive exclusion have been developed (Dorner et al. 2003; Dorner and Lamb 2006; Pitt and Hocking 2006; Khanafari et al. 2007; Atehnkeng et al. 2008b; Alaniz-Zanon et al. 2013). The International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) has used isolates of *Trichoderma* spp. and strains of *Pseudomonas* and *Actinomycetes* for reducing groundnut seed colonization by competitive exclusion or inhibition of aspergilli (Waliyar et al. 2007a).

Soil moisture and temperature

Soil surface temperature greatly influences fungal communities, with densities decreasing when daily average soil temperature is either below 18°C or above 30°C and the proportion of *A. flavus* belonging to the S strain, which produces more aflatoxins, increasing as soil temperature increases (Jaime-Garcia and Cotty 2010). Another study found similar results. Growth of *A. flavus* was optimal between 25°C and 30°C while aflatoxin B_1 production was optimal at 25°C (Giorni et al. 2007). Infection and aflatoxin concentration in groundnuts can be related to the occurrence of soil moisture stress during pod-filling when soil temperatures are near optimal for *A. flavus* (Craufurd et al. 2006).

Insect damage

The most important insects that spread *A. flavus* in pre-harvest maize are in the lepidopteran family: ear borer *Mussidia nigrivenell* (snout moth), *Sitophilus zeamais* (maize weevil), *Carpophilus dimidiatus* (cornsap beetle) and *Spodoptera frugiperda* (fall armyworm). Damage by these pests is correlated with higher aflatoxin levels (Hell et al. 2000; Khlangwiset and Wu 2010; Ni et al. 2011).

In groundnuts, *A. flavus* was commonly associated with kernels from broken pods. Damage to shells which occurs while the crop is in the ground was found to predispose the kernels to contamination with aflatoxin (Bankole et al. 2006). Additionally, infection of groundnut plants with the root-knot nematode *Meloidogyne arenaria* can lead to an increase in aflatoxin contamination of groundnut kernels when the plants are subjected to drought stress during pod maturation (Timper et al. 2004).

Farmyard manure, lime and gypsum

The application of lime and farmyard manure significantly reduced aflatoxin contamination, especially in susceptible cultivars. The application of lime alone reduced aflatoxin contamination by 79% and farmyard manure reduced aflatoxin content by 74%. The above practices help ensure that groundnut plants have the nutrients they need for good growth and that healthy plants that are able to withstand *A. flavus* infection. However, the pod yield was not sufficient with the use of lime or farmyard manure, indicating that the technology may face difficulties in adoption. Additionally, the lack of lime and farmyard manure is a major constraint (Waliyar et al. 2007b).

Timely harvesting

Aflatoxin contamination increases with delays in pod removal after lifting the plants and during storage. In addition, traditional heap drying enhances rapid fungal proliferation and toxin production. Small and immature seeds (gleans) contain the highest levels of toxin and segregating such seeds reduces contamination in the final product. Replacement of farmers' traditional practice of 'heap' drying with window drying of lifted plants has dramatically reduced contamination (Waliyar et al. 2007a).

Other practices

In general, early sowing of groundnuts results in greater pod yields, less infection and lower aflatoxin concentration (Craufurd et al. 2006). Practices that prevent fungal infection and proliferation include summer ploughing, selection of appropriate planting dates to take advantage of periods of rainfall to avoid end-season drought effects, seed dressing with systematic fungicides or biocontrol agents, maintaining good plant density in the fields, removal of premature dead plants, managing pest and diseases, timely harvesting, exclusion of damaged and immature pods, quick pod drying, controlling storage pests and storing the pod or seed with less than 10% moisture (Waliyar et al. 2007a). The use of safe and efficient mechanical threshers and seed storage bins are other cultural practices for reducing aflatoxins in groundnuts.

Ultimately, a combination of pre-harvest strategies, as described above, may be needed to adequately prevent aflatoxin contamination in the field. Although most of the options are cost-effective and practical under subsistence farming conditions, they have largely not been adopted by farmers mainly due to lack of access to inputs, lack of education about the risks of aflatoxins and lack of market incentives for aflatoxin-safe groundnuts (Waliyar et al. 2007a).

8.2. Post-harvest management practices

Post-harvest interventions that reduce aflatoxins include early harvesting; rapid and proper drying, sanitation, packaging, sorting, cleaning, drying and smoking; post-harvest insect control; use of botanical or synthetic pesticides as storage protectants and proper transportation. Contamination is increased by rain during harvest, drying on soil, poor handling during harvest and transport and storage in humid, warm, insect-infested containers and structures (Beed 2013). Food is vulnerable to contamination until it is consumed.

8.2.1. Post-harvest handling of groundnuts

Because the common practice of allowing the groundnuts to dry out in the field predisposes the kernels to *A. flavus* infection (Bankole et al. 2006), ICRISAT has tested various groundnut harvesting practices that help reduce aflatoxin levels. Some of the best harvesting and drying techniques tested, such as avoiding damage to pods, harvesting at right maturity and proper drying of pods reduced aflatoxin by 69–88%. The proper drying technique essentially involves lifting the plants and laying them with foliage directly on the ground and the pods sitting on the foliage, facing the sun. With farmers' practice of removing pods nearly one month after harvesting the crop, the aflatoxin content ranged from 77–342 µg/kg. Removing of pods immediately after lifting reduced aflatoxin contamination by 60% while removal of pods two weeks after harvest reduced contamination by 30%. Pods left in the soil (gleaned pods) had the highest aflatoxin contamination, ranging from 99–413 µg/kg in susceptible varieties compared to 7–11 µg/kg in resistant cultivars. In Nigeria, the recommended method of drying the pods facing the sun reduced aflatoxin contamination by as much as 97% compared to the farmers' method of window drying (Waliyar et al. 2007b).

A package of groundnut post-harvest interventions studied in Guinea included hand sorting to remove visibly mouldy and damaged shells and kernels, drying on natural fibre mats instead of on the ground, training on how to determine the completeness of sun drying, storage of dried groundnuts in natural fibre bags, storage of groundnut bags on wooden pallets and use of insecticide on the floor of the storage facility under the wooden pallets. The cost of these locally available materials for the intervention package was USD 50 per household. Exposure was more than halved five months after harvest in individuals from the intervention villages (Turner et al. 2005).

8.2.2. Post-harvest handling of maize

In maize, removing existing aflatoxin contamination is possible by sorting aflatoxin-contaminated kernels from relatively cleaner ones. This can be done by either simple physical methods (e.g. hand sorting) or flotation and density segregation methods. These sorting methods have been shown to significantly reduce aflatoxin levels in post-harvest maize (Kabak et al. 2006).

After sorting, there are several methods to prevent the growth of *Aspergillus* and hence reduce post-harvest aflatoxin contamination. These include control of moisture levels in stored crops, storage temperature, insect pests and rodents (Kabak et al. 2006). Combinations of these methods to reduce post-harvest contamination have been tested for efficacy in actual rural village conditions.

In Uganda, field drying of maize is a traditional practice carried out by farmers. However, delayed harvesting is associated with higher aflatoxin levels. A study in 2003 and 2004 found that mould incidence, insect damage and aflatoxin levels significantly increased with delayed harvest time. The results indicated that farmers should harvest no later than three weeks after maize has attained physiological maturity (Kaaya and Kyamuhangire 2006). In another study in Uganda,

aflatoxin contamination of harvested maize was positively related to leaving maize to dry in the field for more than three weeks, drying maize without husks, drying maize on the bare ground, shelling maize by beating, heaping maize on the floor during storage and storing maize in baskets. Sorting before storage, storage of maize in shelled form, storage of maize in bags, used of improved granaries as storage structures, storage of maize above the fireplace and use of synthetic pesticides during storage were all practices negatively related to aflatoxin development (Kaaya et al. 2006).

In a study in Benin, higher aflatoxin levels in maize were associated with storage for 3–5 months, insect damage and use of *Khaya sengelensis* bark or other local plants as storage protectants. Additionally, storage of maize in locally made baskets ('ago' made from woven raffia palms, tree branches or bamboo and 'secco', a giant basket made from *Hypparhenia diplandra*) or 'zingo', a granary with a wooden conical based posed on a stone and a thatched roof, were all associated with higher levels of aflatoxins. Lower aflatoxin levels were related to the use of storage or cotton insecticides, mechanical means or smoke to protect against pests and cleaning of stores before loading them with the new harvest (Hell et al. 2000; Gnonlonfin et al. 2013).

8.2.3. Post-harvest management in the value chain

Since cross-contamination and further fungal growth are possible further up the value chain, possible solutions at the trader, warehouse and processor levels are highlighted in Table 11.

Table 11: Possible solutions at the trader, warehouse and processor levels

Application point	Procedure
Trade/warehouse/processor	Awareness building
Trader/warehouse/processor	Monitor mycotoxin levels in stores, remove damaged corn, promote the drying of corn to optimal moisture content before storage
Warehouse/storage	Frequent cleaning of feed delivery systems and short-term storage areas; drying techniques to achieve adequate storage moisture and store product on a clean, dry surface; promote appropriate storage structures of different sizes; monitor and control pests and moisture levels
Warehouse/storage/processor	Separation of damaged and mould-infested kernels by one method or combination of methods
Processor	Enterosorption; chemical inactivation by ammonization, nixtamalization with addition of hydrogen peroxide and sodium bicarbonate, thermal processing

Source: Narrod (2011).

8.3. Dietary and food interventions

When it is not possible to control aflatoxin contamination in the field, after harvest, during outbreaks of acute aflatoxicosis or when contamination levels are uncertain, several dietary interventions are available to reduce aflatoxin-related health risks. One simple dietary intervention, where feasible, is to consume less maize and groundnuts, in favour of other food crops that have significantly lower aflatoxin contamination, such as sorghum and pearl millet (Bandyopadhyay et al. 2007). However, where it is not easy to make such a dietary shift (e.g. where maize and groundnuts have traditionally been staples), other dietary interventions may be helpful.

One class of dietary interventions involves absorption of aflatoxin. Adsorbent compounds can be included in food or feed or taken separately during mealtimes to bind aflatoxin in the gastrointestinal tract, resulting in reduced aflatoxin bioavailability. Several materials have varying degrees of ability to bind aflatoxin, including bentonites, zeolites, diatomaceous earth, activated charcoal and fibres from plant sources (Khlangwiset and Wu 2010). One material that has proven effective in animal feed and is showing promise in human trials is calcium montmorillonite, marketed as NovaSil™ clay.

NovaSil™ clay, a naturally occurring and heat processed calcium montmorillonite, is commonly used as an anticaking additive in animal feed. Studies have shown that capsules containing NovaSil™ clay can be used to effectively reduce the bioavailability of dietary aflatoxin based on a reduction of aflatoxin-specific biomarkers (Wang et al. 2008). It has also been used to prevent aflatoxicosis in a variety of animals by tightly binding aflatoxins in the stomach and intestines, resulting in decreased bioavailability (Phillips et al. 2008). NovaSil™ clay has been shown to reduce aflatoxin toxicity on body and organ weights, feed intake and hepatic vitamin A when tested in broiler chicks. No toxicity has been found in a dose as high as 0.5% w/w in the diet (Pimpukdee et al. 2004).

Green tea polyphenols have been shown to inhibit chemically-induced cancers in animal and epidemiological studies (Groopman et al. 2008; Phillips et al. 2008). Green tea polyphenols inhibit initiation of aflatoxin-induced liver cancer in rats by modulating aflatoxin metabolism (Qin et al. 1997) and in humans, there are inverse associations between green tea consumption and cancer risk (Fujiki and Suganuma 2012).

Chlorophyllin, a derivative of chlorophyll, is a natural constituent of green vegetables in the human diet that has shown anti-carcinogenic effects in animals (Dashwood et al. 1998). Chlorophyllin appears to protect against aflatoxin by sequestering it during the digestive process and hence impeding its absorption. In addition, chlorophyllin may have enzyme-inducing properties that contribute to its mechanism of detoxification (Egner et al. 2001; Groopman et al. 2008). Side effects of chlorophyllin are rare, but may include diarrhoea and discolouration in urine and faeces.

There is increasing evidence that some lactic acid bacteria have the ability to bind aflatoxin B_1 (El-Nezami et al. 2000; Lahtinen et al. 2004; Kabak and Var 2008; Hernandez-Mendoza et al. 2009). These bacteria are important in the fermentation of many foods, including vegetables, fruits and dairy products. The main purpose of *Lactobacillus* inclusion in food has typically been fermentation, not the prevention of aflatoxin risk. Hence, inclusion of culturally appropriate fermented foods in the diet may be a feasible method of partially reducing aflatoxin risk.

For example, in KwaZulu-Natal, South Africa, the fermentation process used to produce a sour maize-based gruel or beverage called amahewu has also been shown to reduce the levels of aflatoxin B_1 while improving nutritional quality (Chelule et al. 2010). However, another study has shown that fermentation and cooking have little effect on reducing aflatoxins (Fandohan et al. 2005). These studies also highlight the risk of reusing the water used to wash contaminated grains, as the water was contaminated with aflatoxins from the maize.

The microorganisms *Nocardia corynebacteroides*, *Mycobacterium fluoranthenivorans*, *Corynebacterium rubrum* and *Rhodococcus erythropolis* have all been evaluated for their ability to degrade aflatoxin B_1 (Hormisch et al. 2004; Teniola et al. 2005; Alberts et al. 2006). Additionally, fungal lactase enzymes (Alberts et al. 2009), inorganic salts and organic acids (Shekhar et al. 2009) and methyleugenol (Sudhakar et al. 2009), a naturally occurring substance present in many essential oils and fruits, have also been studied to determine whether they can degrade aflatoxin B_1. Much of the research on biodegradation of aflatoxins has not made it beyond the laboratory. Large-scale use of biological agents to bind or degrade aflatoxins in contaminated foods may not be feasible or economically viable in developing countries.

8.4. Food processing

Physical cleaning—where mould-damaged kernels, seeds or nuts are removed from the intact commodity—may result in 40–80% reduction of aflatoxins (Park 2002). While sorting, trimming and cleaning may reduce aflatoxin concentrations in commodities, these operations may not completely remove all of the contamination. The initial condition of the grain or commodity and extent of the contamination will have an effect on cleaning efficiency.

In the milling process, aflatoxin contamination may be redistributed and concentrated on certain mill fractions, but there is no step or operation that destroys aflatoxins. In the dry milling of wheat, barley and other cereals, deoxynivalenol, zearalenone, aflatoxins and ochratoxin A were found in highest amounts in fractions of the commodity that are less likely to be used for food production (germ and bran fractions) but are diverted into animal feed (Park 2002; Bullerman and Bianchini 2007).

In the wet milling of corn, aflatoxins may be dissolved into the steep water or distributed among the by-products of the process but not destroyed. By the end of the wet milling process, aflatoxin can be found in the steep water, gluten fibre and germ, while the starch tends to be relatively free of aflatoxins (Park 2002).

In the brewing process, aflatoxin B_1 may be transferred from contaminated grains into beer. Aflatoxin B_1 was relatively stable at boiling temperatures of the mash cooking step, but was more sensitive to mash malting (protein hydrolysis), wort boiling and final fermentation with a removal of 12–27%, 20–30% and 20–30%, respectively, in these steps (Chu et al. 1975).

In a study of corn muffins made from cornmeal naturally contaminated with aflatoxins, 87% ± 4% of the initial amount of aflatoxin B_1 in the cornmeal was found in the baked muffins (Stoloff and Trucksess 1981). However, ordinary cooking of rice contaminated with aflatoxin B_1 showed an average reduction of 34%. Even further reduction was obtained with pressure cooking (78–88%) (Park et al. 2005; Park and Kim 2006). In another study, boiling corn grits resulted in an average reduction of aflatoxins of 28%, while frying the boiled grits resulted in 34–53% total reduction (Stoloff and Trucksess 1981). Production of tortillas by alkaline cooking and steeping of the corn followed by further processing into tortilla chips and corn chips led to reduction of initial aflatoxin contamination by approximately 52% in the tortillas, 84% in the tortilla chips and 79% in the corn chips (Torres et al. 2001).

The effect of the corn flake process on aflatoxin has been studied. Cooking the grits with and without sugars resulted in 64–67% reduction of aflatoxin. After toasting the flakes with and without sugar, the reduction in aflatoxin ranged from 78–85% (Lu et al. 1997).

The effect of extrusion on aflatoxin content appears to be influenced by moisture content, temperature and the presence or absence of additives. Hameed (1993) showed that extrusion alone was able to reduce aflatoxin content by 50–80% and with the addition of ammonia, either as hydroxide (0.7% and 1.0%) or as bicarbonate (0.4%), the aflatoxin reduction achieved was over 95%. Similar results were obtained when peanut meal was subjected to extrusion cooking in the absence (23–66% reduction) or presence (87% reduction) of 2–2.5% ammonium hydroxide (Cheftel 1989).

Other methods of food processing, such as extrusion processing at temperatures above 150°C, have moderate ability to reduce aflatoxin and other mycotoxins (Bullerman and Bianchini 2007). Total aflatoxin levels in peanuts were reduced by 51% after roasting, 27% after blanching and skin removal and a further 11% after grinding to make peanut butter. Overall, there was an 89%

reduction of aflatoxin concentration during the process of peanut butter production (Siwela et al. 2011). Dehulling, following 24 hours of soaking (steeping) and subsequent washing, significantly ($p < 0.05$) reduced aflatoxin B_1 content of corn flour from 900 to 150 µg/kg (Njapau et al. 1998). This same study found that roasting of whole peanut kernels significantly ($p < 0.001$) reduced the levels of aflatoxin B_1 and aflatoxin G_1 in the raw kernels from 8600 µg/kg to 1300 µg/kg and from 6200 µg/kg to 1200 µg/kg, respectively.

However, the aflatoxin levels in the corn flour and roasted peanut kernels were still unacceptably high after these traditional processing techniques. The process of producing refined peanut oil destroys aflatoxins but the peanut cake left over after oil production is contaminated (F. Waliyar, personal communication).

8.5. Hepatitis vaccination

Liver cancer is the fifth most common cancer in the world, with 80% of cases occurring in developing countries. The major risk factors for this cancer have been identified as chronic viral infections, such as hepatitis B and hepatitis C, and dietary exposure to aflatoxin. Given estimates that approximately 70% of liver cancer cases in developing countries can be attributed to hepatitis B, a safe and effective vaccine to prevent chronic hepatitis B infection could prevent more than 250,000 cases per year (Wild and Hall 2000). Hepatitis B vaccination has not been formally considered as an aflatoxin control intervention, as the vaccine itself has no impact on actual aflatoxin levels in diets. However, it reduces the synergistic impact of hepatitis B and aflatoxin in inducing liver cancer (Wu and Khlangwiset 2010b). According to the CDC website (http://www.cdc.gov), a dose of hepatitis B virus vaccine currently costs just over USD 1. It is estimated that hepatitis B vaccination costs USD 910 for every death averted and USD 23 for every DALY averted (Griffiths et al. 2005).

A regular practice now in the United States of America and some other developed nations, hepatitis B vaccination in children is still rare in many parts of the world. Vaccinating children against hepatitis B virus has shown, over the last three decades, to significantly decrease hepatitis B virus infection in several regions including Europe (Williams et al. 1996; Bonanni et al. 2003), Taiwan (Chen et al. 1996) and Thailand (Jutavijittum et al. 2005). This vaccine has already had, and will continue to have, significant impacts on liver cancer incidence, particularly in Africa and East Asia, considering that roughly 65 million out of the 360 million individuals who are chronically infected live in Africa (Kramvis and Kew 2007).

In Nigeria, aflatoxin and chronic hepatitis B infection account for approximately 8–27% and 59–62% of total liver cancers, respectively. Of the three aflatoxin control strategies tested in Nigeria (hepatitis B vaccination, biocontrol and NovaSil™ clay), hepatitis B vaccination resulted in the greatest reduction in number of total liver cancer cases. Out of 43,000 total liver cancer cases, it was calculated that hepatitis B vaccination, biocontrol and NovaSil™ would reduce liver cancer by 49%, 5–19% and 3–10%, respectively (Khlangwiset and Wu 2010).

9. Risk reduction challenges in Africa

Aflatoxin regulations in many least developed countries do little to protect public health, as there is limited enforcement of food safety regulations, especially among rural communities where food quality is rarely formally inspected (Shephard 2008). Subsistence farmers and local traders sometimes have the luxury of discarding obviously mouldy maize and groundnuts. In conjunction with drought, poverty and lack of other food options, people often have no choice but to eat

mouldy food or starve. Thus, regulations do little to help reduce aflatoxin and its related health effects in least developed countries (Shephard 2008; Williams 2008).

Mitigation technologies and practices can be highly effective. However, they are generally complex and require multiple steps not only to reduce prevalence of aflatoxin but also, in some cases (e.g. biocontrol), to ensure a high enough yield to cover the costs of investing in mitigation. Preliminary data suggest that some low-cost mitigation options (e.g. drying technologies) can be as cost-effective as more expensive ones (e.g. biocontrol) (Turner et al. 2005; Wu and Khlangwiset 2010b; Narrod 2011). Pre-harvest biocontrol and post-harvest interventions show the most promise for reducing exposure because they abate the greatest risk (exposure) and have been shown to be effective under conditions found throughout Africa. In the absence of low-cost testing, however, it would be difficult for large numbers of small-scale farmers and traders to sustain a market for premium grain without external support (Waliyar et al. 2007b). In many pilot projects that link small-scale farmers to markets for low-aflatoxin grain, farmers are provided with technical support and in some cases (e.g. AgResults) provided with services directly to ensure a large amount of high-quality production (Bandyopadhyay et al. 2013a). The sustainability of this type of support over time and at scale needs to be assessed. Additional market models will need to be evaluated to ensure access and sustainability.

A major incentive for farmers to invest in technologies and practices that reduce aflatoxin contamination is provided when maize, groundnuts or other crops with low levels of aflatoxin can be sold for a higher price than the same crops with high or unknown levels of aflatoxin. This difference in price is the 'price premium' or the reward to the farmer for producing a higher quality product. In one type of differentiated market, maize or groundnuts have to meet a standard based on human health consequences of aflatoxin consumption. These markets are rare in developing country domestic markets but common for exports or international food aid programs such as the World Food Programme (Méaux et al. 2013). A second type of differentiated market is where grains are used as inputs into other production processes, for example, as animal feed in the production of meat or milk, where there is a perceptible economic benefit associated with use of low-aflatoxin maize or groundnuts.

