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Aflatoxin Contamination in Foods and Feeds: A Special Focus on Africa

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1. Introduction

A groan can almost be audible from scientists who work or have an interest in the field of mycotoxicology (fungi and mycotoxins) to the effect of "not another review publication on AF". It is true that since the discovery of AF in the early 1960s, a huge literature on AF has developed. In one way, this literature can be considered appropriate, as these mycotoxins set the scene for a new burst of activity in the contamination of feeds and foods with mycotoxins, those produced by filamentous fungi, which has shown that these substances are not merely academic novelties, but have important effects in questions of food quality and human and animal health. Further, the principal member of this mycotoxin group, AFB_1, one of the most carcinogenic natural products formed in nature (D'Mello, 2003), a major cause of hepatocellular carcinoma (liver cancer) in both animals and humans is rated as a Class IB carcinogen by the International Agency for Research on Cancer (IARC) (IARC, 1993) meaning that it is a proven cancer-inducing agent. It also occurs ubiquitously in the environment contaminating many different food and feed commodities. Rather interestingly, critics of the excessive focus on AF may perhaps, point out that one of the reasons it is intensely investigated is the ease with which it can be detected and measured due to its strong fluorescence and ultra violet (UV) light absorbing properties (Bhatnagar and Ehrlich, 2002) which skews attention away from other important mycotoxins not containing a chromophore such as the trichothecenes (TH) and fumonisins (FB). Another irony of the kudos given to AF is because of it discovery stemming mainly from the condition observed in Britain called Turkey X disease, where it was associated with deaths of tens of thousands of turkey poults (Blount, 1961) is that many of the symptoms of the disease fit those of cyclopiazonic acid (CPA) and later analysis of the groundnut used to produce feed for these birds was found to contain (CPA) in addition to AFB_1.

Whatever the merits and demerits of again reviewing literature on AF, this review has a specific purpose which has not been thoroughly explored in the past. This is to look at the ravages that this toxin has caused in Africa, not only from an animal and human health point of view, but also from the economic consequence of having agricultural commodities contaminated with these toxins. Although most countries of the world can be affected by AF, it is sub-Saharan Africa (SSA) that has suffered the most. It is difficult to defend this statement because of the lack of detailed information on the occurrence of AF in African crops. This is because, much of SSA agriculture occurs in impoverished rural areas and the lack of technical infrastructure in many African countries does not allow for routine quality control of even commercially produced commodities, never mind those produced by rural populations for their own consumption. Much of the data on the incidence of mycotoxins in SSA countries is generated by non-African agencies (e.g., IARC) (IARC 1993) or by academic institutions. This review attempts to bring together information from studies conducted over the years and attempts to discuss it in a way that will give some idea on the mycotoxin problem in the continent both from an agricultural, economic and health points of view. For reasons already highlighted, occurrence and effects of AF, principally AFB_1, is the best representative in doing this.

2. Factors enchancing the prevalence of aflatoxigenic fungi and aflatoxins in Africa

Prior to AF contamination, the food material must be infected with fungi that have the genetic capacity to synthesize and deposit the toxins on the foods and feeds before or after harvest. Only species of the genus *Aspergillus* are endowed with the 23 genes responsible for synthesis of AF. Members belonging to this genus are most abundant in the tropics and as such, are major food spoilage agents in warmer climates. The genus is metabolically versatile producing over twenty mycotoxins. Of the over 180 species of *Aspergillus*, only a few are aflatoxigenic. After the discovery of AF in the 1960s, *A. flavus* and *A. parasiticus* of the section *Flavi* were the only known AF producers producing the B and B/G types of AF, respectively (Blount, 1961). Other aflatoxigenic species that subsequently emerged are *A. nomius* (B and G types), *A. bombycis* (B and G AF), *A. ochraceoroseus,* and *A. pseudotamarii (*B type), but they occur less frequently (Peterson *et al*., 2001, Ito *et al*. 2001)). *A. tamarii, A. parvisclerotigenus* (B types), *A. rambellii* and certain members of *Aspergillus* subgenus *Nidulantes* namely: *Emericella venezualensis* (Frisvad *et al*., 2005) and *E. astellata* (Frisvad *et al*., 2004), have now been included in the growing list of aflatoxigenic species. *A. arachidicola* sp. Nov. and *A. minisclerotigenes* sp. Nov that produce both forms of the toxin, are the latest emerging aflatoxigenic species (Pildain *et al*. 2008). The unexpected new comer is A. *niger,* an ochratoxin (OT) producer which was discovered over four decades ago but was never associated with AF synthesis. However, in a search for aflatoxigenic fungi in Romanian medicinal herbs, Mircea *et al.* (2008) showed the capacity of some strains of *A. niger* to produce AFB_1. All these known AF producing fungi particularly *A. flavus* are common and widespread in nature, and have been shown as fungal contaminants of African foods and feeds according to Atehnkeng *et al*. (2008), Essono *et al*. (2009), Njobeh *et al*. (2009) and Makun *et al*. (2011).