9.1. Biocontrol

The most promising biological control technique in maize and other grains involves the application of competitive atoxigenic strains of the fungus to the field to displace toxigenic strains that produce aflatoxins. The atoxigenic strains inhibit the development of the toxigenic strains, reducing aflatoxin contamination. Products based on this approach are widely used in the United States of America and have been adapted for Africa. IITA has developed Aflasafe™, a biocontrol product, for use in maize, groundnuts, chili peppers and cassava using native atoxigenic strains of *A. flavus*. The first large-scale production plant for Aflasafe™ opened in Nigeria in 2013. In Nigerian maize, using local atoxigenic strains of *A. flavus* has shown efficacy levels as high as 90%, with a cost of about USD 18 per hectare (Bandyopadhyay et al. 2013a). While the cost of Aflasafe™ is high, it has been shown under certain conditions to be profitable (Bandyopadhyay et al. 2013a). It may also provide spillover benefits in terms of protection during storage and transport and in subsequent cropping seasons (Bandyopadhyay et al. 2013b; Cotty and Bandyopadhyay 2013). In an analysis of cost-effectiveness of risk mitigating practices, biocontrol was the most cost-effective per unit of risk reduction (Narrod 2011). Other biological control options are also available and have been proven to be effective. Application of *Trichoderma* can reduce aflatoxin contamination significantly (Waliyar et al. 2005). Because of the cost, however, there is very little adoption by poor farmers.

9.1.1. Cyclopiazonic acid

In addition to aflatoxins, *A. flavus* also produces cyclopiazonic acid (CPA). CPA is found in corn, cottonseed and groundnuts, and their products. At high concentrations, CPA is toxic to humans and livestock. The levels of aflatoxins and CPA produced by various *A. flavus* isolates vary widely, ranging from isolates that produce high levels of both CPA and aflatoxins to other isolates that produce undetectable levels of the toxins. A problem with some of these biocontrol strains is that while they do not produce aflatoxins, they still produce CPA (Abbas et al. 2011). The implication of aflatoxin biocontrol on *A. flavus* ecology and population biology is a research gap.

9.2. Post-harvest intervention packages

In industrial nations, food storage and processing practices usually prevent post-harvest development of aflatoxins, but post-harvest aflatoxin accumulation remains a threat in many least developed countries. Therefore, attention to key critical control points during crop growth, harvesting, drying and storage of food is essential to reduce post-harvest aflatoxin accumulation in least developed countries (Wagacha and Muthomi 2008).

The post-harvest intervention package of Turner et al. (2005) reduced aflatoxin levels in groundnuts by 69% compared to control groundnuts. Moreover, mean serum aflatoxin albumin adducts in villagers adopting the package were 57.2% lower than in the control villagers five months after harvest. While the initial cost of this package was about USD 50 per household in 2005 to improve the storage condition of 25 groundnut bags, many components of the package last for several years (e.g. wooden drying pallets, storage bags and insecticide).

A beneficial feature of successful post-harvest intervention packages is that most aspects are a simple modification of already existing, culturally appropriate practices. In order to deliver effective post-harvest intervention packages to groundnut growers, provisions must be made for both facilities and human resources. A network of agricultural extension workers is needed to provide education in rural groundnut-growing villages of Africa to ensure broader adoption that can lead to population health benefits. With proper training from extension staff, individuals in communities may be able to educate and train other farmers in their communities to apply post-harvest intervention packages properly. For such an intervention to succeed, it is crucial to develop community interest and support. However, there may be difficulties in changing current practices. As with biocontrol, the challenge to large-scale adoption of post-harvest intervention packages is providing the right economic incentives, especially in the absence of market incentives for aflatoxin-safe products. Individual groundnut growers need motivation to undergo training and bear all the costs needed to implement this package, which can be difficult if aflatoxin is not recognized as a significant public health or market problem. In this case, unlike biocontrol, the packages cannot be applied by agricultural staff going from household to household; the growers themselves must implement the intervention.

Consideration of the fate of groundnuts sorted out because of high aflatoxin levels must also be addressed. If they are consumed by poor households who cannot afford to discard the nuts, then the poorest people in Africa would still suffer the greatest burden of aflatoxin-induced risk. Hence, as part of intervention packages, public education on health risks of aflatoxin is absolutely crucial to ensure the right economic and health incentives for groundnut growers to adopt interventions and to remove highly contaminated nuts from the human food chain. Additionally, if wood is a scarce resource in poor households, the wooden pallets may be destroyed for alternative uses (such as firewood) rather than used for their intended purpose, namely, to elevate the stored groundnut bags for post-harvest protection against aflatoxin accumulation.

Presuming that the insecticides used are already registered in the target countries, no special regulation is required for wide-scale adoption of the intervention package anywhere in Africa. Funding from external agencies may be desirable to aid in the public education efforts, as well as to offset the initial costs of the packages.

9.3. NovaSil™

A variety of dietary interventions can reduce aflatoxin-related health risks. Enterosorbents can be blended into food or feed, or taken separately (e.g. in capsule form) during mealtimes to bind aflatoxin in the gastrointestinal tract, resulting in reduced aflatoxin bioavailability in the body. One advantage of including NovaSil™ (or other effective enterosorbents) in a comprehensive plan to reduce aflatoxin risk is that it can mitigate adverse health effects even if pre-harvest and post-harvest conditions were conducive to high aflatoxin levels in food. NovaSil™ could conceivably be used in 'emergency' situations when aflatoxin levels are determined to be high in foodstuffs. In these situations it is too late to change pre-harvest or post-harvest practices to improve the food available to people at that moment, and few other options to reduce aflatoxin risk are possible. While NovaSil™ does not directly reduce aflatoxin levels in food, it can reduce aflatoxin bioavailability.

Depending on the delivery method to consumers (capsules, blended into meal or other options), NovaSil™ can be purchased or distributed in food markets or local health centres. If any part of the production chain is carried out locally, including blending the clay into meal, trained personnel are required. If NovaSil™ must be imported, transportation and delivery issues to at-risk populations are among the top priorities that need to be planned in advance.

NovaSil™ may be subject to regulations governing food additives in target nations. National and local governments, in collaboration with outside partners, need to make a financial investment for the initial subsidy of NovaSil™, as many of the most aflatoxin-vulnerable populations do not have sufficient funds to purchase quantities necessary to reduce risk through NovaSil™ consumption on a regular basis.

Additionally, it is important to consider the likelihood of adherence to a demanding regimen. For optimal effectiveness, consumers should take NovaSil™ at every meal in which aflatoxin-contaminated foodstuffs (such as maize or groundnuts) are present. NovaSil™ may be unaffordable on a daily basis in certain parts of the world where poverty is rampant and aflatoxin is a significant problem. NovaSil™ proves most cost-effective when other pre-harvest and post-harvest methods fail to prevent dangerously high levels of aflatoxin from entering the food supply (Wu and Khlangwiset 2010a).

9.4. Hepatitis B vaccination

Though the hepatitis B vaccine itself does not affect actual aflatoxin levels in diets, it reduces aflatoxin-induced liver cancer by lowering the risk of hepatitis B virus, thereby preventing the synergistic impact of hepatitis B virus and aflatoxin in inducing liver cancer. To maintain product stability, the vaccine should be stored at 2–7°C (refrigerated but not frozen). Generally, vaccines have been standardized during their manufacturing processes. The hepatitis B vaccine has been used safely for decades with low risk of significant side effects. One main technological challenge for many parts of rural Africa lies in providing and maintaining cold storage for the vaccines. It is also not optimal for individuals to have to travel too far in order to receive the vaccine.

Administration of the vaccine is another consideration. One option is to deliver the vaccine to existing hospitals, clinics and other health care centres. Another option is to deliver the vaccine

through a mobile vaccination service, travelling door-to-door as necessary, with cold storage in the medical vehicle; focusing at first on reaching everyone who had never been previously vaccinated, then focusing primarily on reaching newborn babies, if possible. Even if it were impossible to perfectly target the households with newborn babies, simply vaccinating the mothers in a broad vaccination outreach could dramatically reduce the risk of hepatitis B virus transmission to babies. Nurses, medical assistants or other trained personnel can administer the vaccines. Aside from administering the vaccine, outreach services should also be provided to educate the public on the importance of vaccination and completing the recommended regimen (a series of three or four shots).

Initiating, preparing and maintaining a vaccination program is an extremely complex task that requires governmental coordination at administrative, technical, medical, logistical, educational, financial and political levels. Fortunately, the Global Advisory Group of the Global Alliance for Vaccines and Immunization has specifically recommended that hepatitis B vaccination be integrated into national immunization programs in all countries of the world. The vaccine itself is extremely inexpensive, considering its lifetime benefit: less than USD 1 per dose, with three doses recommended per individual to provide up to 95% efficacy in protection from hepatitis B virus. However, economic issues surrounding hepatitis B vaccination in Africa are largely out of the hands of individuals (Wu and Khlangwiset 2010a).

There are many competing demands for scarce resources and availability of food is often more important than the quality of that food. Moreover, a major challenge for any intervention in food-insecure countries is that there is little price differential for quality; hence, producers may have no incentive to invest in quality enhancement (Khlangwiset and Wu 2010). Overall efficacy to reduce aflatoxin-related health risks tends to be higher for agricultural interventions (pre-harvest and post-harvest) and for hepatitis B vaccination than for dietary interventions. However, there are many times in which only dietary interventions would be helpful, such as in the case of an emergency (Khlangwiset and Wu 2010).

No single intervention to reduce aflatoxin risk in Africa emerges as being 'most feasible' in all categories. It is worth noting that aflatoxin exposure in Ghana has been shown to be significantly correlated with farmers' knowledge of aflatoxin risk (Jolly et al. 2006), while farmers' knowledge of aflatoxin risk in Benin has been correlated with motivation to implement aflatoxin-reduction interventions (Jolly et al. 2009).

In undifferentiated markets, the adoption of risk mitigating practices based solely on health benefits rather than economic incentive will be a hard sell. It will be worthwhile to explore different push mechanisms from other public health interventions (for example, HIV/AIDS testing, condom use and hand washing) to evaluate their inclusion in messaging and adoption of risk mitigating practices.

Risk mitigating practices that increase productivity will face fewer hurdles to adoption. Identifying the different practices or packages of practices will be important. It is also necessary to keep the practices to a minimum to achieve better farmer adoption and success. Practices that show immediate or near immediate benefits will also be easier to adopt. The cost of these practices must be offset by increased yield for adoption at national and regional scale to be feasible.

9.5. Willingness to pay

The risk of aflatoxin contamination and prevalence changes as products move along the value chain, as do strategies to mitigate that risk. When evaluating pre- and post-harvest interventions to

control aflatoxins, what consumers are willing to pay for the different intervention options and aflatoxin-free foods is an important determinant of what interventions are more likely to be adopted. For instance, local food procurement by the World Food Programme in Africa in 2007 encountered significant levels of aflatoxins, thus hindering its ability to access needed foods for feeding programs on the continent. Some work on willingness to pay was done in Kenya as part of the Aflacontrol project headed by IFPRI. The results are summarized in Table 12.

Table 12: Willingness to pay for aflatoxin-reduction technology in Kenya

Technology	Initial starting price (KES)	Mean price WTP (KES)	Premium/discount (-) of initial starting price	Aflatoxin reduction	Factors that impact demand for technology
Improved seed (2 kg)	240	425	77		Land assets, age of HH head (-), HHs with children aged below 5, HH head's primary occupation is non-agricultural, knowledge and perceptions of risk of aflatoxins
Metal silo (capacity 2.5 bags x 90 kg)	6500	5734	-12	60% compared to standard bags	Land owned, age of HH head (-), HHs with children aged below 5, HH head's primary occupation is non-agricultural, knowledge and perceptions of risk of aflatoxins
Drying on tarpaulin 4 x 4 m (8 bags x 90 kg)	2000	2591	30	50% compared to drying on ground	Land owned and assets, knowledge and perceptions of risks of aflatoxin
Plastic silo (capacity 2.5 bags x 90 kg)	2800	2800	0		
Biocontrol (2.5 acres)	1000	1575	58	60%	Land owned and assets

Source: Tiongco (2011)
Note: KES = Kenya shillings; WTP = willing to pay; HH = household

Some of the key findings from this study were: (1) the mean willingness to pay was higher for more cost-effective aflatoxin reduction technologies, (2) the demand for aflatoxin-reduction technologies was higher among younger farmers and those with more assets or income, (3) the perception on risk of aflatoxin contamination drove demand for improved seeds, metal silos and tarpaulins and (4) knowledge on attributes associated with moulds affected demand for improved seeds (Tiongco 2011). It is important to note that this study asked farmers what they would be willing to pay without requiring that payment.

Another study in Kenya found that consumers were clearly willing to pay a premium for visibly clean maize and maize tested and labelled aflatoxin-free. The willingness to pay was higher among younger participants and those with higher income (De Groote et al. 2011).

The evidence on consumer willingness to pay for low-aflatoxin products suggests that where farmers are also consumers, they may take health benefits into consideration when making production decisions. In addition, results from a study by IITA suggest that farmers in Nigeria are willing to pay USD 12–15 per hectare for biocontrol treatments (Bandyopadhyay et al. 2013b) and studies from Kenya and Mali also found that farmers were hypothetically willing to pay more for aflatoxin-reducing technologies (Narrod 2011).

10. Alternative uses

Even with control strategies, the issue of what to do with highly contaminated foods still needs to be addressed. In many countries there are different strategies for this. The most important of these strategies, along with their advantages, disadvantages and costs, are discussed below:

- **Diversion from feed use**: In countries like the United States of America where biofuel is a major consumer of maize, contaminated crops may be used for this purpose. Even though biofuel production may be one way to divert contaminated crops from the food market, mycotoxins are concentrated in the by-products, and since dried distiller's grain and solubles are important animal feed components, mycotoxins may still end up being sold as commercial feed and cause production losses (Wu and Munkvold 2008; Khatibi et al. 2014).
- **Destruction**: Highly contaminated cereals and feeds that cannot be safely used should be destroyed.
- **Sorting, trimming and cleaning**: Physical sorting can reduce contamination significantly. In some studies, 40–80% reductions in aflatoxins were achieved (Park 2002).
- **Extrusion and heating**: The greatest reduction in mycotoxin concentrations in extruded products seems to occur at temperatures greater than 160°C.
- **Binding**: The addition of binding agents such as zeolite clays and aluminosilicates is effective in reducing toxicity. Studies in the United States of America found that when zeolite clays were included in feed at a ratio of 200 parts feed to one part binding agent, they reduced most of the harmful effects of aflatoxins at levels of 1000 ppb for pigs and 7000 ppb for poultry. The cost was around USD 0.25 per tonne of feed (Grace 2013).
- **Charcoal, yeasts and alumina silicates** are capable of binding mycotoxins and are allowed in some countries to be used in feeds (Huwig et al. 2001). Although not common on a large-scale global level, binding and detoxifying techniques are a promising way of utilizing contaminated crops to increase the availability of safe foods. Yeast derivatives such as glucomannans and mannanoligosaccharides can increase growth in animals independent of aflatoxin levels, but can also reduce the pathogenic effects of the toxins (Aravind et al. 2003; Ghahri et al. 2009; Taklimi et al. 2012). Humic acid has also been shown to reduce the toxic effects of aflatoxins (Ghahri et al. 2009; Taklimi et al. 2012).
- **Lactic acid bacteria** are generally considered harmless food additives and are used traditionally in fermented milk products, sourdough and silage. Some strains have the ability to bind aflatoxins and may prevent the fungi from producing toxins (El-Nezami et al. 1998; Pierides et al. 2000).
- **Blending**: One method of reducing moderate levels of aflatoxin contamination is to blend contaminated grain with clean grain (blending one kilogram of grain with aflatoxin contamination five times above the limits with nine kilogram of grain with no detectable aflatoxin would result in 10 kg of grain with aflatoxins at 50% of the permissible amount). Blending of contaminated crops has been practised where highly contaminated crops are mixed with non-contaminated crops to produce a mix that has an average aflatoxin level below the legal limits. This is not allowed in the United States of America since the feed would be considered adulterated, but has been allowed on exception during unusually contaminated harvests (Bagley 1979; Price et al. 1993).
- **Ammoniation**: Other interventions aim to detoxify the contaminated products (Bata 1999; Peltonen et al. 2001). Treatment with gaseous ammonium can reduce aflatoxin levels dramatically and make feed safe and tolerated by animals (Bagley 1979). Ammoniation is a safe and effective way to decontaminate aflatoxins; it has been used with success in many countries but is not legal in others. The average costs are 5–20% of the value of the commodity.
- **Gaseous ozonisation** has also been applied and shown to have effect, especially on reducing aflatoxin B_1 (Proctor et al. 2004). However, it is not in use commercially.
- **Nixtamalization**, the traditional alkaline treatment of maize in Latin America, can reduce toxicity and has potential for wider applications.
- **Experimental treatments**: A large number of chemical, physical and microbiological methods have shown promise under experimental conditions.

- **Flexible feeding of aflatoxin-contaminated cereals to livestock**: Flexible levels of feed means that highly contaminated crops can be diverted from sensitive species to animals that are less susceptible, but only up to a certain level. Feeding to appropriate livestock is probably the best use of most aflatoxin-contaminated cereals, provided levels can be reduced to acceptable limits. There are currently no established levels at which aflatoxins can be guaranteed safe for livestock, but many animals, especially mature ones, can tolerate aflatoxins well. Indeed, many experimental studies do not show statistically significant effects of low doses of aflatoxins and there is a consistent pattern of fewer or no signs at lower doses of aflatoxins and increasing effects at higher doses. Moreover, there appear to be no scientific papers describing toxic effects of mycotoxin when present at very low levels (Boudergue et al. 2009). Growth depression associated with aflatoxins is affected by other factors than species and age. Rats on high-protein diets with 500 ppb aflatoxins had better growth than rats on low-protein diets without aflatoxins. Depending on species, age and length of trial, experiments have found no effects from aflatoxins at levels from 200–5000 ppb and significant effects at levels from 20–10,000 ppb (Grace 2013). Table 13 highlights the acceptable levels of aflatoxin for maize used in animal feeds in the United States of America.

Table 13: Guidelines for acceptable aflatoxin levels in maize for animal feed

Animal	Feed	Aflatoxin level (ppb)
Finishing beef cattle	Corn and groundnut products	300
Beef cattle, swine or poultry	Cottonseed meal	300
Finishing swine of 45 kg or greater	Corn and groundnut products	200
Breeding beef cattle, breeding swine or mature poultry	Corn and groundnut products	100
Immature animals	Corn, groundnut products and other animal feeds and feed ingredients, but excluding cottonseed meal	20

Source: FDA (1994).

- **Palliative**: If aflatoxin-contaminated feed is given to livestock, then palliative measures can reduce some of the risk. Levels of protein in feed and vitamins A, D, E, K and B should be increased as the toxin binds vitamins and affects protein synthesis. Exercise may help.

Optimally, interventions should be focused on different levels. The most effective way of reducing mycotoxins in feed and food is to avoid or minimize contamination in the crops. For this, different strategies have been developed, including drying techniques, addition of preservatives or treatments, genetically modified crops that are more resistant, breeding for more resistant variants and applying non-toxigenic strains of moulds to the crops (Dorner and Lamb 2006; Magan and Aldred 2007; Wu et al. 2008). However, in spite of decades of research and new technologies, mycotoxin continues to contaminate crops and most of the technologies available today are still inaccessible by the poorest producers.

11. Case study: Groundnuts in West Africa

This case study is from the Aflacontrol Project (2012b). Worldwide, approximately 25.7 million tonnes of groundnuts are produced annually from 21 million hectares of cropped land. Twenty-three percent of the world's groundnut production comes from sub-Saharan Africa, of which about 78% comes from West Africa. It is estimated that 95% of groundnuts produced in West Africa are consumed by the household or traded locally. The risk of aflatoxin contamination and prevalence changes as products move along the value chain, as do strategies to mitigate that risk.

Groundnuts play an important role in terms of nutrition and income for people in rural Mali who consume an average of 5–12 kg of groundnuts or groundnut paste per month. Growing of

groundnuts is a major livelihood activity for men and women throughout Mali and the share of total household income from groundnuts is relatively high. Results indicate that aflatoxin contamination results in losses of 9–11% on average. Reduction in income and wealth from aflatoxin contamination of groundnuts is higher for groundnut-producing households in high-risk areas (those areas with aflatoxin levels over 100 ppb) and more so for smallholders; losses are doubled for market-oriented households.

In 2009–10, 33–59% of groundnut samples taken from farmers' fields across the three study areas in Mali (Kayes, Koulokani, and Kita) had aflatoxin levels greater than 20 ppb. Current storage practices—such as use of gunny or plastic bags or single sacks, storage inside farmers' houses or storage in pods without bags in farmers' granaries—are a significant risk factor for contamination. Monthly sampling of farmers' granaries revealed that the aflatoxin content of groundnuts continuously increased from December 2010 (after harvest) to April 2011 in the three regions. There was a positive linear relationship between aflatoxin contamination levels and the number of months that grains were stored in farmers' granaries. Observations showed that the increases in contamination were due to poor facilities, pest damage, inappropriate cultural practices and lack of knowledge of proper storage methods.

Analysis of more than 2500 groundnut samples collected at regular intervals from traders, processors, wholesalers and retail markets revealed no exception in the prevalence of unacceptably high levels of aflatoxins. Furthermore, groundnut paste showed an extremely high level of contamination (1746 ppb and 3135 ppb in 2010 and 2011, respectively). This confirms farmers' observations that groundnuts of poor quality are set aside of making paste after sorting good kernels for markets, leading to high aflatoxin concentrations in this processed product that is commonly used and marketed as an important source of protein.

Overall, aflatoxin levels were generally higher in 2009. There was also a trend of aflatoxin remaining high in some fields from 2009 to 2010. The overall decrease in aflatoxin levels in 2010 suggests an association with weather from year to year. The tendency for some fields to retain high aflatoxin levels from year to year suggests a more local controlling factor such as soil texture, crop rotation or other farming methods. Key findings from focus groups show that knowledge regarding aflatoxin was generally poor and inadequate. Farmers generally did not consume mouldy groundnuts, as they tasted bad, and would instead use these nuts for making soap. There are, however, instances in which farmers mixed mouldy groundnuts with good ones and ground them into paste. Other key findings include (1) female decision-makers have lower pre-storage knowledge, i.e. practices like sorting and discarding bad groundnuts after drying and before storing them, (2) market-oriented households are more likely to take actions to ensure they have a better crop and to mitigate risks of aflatoxin by using storage facilities, (3) households that engage in non-agricultural jobs as a primary occupation have a higher concern about the risks of aflatoxin in purchased food, (4) households that are wealthier have more access to information, (5) consumers were willing to pay a premium for groundnuts that were tested for aflatoxins and labelled aflatoxin-free, (6) respondents would pay at least 53% less for contaminated groundnuts compared to clean groundnuts, (7) on average, producers were willing to pay 62% more than the estimated market price for improved seeds, 72% more for drying upside down, 48% more for tarpaulins and 55% more for improved granaries and (8) producers with more assets were more willing to pay for aflatoxin-reducing technologies.

Overall, the work to reduce aflatoxins in the groundnut value chain in West Africa cannot be driven by public health consequences, although public health messaging is very helpful for donor funding. However, for consumers throughout West Africa, there are too many things that can kill

them and the uncertain nature of when they will die makes it difficult to 'scare' people into adoption of aflatoxin-reduction strategies. The adoption of aflatoxin-reduction strategies in groundnuts is linked to better quality harvest, e.g. the groundnuts taste better, have a higher yield, have a shorter duration to maturity or are resistant to foliar leaf rot. Farmers have to see a difference themselves to adopt a new technology (F. Waliyar, personal communication).

12. Case study: Maize in Kenya

This case study is from the Aflacontrol Project (2012a). Maize is the main dietary staple in Kenya and is one of the crops most susceptible to contamination by aflatoxin. Aflatoxin contamination has a significant negative effect on maize production and hence results in 9.5% loss of total household income on average across all regions in Kenya. An even higher reduction of total income (20%) is experienced by smallholder maize-producing households.

A frequent misconception is that the Kenyan maize aflatoxin issue is confined to eastern Kenya. However, a significant number of samples from farmers' fields in both western and eastern Kenya contained aflatoxin levels that were above 10 µg/kg, the legal limit set by Kenyan authorities. In eastern Kenya, the mean aflatoxin level was 273.8 µg/kg and the highest level detected was 9091.8 µg/kg, representing a level 909-fold higher than the legal limit allowed by government authorities. On average, the proportion of samples with aflatoxin levels greater than 10 ppb varied between 25% and 40% in both 2010 and 2011.

During the April 2004 aflatoxicosis outbreak, research to determine characteristics of maize-producing households and traders in the outbreak area concluded that maize trade was primarily local. The majority (88%) of maize was locally grown, sold to vendors by local farmers (70%) and bought by local residents (88%). Once household stores are depleted, local farm families are likely to buy back essentially the same contaminated maize they sold to vendors, thus continuing exposure from the maize consumed at household level and from market maize (Lewis et al. 2005).

In western Kenya, the mean aflatoxin level was 13.94 µg/kg and the sample with the highest aflatoxin level contained 722.2 µg/kg. On average, 17% of samples from western Kenya had aflatoxin levels above 10 µg/kg. Overall, around 40% of all samples taken from farmers' fields in both eastern and western Kenya had aflatoxin levels of 10 ppb in February 2010. The proportion of maize with aflatoxin levels greater than 10 ppb was higher in samples taken from farmers' stores and markets than maize samples taken from farmers' fields, suggesting that current storage practices are a significant factor in high levels of aflatoxin contamination.

Analysis of the economic efficiency (measured in terms of storage costs per tonne) of the different storage structures revealed that storing maize using separate structures is the most efficient method at about 1650 Kenya shillings (KES) (about USD 17) per tonne of maize, followed by traditional cribs with round bottoms, traditional granaries with flat bottoms and improved granaries with wooden walls. On the other hand, it is costly and inefficient to store maize in baskets, metal silos or large pots.

Results from the risk assessment indicate that adding pre-harvest operational practices (such as use of drought- and pest-resistant seeds, irrigation and insecticides, and adoption of good agricultural practices and cropping system management), both singularly and in combination, reduced aflatoxin concentrations; the more operational practices added, the more reduction was seen. Findings on environmental conditions (temperature, rainfall and elevation) indicated that average temperature seemed to be more important in predicting aflatoxin prevalence in maize.

The biocontrol option was the first to be employed as it provides the greatest aflatoxin reduction at the lowest cost. The second option was the use of plastic containers. While this option is not as effective at reducing contamination as the other two remaining options (metal silos and tarpaulins), it is significantly less expensive.

Knowledge of the causes and nature of aflatoxin contamination and how to limit it or evaluate risk is generally poor. Other key findings included:

1. General level of education of households had a significant positive effect on knowledge of the risk and spread of aflatoxin contamination. However, education also had a negative effect on the degree of concern about high levels of aflatoxin prevalence and aflatoxicosis outbreak in the village. This finding suggests that households with more education are able to make decisions to take appropriate actions to prevent or reduce exposure to aflatoxin contamination.
2. Most farmers had heard about aflatoxins through local radio. Efforts to expand the effectiveness of such media are needed, especially to target farmers who have not received formal education.
3. Having children under five years of age in the household has a positive effect on knowledge of the attributes associated with safe consumption of food for humans and animals and on storage practices that minimize the formation and growth of fungi/mould.
4. Female household heads have higher knowledge about the harmful effects of feeding mouldy grains to animals and eating mouldy maize products.
5. Households in eastern Kenya (drylands), where aflatoxicosis outbreaks occurred in 2004, had a higher perception of risk (which was expected) but less knowledge of safety attributes and recommended storage practices such as use of plastic and metal silos, clay pots and plastic bags.
6. Consumers showed a high discount for contaminated maize (KES 20–30 per 2 kg) and a high premium for labelled and tested maize (KES 10–15 per 2 kg). The premium is positively associated with increased schooling and is higher in regions where aflatoxin awareness is high and where fatalities have been reported.

13. Research gaps

The immediate research needs are:

1. Human and animal health
    a. Quantifying the human health impacts and burden of disease due to aflatoxin exposure
    b. Threshold aflatoxin levels associated with adverse health effects
    c. Impacts of aflatoxin on malnutrition and stunting
    d. Whole diet assessment of aflatoxin exposure
    e. Allergenic and human health aspects of the use of atoxigenic strains of *A. flavus* in biocontrol
    f. Interpretation and application of aflatoxin B_1 adducts and urine immunoassays (aflatoxin metabolites or adducts in urine and semen indicate exposure but do not necessarily equate to adverse health effects)
    g. Interaction of toxins, malnutrition, zoonoses and stunting
    h. Impact of aflatoxins on intensive livestock and options for control
    i. The relationship between aflatoxin levels in biological specimens and levels in food

j. An early warning system designed to detect food contamination that could cause illness. To create an effective and sustainable system, health surveillance and food and biological monitoring strategies must be adapted to meet the needs of developing countries. Early warning signs need to be validated and response protocols need to be developed.

2. Risk mitigation
    a. Where risk-mitigating technologies and practices provide sufficient economic benefits to justify their adoption, more research is needed to document the extent of adoption, the factors that promote and constrain adoption within farm households in the context of risk and imperfect information, and the magnitude of the potential impacts of their adoption on aflatoxin exposure in producer households as well as local markets.
    b. Further evaluation on the sustainability, cultural acceptability, ethical implication and overall effectiveness of potential interventions
    c. Identification of critical control points for aflatoxins in food and feed
    d. Alternative uses for highly contaminated foods and feeds
    e. Small-scale decontamination of aflatoxins
    f. Gender analysis of risk-mitigating technologies especially related to access, messaging and adoption
    g. Negative or unintended consequences of risk-mitigating technologies (e.g. more income in the household and the impact this may have on women)

3. Diagnostics
    a. A simple screening method, adapted for developing countries, would benefit subsistence farmers and be useful to public health and agriculture institutions. Current field methods lack applicability and suitable action levels for developing countries.
    b. Reducing the cost and improving durability, ease of transport and usability of field methods
    c. Standard analytical protocols to enable comparison of results across laboratories and studies

14. Conclusion

Preventing aflatoxin contamination is a hidden challenge. Because aflatoxins are invisible and odourless, have an extended timeframe for the health impacts from chronic consumption to become manifest and the fact that aflatoxin consumption works in synergy with nutritional and health conditions in poor communities, the risk of aflatoxin exposure is overshadowed by larger issues of poverty, food security, child health, maternal health, infectious disease and fragmented health services. However, the risk of aflatoxin consumption is real to billions of people throughout the developing world and initial research has shown links between aflatoxin exposure and some of the larger issues of health and disease. Many studies have already established widespread exposure in populations throughout the developing world. More research is needed to strengthen the links between health and exposure to make a stronger case for scaling up research, interventions and policy.

Additional challenges include the need for diagnostics to determine contamination levels in areas where laboratory capacity is still being developed. Awareness of aflatoxins and their risks is still very low, particularly in the areas that are at highest risk for consumption of highly contaminated

food products. In addition to poor laboratory infrastructure and lack of inexpensive rapid farm-level diagnostics, aflatoxin regulations are either non-existent or poorly enforced in these high-risk areas. The need for alternative uses of highly contaminated products will need to be addressed, especially as regulations are developed and enforced.

Furthermore, the market does not usually discriminate between contaminated and aflatoxin-free products. At present, there is no price incentive for many farmers to adopt aflatoxin-reduction strategies. It can be hard to demonstrate product value to consumer health and farmer income, making it difficult to create more than niche markets for aflatoxin-safe foods.

Interventions would ideally be combined in a suite to solve aflatoxin problems in least developed countries. Delivery of the intervention to people and places in need may be the most significant challenge to implementing aflatoxin risk-reduction interventions. Understanding constraints to feasibility helps scientists and policymakers to think beyond efficacy and material costs. For interventions to succeed in less developed countries, governments, scientists, international organizations, farmers and consumers must work collaboratively to overcome challenges in implementing the interventions, namely, human resource, equipment, technology and transportation and financial requirements as well as constraints to adoption.

15. References

Abbas, H., Weaver, M., Horn, B., Carbone, I., Monacell, J. and Shier T. 2011. *Aspergillus*, aflatoxins, cyclopiazonic acid and biological control of aflatoxins. *Toxin Reviews* 30(2–3): 59–70.

Abdel-Hafez, A. and Saber, S.M. 1993. Mycoflora and mycotoxin of hazelnut (*Corylus avellana* L.) and walnut (*Juglans regia* L.) seeds in Egypt. *Zentralblatt für Mikrobiologie* 148(2): 137–147.

Abdulrazzaq, Y.M., Osman, N. and Ibrahim A. 2002. Fetal exposure to aflatoxins in the United Arab Emirates. *Annals of Tropical Paediatrics: International Child Health* 22(1): 3–9.

Abdulrazzaq, Y.M., Osman, N., Yousif, Z.M. and Al-Falahi, S. 2003. Aflatoxin M1 in breast-milk of UAE women. *Annals of Tropical Paediatrics: International Child Health* 23(3): 173–179.

Abdulrazzaq, Y.M., Osman, N., Yousif, Z.M. and Trad, O. 2004. Morbidity in neonates of mothers who have ingested aflatoxins. *Annals of Tropical Paediatrics: International Child Health* 24(2): 145–151.

Abulu, E., Uriah, N., Aigbefo, H., Oboh, P. and Agbonlahor, D. 1998. Preliminary investigation on aflatoxin in cord blood of jaundiced neonates. *West African Journal of Medicine* 17(3): 184–187.

Adebayo-Tayo, B.C., Onilude, A.A. and Patrick, U.G. 2008. Mycofloral of smoke-dried fishes sold in Uyo, Eastern Nigeria. *World Journal of Agricultural Sciences* 4(3): 346–350.

Aflacontrol Project. 2012a. *Kenya: Reducing the spread of aflatoxins in Kenya.* Aflacontrol Project Note 5. Washington, DC: IFPRI.

Aflacontrol Project. 2012b. *Mali: Reducing the spread of aflatoxins in Mali.* Aflacontrol Project Note 4. Washington, DC: IFPRI.

Afla-guard. 2005. *Aflatoxin in Africa.* (Available from www.circleoneglobal.com/aflatoxin_africa.htm) (Accessed 28 August 2014)

Afshar, P., Shokrzadeh, M., Kalhori, S., Babaee, Z. and Saravi, S.S. 2013. Occurrence of ochratoxin A and aflatoxin M1 in human breast milk in Sari, Iran. *Food Control* 31: 525–529.

Ahene, R., Odamtten, G. and Owusu, E. 2011. Fungal and bacterial contaminants of six spices and spice products in Ghana. *African Journal of Environmental Science and Technology* 5(9): 633–640.

Ahmed, H., Hendrickse, R., Maxwell, S. and Yakubu, A. 1995. Neonatal jaundice with reference to aflatoxins: an aetiological study in Zaria, northern Nigeria. *Annals of Tropical Paediatrics: International Child Health* 15(1): 11–20.

Alaniz-Zanon, M.S., Chiotta, M.L., Giaj-Merlera, G., Barros, G. and Chulze, S. 2013. Evaluation of potential biocontrol agent for aflatoxin in Argentinean peanuts. *International Journal of Food Microbiology* 162(3): 220–225.

Alberts, J.F., Engelbrecht, Y., Steyn, P.S., Holzapfel, W.H. and van Zyl, W.H. 2006. Biological degradation of aflatoxin B1 by *Rhodococcus erythropolis* cultures. *International Journal of Food Microbiology* 109(1–2): 121–126.

Alberts, J.F., Gelderblom, W.C, Botha, A. and van Zyl, W.H. 2009. Degradation of aflatoxin B(1) by fungal laccase enzymes. *International Journal of Food Microbiology* 135(1): 47–52.

Andretta, I., Kipper, M., Lehnen, C., Hauschild, L., Vale, M. and Lovatto, P. 2011. Meta-analytical study of productive and nutritional interactions of mycotoxins in growing pigs. *Animal* 6(9): 1476.

Aravind, K.L., Patil, V.S., Devegowda, G., Umakantha, B. and Ganpule, S.P. 2003. Efficacy of esterified glucomannan to counteract mycotoxicosis in naturally contaminated feed on performance and serum biochemical and hematological parameters in broilers. *Poultry Science* 82(4): 571–576.

Armbrecht, B., Wiseman, H., Shalkop, W. and Geleta, J. 1971. Swine aflatoxicosis. 1. An assessment of growth efficiency and other responses in growing pigs fed aflatoxin. *Environmental Physiology* 1: 198–208.

Atanda, O., Oguntubo, A., Adejumo, O., Ikeorah, J. and Akpan, I. 2007. Aflatoxin M1 contamination of milk and ice cream in Abeokuta and Odeda local governments of Ogun State, Nigeria. *Chemosphere* 68(8): 1455–1458.

Atehnkeng, J., Ojiambo, P.S., Donner, M., Ikotun, T., Sikora, R.A., Cotty, P.J. and Bandyopadhyay, R. 2008a. Distribution and toxigenicity of *Aspergillus* species isolated from maize kernels from three agro-ecological zones in Nigeria. *International Journal of Food Microbiology* 122(1–2): 74–84.

Atehnkeng, J., Ojiambo, P.S., Ikotun, T., Sikora, R.A., Cotty, P.J. and Bandyopadhyay, R. 2008b. Evaluation of atoxigenic isolates of *Aspergillus flavus* as potential biocontrol agents for aflatoxin in maize. *Food Additives & Contaminants: Part A* 25(10): 1264–1271.

Ayalew, A., Fehrmann, H., Lepschy, J., Beck, R. and Abate, D. 2006. Natural occurrence of mycotoxins in staple cereals from Ethiopia. *Mycopathologia* 162(1): 57–63.

Azzam, A. and Gabal, M. 1998. Aflatoxin and immunity in layer hens. *Avian Pathology* 27(6): 570–577.

Azziz-Baumgartner, E., Lindblade, K., Gieseker, K., Rogers, H.S., Kieszak, S., Njapau, H., Schleicher, R., McCoy, L.F., Misore, A., DeCock, K., Rubin, C., Slutsker, L. and Aflatoxin Investigative Group. 2005. Case-control study of an acute aflatoxicosis outbreak, Kenya, 2004. *Environmental Health Perspectives* 113(12): 1779–1783.

Bagley, E.B. 1979. Decontamination of corn containing aflatoxin by treatment with ammonia. *Journal of the American Oil Chemists' Society* 56(9): 808–811.

Bailly, J.-D. and Guerre, P. 2009. Mycotoxins in meat and processed meat products. In: Toldrá, F. (ed), *Safety of meat and processed meat*. New York, NY: Springer. pp. 83–124.

Bakker, G.J., Sizoo, E., Jekel, A., Pereboom-de Fauw, D.K., Schothorst, R. and Van Egmond, H. 2009. Determination of mean daily intakes of aflatoxin B 1, aflatoxin M 1, ochratoxin A, trichothecenes and fumonisins in 24-hour diets of children in the Netherlands. *World Mycotoxin Journal* 2(4): 451–459.

Bandyopadhyay, R., Atehnkeng, J., Mutegi, C., Augusto, J., Akello, J., Akande, A., Kaptoge, L., Beed, F., Olasupo, O., Abdoulaye, T., Cotty, P., Menkir, A. and Masha, K. 2013a. *Ensuring the food safety of African food crops.* IITA R4D Review 10 (March): 11–16.

Bandyopadhyay, R., Cotty, P. and Mutegi, C. 2013b. *Biological control of aflatoxins: Outcompeting harmful aflatoxin producers.* ILRI Media Briefing 6. Nairobi, Kenya: ILRI. (Available from http://hdl.handle.net/10568/34288) (Accessed 28 August 2014)

Bandyopadhyay, R., Kumar, M. and Leslie, J.F. 2007. Relative severity of aflatoxin contamination of cereal crops in West Africa. *Food Additives & Contaminants* 24(10): 1109–1114.

Bankole, S. and Adebanjo, A. 2004. Mycotoxins in food in West Africa: Current situation and possibilities of controlling it. *African Journal of Biotechnology* 2(9): 254–263.

Bankole, S., Schollenberger, M. and Drochner, W. 2006. Mycotoxins in food systems in Sub Saharan Africa: A review. *Mycotoxin Research* 22(3): 163–169.

Bankole, S.A. and Eseigbe, D.A. 1996. Occurrence of mycoflora and aflatoxin in market tiger nut. *Crop Research* 11: 219–223.

Bankole, S.A. and Mabekoje, O.O. 2004. Mycoflora and occurrence of aflatoxin B1 in dried yam chips from markets in Ogun and Oyo States, Nigeria. *Mycopathologia* 157(1): 111–115.

Bassa, S., Mestres, C., Hell, K., Vernia, P. and Cardwell, K. 2001. First report of aflatoxin in dried yam chips in Benin. *Plant Disease* 85: 1032.

Bata, A. 1999. Detoxification of mycotoxin-contaminated food and feed by microorganisms. *Trends in Food Science & Technology* 10(6-7): 223–228.

Beaver, R.W., Wilson, D.M., James, M.A., Haydon, K.D., Colvin, B.M., Sangster, L.T., Pikul, A.H. and Groopman, J.D. 1990. Distribution of aflatoxins in tissues of growing pigs fed an aflatoxin-contaminated diet amended with a high affinity aluminosilicate sorbent. *Veterinary and Human Toxicology* 32(1): 16–18.

Beed, F. 2013. *What are aflatoxins?* Presentation at the Science Forum 2013, Bonn, Germany, 23–25 September 2013. (Available from http://www.slideshare.net/ILRI/what-are-aflatoxins) (Accessed 28 August 2014)

Bhat, R.V. and Vasanthi, S. 2003. Mycotoxin food safety risk in developing countries. In: Unnevehr, L.J. (ed), *Food safety in food security and food trade*. 2020 Vision Focus 10, Brief 3.

Black, R.E., Allen, L.H., Bhutta, Z.A., Caulfield, L.E., De Onis, M., Ezzati, M., Mathers, C. and Rivera, J. 2008. Maternal and child undernutrition: Global and regional exposures and health consequences. *Lancet* 371(9608): 243–260.

Bonanni, P., Pesavento, G., Bechini, A., Tiscione, E., Mannelli, F., Benucci, C. and Nostro, A.L. 2003. Impact of universal vaccination programmes on the epidemiology of hepatitis B: 10 years of experience in Italy. *Vaccine* 21(7–8): 685–691.

Boudergue, C., Burel, C., Dragacci, S., Favrot, M.-C., Fremy, J.-M., Massimi, C., Prigent, P., Debongnie, P., Pussemier, L., Boudra, H., Morgavi, D., Oswald, I., Perez, A. and Avantaggiato, G. 2009. Review of mycotoxin-detoxifying agents used as feed additives: Mode of action, efficacy and feed/food safety. *EFSA Supporting Publications* 6(9). doi:10.2903/sp.efsa.2009

Boutrif, E. 1998. Prevention of aflatoxin in pistachios. In: Albert, J.L. (ed), *Food, Nutrition and Agriculture 21*. Rome, Italy: FAO. pp. 32–39.

Bryden, W., Cumming, R. and Balnave, D. 1979. The influence of vitamin A status on the response of chickens to aflatoxin B1 and changes in liver lipid metabolism associated with aflatoxicosis. *British Journal of Nutrition* 41(03): 529–540.

Bullerman, L.B. and Bianchini, A. 2007. Stability of mycotoxins during food processing. *International Journal of Food Microbiology* 119(1–2): 140–146.

Cano-Sancho, G., Sanchis, V., Marín, S. and Ramos, A.J. 2013. Occurrence and exposure assessment of aflatoxins in Catalonia (Spain). *Food and Chemical Toxicology* 51: 188–193.

Cardwell, K.F. and Hendy, S.H. 2004. Risk of exposure to and mitigation of effect of aflatoxin on human health: A West African example. *Toxin Reviews* 23: 217–247.

Cassel, K., Campbell, B., Draper, M. and Epperson, B. 2012. *Aflatoxins: Hazards in grain/aflatoxicosis in livestock*. (Available from http://pubstorage.sdstate.edu/AgBio_Publications/articles/FS907.pdf) (Accessed 28 August 2014)

CAST (Council for Agricultural Science and Technology). 2003. *Mycotoxins: risks in plant, animal, and human systems.* Ames, Iowa: CAST.

Caulfield, L.E., de Onis, M., Blössner, M. and Black, R.E. 2004. Undernutrition as an underlying cause of child deaths associated with diarrhea, pneumonia, malaria, and measles. *American Journal of Clinical Nutrition* 80(1): 193–198.

CDC (Centers for Disease Control and Prevention). 2012. *Aflatoxin.* (Available from http://www.cdc.gov/nceh/hsb/chemicals/aflatoxin.htm) (Accessed 28 August 2014)

Chao, T.C., Maxwell, S.M. and Wong, S.Y. 1991. An outbreak of aflatoxicosis and boric acid poisoning in Malaysia: A clinicopathological study. *Journal of Pathology* 164(3): 225–233.

Chavez-Sanchez, M.C., Martínez Palacios, C. and Osorio Moreno, I. 1994. Pathological effects of feeding young *Oreochromis niloticus* diets supplemented with different levels of aflatoxin B1. *Aquaculture* 127(1): 49–60.

Cheftel, J.C. 1989. Extrusion cooking and food safety. In: Mercier, C., Linko, P. and Harper, J.M. (eds), *Extrusion cooking*. St. Paul, Minnesota: American Association of Cereal Chemists. pp. 435–461.

Chelule, P.K., Mbongwa, H.P., Carries, S. and Gqaleni, N. 2010. Lactic acid fermentation improves the quality of *amahewu*, a traditional South African maize-based porridge. *Food Chemistry* 122: 656–661.

Chen, H.L., Chang, M.H., Ni, Y.H., Hsu, H.Y., Lee, P.I., Lee, C.Y. and Chen, D.S. 1996. Seroepidemiology of hepatitis B virus infection in children: Ten years of mass vaccination in Taiwan. *Journal of the American Medical Association* 276(11): 906–908.

Chu, F., Chang, C., Ashoor, S.H. and Prentice, N. 1975. Stability of aflatoxin B1 and ochratoxin A in brewing. *Applied Microbiology* 29(3): 313–316.