Despite the fact that a strain of mould has the genetic potential to produce a particular mycotoxin, the level of production would in part be influenced by the nutrients available.

Typically, moulds require a source of energy in the form of carbohydrates or vegetable oils in addition to a source of nitrogen either organic or inorganic, trace elements and available moisture for growth and toxin production. Cereals particularly oilseeds adequately provide all these nutrients and so are considered ideal substrates for growth of fungi and consequently, toxin synthesis (FAO, 1983). However, even amongst the cereals, mycotoxins contamination varies with size and integrity of seed the coat with the small, compact grains (wheat, rice, oat, sorghum) and those encapsulated in hard seed coats (beans and soybeans) being less susceptible to fungal infection and mycotoxin formation than larger grains such as maize (Stössel, 1986). In Africa, grains contribute about 46% of the total energy intake (FAOSTAT, 2010), this figure may even be higher in rural SSA, where cereals and tubers and roots are virtually the sources of nutrition. This over dependence on grains, an ideal substrate for AF synthesis in rural Africa is a major reason for the high AF load in the continent.

Probably the two most important environmental components favouring mould growth and AF production are hot and humid conditions. Although the optimum temperature and moisture content for growth and toxin production for the various aflatoxigenic fungi varies, many of them achieve best growth and toxin synthesis between 24^{O}C and 28^{O}C (Schindler *et al*., 1977) and seed moisture content of at least 17.5% (Trenk and Hartman, 1970; Ominski *et al*., 1994). These conditions approximate the ambient climatic conditions in most parts of Africa and hence also account for the high prevalence of the toxins in the continent. As to how climate change will alter the AF situation in the continent, is still a matter of public debate. However, in an attempt to predict how extreme climatic conditions associated with climate change can affect AF contamination, Paterson and Lima (2010) adduced that while anticipated warmer (33^{O}C) and more humid conditions might increase AF prevalence in Europe, perhaps the reverse might be the case in tropical Africa as most aflatoxigenic fungi will not survive the expected 40^{O}C. Even though this prediction is plausible, it is pertinent to state herein that if there will be a rise in temperature and a rainfall reduction in all parts of Africa except for East Africa where an increase in mean annual rainfall is expected (IPCC, 2007), it is only reasonable to assume that many regions of the continent will experience droughts while East Africa would be hotter and more humid and given that these conditions favour AF production (Mutegi *et al*. 2009)), climate change might exacerbate the AF crisis in Africa. In agreement with this deduction, Paterson and Lima (2010) also speculated that if temperature does not increase as envisaged, then droughts might be more frequent. Drought conditions actually constitute stress factors to plants rendering them vulnerable to *Aspergillus* infection (Robertson, 2005; Holbrook *et al*., 2004) with ensuing increase in AF pollution. An indelible sign that droughts prop up AF contamination is the fact that these conditions preceded the fatal outbreak of acute human aflatoxicosis that occurred in Kenya in 2004 (Afla-guard, 2005; CDC, 2004).