CIMMYT (International Maize and Wheat Improvement Center). 2004. *Food safety: Ensuring safe, healthy, nutritious food.* Mexico City, Mexico: CIMMYT.

Clarke, R. and Fattori, V. 2013. *Codex standards: A global tool for aflatoxin management.* IFPRI 2020 Focus 20, Brief 13. Washington, DC: IFPRI.

Coker, R.D., Nagler, M.J., Defize, P.R., Derksen, G.B., Buchholz, H., Putzka, H.A., Hoogland, H.P., Roos, A.H. and Boenke, A. 2000. Sampling plans for the determination of aflatoxin B1 in large shipments of animal feedstuffs. *Journal of AOAC International* 83(5): 1252–1258.

Coppock, R.W., Christian, R.R.G. and Jacobsen, B.J. 2012. Aflatoxins. In: Gupta, R.C. (ed), *Veterinary toxicology: Basic and clinical principles*. Cambridge, Massachusetts: Academic Press.

Cotty, P.J. and Bandyopadhyay, R. 2013. *Biological control of aflatoxins.* Presentation at the Science Forum 2013, Bonn, Germany, 23–25 September 2013. (Available from http://www.slideshare.net/ILRI/biological-control-of-aflatoxins-26531914) (Accessed 28 August 2014)

Cotty, P.J. and Jaime-Garcia, R. 2007. Influences of climate on aflatoxin producing fungi and aflatoxin contamination. *International Journal of Food Microbiology* 119(1–2): 109–115.

Coulter, J., Lamplugh, S., Suliman, G., Omer, M. and Hendrickse, R. 1984. Aflatoxins in human breast milk. *Annals of Tropical Paediatrics: International Child Health* 4(2): 61–66.

Craufurd, P., Prasad, P., Waliyar, F. and Taheri, A. 2006. Drought, pod yield, pre-harvest *Aspergillus* infection and aflatoxin contamination on peanut in Niger. *Field Crops Research* 98(1): 20–29.

Daniel, J.H., Lewis, L.W., Redwood, Y.A., Kieszak, S., Breiman, R.F., Flanders, W.D., Bell, C., Mwihia, J., Ogana, G., Likimani, S., Straetemans, M. and McGeehin, M.A. 2011. Comprehensive assessment of maize aflatoxin levels in Eastern Kenya, 2005–2007. *Environmental Health Perspectives* 119(12): 1794–1799.

Dashwood, R., Negishi, T., Hayatsu, H., Breinholt, V., Hendricks, J. and Bailey, G. 1998. Chemopreventive properties of chlorophylls towards aflatoxin B1: A review of the antimutagenicity and anticarcinogenicity data in rainbow trout. *Mutation Research* 399(2): 245–253.

De Groote, H., Bett, C., Kimenju, S., Narrod, C., Tiongco, M. and Scott, R. 2011. *Estimating consumer willingness to pay for aflatoxin-free food in Kenya.* Presentation at the Closing Workshop: Aflacontrol Conference, Nairobi, Kenya, 30 November 2011. (Available from www.slideshare.net/pchenevixtrench/estimating-consumer-willingness-to-pay-for-aflatoxin-free-food) (Accessed 28 August 2014)

Decastelli, L., Lai J., Gramaglia, M., Monaco, A., Nachtmann, C., Oldano, F., Ruffier, M., Sezian, A., Bandirola, C. 2007. Aflatoxins occurrence in milk and feed in Northern Italy during 2004–2005. *Food Control* 18(10): 1263–1266.

Dersjant-Li, Y., Verstegen, M.W. and Gerrits, W.J. 2003. The impact of low concentrations of aflatoxin, deoxynivalenol or fumonisin in diets on growing pigs and poultry. *Nutrition Research Reviews* 16(02): 223–239.

Dhanasekaran, D., Panneerselvam, A. and Thajuddin, N. 2009. Evaluation of aflatoxicosis in hens fed with commercial poultry feed. *Turkish Journal of Veterinary and Animal Sciences* 33(5): 385–391.

Ding, X., Li, P., Bai, Y. and Zhou, H. 2012. Aflatoxin B1 in post-harvest peanuts and dietary risk in China. *Food Control* 23: 143–148.

Doerr, J., Huff, W., Wabeck, C., Chaloupka, G., May, J., Merkley, J. 1983. Effects of low level chronic aflatoxicosis in broiler chickens. *Poultry Science* 62(10): 1971–1977.

Dorner, J.W. and Horn, B.W. 2007. Separate and combined applications of nontoxigenic *Aspergillus flavus* and *A. parasiticus* for biocontrol of aflatoxin in peanuts. *Mycopathologia* 163(4): 215–223.

Dorner, J.W. and Lamb, M.C. 2006. Development and commercial use of afla-Guard™, an aflatoxin biocontrol agent. *Mycotoxin Research* 22(1): 33–38.

Dorner, J.W., Cole, R.J., Connick, W.J., Daigle, D.J., McGuire, M.R. and Shasha, B.S. 2003. Evaluation of biological control formulations to reduce aflatoxin contamination in peanuts. *Biological Control* 26: 318–324.

Ediage, E.N., Di Mavungu, J.D., Song, S., Sioen, I. and De Saeger, S. 2013. Multimycotoxin analysis in urines to assess infant exposure: A case study in Cameroon. *Environment International* 57–58: 50–59.

Edrington, T., Harvey, R. and Kubena, L. 1994. Effect of aflatoxin in growing lambs fed ruminally degradable or escape protein sources. *Journal of Animal Science* 72(5): 1274–1281.

Egal, S., Hounsa, A., Gong, Y.Y., Turner, P.C., Wild, C.P., Hall, A.J., Hell, K. and Cardwell, K.F. 2005. Dietary exposure to aflatoxin from maize and groundnut in young children from Benin and Togo, West Africa. *International Journal of Food Microbiology* 104(2): 215–224.

Egner, P.A., Wang, J.B., Zhu, Y.R., Zhang, B.C., Wu, Y., Zhang, Q.N., Qian, G.S., Kuang, S.Y., Gange, S.J., Jacobson, L.P., Helzlsouer, K.J., Bailey, G.S., Groopman, J.D. and Kensle, T.W. 2001. Chlorophyllin intervention reduces aflatoxin-DNA adducts in individuals at high risk for liver cancer. *Proceedings of the National Academy of Sciences of the United States of America* 98(25): 14601–14606.

El-Nezami, H., Kankaanpaa, P., Salminen, S. and Ahokas, J. 1998. Ability of dairy strains of lactic acid bacteria to bind a common food carcinogen, aflatoxin B_1. *Food and Chemical Toxicology* 36(4): 321–326.

El-Nezami, H., Mykkänen, H., Kankaanpää, P., Salminen, S. and Ahokas, J. 2000. Ability of *Lactobacillus* and *Propionibacterium* strains to remove aflatoxin B, from the chicken duodenum. *Journal of Food Protection* 63(4): 549–552.

El-Nezami, H., Nicoletti, G., Neal, G., Donohue, D. and Ahokas, J. 1995. Aflatoxin M1 in human breast milk samples from Victoria, Australia and Thailand. *Food and Chemical Toxicology* 33(3): 173–179.

El-Tras, W.F., El-Kady, N.N. and Tayel, A.A. 2011. Infants exposure to aflatoxin M_1 as a novel foodborne zoonosis. *Food and Chemical Toxicology* 49(11): 2816–2819.

Elgerbi, A.M., Aidoo, K.E., Candlish, A.A. and Tester, R.F. 2004. Occurrence of aflatoxin M1 in randomly selected North African milk and cheese samples. *Food Additives & Contaminants* 21(6): 592–597.

Elzupir, A.O. and Elhussein, A.M. 2010. Determination of aflatoxin M1 in dairy cattle milk in Khartoum State, Sudan. *Food Control* 21(6): 945–946.

Essono, G., Ayodele, M., Akoa, A., Foko, J., Filtenborg, O. and Olembo, S. 2009. Aflatoxin-producing *Aspergillus* spp. and aflatoxin levels in stored cassava chips as affected by processing practices. *Food Control* 20(7): 648–654.

Ewuola, E., Jimoh, O. and Bello, A. 2013. Growth response and nutrient digestibility of West African Dwarf goats fed micro doses of dietary aflatoxin. *Scientific Journal of Animal Science* 2(11): 316–322.

Ezekiel, C., Sulyok, M., Warth, B., Odebode, A. and Krska, R. 2012. Natural occurrence of mycotoxins in peanut cake from Nigeria. *Food Control* 27(2): 338–342.

Fallah, A., Rahnama, M., Jafari, T. and Saei-Dehkordi, S.S. 2011. Seasonal variation of aflatoxin M1 contamination in industrial and traditional Iranian dairy products. *Food Control* 22: 1653–1656.

Fandohan, P., Zoumenou, D., Hounhouigan, D.J., Marasas, W.F., Wingfield, M.J. and Hell, K. 2005. Fate of aflatoxins and fumonisins during the processing of maize into food products in Benin. *International Journal of Food Microbiology* 98(3): 249–259.

Fapohunda, S. 2011. *Impact of mycotoxins on sub-Saharan Africa: Nigeria as a case study.* European Mycotoxin Awareness Network. (Available from http://www.mycotoxins.com) (Accessed 7 July 2011)

Feddern, V., Dors, G.C., Tavernari, F. de C., Mazzuco, H., Cunha, A., Krabbe, E.L. and Scheuermann, G.N. 2013. Aflatoxins importance on animal nutrition. In: Razzaghi-Abyaneh, M. (ed), *Aflatoxins – Recent advances and future prospects*. InTech, DOI: 10.5772/51952. (Available from http://www.intechopen.com/books/aflatoxins-recent-advances-and-future-prospects/aflatoxins-importance-on-animal-nutrition) (Accessed 28 August 2014)

Fellinger, A. 2006. *Worldwide mycotoxin regulations and analytical challenges*. World Grain Summit: Food and Beverages, San Francisco, CA, 17–20 September 2006.

Fernández, A., Belío, R., Ramos, J.J., Sanz, M.C. and Sáez, T. 1997. Aflatoxins and their metabolites in the tissues, faeces and urine from lambs feeding on an aflatoxin-contaminated diet. *Journal of the Science of Food and Agriculture* 74(2): 161–168.

Fink-Gremmels, J. 2008. The role of mycotoxins in the health and performance of dairy cows. *Veterinary Journal* 176(1): 84–92.

FAO (Food and Agriculture Organization of the United Nations). 2000. *Drought in Kenya: Food situation is deteriorating rapidly - first starvation related deaths.* (Available from http://reliefweb.int/report/kenya/drought-kenya-food-situation-deteriorating-rapidly-first-starvation-related-deaths) (Accessed 28 August 2014)

FDA (Food and Drug Administration). 1994. *CPG Sec. 683.100 Action levels for aflatoxins in animal feeds*. (Available from http://www.fda.gov/ICECI/ComplianceManuals/CompliancePolicyGuidanceManual/ucm074703.htm) (Accessed 28 August 2014)

Fufa, H. and Urga, K. 1996. Screening of aflatoxins in Shiro and ground red pepper in Addis Ababa. *Ethiopian Medical Journal* 34(4): 243–249.

Fujiki, H. and Suganuma, M. 2012. Green tea: An effective synergist with anticancer drugs for tertiary cancer prevention. *Cancer Letters* 324(2): 119–125.

Galvano, F., Pietri, A., Bertuzzi, T., Gagliardi, L., Ciotti, S., Luisi, S., Bognanno, M., La Fauci, L., Iacopino, A.M., Nigro, F., Li Volti, G., Vanella, L., Giammanco, G., Tina, G.L and Gazzolo D. 2008. Maternal dietary habits and mycotoxin occurrence in human mature milk. *Molecular Nutrition and Food Research* 52(4): 496–501.

Ghahri, H., Talebi, A., Chamani, M., Lotfollahian, H. and Afzali, N. 2009. Ameliorative effect of esterified glucomannan, sodium bentonite, and humic acid on humoral immunity of broilers during chronic aflatoxicosis. *Turkish Journal of Veterinary and Animal Sciences* 33(5): 419–425.

Ghali, R., Hmaissia-Khlifa, K., Ghorbel, H., Maaroufi K. and Hedili A. 2008. Incidence of aflatoxins, ochratoxin A and zearalenone in Tunisian foods. *Food Control* 19(9): 921–924.

Ghali, R., Khlifa, K.H., Ghorbel, H., Maaroufi, K. and Hedilli, A. 2010. Aflatoxin determination in commonly consumed foods in Tunisia. *Journal of the Science of Food and Agriculture* 90(14): 2347–2351.

Ghiasian, S.A., Maghsood, A.H., Neyestani, T.R. and Mirhendi, S.H. 2007. Occurrence of aflatoxin M1 in raw milk during the summer and winter seasons in Hamedan, Iran. *Journal of Food Safety* 27(2): 188–198.

Giambrone, J., Diener, U., Davis, N., Panangala, V. and Hoerr, F. 1985. Effects of aflatoxin on young turkeys and broiler chickens. *Poultry Science* 64(9): 1678–1684.

Giorni, P., Magan, N., Pietri, A., Bertuzzi, T. and Battilani, P. 2007. Studies on *Aspergillus* section *Flavi* isolated from maize in northern Italy. *International Journal of Food Microbiology* 113(3): 330–338.

Gnonlonfin, G.J., Hell, K., Adjovi, Y., Fandohan, P., Koudande, D.O., Mensah, G.A., Sanni, A. and Brimer, L. 2013. A review on aflatoxin contamination and its implications in the developing world: a sub-Saharan African perspective. *Critical Reviews in Food Science and Nutrition* 53(4): 349–365.

Gnonlonfin, G.J., Hell, K., Fandohan, P. and Siame, A.B. 2008. Mycoflora and natural occurrence of aflatoxins and fumonisin B1 in cassava and yam chips from Benin, West Africa. *International Journal of Food Microbiology* 122(1–2): 140–147.

Gong, Y., Hounsa, A., Egal, S., Turner, P.C., Sutcliffe, A.E., Hall, A.J., Cardwell, K. and Wild, CP. 2004. Postweaning exposure to aflatoxin results in impaired child growth: a longitudinal study in Benin, West Africa. *Environmental Health Perspectives* 112(13): 1334–1338.

Gong, Y.Y., Cardwell, K., Hounsa, A., Egal, S., Turner, P.C., Hall, A.J. and Wild, CP. 2002. Dietary aflatoxin exposure and impaired growth in young children from Benin and Togo: Cross sectional study. *British Medical Journal* 325(7354): 20–21.

Gong, Y.Y., Egal, S., Hounsa, A., Turner, P.C., Hall, A.J., Cardwell, K.F. and Wild, CP. 2003. Determinants of aflatoxin exposure in young children from Benin and Togo, West Africa: The critical role of weaning. *International Journal of Epidemiology* 32(4): 556–562.

Gong, Y.Y., Wilson, S., Mwatha, J.K., Routledge, M.N., Castelino, J.M., Zhao, B., Kimani, G., Kariuki, H.C., Vennervald, B.J., Dunne, D.W. and Wild, C.P. 2012. Aflatoxin exposure may contribute to chronic hepatomegaly in Kenyan school children. *Environmental Health Perspectives* 120(6): 893–896.

Grace, D. 2013. *Animals and aflatoxins.* IFPRI 2020 Focus 20, Brief 5. Washington, DC: IFPRI.

Griffiths, U.K., Hutton, G. and Das Dores Pascoal, E. 2005. The cost-effectiveness of introducing hepatitis B vaccine into infant immunization services in Mozambique. *Health Policy Plan* 20(1): 50–59.

Groopman, J.D. and Kensler, T.W. 2005. Role of metabolism and viruses in aflatoxin-induced liver cancer. *Toxicology and Applied Pharmacology* 206(2): 131–137.

Groopman, J.D., Kensler, T.W. and Wild, C.P. 2008. Protective interventions to prevent aflatoxin-induced carcinogenesis in developing countries. *Annual Review of Public Health* 29: 187–203.

Guo, B., Chen, Z.Y., Lee, R.D. and Scully, B.T. 2008. Drought stress and preharvest aflatoxin contamination in agricultural commodity: genetics, genomics and proteomics. *Journal of Integrative Plant Biology* 50(10): 1281–1291.

Guo, B.Z., Russin, J.S., Brown, R.L., Cleveland, T.E. and Widstrom, N.W. 1996. Resistance to aflatoxin contamination in corn as influenced by relative humidity and kernel germination. *Journal of Food Protection* 59(3): 276–281.

Gürbay, A., Sabuncuoğlu, S.A., Girgin, G., Sahin, G., Yiğit, S., Yurdakök, M. and Tekinalp, G. 2010. Exposure of newborns to aflatoxin M1 and B1 from mothers' breast milk in Ankara, Turkey. *Food and Chemical Toxicology* 48(1): 314–319.

Hameed, H.G. 1993. *Extrusion and chemical treatments for destruction of aflatoxin in naturally-contaminated corn.* PhD dissertation. Tuscon, Arizona: University of Arizona. (Available from http://hdl.handle.net/10150/186457) (Accessed 28 August 2014)

Harvey, J., Gnonlonfin, B., Fletcher, M., Fox, G., Trowell, S., Berna, A., Nelson, R. and Darnell, R. 2013. *Improving diagnostics for aflatoxin detection.* IFPRI 2020 Focus 20, Brief 19. Washington, DC: IFPRI.

Harvey, R., Kubena, L. and Elissalde, M. 1995a. Effects of aflatoxin on tocopherol and retinol concentrations in growing barrows. *Agri-Practice* 16.

Harvey, R., Edrington, T., Kubena, L., Elissalde, M. and Rottinghaus, G. 1995b. Influence of aflatoxin and fumonisin B1-containing culture material on growing barrows. *American Journal of Veterinary Research* 56(12): 1668–1672.

Harvey, R., Huff, W., Kubena, L. and Phillips, T. 1989. Evaluation of diets contaminated with aflatoxin and ochratoxin fed to growing pigs. *American Journal of Veterinary Research* 50(8): 1400–1405.

Harvey, R.B., Kubena, L.F., Elissalde, M.H., Corrier, D.E. and Phillips, T.D. 1994. Comparison of two hydrated sodium calcium aluminosilicate compounds to experimentally protect growing barrows from aflatoxicosis. *Journal of Veterinary Diagnostic Investigation* 6(1): 88–92.

Hassan, A.A., Hassan, M.A., El Shafei, H.M., El Ahl, R.M.H.S. and Abd El-Dayem, R.H. 2011. Detection of aflatoxigenic moulds isolated from fish and their products and its public health significance. *Nature and Science* 9(9): 106.

Hell, K., Bandyopadhyay, R., Kiewnick, S., Coulibaly, O., Menkir, A. and Cotty, P. 2005. *Optimal management of mycotoxins for improving food safety and trade of maize in West Africa.* Presented at the Tropentag 2005 Conference, Hohenheim, Germany, 11–13 October 2005.

Hell, K., Cardwell, K.F., Setamou, M. and Poehling H.-M. 2000. The influence of storage practices on aflatoxin contamination in maize in four agroecological zones of Benin, West Africa. *Journal of Stored Products Research* 36(4): 365–382.

Hell, K., Gnonlonfin, B.G., Kodjogbe, G., Lamboni, Y. and Abdourhamane, I.K. 2009. Mycoflora and occurrence of aflatoxin in dried vegetables in Benin, Mali and Togo, West Africa. *International Journal of Food Microbiology* 135(2): 99–104.

Hendrickse, R.G. 1984. The influence of aflatoxins on child health in the tropics with particular reference to kwashiorkor. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 78(4): 427–435.

Hendrickse, R.G., Coulter, J.B., Lamplugh, S.M., MacFarlane, S.B., Williams, T.E., Omer, M.I., Suliman, G.I. and El-Zorgani, G.A. 1983. Aflatoxins and kwashiorkor. Epidemiology and clinical studies in Sudanese children and findings in autopsy liver samples from Nigeria and South Africa. *Bulletin de la Société de Pathologie Exotique et de ses Filiales* 76(5): 559–566.

Hernandez-Mendoza, A., Guzman-de-Peña, D. and Garcia, H.S. 2009. Key role of teichoic acids on aflatoxin B binding by probiotic bacteria. *Journal of Applied Microbiology* 107(2): 395–403.

Hernández-Martínez, R. and Navarro-Blasco, I. 2010. Aflatoxin levels and exposure assessment of Spanish infant cereals. *Food Additives & Contaminants* 3(4): 275–288.

Holbrook, C.C., Guo, B.Z., Wilson, D.M. and Timper, P. 2006. *The U.S. breeding program to develop peanut with drought tolerance and reduced aflatoxin contamination.* Presented at the International Conference on Groundnut Aflatoxin Management and Genomics, Guangzhou, China, 5–9 November 2006.

Hormisch, D., Brost, I., Kohring, G.W., Giffhorn, F., Kroppenstedt, R.M., Stackebrandt, E., Färber, P. and Holzapfel W.H. 2004. *Mycobacterium fluoranthenivorans* sp. nov., a fluoranthene and aflatoxin B1 degrading bacterium from contaminated soil of a former coal gas plant. *Systematic and Applied Microbiology* 27(6): 653–660.

Houssou, P., Ahohuendo, B., Fandohan, P., Kpodo, K., Hounhouigan, D., Jakobsen, M. 2009. Natural infection of cowpea (*Vigna unguiculata* (L.) Walp.) by toxigenic fungi and mycotoxin contamination in Benin, West Africa. *Journal of Stored Products Research* 45(1): 40–44.

Huff, W. 1980. Evaluation of tibial dyschondroplasia during aflatoxicosis and feed restriction in young broiler chickens. *Poultry Science* 59(5): 991–995.

Huff, W., Kubena, L., Harvey, R., Hagler, W., Swanson, S., Phillips, T. and Creger C. 1986. Individual and combined effects of aflatoxin and deoxynivalenol (DON, vomitoxin) in broiler chickens. *Poultry Science* 65(7): 1291–1298.

Hussain, Z., Khan, M.Z., Khan, A., Javed, I., Saleemi, M.K., Mahmood, S. and Asi, M.R. 2010. Residues of aflatoxin B1 in broiler meat: effect of age and dietary aflatoxin B1 levels. *Food and Chemical Toxicology* 48(12): 3304–3307.

Huwig, A., Freimund, S., Käppeli, O. and Dutler, H. 2001. Mycotoxin detoxication of animal feed by different adsorbents. *Toxicology Letters* 122(2): 179–188.

IARC (International Agency for Research on Cancer). 1982. *IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans: Some aromatic amines, anthraquinones and nitroso compounds, and inorganic fluorides used in drinking-water and dental preparations.* Volume 27. Lyons, France: IARC.

Ibeh, I.N., Uraih, N. and Ogonar, J.I. 1994. Dietary exposure to aflatoxin in human male infertility in Benin City, Nigeria. *International Journal of Fertility and Menopausal Studies* 39(4): 208–214.

Idris, Y.M., Mariod, A.A., Elnour, I.A. and Mohamed, A.A. 2010. Determination of aflatoxin levels in Sudanese edible oils. *Food and Chemical Toxicology* 48(8–9): 2539–2541.