Soil is another natural factor that exerts a powerful influence on the incidence of fungi. Crops grown in different soil types may have significantly different levels of AF contamination. For example, peanuts grown in light sandy soils support rapid growth of the fungi, particularly under dry conditions, while heavier soils result in less contamination of peanuts due to their high water holding capacity which helps the plant to prevent drought stress (Codex Alimentarius Commission, 2004). Produce harvested from land on which groundnut has been planted the previous year were more highly infested by *A. flavus* and

contained more AF than crops grown on land previously planted with rye, oats, melon or potatoes (Martin and Gilman, 1976). Likewise, previously fungicide treated soil has been shown to reduce incidence of *A. flavus* in groundnuts to very low levels. Accordingly, it might also be useful to add recommendations on appropriate crop rotation programmes and soil treatments in order to reduce the hazards from mycotoxins.

The presence of other microorganisms either bacteria or other fungi may alter AF elaboration on food materials. When *A. parasiticus* was grown in the presence of some bacteria; *Streptococcus lactis* and *Lactobacillius casei,* AF production was reduced (Ominski *et al*., 1994). Meanwhile, fungal metabolites such as rubratoxins from *Penicillium purpurogenum;* cerulenin from *Cephalosporium caerulens;* and *Acrocylindrium oryzae* enhance AF production even though they repress growth of AF producing fungi (Smith and Moss, 1985). This type of positive interaction between fungi in the same food matrix with regards to AF synthesis couple with multi-occurrence of mycotoxins from the different fungi which could at the least have additive if not synergistic health impact on the host (Speijer and Speijer, 2004) worsen the AF plight in Africa because such simultaneous co-occurrence of fungi and mycotoxins in African agricultural commodities is very common phenomenon as indicated by many workers including Makun *et al*. (2007), Makun *et al*. (2009) and Njobeh *et al*. (2009).

Much more than in other parts of the world, insects, termites, rodents and birds constitute a major problem to food safety and availability in Africa. The accessibility of these pests and predators to crops is made possible by the available deplorable storage facilities (bags, 'rhumbu') and traditional postharvest preservation techniques (drying grains on rocks and bare floor) applied in the region. Besides eating up parts of the human rations, they boost the susceptibility of crops to microbial infestation and infection with a correlating swell in AF occurrence (Hell *et al*. 2000). In the course of feeding on crops in the field or during storage, these animals physically wound kernels and tubers. Mechanical damage resulting from such actions of pests disrupts the seed coat and facilitates the penetration of fungal inoculum into the interior of the grains. In addition to the physical damage, these animals transmit spores from other plants and environmental surfaces to inoculate the already defective kernels and tubers and as such, help to distribute moulds widely throughout a bulk mass of grain or feed. The metabolic activities of pests especially insect larvae also produce metabolic water and heat (Sinha, 1984) that are beneficial for mould growth. The activities of these insects and birds occasioned by poor storage amenities (Hell et al., 2000; Udoh *et al*., 2000) provoke fungal growth and toxin production (Agboola, 1992; Wagacha and Muthomi, 2008). According to the review of Bankole and Adebanjo (2003) on the mycotoxin situation in West Africa, the commonest insects that spread *A. flavus* in preharvest maize in the region are lepidopteran ear borer *Mussidia nigrivenella*, *Sitophilus zeamais* and *Carpophilus dimidiatus.*

Agricultural practices have profound impact on AF contamination of foods. Population drift from agrarian rural areas to urban settlements in search for employment has led to reduced workers on the farm and so 'off season' harvesting during early rains is increasingly common. Also because farmers have improved varieties of grains particularly maize, crops are now grown and harvested twice during the planting season and so harvesting of the first set of these crops is done in the wet months of July and August. Such 'off season' harvesting promotes growth of aflatoxigenic fungi and toxin synthesis (Kaaya and Warren, 2005). Harvesting methods that enhance seed breakage would also increase the degree of

mycotoxin formation. *Aspergillius flavus* was observed to be more abundant in kernels from pods gathered by combined harvesting than from hand harvested pods and the respective AFB_1 content were 1780 and 140ppb (Martin and Gilman, 1976). This is in agreement with the suggestion that certain modern agricultural management practices may create unique ecological niches which select toxigenic fungi (Bilgrami et al., 1981). Meanwhile, as Africa is experiencing a boost in mechanized farming, there are no commiserate control measures put in place to reduce the negative impact of this agricultural revolution on mycotoxin pollution.

Fungi are generally aerobic organisms; therefore storage atmosphere deficient in oxygen would lead to reduced metabolism and consequently mycotoxin production. Javis (1971) and Agboola (1992) reported that a reduction in oxygen content of storage environment from 5% to 1% and increase in carbon dioxide content to above 20% dramatically reduced the growth of *A. flavus* and AF production. Commodities are better stored anaerobically with the addition of organic acids such as propionic acid as preservatives in storage systems which do not absorb moisture or enhance moisture migration (Ominski *et al*. 1994). Unfortunately traditional storage facilities in Africa are devoid of such standard storage environments (Hell *et al*., 2000; Udoh *et al*., 2000).

Unwholesome trade practices have become problematic in guaranteeing food safety in the developing world. Since fungal proliferation and mycotoxin formation increase with duration of storage (Hell *et al*. 2000) when favourable conditions prevail, the hoarding of commodities under poor storage circumstances from October to July/August for sale during period of scarcity in order to maximize profits which has become a common practice in rural Africa exaggerates AF contamination problem. Similarly, the tradition of mixing grains of different grades in order to improve the quality of contaminated grains especially when one contains a large number of fungi spores will provide inoculum for the good grade and probably contaminate the toxin-free grain with AF. Other compelling factors that worsen the AF burden in Africa are public ignorance of the existence of the toxins; complete absence or lack of enforcement of regulatory limits; and introduction of contaminated food into the food chain which has become inevitable due to shortage of food supply caused by drought, wars and other socioeconomic and political insecurity (Wagacha and Muthomi, 2008).

3. Occurrence of AF in foods and feeds

AF producing fungi particularly *A. flavus* are common and widespread in nature and most often found when certain grains are grown under stressful conditions such as drought. The moulds occurs in soil, decaying vegetation, hay and grains undergoing microbiological deterioration and invades all types of organic substrates when the conditions are favourable for growth particularly in hot and humid situations (Ominski *et al*.,1994). Under such a suitable environment, aflatoxigenic fungi contaminate foods and feeds directly or indirectly. In direct contamination, the product is infected with aflatoxigenic fungi with subsequent toxin production. Indirect contamination occurs when food or feed was previously contaminated with AF producing fungi and although the fungi has been removed or killed during processing, AF still remains in the final product. Such contamination of cereals and oilseeds is the main point of entry of many mycotoxins in the human and animal dietary systems particularly in Africa (Smith and Moss, 1985). Fungal infection of agricultural

produce is inevitable but while in developed countries, they get removed from the food chain, in most parts of Africa, moulded foods are part of the daily diets. All foods and feedstuffs are vulnerable to fungal contamination but the nature and degree of aflatoxigenic fungal contamination will depend on the presence or absence of AF in the product. Whereas the identification of toxigenic fungal contaminants is an undoubted pointer to a potential risk, positive conclusions can only be made with certainty by quantifying the suspected toxins which is why this critical review of levels of AF in food commodities is an empirical assessment of Africa's health condition with regards to mycotoxicoses. The crops that frequently support growth of AF producing fungi and subsequent toxin production are thus principal sources of exposure to AF that include but not limited to cereals (maize, sorghum, pearl millet, rice, wheat), oilseeds (peanut, soybean, sunflower, cotton), spices (chile peppers, black pepper, coriander, turmeric, ginger), and tree nuts (almond, pistachio, walnut, coconut, brazil nuts) (FAO, 1983). The other possible sources of entry of mycotoxin into animal and human systems include fruits, vegetables, animal tissues and animal products and fermented products (Jarvis, 1976).