ICRISAT (International Crops Research Institute for the Semi-Arid Tropics). 2006. *The seedlings of success in the semi-arid tropics nurtured.* Andhra Pradesh, India: ICRISAT.

Ismail, M.A., Taligoola, H.K. and Nakamya, R. 2008. Mycobiota associated with baby food products imported into Uganda with special reference to aflatoxigenic aspergilli and aflatoxins. *Czech Mycology* 60(1): 75–89.

Jacobson, W.C., Harmeyer, W.C., Jackson, J.E., Armbrecht, B. and Wiseman, H.G. 1978. Transmission of aflatoxin B into the tissues of growing pigs. *Bulletin of Environmental Contamination and Toxicology* 19(2): 156–161.

Jager, A., Tedesco, M., Souto, P. and Oliveira, C. 2013. Assessment of aflatoxin intake in São Paulo, Brazil. *Food Control* 33(1): 87–92.

Jaime-Garcia, R. and Cotty, P.J. 2010. Crop rotation and soil temperature influence the community structure of *Aspergillus flavus* in soil. *Soil Biology & Biochemistry* 42: 1842–1847.

James, B., Adda, C., Cardwell, K., Annang, D., Hell, K., Korie, S., Edorh, M., Gbeassor, F., Nagatey, K. and Houenou, G. 2007. Public information campaign on aflatoxin contamination of maize grains in market stores in Benin, Ghana and Togo. *Food Additives & Contaminants* 24(11): 1283–1291.

Jantrarotai, W. and Lovell, R.T. 1990. Subchronic toxicity of dietary aflatoxin B1 to channel catfish. *Journal of Aquatic Animal Health* 2(4): 248–254.

Jiang, Y., Jolly, P.E., Ellis, W.O., Wang, J.S., Phillips, T.D. and Williams, JH. 2005. Aflatoxin B1 albumin adduct levels and cellular immune status in Ghanaians. *International Immunology* 17(6): 807–814.

Jiang, Y., Jolly, P.E., Preko, P., Wang, J.S., Ellis, W.O., Phillips, T.D. and Williams, J.H. 2008. Aflatoxin-related immune dysfunction in health and in human immunodeficiency virus disease. *Clinical and Developmental Immunology* 2008: Article ID 790309.

Jimoh, K. and Kolapo, A. 2008. Mycoflora and aflatoxin production in market samples of some selected Nigerian foodstuffs. *Research Journal of Microbiology* 3(3): 169–174.

Jolly, C.M., Awuah, R.T., Fialor, S.C., Agyemang, K.O., Kagochi, J.M. and Binns, A.D. 2008. Groundnut consumption frequency in Ghana. *International Journal of Consumer Studies* 32(6): 675–686.

Jolly, C.M., Bayard, B. and Vodouhe, S. 2009. Risks of ingestion of aflatoxin-contaminated groundnuts in Benin: Scale measurements, beliefs, and socioeconomic factors. *Risk Analysis* 29(10): 1395–1409.

Jolly, P., Jiang, Y., Ellis, W., Awuah, R., Nnedu, O., Phillips, T., Wang, J.S., Afriyie-Gyawu, E., Tang, L., Person, S., Williams, J. and Jolly, C. 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(4): 345–358.

Jolly, P.E., Jiang, Y., Ellis, W.O., Awuah, R.T., Appawu, J., Nnedu, O., Stiles, J.K., Wang, J., Adjei, O., Jolly, C.M. and Williams, J.H. 2007. Association between aflatoxin exposure and health characteristics, liver function, hepatitis and malaria infections in Ghanaians. *Journal of Nutritional & Environmental Medicine* 16(3–4): 242–257.

Jones, M., Tanya, V., Mbofung, C., Fonkem, D. and Silverside, D. 2001. A microbiological and nutritional evaluation of the West African dried meat product, kilishi. *Journal of Food Technology in Africa* 6(4): 126–129.

Jonsyn, F., Maxwell, S. and Hendrickse, R. 1995a. Human fetal exposure to ochratoxin A and aflatoxins. *Annals of Tropical Paediatrics: International Child Health* 15(1): 3–9.

Jonsyn, F.E., Maxwell S.M. and Hendrickse R.G. 1995b. Ochratoxin A and aflatoxins in breast milk samples from Sierra Leone. *Mycopathologia* 131(2): 121–126.

Jutavijittum, P., Jiviriyawat, Y., Yousukh, A., Hayashi, S. and Toriyama, K. 2005. Evaluation of a hepatitis B vaccination program in Chiang Mai, Thailand. *Southeast Asian Journal of Tropical Medicine and Public Health* 36(1): 207–212.

Kaaya, A.N. and Kyamuhangire, W. 2006. The effect of storage time and agroecological zone on mould incidence and aflatoxin contamination of maize from traders in Uganda. *International Journal of Food Microbiology* 110(3): 217–223.

Kaaya, A.N., Kyamuhangire, W. and Kyamanywa, S. 2006. Factors affecting aflatoxin contamination of harvested maize in the three agroecological zones of Uganda. *Journal of Applied Sciences* 6(11): 2401–2407.

Kaaya, N.A. and Warren, HL. 2005. A review of past and present research on aflatoxin in Uganda. *African Journal of Food, Agriculture, Nutrition and Development* 5(1).

Kabak, B. and Var I. 2008. Factors affecting the removal of aflatoxin M1 from food model by *Lactobacillus* and *Bifidobacterium* strains. *Journal of Environmental Science and Health, Part B* 43(7): 617–624.

Kabak, B., Dobson, A.D. and Var, I. 2006. Strategies to prevent mycotoxin contamination of food and animal feed: A review. *Critical Reviews in Food Science and Nutrition* 46(8): 593–619.

Kamika, I. and Takoy, L.L. 2011. Natural occurrence of aflatoxin B1 in peanut collected from Kinshasa, Democratic Republic of Congo. *Food Control* 22(11): 1760–1764.

Kamkar, A. 2005. A study on the occurrence of aflatoxin M1 in raw milk produced in Sarab city of Iran. *Food Control* 16(7): 593–599.

Kang'ethe, E.K. and Lang'a, KA. 2009. Aflatoxin B1 and M1 contamination of animal feeds and milk from urban centers in Kenya. *African Health Sciences* 9(4): 218–226.

Kanora, A. and Maes, D. 2009. The role of mycotoxins in pig reproduction: a review. *Veterinarni Medicina* 54(12): 565–576.

Katerere, D.R., Stockenström, S., Thembo, K.M., Rheeder, J.P., Shephard, G.S. and Vismer, H.F. 2008. A preliminary survey of mycological and fumonisin and aflatoxin contamination of African traditional herbal medicines sold in South Africa. *Human & Experimental Toxicology* 27(11): 793–798.

Keenan, J., Jolly, P., Preko, P., Baidoo, J., Wang, J.S., Phillips, T.D. and McGwin, G. 2011. Association between aflatoxin b1 albumin adduct levels and tuberculosis infection among HIV+ Ghanaians. *Archives of Clinical Microbiology* 2(3).

Kenji, G.M., Mvula, M.A., Koaze, H. and Baba, N. 2000. Aflatoxin contamination of Kenyan maize flour and malted Kenyan and Malawian grains. *Scientific Reports of the Faculty of Agriculture, Okayama University* 89: 5–7.

Keyl, A. and Booth, A. 1971. Aflatoxin effects in livestock. *Journal of the American Oil Chemists' Society* 48(10): 599–604.

Khanafari, A., Soudi, H. and Miraboulfathi, M. 2007. Biocontrol of *Aspergillus flavus* and aflatoxin B1 production in corn. *Iranian Journal of Environmental Health* 4(3): 163–168.

Khatibi, P.A., McMaster, N.J., Musser, R. and Schmale, D.G., III. 2014. Survey of mycotoxins in corn distillers' dried grains with solubles from seventy-eight ethanol plants in twelve states in the U.S. in 2011. *Toxins* 6: 1155–1168.

Khlangwiset, P., Shephard, G.S. and Wu, F. 2011. Aflatoxins and growth impairment: A review. *Critical Reviews in Toxicology* 41(9): 740–755.

Khlangwiset, P. and Wu, F. 2010. Costs and efficacy of public health interventions to reduce aflatoxin-induced human disease. *Food Additives & Contaminants, Part A* 27(7): 998–1014.

Kirk, G.D., Bah, E., Montesano, R. 2006. Molecular epidemiology of human liver cancer: Insights into etiology, pathogenesis and prevention from the Gambia, West Africa. *Carcinogenesis* 27(10): 2070–2082.

Kpodo, K.A. 1995. Mycotoxins in maize and fermented maize products in Southern Ghana. In: Cardwell K.F. (ed), *Proceedings of a workshop on mycotoxins in food in Africa*. Cotonou, Benin: IITA. p. 33.

Kramvis, A. and Kew, M.C. 2007. Epidemiology of hepatitis B virus in Africa, its genotypes and clinical associations of genotypes. *Hepatology Research* 37(s1): S9–S19.

Krishnamachari, K.A., Bhat, R.V., Nagarajan, V., Tilak, T.B. 1975. Hepatitis due to aflatoxicosis. An outbreak in Western India. Lancet 1(7915): 1061–1063.

Kumagai, S., Nakajima, M., Tabata, S., Ishikuro, E., Tanaka, T., Norizuki, H., Itoh, Y., Aoyama, K., Fujita, K., Kai, S., Sato, T., Saito, S., Yoshiike, N. and Sugita-Konishi, Y. 2008. Aflatoxin and ochratoxin A contamination of retail foods and intake of these mycotoxins in Japan. *Food Additives & Contaminants, Part A* 25(9): 1101–1106.

Kuniholm, M.H., Lesi, O.A., Mendy, M., Akano, A.O., Sam, O., Hall, A.J., Whittle, H., Bah, E., Goedert, J.J., Hainaut, P. and Kirk, G.D. 2008. Aflatoxin exposure and viral hepatitis in the etiology of liver cirrhosis in the Gambia, West Africa. *Environmental Health Perspectives* 116(11): 1553–1557.

Lahtinen, S.J., Haskard, C.A., Ouwehand, A.C., Salminen, S.J. and Ahokas, J.T. 2004. Binding of aflatoxin B1 to cell wall components of *Lactobacillus rhamnosus* strain GG. *Food Additives & Contaminants* 21(2): 158–164.

Lamplugh, S., Hendrickse, R.G., Apeagyei, F. and Mwanmut, D.D. 1988. Aflatoxins in breast milk, neonatal cord blood, and serum of pregnant women. *British Medical Journal (Clinical Research Edition)* 296(6627): 968.

Lanyasunya, T.P., Wamae, L.M., Musa, H.H., Olowofeso, O. and Lokwaleput, I.K. 2005. The risk of mycotoxins contamination of dairy feed and milk on smallholder dairy farms in Kenya. *Journal of Nutrition* 4(3): 162–169.

Leblanc, J.C., Tard, A., Volatier, J.L. and Verger, P. 2005. Estimated dietary exposure to principal food mycotoxins from the first French Total Diet Study. *Food Additives & Contaminants* 22(7): 652–272.

Lewis, L., Onsongo, M., Njapau, H., Schurz-Rogers, H., Luber, G., Kieszak, S., Nyamongo, J., Backer, L., Dahiye, A.M., Misore, A., DeCock, K., Rubin, C. and Kenya Aflatoxicosis Investigation Group. 2005. Aflatoxin contamination of commercial maize products during an outbreak of acute aflatoxicosis in eastern and central Kenya. *Environmental Health Perspectives* 113(12): 1763–1767.

Lindemann, M., Blodgett, D., Kornegay, E. and Schurig, G. 1993. Potential ameliorators of aflatoxicosis in weanling/growing swine. *Journal of Animal Science* 71(1): 171–178.

Linsell, C. and Peers, F. 1977. Aflatoxin and liver cell cancer. *Transactions of the Royal Society of Tropical Medicine and Hygiene* 71(6): 471–473.

Liu, Y. and Wu, F. 2010. Global burden of aflatoxin-induced hepatocellular carcinoma: a risk assessment. *Environmental Health Perspectives* 118(6): 818–824.

Liu, Y., Chang, C.C., Marsh, G.M. and Wu, F. 2012. Population attributable risk of aflatoxin-related liver cancer: Systematic review and meta-analysis. *European Journal of Cancer* 48(14): 2125–2126.

Lu, Z., Dantzer, W., Hopmans, E., Prisk, V., Cunnick, J., Murphy, P. and Hendrich, S. 1997. Reaction with fructose detoxifies fumonisin B1 while stimulating liver-associated natural killer cell activity in rats. *Journal of Agricultural and Food Chemistry* 45(3): 803–809.

Lubulwa, A. and Davis, J. 1994. Estimating the social costs of the impacts of fungi and aflatoxins in maize and peanuts. In: Highley, E., Wright, E., Banks, H. and Champ, B. (eds), *Proceedings of the 6th International Working Conference on Stored Product Protection*. Canberra, Australia: CAB International. pp. 1017–1042.

Madden, U. and Stahr, H. 1992. Effect of soil on aflatoxin tissue retention in chicks when added to aflatoxin-contaminated poultry rations. *Veterinary and Human Toxicology* 34(6): 521–523.

Magan, N. and Aldred, D. 2007. Post-harvest control strategies: Minimizing mycotoxins in the food chain. *International Journal of Food Microbiology* 119(1-2): 131–139.

Makun, H.A., Dutton, M.F., Njobeh, P.B., Mwanza, M. and Kabiru, A.Y. 2011. Natural multi-occurrence of mycotoxins in rice from Niger State, Nigeria. *Mycotoxin Research* 27(2): 97–104.

Makun, H.A., Gbodi, T.A., Akanya, O.H., Salako, E.A. and Ogbadu, G.H. 2007. Fungi and some mycotoxins contaminating rice (*Oryza sativa*) in Niger state, Nigeria. *African Journal of Biotechnology* 6(2).

Marin, D.E., Taranu, I., Bunaciu, R.P., Pascale, F., Tudor, D.S., Avram, N., Sarca, M., Cureu, I., Criste, R.D., Suta, V. and Oswald, I.P. 2002. Changes in performance, blood parameters, humoral and cellular immune responses in weanling piglets exposed to low doses of aflatoxin. *Journal of Animal Science* 80(5): 1250–1257.

Maxwell, S., Apeagyei, F., De Vries, H., Mwanmut, D. and Hendrickse, R. 1989. Aflatoxins in breast milk, neonatal cord blood and sera of pregnant women. *Toxin Reviews* 8(1–2): 19–29.

Maxwell, S., Familusi, J., Sodeinde, O., Chan, M. and Hendrickse, R. 1994. Detection of naphthols and aflatoxins in Nigerian cord blood. *Annals of Tropical Paediatrics: International Child Health* 14(1): 3–5.

Mbah, M. and Akueshi, C. 2009. Aflatoxin in mould infested sesame seeds. *African Journal of Biotechnology* 8(3).

McLean, M. 1995. The phytotoxicity of selected mycotoxins on mature, germinating *Zea mays* embryos. *Mycopathologia* 132(3): 173–183.

Méaux, S., Pantiora, E. and Schneider, S. 2013. *Aflatoxin management in the World Food Programme through P4P local procurement.* IFPRI 2020 Focus 20, Brief 9. Washington, DC: IFPRI.

Menkir, A., Brown, R.L., Bandyopadhyay, R., Chen, Z.Y. and Cleveland, T.E. 2006. A USA-Africa collaborative strategy for identifying, characterizing, and developing maize germplasm with resistance to aflatoxin contamination. *Mycopathologia* 162(3): 225–232.

MRC (South African Medical Research Council). 2006. *Aflatoxin in peanut butter: MRC Policy Brief.* (Available from http://www.scienceinafrica.com/health/aflatoxin-peanut-butter-mrc-policy-brief) (Accessed 28 August 2014)

Miele, M., Donato, F., Hall, A.J., Whittle, H., Chapot, B., Bonatti, S., De Ferrari, M., Artuso, M., Gallerani, E. and Abbondandolo, A. 1996. Aflatoxin exposure and cytogenetic alterations in individuals from the Gambia, West Africa. *Mutation Research/Fundamental and Molecular Mechanisms of Mutagenesis* 349(2): 209–217.

Miller, D.M., Wilson, D.M., Wyatt, R.D., McKinney, J., Crowell, W. and Stuart B. 1982. High performance liquid chromatographic determination and clearance time of aflatoxin residues in swine tissues. *Journal – Association of Official Analytical Chemists* 65(1): 1–4.

Miller, J.D. 1995. Mycotoxins. In: Cardwell KF (ed), *Proceedings of the workshop on mycotoxins in food in Africa*. Cotonou, Benin: IITA. pp. 18–22.

Mocchegiani, E., Corradi, A., Santarelli, L., Tibaldi, A., DeAngelis, E., Borghetti, P., Bonomi, A., Fabris, N. and Cabassi, E. 1998. Zinc, thymic endocrine activity and mitogen responsiveness (PHA) in piglets exposed to maternal aflatoxicosis B_1 and G_1. *Veterinary Immunology and Immunopathology* 62(3): 245–260.

Motawee, M.M., Bauer, J. and McMahon, D.J. 2009. Survey of aflatoxin M(1) in cow, goat, buffalo and camel milks in Ismailia-Egypt. *Bulletin of Environmental Contamination and Toxicology* 83(5): 766–769.

Mphande, F.A., Siame, B.A. and Taylor, J.E. 2004. Fungi, aflatoxins, and cyclopiazonic acid associated with peanut retailing in Botswana. *Journal of Food Protection* 67(1): 96–102.

Muriuki, G.K. and Siboe, G.M. 1995. Maize flour contaminated with toxigenic fungi and mycotoxins in Kenya. *African Journal of Health Sciences* 2(1): 236–241.

Mutegi, C.K., Hendriks, S.L., Jones, R.B., Okello, J.J. and Ngugi, H.K. 2007. *Role of collective action and handling practices on aflatoxin contamination of groundnuts: Evidence from Kenya.* Presented at the 8th African Crop Science Society Conference, El-Minia, Egypt, 27–31 October 2007.

Mutegi, C.K., Ngugi, H.K., Hendriks, S.L., Jones, R.B. 2009. Prevalence and factors associated with aflatoxin contamination of peanuts from Western Kenya. *International Journal of Food Microbiology* 130(1): 27–34.

Muture, B.N. and Ogana, G. 2005. Aflatoxin levels in maize and maize products during the 2004 food poisoning outbreak in Eastern Province of Kenya. *East African Medical Journal* 82(6): 275–279.

Mwihia, J.T., Straetmans, M., Ibrahim, A., Njau, J., Muhenje, O., Guracha, A., Gikundi, S., Mutonga, D., Tetteh, C., Likimani, S., Breiman, R.F., Njenga, K. and Lewis, L. 2008. Aflatoxin levels in locally grown maize from Makueni District, Kenya. *East African Medical Journal* 85(7): 311–317.

Narrod, C. 2011. *Findings from the cost effectiveness analysis.* Presentation at the Closing Workshop: Aflacontrol Conference, Nairobi, Kenya, 30 November 2011. (Available from http://www.slideshare.net/pchenevixtrench/findings-from-the-cost-effectiveness-analysis) (Accessed 28 August 2014)

Navas, S.A., Sabino, M. and Rodriguez-Amaya, D.B. 2005. Aflatoxin M(1) and ochratoxin A in a human milk bank in the city of São Paulo, Brazil. *Food Additives & Contaminants* 22(5): 457–462.

Ndung'u, J.W., Makokha, A.O., Onyango, C.A., Mutegi, C.K., Wagacha, J.M., Christie, M.E., Wanjoya, A.K. 2013. Prevalence and potential for aflatoxin contamination in groundnuts and peanut butter from farmers and traders in Nairobi and Nyanza provinces of Kenya. *Journal of Applied Biosciences* 65: 4922–4934.

Ngindu, A., Kenya, P.R., Ngira, J.A., Ocheng, D.M., Omondi, T.N., Ngare, W., Gatei, D., Johnson, B.K., Ngira, J.A., Nandwa, H., Jansen, A.J., Kaviti, J.N. and Siongok, T.A. 1982. Outbreak of acute hepatitis caused by aflatoxin poisoning in Kenya. *Lancet* 1(8285): 1346–1348.

Ni, X., Wilson, J.P., Buntin, G.D., Guo, B., Krakowsky, M.D., Lee, R.D., Cottrell, T.E., Scully, B.T., Huffaker, A. and Schmelz, E.A. 2011. Spatial patterns of aflatoxin levels in relation to ear-feeding insect damage in pre-harvest corn. *Toxins* 3(7): 920–931.

Nigam, S., Waliyar, F., Aruna, R., Reddy, S., Kumar, P.L., Craufurd, P., Diallo, A., Ntare, B. and Upadhyaya, H. 2009. Breeding peanut for resistance to aflatoxin contamination at ICRISAT. *Peanut Science* 36(1): 42–49.

Njapau, H., Muzungaile, E.M. and Changa, R.C. 1998. The effect of village processing techniques on the content of aflatoxins in corn and peanuts in Zambia. *Journal of the Science of Food and Agriculture* 76(3): 450–456.

Njobeh, P.B., Dutton, M.F., Koch, S.H., Chuturgoon, A.A., Stoev, S.D. and Mosonik, J.S. 2010. Simultaneous occurrence of mycotoxins in human food commodities from Cameroon. *Mycotoxin Research* 26(1): 47–57.

Nyathi, C., Mutiro, C., Hasler, J. and Chetsanga, C. 1989. Human exposure to aflatoxins in Zimbabwe. *Central African Journal of Medicine* 35(12): 542–545.

Obura, A. 2013. *Aflatoxicosis: Evidence from Kenya.* IFPRI 2020 Focus 20, Brief 2. Washington, DC: IFPRI.

Obuseh, F.A., Jolly, P.E., Kulczycki, A., Ehiri, J., Waterbor, J., Desmond, R.A., Preko, P.O., Jiang, Y. and Piyathilake, C.J. 2011. Aflatoxin levels, plasma vitamins A and E concentrations, and their association with HIV and hepatitis B virus infections in Ghanaians: A cross-sectional study. *Journal of the International AIDS Society* 14: 53.

Odhav, B. and Naicker, V. 2002. Mycotoxins in South African traditionally brewed beers. *Food Additives & Contaminants* 19(1): 55–61.

Odoemelam, S.A. and Osu, C.I. 2009. Aflatoxin B_1 contamination of some edible grains marketed in Nigeria. *Journal of Chemistry* 6(2): 308–314.

Ogiehor, I.S., Ikenebomeh, M.J. and Ekundayo, A.O. 2007. The bioload and aflatoxin content of market garri from some selected states in southern Nigeria: Public health significance. *African Health Sciences* 7(4): 223–227.

Okoth, S.A. and Ohingo, M. 2004. Dietary aflatoxin exposure and impaired growth in young children from Kisumu District, Kenya: Cross sectional study. *African Journal of Health Sciences* 11(1–2): 43–54.