There are at least 14 natural occurring AF known to exist, however, only six have public health and agricultural significance and they include AFB_1, B_2, G_1 and G_2. The other two are AFM_1 and M_2 which are the hydroxylated forms of AFB_1 and B_2, respectively that are secreted in animal (including humans) tissues and fluids. There is no region of the world that is free from AF problem but the strict food and feed quality control programmes put in place in the developed countries greatly reduce the AF burden in those countries. This however, is not the case in Africa and to some extent, in Asia. Besides the lack of regulation of mycotoxins in the continent, the prevailing hot, humid climate and subsistence on foods suitable for AF contamination and other factors discussed in Section 2.0, make Africa the most AF vulnerable region in the world. Hence, a current overview of its natural occurrence in different raw and processed food commodities that are major sources of the toxins in the continent which will not only reflect the disparity in human exposure to AF worldwide, but also contributes to a better understanding of Africa's health afflictions is the primary objective of this section.

3.1 Raw agricultural products

3.1.1 Nuts and oilseeds

These products are the most investigated of all foods and feeds with regards to AF contamination. The reasons being that they are the most susceptible to AF in addition to their high protein content particularly in the case of groundnut which has made them priceless components of many animal and human diets. Data on AF incidence and concentrations in groundnut are consistently incriminating with several reports regularly accounting for extremely high levels of the toxins (Table 1) in all regions of the continent. Table 1 shows over 60% prevalence of AF across Africa at very critical levels. The incidence and levels reported vary with season. Highly contaminated samples were obtained during the rainy season (90% frequency at 12-937 µg/kg) than in the dry season (53.2% prevalence at 15-390 µg/kg) (Kamika and Takoy, 2011). The highest contaminations noted in Malawi (ICRISAT, 2010) were in districts that were prone to late session rains which enhance favourable conditions for post harvest contamination. Similarly, Mutegi *et al*. (2009)

demonstrated that wet and humid weathers are associated with severe AF contamination than drier locations. Serious contamination of Nigerian groundnut is a recurring problem since the 1960s when up to 8000 µg/kg were reported in 1976 (Opadokun, 1992) from the Northern region of the country. Even after heat treatment that should reduce AF levels in contaminated samples (Ogunsanwo *et al*. 2004), dry-roasted groundnuts from South Western part of the country contain as high as 165 µg/kg (Bankole *et al*.2005). Bankole and Adebanjo (2003) reviewed the AF contents in groundnut cake (range: 20 – 455 µg/kg) and snack (30 µg/kg) from Nigeria and concluded that the levels of contamination in these products were toxicologically unsafe. Homemade and unrefined groundnut oil contains AF at levels ranging between 20 and 2000 µg/kg in Nigeria (Obidoa and Gugnani, 1992). It is pertinent to state herein that in 1988, the deaths of some primary school children in Nigeria were associated with incriminating levels of AF in groundnut cake '*kulikuli*' (Fapohunda, 2011). The AF problem of peanuts persists even in South Africa where food safety standards are adhered to. This came to limelight in 2001 when the South African Primary School Nutrition Programme received substantial media coverage on unacceptably high AF content in sandwich containing peanut butter given to school children. The government then established a national monitoring programme to survey AF in groundnuts and peanut products. Between July, 2003 and March 2004, 1140 peanut and groundnut products samples were analyzed and accordingly, about 30% of analyzed samples did not comply with the legal limit of 10 µg/kg (5 µg/kg for AFB_1) and concentrations as high as 560µg/kg were obtained (MRC, 2006).

AF contamination of cottonseed has been a major concern worldwide as extremely high contents ranging between 200,000 to 300,000µg/kg were reported much earlier in samples exported to the European markets (Smith and Moss, 1985). A survey of cottonseed for AF during such periods (between 1976 and 1979) when Nigeria was a major exporter of the crop, revealed over 60% (17/28) incidence rate with mean value of 105µg/kg recorded (Opadokun, 1992). Gbodi (1986) subsequently showed moderate contamination of the cash crop grown in the semi temperate climate of Plateau State, Nigeria (Table 1). Mycotoxin research on cottonseed in Nigeria has since ceased to exist as the country no longer depends on agriculture as its main source of revenue. When AF contaminated cottonseed is processed into oil, the toxins are concentrated in the residual cottonseed meal and cake which are often used as feed components of livestock feeds. The cottonseed oil also contains residual amount of the toxins at certain levels but this usually depends on the extent of contamination of the seeds from which it was obtained.