Oliveira, C.A., Kobashigawa, E., Reis, T.A., Mestieri, L., Albuquerque, R. and Corrêa, L.M.B. 2000. Aflatoxin B_1 residues in eggs of laying hens fed a diet containing different levels of the mycotoxin. *Food Additives & Contaminants* 17(6): 459–462.

Oluwafemi, F. and Ibeh, I.N. 2011. Microbial contamination of seven major weaning foods in Nigeria. *Journal of Health, Population and Nutrition* 29(4): 415–419.

Ortblad, K.F., Lozano, R. and Murray, C.J. 2013. The burden of HIV: Insights from the Global Burden of Disease Study 2010. *AIDS* 27(13): 2003–2017.

Osweiler, G.D. 2006. Occurrence of mycotoxins in grains and feeds. In: Straw, B., Zimmerman, J., D'Allaire, S. and Taylor, D. (eds), *Diseases of swine*. 9th edition. Ames, Iowa: Blackwell Publishing. pp. 915–929.

Otim, M.O., Mukiibi-Muka, G., Christensen, H., Bisgaard, M. 2005. Aflatoxicosis, infectious bursal disease and immune response to Newcastle disease vaccination in rural chickens. *Avian Pathology* 34(4): 319–323.

Otsuki, T., Wilson, J.S. and Sewadeh, M. 2001. Saving two in a billion: Quantifying the trade effect of European food safety standards on African exports. *Food Policy* 26(5): 495–514.

Oveisi, M.-R., Jannat, B., Sadeghi, N., Hajimahmoodi, M. and Nikzad, A. 2007. Presence of aflatoxin M_1 in milk and infant milk products in Tehran, Iran. *Food Control* 18(10): 1216–1218.

Oyelami, O., Maxwell, S. and Adeoba, E. 1996. Aflatoxins and ochratoxin A in the weaning food of Nigerian children. *Annals of Tropical Paediatrics: International Child Health* 16(2): 137–140.

Olufunmilayo, G.O. and Oyefolu, A.B. 2010. Natural occurrence of aflatoxin residues in fresh and sun-dried meat in Nigeria. *Pan African Medical Journal* 7: 14.

Panangala, V.S., Giambrone, J.J., Diener, U.L., Davis, N.D., Hoerr, F.J., Mitra, A., Schultz, R.D. and Wilt, G.R. 1986. Effects of aflatoxin on the growth performance and immune responses of weanling swine. *American Journal of Veterinary Research* 47(9): 2062–2067.

Pandey, I. and Chauhan S.S. 2007. Studies on production performance and toxin residues in tissues and eggs of layer chickens fed on diets with various concentrations of aflatoxin AFB1. *British Poultry Science* 48(6): 713–723.

Park, D.L. 2002. Effect of processing on aflatoxin. In: DeVries, J.W., Trucksess, M.W. and Jackson, L.S. (eds), *Mycotoxins and food safety*. New York, NY: Springer. pp. 173–179.

Park J.W. and Kim, Y.-B. 2006. Effect of pressure cooking on aflatoxin B_1 in rice. *Journal of Agricultural and Food Chemistry* 54(6): 2431–2435.

Park, J.W., Kim, E.K. and Kim, Y.B. 2004. Estimation of the daily exposure of Koreans to aflatoxin B_1 through food consumption. *Food Additives & Contaminants* 21(1): 70–75.

Park J.W., Lee, C. and Kim, Y.-B. 2005. Fate of aflatoxin B_1 during the cooking of Korean polished rice. *Journal of Food Protection* 68(7): 1431–1434.

Patten, R.C. 1981. Aflatoxins and disease. *American Journal of Tropical Medicine and Hygiene* 30(2): 422–425.

Peers F.G. and Linsell C.A. 1973. Dietary aflatoxins and liver cancer – A population based study in Kenya. *British Journal of Cancer* 27(6): 473–484.

Peers, F., Bosch, X., Kaldor, J., Linsell, A. and Pluijmen, M. 1987. Aflatoxin exposure, hepatitis B virus infection and liver cancer in Swaziland. *International Journal of Cancer* 39(5): 545–553.

Peltonen, K., El-Nezami, H., Haskard, C., Ahokas, J. and Salminen, S. 2001. Aflatoxin B_1 binding by dairy strains of lactic acid bacteria and bifidobacteria. *Journal of Dairy Science* 84(10): 2152–2156.

Phillips, T.D., Afriyie-Gyawu, E., Williams, J., Huebner, H., Ankrah, N.-A., Ofori-Adjei, D., Jolly, P., Johnson, N., Taylor, J., Marroquin-Cardona, A., Xu, L., Tang, L. and Wang, J.-S. 2008. Reducing human exposure to aflatoxin through the use of clay: A review. *Food Additives & Contaminants : Part A* 25(2): 134–145.

Pierides, M., El-Nezami, H., Peltonen, K., Salminen, S. and Ahokas, J. 2000. Ability of dairy strains of lactic acid bacteria to bind aflatoxin M_1 in a food model. *Journal of Food Protection* 63(5): 645–650.

Pimpukdee, K., Kubena, L.F., Bailey, C.A., Huebner, H.J., Afriyie-Gyawu, E. and Phillips, T.D. 2004. Aflatoxin-induced toxicity and depletion of hepatic vitamin A in young broiler chicks: protection of chicks in the presence of low levels of NovaSil PLUS in the diet. *Poultry Science* 83(5): 737–744.

Pitt, J.I. and Hocking, A.D. 2006. Mycotoxins in Australia: Biocontrol of aflatoxin in peanuts. *Mycopathologia* 162(3): 233–243.

Pitt, J.I., Wild, C.P., Baan, R.A., Gelderblom, W.C.A., Miller, J.D., Riley, R.T. and Wu, F. 2012. *Improving public health through mycotoxin control.* IARC Scientific Publication No. 158. Lyons, France: IARC.

Polychronaki, N., Turner P.C., Mykkänen, H., Gong, Y., Amra, H., Abdel-Wahhab, M. and El-Nezami, H. 2006. Determinants of aflatoxin M_1 in breast milk in a selected group of Egyptian mothers. *Food Additives & Contaminants* 23(7): 700–708.

Polychronaki, N., West, R.M., Turner, P.C., Amra, H., Abdel-Wahhab, M., Mykkänen, H. and El-Nezami, H. 2007. A longitudinal assessment of aflatoxin M_1 excretion in breast milk of selected Egyptian mothers. *Food and Chemical Toxicology* 45(7): 1210–1215.

Polychronaki, N., Wild, C.P., Mykkänen, H., Amra, H., Abdel-Wahhab, M., Sylla, A., Diallo, M., El-Nezami, H. and Turner, P.C. 2008. Urinary biomarkers of aflatoxin exposure in young children from Egypt and Guinea. *Food and Chemical Toxicology* 46(2): 519–526.

Prabaharan, S., George, V. and Balasubramaniam, G. 1999. Influence of dietary aflatoxin and coccidiosis on growth rate in broiler chicken. *Indian Veterinary Journal* 76(9): 827–828.

Prandini, A., Tansini, G., Sigolo, S., Filippi, L., Laporta, M. and Piva, G. 2009. On the occurrence of aflatoxin M_1 in milk and dairy products. *Food and Chemical Toxicology* 47(5):984–991.

Price, W.D., Lovell, R.A., and McChesney, D.G. 1993. Naturally occurring toxins in feedstuffs: Center for Veterinary Medicine Perspective. *Journal of Animal Science* 71(9): 2556–2562.

Probst, C., Schulthess, F. and Cotty, P.J. 2010. Impact of *Aspergillus* section *Flavi* community structure on the development of lethal levels of aflatoxins in Kenyan maize (*Zea mays*). *Journal of Applied Microbiology* 108(2): 600–610.

Proctor, A.D., Ahmedna, M., Kumar, J.V. and Goktepe, I. 2004. Degradation of aflatoxins in peanut kernels/flour by gaseous ozonation and mild heat treatment. *Food Additives & Contaminants* 21(8): 786–793.

Qin, G., Gopalan-Kriczky, P., Su, J., Ning, Y. and Lotlikar, P.D. 1997. Inhibition of aflatoxin B_1-induced initiation of hepatocarcinogenesis in the rat by green tea. *Cancer Letters* 112(2): 149–154.

Qureshi, M., Brake, J., Hamilton, P., Hagler, W. and Nesheim, S. 1998. Dietary exposure of broiler breeders to aflatoxin results in immune dysfunction in progeny chicks. *Poultry Science* 77(6): 812–819.

Rahimi, E., Bonyadian, M., Rafei, M. and Kazemeini, H.R. 2010. Occurrence of aflatoxin M1 in raw milk of five dairy species in Ahvaz, Iran. *Food and Chemical Toxicology* 48(1): 129–131.

Ram, K., Rao, D. and Rao, P. 1988. Effect of aflatoxin feeding and its withdrawal effect on the growth-rate of broilers and layers under long-term feeding trials. *Indian Veterinary Journal* 65(2): 113–116.

Ramjee, G., Berjak, P., Adhikari, M. and Dutton, M.F. 1992. Aflatoxins and kwashiorkor in Durban, South Africa. *Annals of Tropical Paediatrics: International Child Health* 12(3): 241–247.

Randall, G. and Bird, F. 1979. The effect of exercise on the toxicity of aflatoxin B1 in chickens. *Poultry Science* 58(5): 1284–1288.

Reddy, K., Salleh, B., Saad, B., Abbas, H., Abel, C. and Shier, W. 2010. An overview of mycotoxin contamination in foods and its implications for human health. *Toxin Reviews* 29(1): 3–26.

Ricci, K.A., Girosi, F., Tarr, P.I., Lim, Y.-W., Mason, C., Miller, M., Hughes, J., Von Seidlein, L., Agosti, J.M. and Guerrant, R.L. 2006. Reducing stunting among children: The potential contribution of diagnostics. *Nature* 444: 29–38.

Richard, J.L., Pier, A.C., Stubblefield, R.D., Shotwell, O.L., Lyon, R.L. and Cutlip, R.C. 1983. Effect of feeding corn naturally contaminated with aflatoxin on feed efficiency, on physiologic, immunologic, and pathologic changes, and on tissue residues in steers. *American Journal of Veterinary Research* 44(7): 1294–1299.

Richard, J.L., Stubblefield, R.D., Lyon, R.L., Peden, W.M., Thurston, J.R. and Rimler, R.B. 1986. Distribution and clearance of aflatoxins B_1 and M_1 in turkeys fed diets containing 50 or 150 ppb aflatoxin from naturally contaminated corn. *Avian Diseases* 30(4): 788–793.

Roy, D. 2013. *Trade impacts of aflatoxin standards*. IFPRI 2020 Focus 20, Brief 12. Washington, DC: IFPRI.

Ruangwises, N. and Ruangwises, S. 2010. Aflatoxin M(1) contamination in raw milk within the central region of Thailand. *Bulletin of Environmental Contamination and Toxicology* 85(2): 195–198.

Rubert, J., Fapohunda, S., Soler, C., Ezekiel, C., Mañes, J. and Kayode, F. 2013. A survey of mycotoxins in random street-vended snacks from Lagos, Nigeria, using QuEChERS-HPLC-MS/MS. *Food Control* 32(2): 673–677.

Saad, A.M., Abdelgadirt, A.M. and Moss, M.O. 1995. Exposure of infants to aflatoxin M_1 from mothers' breast milk in Abu Dhabi, UAE. *Food Additives & Contaminants* 12(2): 255–261.

Sadeghi, N., Oveisi, M., Jannat, B., Hajimahmoodi, M., Bonyani, H. and Jannat, F. 2009. Incidence of aflatoxin M1 in human breast milk in Tehran, Iran. *Food Control* 20: 75–78.

Sassahara, M., Pontes Netto, D. and Yanaka, E.K. 2005. Aflatoxin occurrence in foodstuff supplied to dairy cattle and aflatoxin M_1 in raw milk in the North of Paraná state. *Food and Chemical Toxicology* 43(6): 981–984.

Schell, T., Lindemann, M., Kornegay, E. and Blodgett, D. 1993a. Effects of feeding aflatoxin-contaminated diets with and without clay to weanling and growing pigs on performance, liver function, and mineral metabolism. *Journal of Animal Science* 71(5): 1209–1218.

Schell, T., Lindemann, M., Kornegay, E., Blodgett, D. and Doerr, J. 1993b. Effectiveness of different types of clay for reducing the detrimental effects of aflatoxin-contaminated diets on

performance and serum profiles of weanling pigs. *Journal of Animal Science* 71(5): 1226–1231.

Shank, R.C., Gordon, J.E., Wogan, G.N., Nondasuta, A. and Subhamani, B. 1972. Dietary aflatoxins and human liver cancer. III. Field survey of rural Thai families for ingested aflatoxins. *Food and Cosmetics Toxicology* 10(1): 71–84.

Shekhar, M., Singh, S., Khan, A.A.A. and Kumar, S. 2009. Efficacy of inorganic salts and organic acids against colony growth of *Aspergillus flavus* and their use to control aflatoxin level in post harvest maize. *Internet Journal of Food Safety* 11: 4–10.

Shephard, G.S. 2008. Risk assessment of aflatoxins in food in Africa. *Food Additives & Contaminants: Part A* 25(10): 1246–1256.

Shephard, G., Leggott, N. and Stockenstrom, S. 2002. Preparation of South African maize porridge: Effect on fumonisin mycotoxin levels. *South African Journal of Science* 98(7–8): 393–396.

Shuaib, F.M.B., Jolly, P.E., Ehiri, J.E., Jiang, Y., Ellis, W.O., Stiles, J.K., Yatich, N.J., Funkhouser, E., Person, S.D., Wilson, C. and Williams, J.H. 2010a. Association between anemia and aflatoxin B_1 biomarker levels among pregnant women in Kumasi, Ghana. *American Journal of Tropical Medicine and Hygiene* 83(5): 1077–1083.

Shuaib, F.M., Ehiri, J., Abdullahi, A., Williams, J.H. and Jolly, P.E. 2010b. Reproductive health effects of aflatoxins: A review of the literature. *Reproductive Toxicology* 29(3): 262–270.

Shuaib, F.M., Jolly, P.E., Ehiri, J.E., Ellis, W.O., Yatich, N.J., Funkhouser, E., Person, S.D., Williams, J.H., Qian, G. and Wang, J.S. 2012. Socio-demographic determinants of aflatoxin B1-lysine adduct levels among pregnant women in Kumasi, Ghana. *Ghana Medical Journal* 46(4): 179–188.

Shukla, S. and Pachauri, S. 1985. Effect of aflatoxicosis on growth and development in cockerels. *Indian Veterinary Journal* 62(4): 341–342.

Shundo, L., Navas, S.A., Lamardo, L.C.A., Ruvieri, V. and Sabino, M. 2009. Estimate of aflatoxin M_1 exposure in milk and occurrence in Brazil. *Food Control* 20(7): 655–657.

Sirot, V., Fremy, J.-M. and Leblanc, J.-C. 2013. Dietary exposure to mycotoxins and health risk assessment in the second French total diet study. *Food and Chemical Toxicology* 52: 1–11.

Siwela, A.H., Mukaro, K.J. and Nziramasanga, N. 2011. Aflatoxin carryover during large scale peanut butter production. *Food and Nutrition Sciences* 2: 105–108.

Sodeinde, O., Chan, M.C., Maxwell, S.M., Familusi, J.B. and Hendrickse, R.G. 1995. Neonatal jaundice, aflatoxins and naphthols: Report of a study in Ibadan, Nigeria. *Annals of Tropical Paediatrics: International Child Health* 15(2): 107–113.

Somorin, Y.M., Bertuzzi, T., Battilani, P. and Pietri, A. 2012. Aflatoxin and fumonisin contamination of yam flour from markets in Nigeria. *Food Control* 25(1): 53–58.

Southern, L. and Clawson, A. 1979. Effects of aflatoxins on finishing swine. *Journal of Animal Science* 49(4): 1006–1011.

Stoloff, L. and Trucksess M. 1981. Effect of boiling, frying, and baking on recovery of aflatoxin from naturally contaminated corn grits or cornmeal. *Journal–Association of Official Analytical Chemists* 64(3): 678–680.

Strosnider, H., Azziz-Baumgartner, E., Banziger, M., Bhat, R.V., Breiman, R., Brune, M.N., DeCock, K., Dilley, A., Groopman, J., Hell, K., Henry, S.H., Jeffers, D., Jolly, C., Jolly, P., Kibata, G.N., Lewis, L., Liu, X., Luber, G., McCoy, L., Mensah, P., Miraglia, M., Misore, A., Njapau, H., Ong, C.N., Onsongo, M.T., Page, S.W., Park, D., Patel, M., Phillips, T., Pineiro, M., Pronczuk, J., Rogers, H.S., Rubin, C., Sabino, M., Schaafsma, A., Shephard, G., Stroka, J., Wild, C., Williams, J.T. and Wilson, D. 2006. Workgroup report: Public health strategies for reducing aflatoxin exposure in developing countries. *Environmental Health Perspectives* 114(12): 1898–1903.

Sudhakar, P., Latha, P., Sreenivasulu, Y., Reddy, B.V., Hemalatha, T.M., Balakrishna, M. and Reddy, K.R. 2009. Inhibition of *Aspergillus flavus* colonization and aflatoxin (AfB1) in peanut by methyleugenol. *Indian Journal of Experimental Biolology* 47(1):63–67.

Sugita-Konishi, Y., Sato, T., Saito, S., Nakajima, M., Tabata, S., Tanaka, T., Norizuki, H., Itoh, Y., Kai, S., Sugiyama, K., Kamata, Y., Yoshiike, N. and Kumagai, S. 2010. Exposure to aflatoxins in Japan: Risk assessment for aflatoxin B_1. *Food Additives & Contaminants: Part A* 27(3): 365–372.

Tajkarimi, M., Aliabadi-Sh, F., Salah Nejad, A., Poursoltani, H., Motallebi, A.A. and Mahdavi, H. 2008. Aflatoxin M_1 contamination in winter and summer milk in 14 states in Iran. *Food Control* 19(11): 1033–1036.

Tajkarimi, M., Shojaee Aliabadi, F., Salah Nejad, M., Pursoltani, H., Motallebi, A.A. and Mahdavi, H. 2007. Seasonal study of aflatoxin M_1 contamination in milk in five regions in Iran. *International Journal of Food Microbiology* 116(3): 346–349.

Taklimi, S.M.S.M., Ghahri, H., and Isakan, M.A. 2012. Influence of different levels of humic acid and esterified glucomannan on growth performance and intestinal morphology of broiler chickens. *Agricultural Sciences* 3(5): 663–668.

Tchana, A.N., Moundipa, P.F. and Tchouanguep, F.M. 2010. Aflatoxin contamination in food and body fluids in relation to malnutrition and cancer status in Cameroon. *International Journal of Environmental Research and Public Health* 7(1): 178–188.

Teniola, O.D., Addo, P.A., Brost, I.M., Färber, P., Jany, K.D., Alberts, J.F., van Zyl, W.H., Steyn, P.S. and Holzapfel ,W.H. 2005. Degradation of aflatoxin B(1) by cell-free extracts of *Rhodococcus erythropolis* and *Mycobacterium fluoranthenivorans* sp. nov. DSM44556(T). *International Journal of Food Microbiology* 105(2): 111–117.

Thomas, A.E., Coker, H.A., Odukoya, O.A., Isamah, G.K. and Adepoju-Bello, A. 2003. Aflatoxin contamination of *Arachis hypogaea* (groundnuts) in Lagos area of Nigeria. *Bulletin of Environmental Contamination and Toxicology* 71(1): 42–45.

Timper, P., Wilson, D.M., Holbrook, C.C. and Maw, B.W. 2004. Relationship between *Meloidogyne arenaria* and aflatoxin contamination in peanut. *Journal of Nematology* 36(2): 167–170.

Tiongco, M. 2011. *Estimating demand for aflatoxin risk reducing strategies in Kenya*. Presentation at the Closing Workshop: Aflacontrol Conference, Nairobi, Kenya, 30 November 2011. (Available from http://www.slideshare.net/pchenevixtrench/estimating-demand-for-aflatoxin-risk-reducing-strategies-in-kenya-2) (Accessed 28 August 2014)

Torres, P., Guzmán-Ortiz, M. and Ramírez-Wong, B. 2001. Revising the role of pH and thermal treatments in aflatoxin content reduction during the tortilla and deep frying processes. *Journal of Agricultural and Food Chemistry* 49(6): 2825–2829.

Trucksess, M.W., Stoloff L., Brumley, W.C., Wilson, D.M., Hale, O.M., Sangster, L.T. and Miller, D.M. 1982. Aflatoxicol and aflatoxins B_1 and M_1 in the tissues of pigs receiving aflatoxin. *Journal–Association of Official Analytical Chemists* 65(4): 884–887.

Trucksess, M.W., Stoloff, L., Young, K., Wyatt, R.D. and Miller, B.L. 1983. Aflatoxicol and aflatoxins B_1 and M_1 in eggs and tissues of laying hens consuming aflatoxin-contaminated feed. *Poultry Science* 62(11): 2176–2182.

Turconi, G., Guarcello, M., Livieri, C., Comizzoli, S., Maccarini, L., Castellazzi, A.M., Pietri, A., Piva, G. and Roggi, C. 2004. Evaluation of xenobiotics in human milk and ingestion by the newborn: An epidemiological survey in Lombardy (Northern Italy). *European Journal of Nutrition* 43(4): 191–197.

Turner, N.W., Subrahmanyam, S. and Piletsky, S.A. 2009. Analytical methods for determination of mycotoxins: A review. *Analytica Chimica Acta* 632(2): 168–180.

Turner, P.C., Collinson, A.C., Cheung, Y.B., Gong, Y., Hall, A.J., Prentice, A.M. and Wild, C.P. 2007. Aflatoxin exposure in utero causes growth faltering in Gambian infants. *International Journal of Epidemiology* 36(5): 1119–1125.

Turner, P.C., Mendy, M., Whittle, H., Fortuin, M., Hall, A.J. and Wild, C.P. 2000. Hepatitis B infection and aflatoxin biomarker levels in Gambian children. *Tropical Medicine & International Health* 5(12): 837–841.

Turner, P.C., Moore, S.E., Hall, A.J., Prentice, A.M. and Wild, C.P. 2003. Modification of immune function through exposure to dietary aflatoxin in Gambian children. *Environmental Health Perspectives* 111(2): 217–220.

Turner, P.C., Sylla, A., Gong, Y.Y., Diallo, M.S., Sutcliffe, A.E., Hall, A.J. and Wild, CP. 2005. Reduction in exposure to carcinogenic aflatoxins by postharvest intervention measures in West Africa: A community-based intervention study. *Lancet* 365(9475): 1950–1956.

Udoh, J.M., Cardwell, K.F. and Ikotun, T. 2000. Storage structures and aflatoxin content of maize in five agro-ecological zones of Nigeria. *Journal of Stored Products* 36(2): 187–201.