Aside from cottonseed, its products are also very important sources of human and animal exposure to AF. Abalaka (1984) found 26.1µg/kg and 12.6 µg/kg of AF in groundnut and cottonseed oil samples from the Guinea Savannah region of Nigeria. All cottonseed meal samples investigated for AF in South Africa (Table 1) contained the toxins in which 42 of the 60 samples analyzed exceeded the maximum level for feeds. The AF menace in cottonseed is not limited to the sub-Saharan region of the continent as aflatoxigenic fungi and the two B forms of the toxin were found naturally contaminating cottonseed, cottonseed meal and cake from Egypt (Mazen *et al.* 1990). Melon seed another important oilseed in West Africa has been shown to be prone to fungal and AF contamination (Table 1) at largely unsafe levels (Bankole *et al*. 2006). Also Opadokun (1992) reported high incidence (73%) of AF in Nigerian melon seed at mean content of 19µg/kg.

Commodity	Country	Type of Aflatoxin	Incidence rate	Range (μg/kg)	Mean level ± SD (μg/kg)	Reference
Barley	Tunisia	AFB1	11/25	3.5-11.5	18.4 ±27.3	Ghali *et al.* (2008)
Cheese	Libya	AFM_1	15/20	0.11-0.52		Elgerbi *et al.* (2004)
Cow milk	Nigeria	AFM_1	3/22	-	≤ 2.04	Atanda *et al.* (2007)
	Sudan	AFM_1	42/44	0.22 – 6.90	2.07	Elzupir and Elhussein, (2010)
	South Africa	AFM_1	42/42	0.04 – 1.32	0.12	Dutton *et al.* (2010)
	Kenya	AFM_1	474/613	0.005-0.78	0.064	Kang'ethe and Lang'a (2009)
	Cameroon	AFM_1	10/63	0.006-0.527	-	Tchana *et al.* (2010)
Cowpea	Benin	AFB1	3/92		3.58	Houssou *et al.* (2009)
	Cameroon	AF	5/15	0.2-6.2	2.4	Njobeh *et al.* (2010)
Dried Beef	Nigeria	AFB	10/10	0.003		Oyero and Oyefolu (2010)
		AFG	10/10	0.004		
Dried Chilli	West Africa	AFB1	1/30	3.2	3.2	Hell *et al.* (2009)
Dried figs	Morocco	AFB1	1/20	0.28	0.28	Juan *et al.* (2008)
Dried okra	West Africa	AFB1	3/30		5.4	Hell *et al.* (2009)
		AFB2	1/30		0.6	
Dried raisins	Morocco	AFB1	4/20	3.2-13.9	10.7±2.3	Juan *et al.* (2008)
Egg	Cameroon	AF	28/62	0.002-7.68	0.82± 1.71	Tchana *et al.* (2010)
Fresh Beef	Nigeria	AFB	10/10	0.02		Oyero and Oyefolu (2010)
		AFG	10/10	0.03		
Groundnut	DR Congo	AFB1	43/60	1.5-937	229.07	Kamika and Takoy, (2011)
Groundnut	Malawi	AFB1	/1189	0-3871		ICRISAT (2010)
Groundnut	Kenya	AF	170/769	0-7525	< 4	Mutegi *et al.*(2009)
Local beer	Malawi	AF	5/5	8.8-34.5	22.3±4.93	Matumba *et al.* (2011)
Maize	Malawi	AFB		0.0-1335		ICRISAT (2010)
Maize	Nigeria	AFB1	55/55	0-1874	257.82	Atehnkeng *et al.* (2008)
		AFB2		0-608		
		AFG1		0-937		
		AFG2		0-286		
Maize	Ghana	AF	30/30	6.20-29.50	13.596	Akrobortu (2008)
Maize	Uganda	AF	22/49	1.00-1000		Kaaya and Warren, (2005)
Maize flour	Morocco	AFB1	16/20	0.23-11.2	1.57± 0.78	Zinedine et al. (2007) [b]
Melon seed	Nigeria	AFB1	37/137	2.3-47.7	14.2	Bankole *et al.*(2006)
Milk[c]	South Africa	AFM1	98/114	Max: 2.07	0.15	Lishia et al. (data unpublished)
Milk[d]	South Africa	AFM1	85/85	Max: 2.48	0.14	
Millet	Nigeria	AFB1	12/49	1370.28-3495	2587.47±78.23	Makun *et al.*(2007)
Mouldy Sorghum	Nigeria	AFB1	93/168	0-1164	199.51-259.90	Makun *et al.* (2009)
Pasteurized milk	Morocco	AFM_1	48/54	0.001-0.117	0.018	Zinedine *et al.* (2007)[a]
Powdered milk	Nigeria	AFM_1	19/100	0.02-0.41	0.136	Makun *et al.* (2010)
Powdered soymilk	Nigeria	AFB1	30/30	4.58-19.76	11.53	Adebayo-Tayo *et al.* (2009)
		AFB2		2.57-11.54	6.04	