UNICEF (United Nations Children's Fund). 2013. *Improving child nutrition: The achievable imperative for global progress.* New York, NY: UNICEF.

USAID (United States Agency for International Development). 2003. *Aflatoxin: A synthesis of the research in health, agriculture and trade.* (Available from http://agarchive.kdid.org/library/aflatoxin-synthesis-research-health-agriculture-and-trade) (Accessed 28 August 2014)

Uriah, N., Ibeh, I.N. and Oluwafemi, E. 2001. A study on the impact of aflatoxin on human reproduction. *African Journal of Reproductive Health* 5: 106–110.

Urio, E.M., Juma, A., Mwanyika, S., Mlingi, N.V., Ndunguru, G.T. and Ndossi, G.D. 2006. The occurrence of aflatoxin M_1 in fresh cow milk retailed in Dar es Salaam, Tanzania. In: Njapau, H., Trujillo, S., van Egmond, H.P. and Park, D.L. (eds), *Mycotoxins and phycotoxins: Advances in determination, toxicology, and exposure management*. Proceedings of the XIth International IUPAC Symposium on Mycotoxins and Phycotoxins, May 17–21, 2004, Bethesda, Maryland, USA. Wageningen, the Netherlands: Wageningen Academic Publishers. pp. 203–208.

Van Egmond, H. and Jonker, M. 2004. *Worldwide regulations for mycotoxins in food and feed in 2003.* Rome, Italy: FAO.

Van Rensburg, S.J., Cook-Mozaffari, P., Van Schalkwyk, D.J., Van der Watt, J.J., Vincent, T.J. and Purchase, I.F. 1985. Hepatocellular carcinoma and dietary aflatoxin in Mozambique and Transkei. *British Journal of Cancer* 51(5): 713–726.

Vardon, P., McLaughlin, C. and Nardinelli, C. 2003. Potential economic costs of mycotoxins in the United States. In: Richard, J. and Payne, G.A. (eds), *Mycotoxins: Risks in plant, animal, and human systems*. Ames, Iowa: CAST. pp. 136–142.

Villa, P. and Markaki, P. 2009. Aflatoxin B_1 and ochratoxin A in breakfast cereals from athens market: Occurrence and risk assessment. *Food Control* 20(5): 455–461.

Vries, H.D., Maxwell, S. and Hendrickse, R. 1989. Foetal and neonatal exposure to aflatoxins. *Acta Paediatrica* 78(3): 373–378.

Wagacha, J.M. and Muthomi, J.W. 2008. Mycotoxin problem in Africa: Current status, implications to food safety and health and possible management strategies. *International Journal of Food Microbiology* 124(1): 1–12.

Wagacha, J.M., Mutegi, C., Karanja, L., Kimani, J. and Christie, M.E. 2013. Fungal species isolated from peanuts in major Kenyan markets: Emphasis on *Aspergillus* section *Flavi*. *Crop Protection* 52: 1–9.

Waliyar, F., Lava Kumar, P., Nigam, S.N., Sharma, K.K., Upadhyaya, H.D., Thakur, R.P., Aruna, R. and Mallikarjuna, N. 2007a. *Integrated management of aflatoxin contamination: Progress towards enhancing food and feed quality and human and animal health.* Andhra Pradesh, India: ICRISAT. (Available from http://oar.icrisat.org/id/eprint/5833) (Accessed 28 August 2014)

Waliyar, F., Ntare, B.R., Diallo, A.T., Kodio, O. and Diarra, B. 2007b. *On-farm management of aflatoxin contamination of groundnut in West Africa: A synthesis report.* Andhra Pradesh, India: ICRISAT. (Available from http://oar.icrisat.org/id/eprint/402) (Accessed 28 August 2014)

Waliyar, F., Osiru, M., Sudini, H.K. and Njoroge, S. 2013. *Reducing aflatoxins in groundnuts through integrated management and biocontrol.* IFPRI 2020 Focus 20, Brief 18. Washington, DC: IFPRI.

Waliyar, F., Reddy, S.V., Subramanyam, K., Craufurd, P.Q., Wheeler, T.R., Rama Devi, K., Thakur, R.P., Nigam, S.N., Upadhyaya, H.D. and Desai, S. 2005. *Management of aflatoxins in groundnut in Southern India.* In: Summary proceedings of the International Peanut Conference 2005: "Prospects and Emerging Opportunities for Peanut Quality and Utilization Technology", Bangkok, Thailand, 9–12 January 2005. pp. 106–108. (Available from http://oar.icrisat.org/id/eprint/5399) (Accessed 28 August 2014)

Wang, P., Afriyie-Gyawu, E., Tang, Y., Johnson, N.M., Xu, L., Tang, L., Huebner, H.J., Ankrah, N.-A., Ofori-Adjei, D., Ellis, W., Jolly, P.E., Williams, J.H., Wang, J.-S. and Phillips, T.D. 2008. NovaSil clay intervention in Ghanaians at high risk for aflatoxicosis: II. Reduction in biomarkers of aflatoxin exposure in blood and urine. *Food Additives & Contaminants: Part A* 25(5): 622–634.

Whitaker, T.B., Dickens, J.W. and Monroe, R.J. 1979. Variability associated with testing corn for aflatoxin. *Journal of the American Oil Chemists Society* 56(9): 789–794.

Whitaker, T.B., Whitten, M.E. and Monroe, R.J. 1976. Variability associated with testing cottonseed for aflatoxin. *Journal of the American Oil Chemists Society* 53(7): 502–505.

Whitlow, L.W. and Hagler, W.M. Jr. 1997. Effects of mycotoxins on the animal: The producer's perspective. In: *Silage: Field to feedbunk*. Hershey, PA: Northeast Regional Agricultural Engineering Service. pp. 222–232.

WHO (World Health Organization). 2002. *Evaluation of certain mycotoxins in food: Fifty-sixth report of the Joint FAO/WHO Expert Committee on Food Additives.* Geneva, Switzerland: WHO.

Wild, C.P. 2007. Aflatoxin exposure in developing countries: The critical interface of agriculture and health. *Food and Nutrition Bulletin* 28(2 Suppl): S372–S380.

Wild, C.P. and Hall, A.J. 2000. Primary prevention of hepatocellular carcinoma in developing countries. *Mutation Research/Reviews in Mutation Research* 462(2–3): 381–393.

Wild, C.P., Fortuin, M., Donato, F., Whittle, H.C., Hall, A.J., Wolf, C.R. and Montesano, R. 1993. Aflatoxin, liver enzymes, and hepatitis B virus infection in Gambian children. *Cancer Epidemiology Biomarkers & Prevention* 2(6): 555–561.

Wild, C.P., Hudson, G.J., Sabbioni, G., Chapot, B., Hall, A.J., Wogan, G.N., Whittle, H., Montesano, R. and Groopman, J.D. 1992. Dietary intake of aflatoxins and the level of albumin-bound aflatoxin in peripheral blood in the Gambia, West Africa. *Cancer Epidemiology Biomarkers & Prevention* 1(3): 229–234.

Wild, C.P., Jiang, Y.-Z., Allen, S.J., Jansen, L.A.M., Hall, A.J. and Montesano, R. 1990. Aflatoxin-albumin adducts in human sera from different regions of the world. *Carcinogenesis* 11(12): 2271–2274.

Wild, C.P., Pionneau, F.A., Montesano, R., Mutiro, C.F. and Chetsanga, C.J. 1987. Aflatoxin detected in human breast milk by immunoassay. *International Journal of Cancer* 40(3): 328–333.

Wild, C.P., Yin, F., Turner, P.C., Chemin, I., Chapot, B., Mendy, M., Whittle, H., Kirk, G.D., Hall, A.J. 2000. Environmental and genetic determinants of aflatoxin-albumin adducts in the Gambia. *International Journal of Cancer* 86(1): 1–7.

Williams, J.H. 2008. Institutional stakeholders in mycotoxin issues – Past, present and future. In: Leslie, J.F., Bandyopadhyay, R. and Visconti A. (eds), *Mycotoxins: Detection methods, management and public health and agricultural trade*. Oxfordshire, UK: CAB International. pp. 349–358.

Williams, J.H., Grubb, J.A., Davis, J.W., Wang, J.-S., Jolly, P.E., Ankrah, N.-A., Ellis, W.O., Afriyie-Gyawu, E., Johnson, N.M., Robinson, A.G. and Phillips, T.D. 2010. HIV and hepatocellular and

esophageal carcinomas related to consumption of mycotoxin-prone foods in sub-Saharan Africa. *American Journal of Clinical Nutrition* 92(1): 154–160.

Williams, J.H., Phillips, T.D., Jolly, P.E., Stiles, J.K., Jolly, C.M. and Aggarwal, D. 2004. Human aflatoxicosis in developing countries: A review of toxicology, exposure, potential health consequences, and interventions. *American Journal of Clinical Nutrition* 80(5): 1106–1122.

Williams, J.R., Nokes, D.J. and Anderson, R.M. 1996. Targeted hepatitis B vaccination – A cost effective immunisation strategy for the UK? *Journal of Epidemiology & Community Health* 50(6): 667–673.

Wu, F. 2004. Mycotoxin risk assessment for the purpose of setting international regulatory standards. *Environmental Science & Technology* 38(15): 4049–4055.

Wu, F. 2013. *Aflatoxin exposure and chronic human diseases: Extimates of burden of disease.* IFPRI 2020 Focus 20, Brief 3. Washington, DC: IFPRI.

Wu, F. and Khlangwiset, P. 2010a. Evaluating the technical feasibility of aflatoxin risk reduction strategies in Africa. *Food Additives & Contaminants: Part A* 27(5): 658–676.

Wu, F. and Khlangwiset, P. 2010b. Health economic impacts and cost-effectiveness of aflatoxin-reduction strategies in Africa: Case studies in biocontrol and post-harvest interventions. *Food Additives & Contaminants: Part A* 27(4): 496–509.

Wu, F. and Munkvold, G.P. 2008. Mycotoxins in ethanol co-products: Modeling economic impacts on the livestock industry and management strategies. *Journal of Agricultural and Food Chemistry* 56(11): 3900–3911.

Wu, F., Bhatnagar, D., Bui-Klimke, T., Carbone, I., Hellmich, R., Munkvold, G., Paul, P., Payne, G. and Takle, E. 2011a. Climate change impacts on mycotoxin risks in US maize. *World Mycotoxin Journal* 4(1): 79–93.

Wu, F., Liu, Y. and Bhatnagar, D. 2008. Cost-effectiveness of aflatoxin control methods: Economic incentives. *Toxin Reviews* 27(3-4): 203–225.

Wu, F., Liu, Y., Narrod, C., Tiongco, M. and Scott, R. 2011b. *Measuring the burden of aflatoxin-induced human disease.* Presentation at the Closing Workshop: Aflacontrol Conference, Nairobi, Kenya, 30 November 2011. (Available from http://www.slideshare.net/pchenevixtrench/measuring-the-burden-of-aflatoxin-induced-human-disease) (Accessed 28 August 2014)

Wu, H.C., Wang, Q., Yang, H.I., Ahsan, H., Tsai, W.Y., Wang, L.Y., Chen, S.Y., Chen, C.J. and Santella, R.M. 2009. Aflatoxin B1 exposure, hepatitis B virus infection, and hepatocellular carcinoma in Taiwan. *Cancer Epidemiology Biomarkers & Prevention* 18(3): 846–853.

Xiong, B. and Beghin, J. 2011. *Aflatoxin redux: Does European aflatoxin regulation hurt groundnut exporters from Africa?* (Available from http://dx.doi.org/10.2139/ssrn.1621123) (Accessed 28 August 2014)

Yard, E.E., Daniel, J.H., Lewis, L.S., Rybak, M.E., Paliakov, E.M., Kim, A.A., Montgomery, J.M., Bunnell, R., Abudo, M.U., Akhwale, W., Breiman, R.F. and Sharif, S.K. 2013. Human aflatoxin exposure in Kenya, 2007: A cross-sectional study. *Food Additives & Contaminants: Part A* 30(7): 1322–1331.

Younis, Y.M.H. and Malik, K.M. 2003. TLC and HPLC assays of aflatoxin contamination in Sudanese peanuts and peanut products. *Kuwait Journal of Science and Engineering* 30(1): 79–94.

Zaghini, A., Martelli, G., Roncada, P., Simioli, M. and Rizzi, L. 2005. Mannanoligosaccharides and aflatoxin B1 in feed for laying hens: Effects on egg quality, aflatoxins B1 and M1 residues in eggs, and aflatoxin B1 levels in liver. *Poultry Science* 84(6): 825–832.

Zarba, A., Wild, C.P., Hall, A.J., Montesano, R., Hudson, G.J. and Groopman, J.D. 1992. Aflatoxin M1 in human breast milk from the Gambia, West Africa, quantified by monoclonal antibody immunoaffinity chromatography and HPLC. *Carcinogenesis* 13(5): 891–894.

Appendix 1 : Aflatoxin regulations by country, as of 2003

Country/region	Aflatoxins regulated	Feeds regulated	Animals regulated	Maximum limits (ppb)
Bangladesh	B1, B2, G1, G2	Mixed feed	Poultry	100
Barbados	B1, B2, G1, G2	All feedstuffs		50
Brazil	B1, B2 ,G1, G2	Feed and ingredients		50
Canada	B1, B2 ,G1, G2	All feeds	All animals	20
Chile	B1, B2, G1, G2	Complete	Poultry, goat, cattle	30
Chile	B1, B2, G1, G2	Complete		10
Chile	B1, B2, G1, G2	Feed ingredients except groundnuts, cottonseed, maize and their derivatives		50
Chile	B1, B2, G1, G2	Groundnuts, cottonseed, maize and their derivatives as feed ingredients		200
Colombia	B1, B2, G1, G2	Feed	Rabbit, trout	10
Colombia	B1, B2, G1, G2	Feed	Poultry, dog, cat, fish	20
Colombia	B1, B2, G1, G2	Feed	Bovine, pig	50
Colombia	B1, B2, G1, G2	Maize and products		20
Colombia	B1, B2, G1, G2	Sorghum		40
Costa Rica	B1, B2, G1, G2	Maize		50
Cote d'Ivoire	B1, B2, G1, G2	Complete	Pigs, poultry (except ducks and young animals)	38
Cote d'Ivoire	B1, B2, G1, G2	Complete	Dairy cattle	50
Cote d'Ivoire	B1, B2, G1, G2	Complete	Cattle, sheep, goats	75
Cote d'Ivoire	B1, B2,G1, G2	Complete		10
Cote d'Ivoire	B1, B2,G1, G2	Straight		100
Cuba	B1, B2,G1, G2	Feed and ingredients		5
Egypt	B1	Feed	Animal, chicken	10
Egypt	B1, B2,G1, G2	Feed	Animal, chicken	20
El Salvador	B1	Supplementary feeds	Pigs, poultry, dairy cattle	20
El Salvador	B1	Composite feedstuffs	Cattle, sheep, goats	20
El Salvador	B1	All feedstuffs		10
Estonia	B1, B2, G1, G2	Ingredients of vegetable origin		100
Estonia	B1, B2,G1, G2	Complete feedstuffs for cattle, pigs and other farm animals		100
Estonia	B1, B2, G1, G2	Complete feedstuffs for young cattle, young pigs and other young farm animals		50
Estonia	B1, B2, G1, G2	Complete feedstuffs for milk producing animals		20
Estonia	B1, B2, G1, G2	Complementary feedstuffs for cattle, pigs and other farm animals		50
Estonia	B1, B2, G1, G2	Complementary feedstuffs for young cattle, young pigs and other young farm animals		10
European Union	B1	Complete	Pigs, poultry (except young animals)	20
European Union	B1	Complementary feedstuffs	Pigs, poultry (except young animals)	20
European Union	B1	Complete	Dairy cattle	5
European Union	B1	Complete	Cattle, sheep, goats (except young animals)	20
European Union	B1	Complementary feedstuffs	Cattle, sheep, goats (except young animals)	20
European Union	B1	Complete	Calves, lambs	10
European Union	B1	Other complete feedstuffs		10

European Union	B1	Other complementary feedstuffs		5
European Union	B1	All feed materials		20
Guatemala	B1, B2, G1, G2	Feed concentrate		20
India	B1	Peanut meal	Export	120
Iran	B1	Fish meal, meat meal, bone meal, blood meal, single cell protein, rice and wheat bran	Sheep, goats, beef cattle	10
Iran	B1	Soya bean meal, sunflower meal, sesame seed meal, olive meal and other meals from oil producing seeds	Sheep, goats, beef cattle	10
Iran	B1, B2, G1, G2	Soya bean meal, sunflower meal, sesame seed meal, olive meal and other meals from oil producing seeds	Sheep, goats, beef cattle	20
Iran	B1, B2, G1, G2	Premixes including vitamins and mineral premixes	Sheep, goats, beef cattle	10
Iran	B1	Complete	Sheep, goats, beef cattle	50
Iran	B1	Soya bean meal, sunflower meal, sesame seed meal, olive meal and other meals from oil producing seeds	Poultry, calf, lamb, kid, dairy sheep, dairy goats, dairy cattle	5
Iran	B1, B2, G1, G2	Soya bean meal, sunflower meal, sesame seed meal, olive meal and other meals from oil producing seeds	Poultry, calf, lamb, kid, dairy sheep, dairy goats, dairy cattle	20
Iran	B1	Fish meal, meat meal, bone meal, blood meal, single cell protein, rice and wheat bran	Poultry, calf, lamb, kid, dairy sheep, dairy goats, dairy cattle	5
Iran	B1, B2, G1, G2	Fish meal, meat meal, bone meal, blood meal, single cell protein, rice and wheat bran	Poultry, calf, lamb, kid, dairy sheep, dairy goats, dairy cattle	20
Iran	B1, B2, G1, G2	Premixes including vitamins and mineral premixes	Poultry	10
Iran	B1	Complete	Layers and broiler parent and grandparent stocks	5
Iran	B1, B2, G1, G2	Complete	Layers and broiler parent and grandparent stocks	20
Iran	B1	Complete	Layers and breeders (broilers and layers)	10
Iran	B1, B2, G1, G2	Complete	Layers and breeders (broilers and layers)	20
Iran	B1, B2, G1, G2	Premixes including vitamins and mineral premixes	Calf, lamb, kid, dairy sheep, dairy goats, dairy cattle	5
Iran	B1	Complete	Calf, lamb, kid, dairy sheep, dairy goats, dairy cattle	5
Iran	B1	Complete	Broilers and pullet	10
Iran	B1	Maize	All animals	5
Iran	B1, B2, G1, G2	Maize	All animals	20
Iran	B1	Cottonseed meal		15
Iran	B1, B2, G1, G2	Cottonseed meal		50
Israel	B1, B2, G1, G2	Grain	All animals	20
Japan	B1	Complete	Cattle, pigs, chicken, quail (except young and dairy cows)	20
Japan	B1	Complete	Calves, dairy cows, piglets, young chicken, broilers	10
Jordan	B1	Feedstuffs	All animals	15
Jordan	B1, B2, G1, G2	Feedstuffs	All animals	30
Latvia	B1	Animal feed		5
Mexico	B1, B2, G1, G2	Cereals	Fattening cows, pigs	200

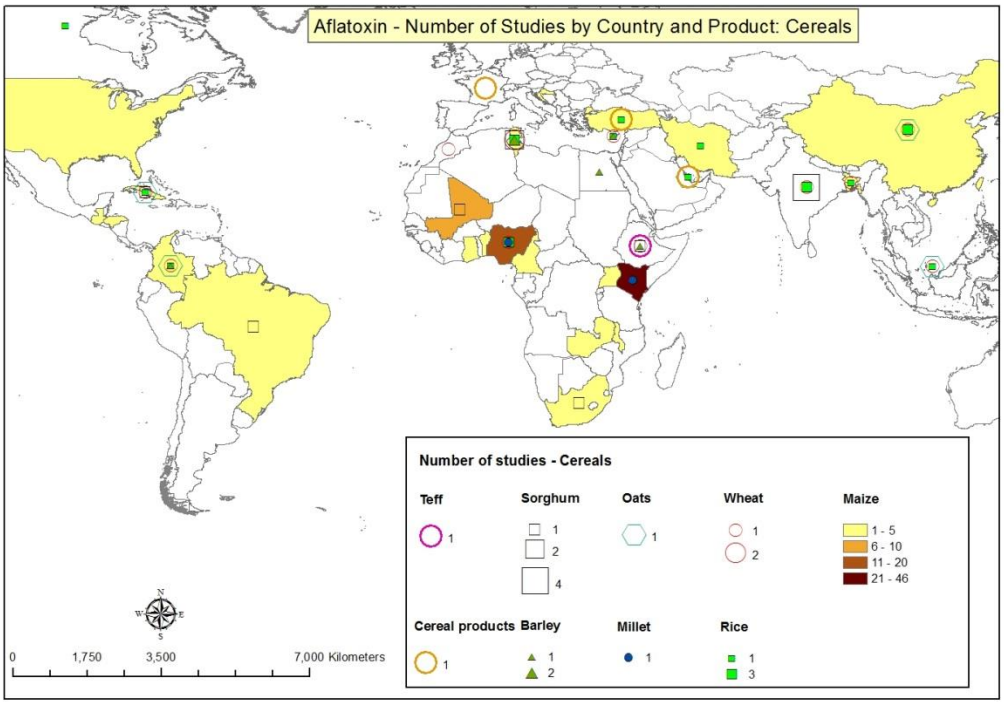
Mexico	B1, B2, G1, G2	Feedstuffs	Dairy cattle, poultry	0
Morocco	B1	Complete feedstuffs	Pigs, poultry (except young animals)	20
Morocco	B1	Complementary feedstuffs	Pigs, poultry (except young animals)	30
Morocco	B1	Complete feedstuffs	Dairy animals	5
Morocco	B1	Other complementary feedstuffs	Dairy animals	10
Morocco	B1	Complete feedstuffs	Cattle, sheep, goats (except dairy and young animals)	50
Morocco	B1	Complementary feedstuffs	Cattle, sheep, goats (except dairy and young animals)	50
Morocco	B1	Complete feedstuffs	Calves, lambs	10
Morocco	B1	Simple feedstuffs (except peanuts, copra, cottonseed, babassu, maize and their products)		50
Morocco	B1	Peanuts, copra, cottonseed, babassu, maize and their products		20
Morocco	B1	Other complete feedstuffs		10
Mozambique	B1, B2, G1, G2	Peanut, maize, peanut butter	All animals	10
Mozambique	B1, B2, G1, G2	Cereals and feedstuffs	All animals	10
Nepal	B1, B2, G1, G2	Feedstuffs	All animals	50
Oman	B1	Complete feedstuffs	Poultry	20
Philippines	B1	Mixed feed	All animals	20
Philippines	B1	Copra and copra products	All animals	20
Republic of Korea	B1	Complete	Other	20
Republic of Korea	B1	Complete	Calves, chicken, piglets, broilers (early stage), dairy cattle	10
Republic of Korea	B1	Feed ingredients: vegetable proteins, grains, by-products of grains and food		50
Senegal	B1	Straight feedstuffs: peanut products	All animals	50
Senegal	B1	Feedstuff ingredients: peanut products	All animals	300
Serbia and Montenegro	B1, B2, G1, G2	Feed	Pigs, poultry	20
Serbia and Montenegro	B1, B2, G1, G2	Feed	Oxen, sheep, goats	50
Serbia and Montenegro	B1, B2, G1, G2	Feed	Chicken, pigs (until 50 kg), calves, young turkeys, ducklings, cows	10
Suriname	B1, B2, G1, G2	Feedstuffs	All animals	30
Sweden	B1	Feedstuff ingredients	Other	50
Sweden	B1	Cereal grains and forages as feedstuff ingredients	Dairy cattle	1
Sweden	B1	Feedstuff ingredients	Dairy cattle	10
Sweden	B1	Complete feedstuff (including forage)	Dairy cattle	1.5
Sweden	B1	Mixed feedstuffs (excluding forages)	Dairy cattle	3
Switzerland	B1	Complementary feeds	Pigs, poultry (except young animals)	30
Switzerland	B1	Complete feeds	Pigs, poultry (except young animals)	20
Switzerland	B1	Complete and complementary feeds	Other	10
Switzerland	B1	Complementary feed	Dairy cows, dairy sheep, dairy goats	5
Switzerland	B1	Complete and complementary	Cattle, sheep, goats (except dairy and young animals)	50