Commodity	Country	Type of Aflatoxin	Incidence rate	Range (μg/kg)	Mean level ± SD (μg/kg)	Reference
Raw cow Milk	Egypt	AFM_1	19/50	0.023 – 0.073	0.049 ± 0.017	Amer and Ibrahim (2010).
Rice	Nigeria	AF	21/21	27.7–371.9	82.5±16.9	Makun *et al.* (2011)
Roasted Groundnut	Nigeria	AFB1	68/106	5-165	25.5	Bankole *et al*. (2005)
		AFB2	28/106	6-26	10.7	
		AFG1	12/106	5-20	7.2	
		AFG2	3/106	7-10	8.0	
Smoke-dried fish	Nigeria	AFB1	11/11	1.505-8.11	3.46	Adebayo-Tayo *et al*. (2008)
		AFG1	11/11	1.810-4.51	2.94	
Sorghum	Tunisia	AFB1	58/93	0.34-52.9	9.9±11.5	Ghali *et al.* (2009)
		AFB2	45/93	0.11-3.7		
		AFG1	3/93	0.45-0.7		
Sorghum	Malawi	AFB1	2/15	1.7-3.0	2.35±0.65	Matumba *et al*.(2011)
Sorghum	Ethiopia	AFB1			0-26	Ayalew *et al*. (2006)
Soybean	Cameroon	AF	2/5	0.2-3.9	2.1	Njobeh *et al*. (2010)
Wheat	Kenya	AFB1	23/50	0-7	1.93	Muthomi *et al*. (2008)
	Tunisia	AFs	15/51	4.0-12.9	6.7±2.4	Ghali *et al*. (2008)
	Nigeria	AFB1		17.10-20.53	19.00±1.67	Odoemelam and Osu, (2009)
Wheat & products	Algeria	AFB1	30/53	0.13-37.42	>5	Riba *et al*. (2010)
	South Africa	AFB1	13/238	0.5-2.0	>2	Mashinini and Dutton, (2006)

[c]Milk obtained from rural communities in South Africa. [d]Milk obtained from commercial farms