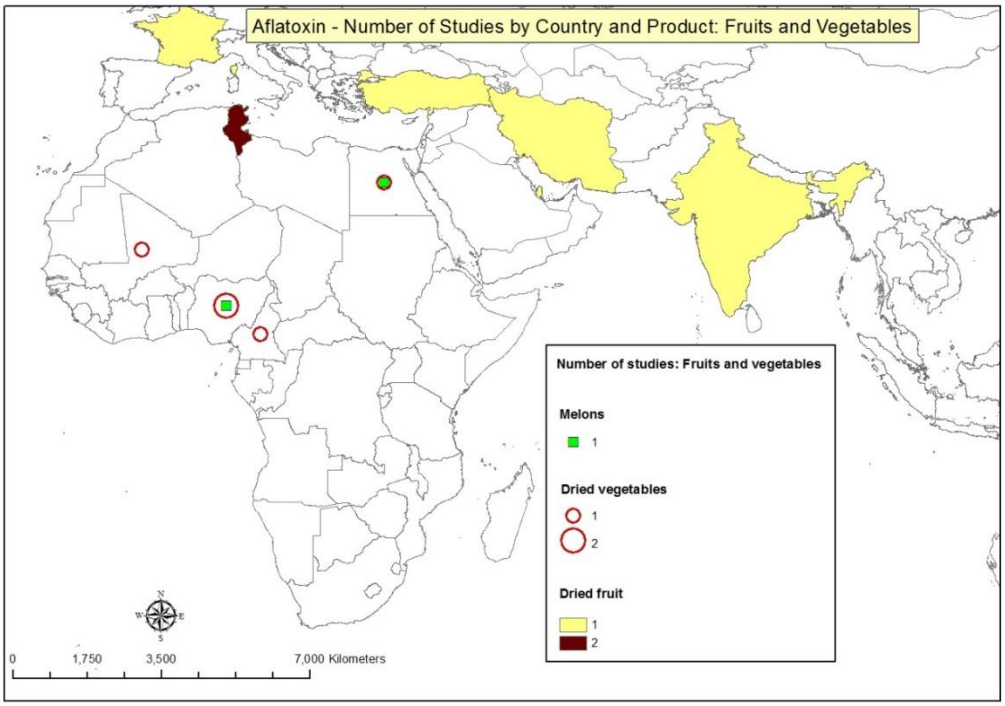
Switzerland	B1	Babassu seed, cotton seed, peanut, coconut, maize kernel, palm kernel and their products as raw materials	All animals	200
Switzerland	B1	Babassu seed, cotton seed, peanut, coconut, maize kernel, palm kernel and their products as single feed materials	All animals	20
Switzerland	B1	Other single feeds/raw materials	All animals	50
Syria	B1, B2, G1, G2	Complete	Other	20
Syria	B1, B2, G1, G2	Complete	Cattle	10
Taiwan	B1, B2, G1, G2	Maize: raw material	All animals	50
Taiwan	B1	Feedstuffs	All animals	25–100
Tanzania	B1	Complete	All animals	5
Tanzania	B1, B2, G1, G2	Complete	All animals	10
Turkey	B1	Mixed feed	Ruminants (except young)	50
Turkey	B1	Mixed feed	Poultry (except young)	20
Turkey	B1	Mixed feed	Other	10
Turkey	B1	Feedstuffs	All animals	50
Ukraine	B1	Combined feed	Poultry	25
Ukraine	B1	Combined feed	Non-productive animals	10
Ukraine	B1	Combined feed	Dairy cows, piglets	50
Ukraine	B1	Combined feed	Calves and sheep older than 4 months, animals for meat, breeding bulls	100
United States of America	B1, B2, G1, G2	Maize, peanut and other products except cottonseed meal	Immature animals	20
United States of America	B1, B2, G1, G2	Maize and peanut products	Finishing swine over 45 kg	200
United States of America	B1, B2, G1, G2	Maize and peanut products	Fattening beef cattle	300
United States of America	B1, B2, G1, G2	Corn, corn products, cottonseed meal, and other animal feeds and feed ingredients	Dairy animals, for animal species or uses not specified above, or when the intended use is not known	20
United States of America	B1, B2, G1, G2	Maize and peanut products	Breeding beef cattle, breeding swine, or mature poultry	100
United States of America	B1, B2, G1, G2	Maize and peanut products	Beef cattle, swine, poultry	300
Zimbabwe	B1, G1	Complete	Poultry	10

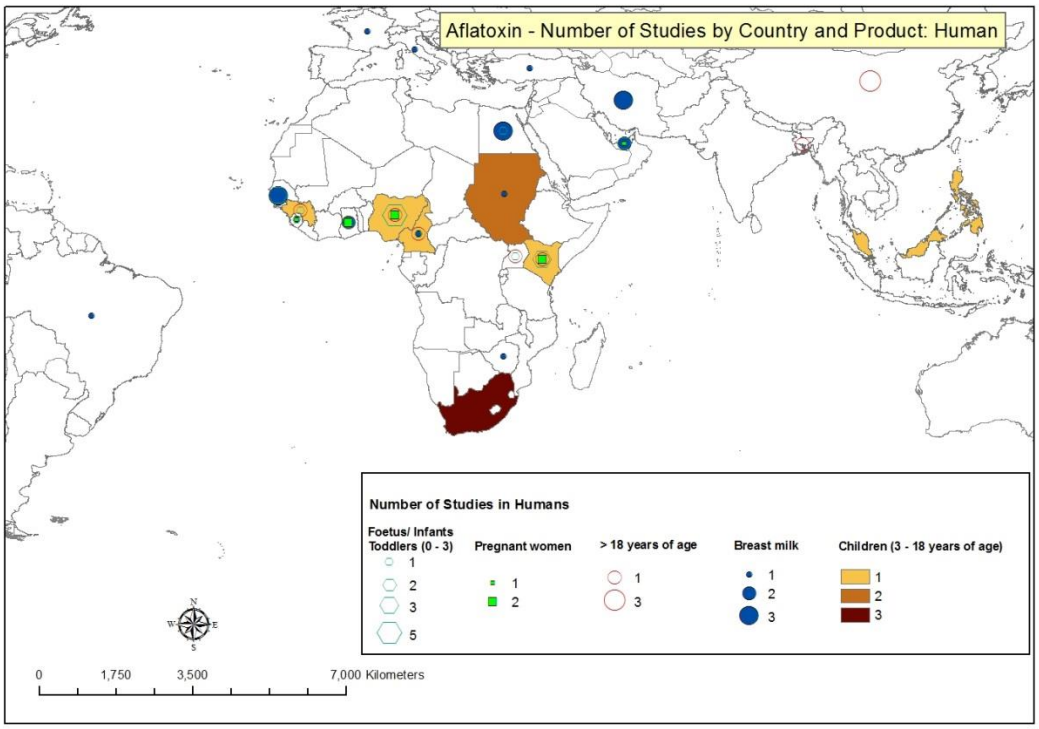
Note: ppb = parts per billion

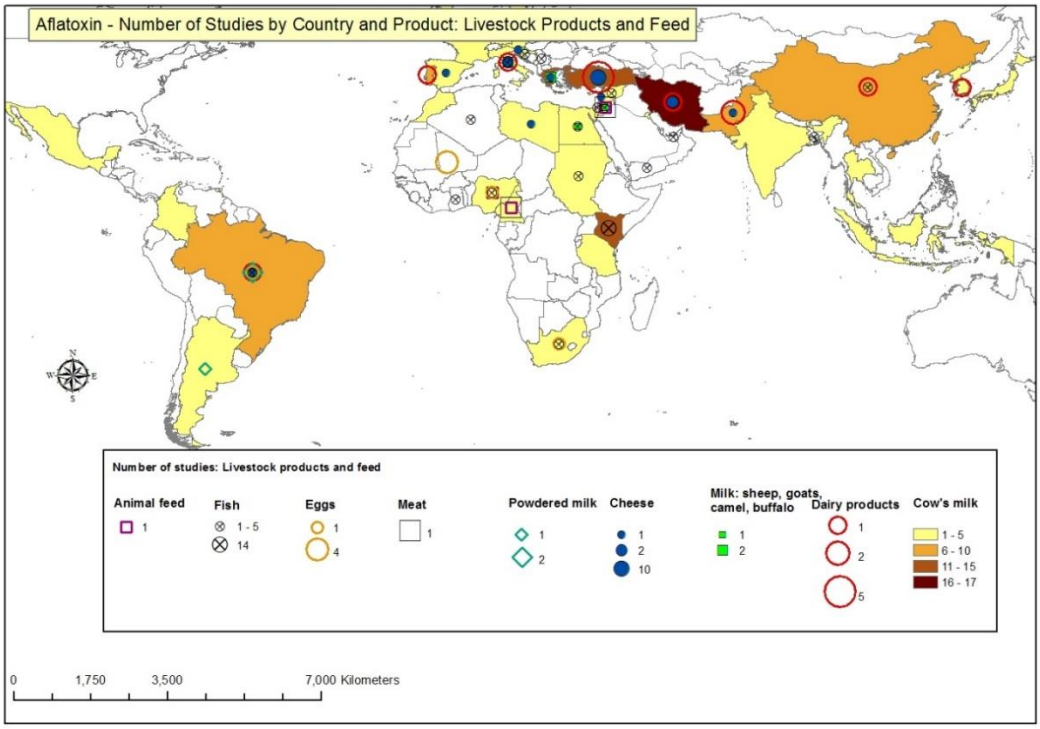
Appendix 2: Aflatoxin prevalence surveys by commodity and country

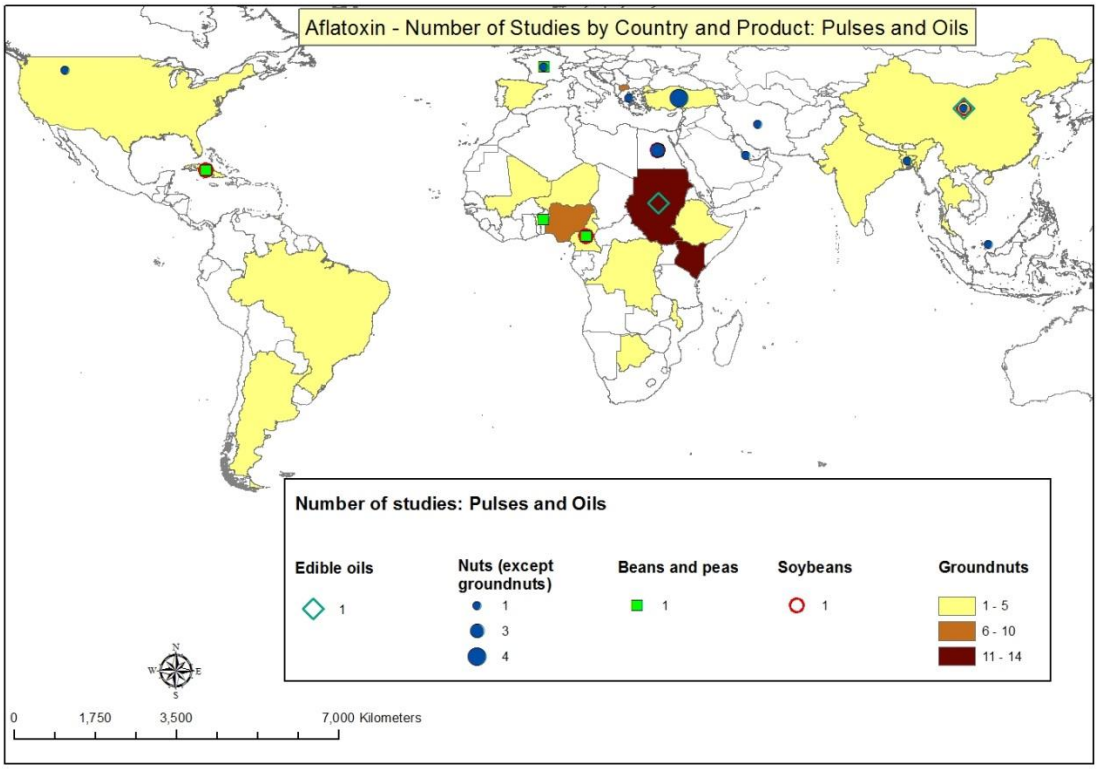
Maps delineate the number of aflatoxin surveys by commodity and country between 2000 and the present, in published literature written in English.

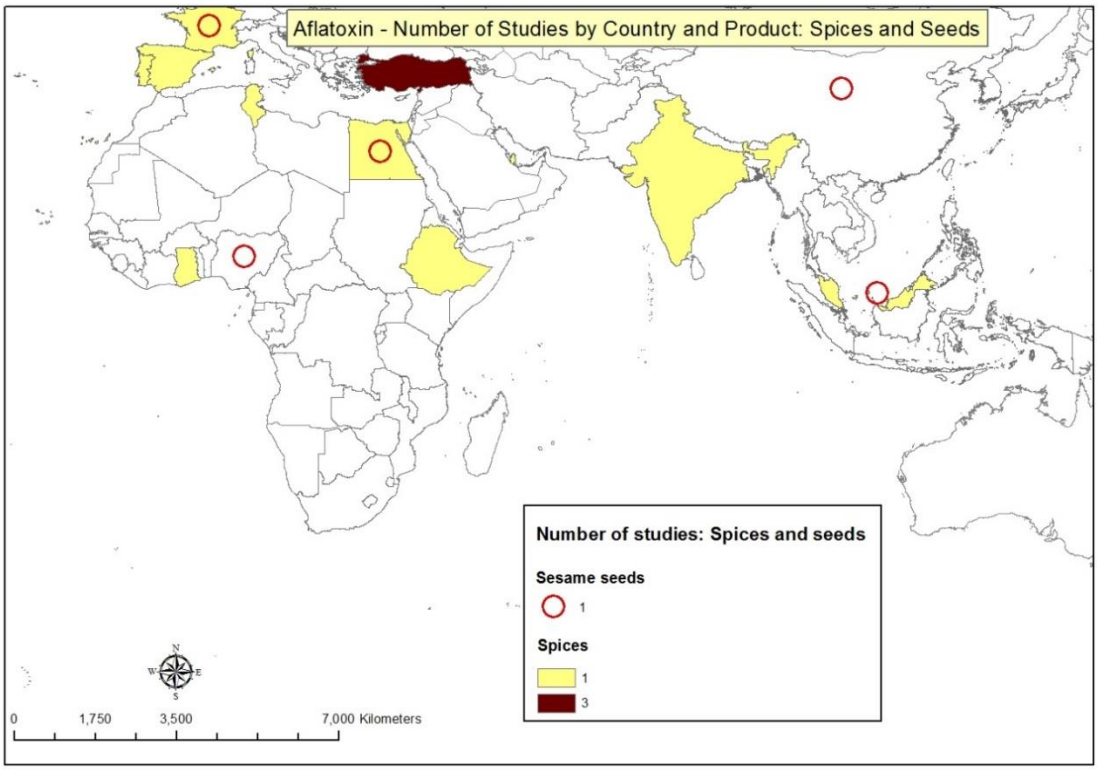


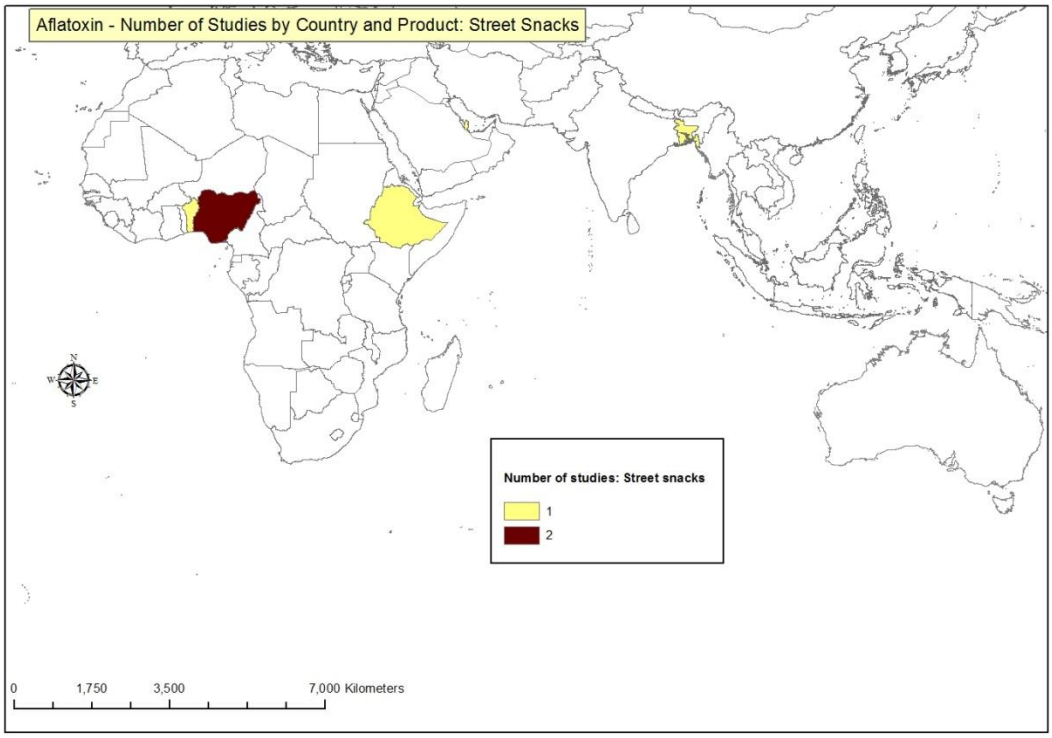


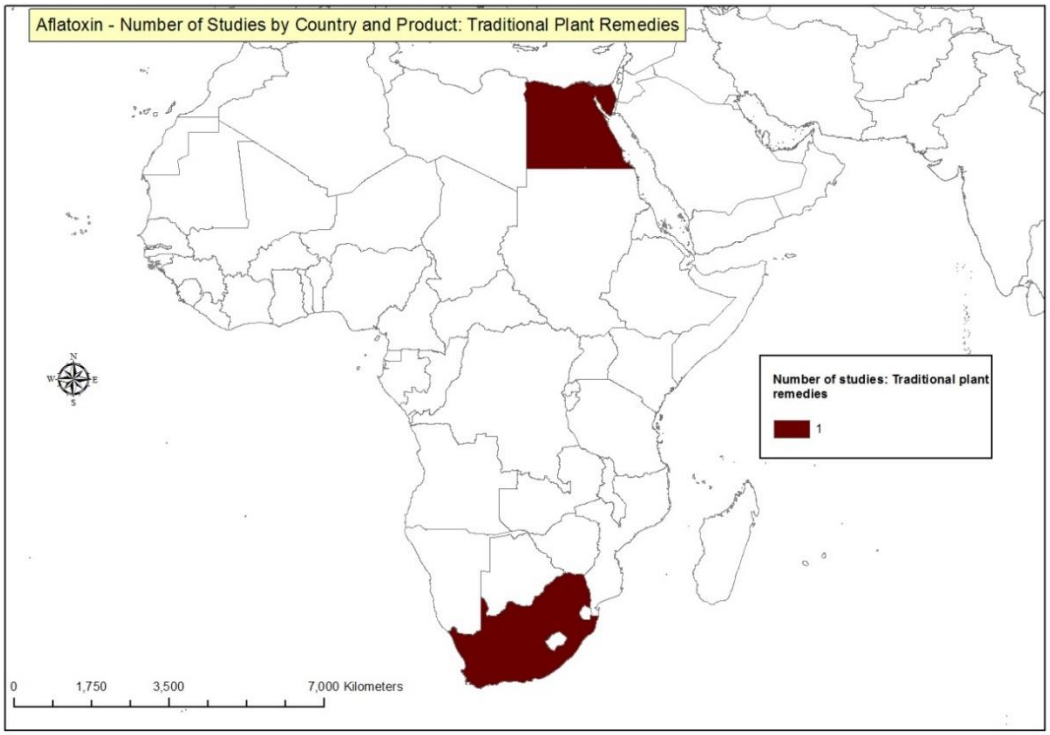

Aflatoxin - Number of Studies by Country and Product: Human
Number of Studies in Humans
Foetus/ Infants Toddlers (0 - 3)
1
2
3
5
Pregnant women
1
2
> 18 years of age
1
3
Breast milk
1
2
3
Children (3 - 18 years of age)
1
2
3
0
1,750
3,500
7,000 Kilometers


Aflatoxin - Number of Studies by Country and Product: Livestock Products and Feed
Number of studies: Livestock products and feed
Animal feed
1
Fish
1 - 5
14
Eggs
1
4
Meat
1
Powdered milk
1
2
Cheese
1
2
10
Milk: sheep, goats, camel, buffalo
1
2
Dairy products
1
2
5
Cow's milk
1 - 5
6 - 10
11 - 15
16 - 17
0
1,750
3,500
7,000 Kilometers


Aflatoxin - Number of Studies by Country and Product: Pulses and Oils
Number of studies: Pulses and Oils
Edible oils
1
Nuts (except groundnuts)
1
3
4
Beans and peas
1
Soybeans
1
Groundnuts
1 - 5
6 - 10
11 - 14
0
1,750
3,500
7,000 Kilometers


Aflatoxin - Number of Studies by Country and Product: Spices and Seeds
Number of studies: Spices and seeds
Sesame seeds
1
Spices
1
3
0
1,750
3,500
7,000 Kilometers


Aflatoxin - Number of Studies by Country and Product: Street Snacks
Number of studies: Street snacks
1
2
0
1,750
3,500
7,000 Kilometers


Aflatoxin - Number of Studies by Country and Product: Traditional Plant Remedies
Number of studies: Traditional plant remedies
1
0
1,750
3,500
7,000 Kilometers