Table 1. Some reports of aflatoxin levels in some foods commodities from Africa

Low AF contamination of cowpea, soybean and their products is reported in many parts of the world. The data on these nuts from Cameroon and Benin (Table 1) showed only trace amount of AF in few samples. Similarly, only 3 positive samples were demonstrated in 268 cowpea samples analyzed between 1975 and 1983 from Nigeria (Opadokun, 1992). In the same review, it was shown that all two samples of soybean oil assayed did not contain AF while 41 of 55 palm kernels were contaminated with low amount of the toxin. High seed coat integrity, ensuring limited access and low moisture content are responsible for the low susceptibilities of these nuts to aflatoxigenic fungi (Stossel, 1986) with consequent rare occurrence of the toxins in them. In spite of the low presence of AF in soybean, intolerable levels of these carcinogens were found in branded and unbranded powdered soymilk, a processed product of soybean in Nigeria (Adebayo-Tayo *et al*. 2009). Soymilk has become a popular infant recipe in Africa because it is a cheap source of water soluble protein, carbohydrates and oil and so human exposure to AF can arise in countries using it for human consumption. Although producing fungi particularly *Aspergillus flavus* are considerably natural contaminants of cocoa bean (Sanchev-Hervas *et al*. 2008), AF are rarely detected in cocoa bean and at very low concentration. In a survey for mycotoxins in cocoa and cocoa products sponsored by the Foundation of German Cocoa and Chocolate Industry, Hamburg, Beucker *et al*. (2005) screened over 200 samples and found AFB_1 at concentrations below 2μg/kg. Literature search for data on high AF in cocoa from Africa was unsuccessful and that could be a clear indication that these toxins are not problematic in this cash crop. However, ochratoxin A is the mycotoxin of interest to the cocoa (Arogeun and Jayeola, 2005) and soybean (Smith and Moss, 1985) industries.

3.1.2 Cereals

Cereals are rich sources of minerals, vitamins, carbohydrates, oils and proteins but when refined majority of the nutrients are lost leaving mostly carbohydrates and are therefore grown mainly for energy. Grains as they are sometimes called provide more food energy worldwide than any other crop and thus are staples. They are staple for two third of the earth's population, providing 85% of the world's food energy and protein intake (FAOSTAT, 2006). Cereal consumption is moderate in developed countries however in Africa and Asia, it is a daily sustenance. In Africa, cereals contribute 46% of the total energy intake; however, this figure could be as high as 78% in some African countries (FAOSTAT, 2010). According to figures made available by the afore mentioned statistics division of FAO, the six most cultivated and hence consumed grains worldwide in order of decreasing production are maize, rice, wheat, barley, Sorghum and millet, and of these major grains maize, wheat and rice together account for 87% of all cereal production worldwide and 43% of all food calories. Because of their rich nutrient composition, cereals support fungal growth and mycotoxin production excellently on the farm, during storage and after processing into foods and feeds with the small grains (sorghum, rice, wheat, millet and rice) being less susceptible to fungal and toxin contaminations than the larger grains like maize. And since these ideal substrates for mycotoxin contamination are highly consumed globally, they constitute the most remarkable sources of mycotoxins (especially the most prevalent of mycotoxins; AF) to animals and human beings. While there are other cereals (oats, rye, triticale, fonio, buckwheat and quinoa) of global significance, this review will focus on the six major ones which are pertinent to Africa.

Maize is one of the important staple foods in Africa and is now widely grown for animal feeds. It is the third most cultivated food commodity in the continent after cassava and sugar cane but it ranks the first in term of food energy supply in Africa. AF are regularly detected in maize throughout the world and the recent serious contamination which was associated with drought led to fatal human aflatoxicosis in Kenya (Afla-guard, 2005; CDC, 2004) as previously stated in Section 2.0. The natural occurrence of AF in maize from some African countries has been reviewed (Table1). A regional effect on the incidence of the toxins has been demonstrated with lower contents observed in the drier North African countries (Morocco; 0.11-11.2 µg/kg) than amounts reported for such SSA countries as Uganda, Nigeria, Ghana and Malawi having up to 1874 4µg/kg. Monitoring for AF in maize samples from different regions of Africa showed disturbingly high levels of contamination above 1000 µg/kg with many of the samples containing AF contents exceeding the CODEX regulatory limit of 20 µg/kg (five times more lenient than the EU guideline of 4µg/kg). Over 77% of the tested samples from Malawi were contaminated with AF with 90% of the positive samples not qualified for the EU export market (ICRISAT, 2010). Atehnkeng *et al*. (2008) previously analyzed 55 samples of maize from 11 districts across three agro-ecological zones of Nigeria for AF and mean values ranging between 30.9 -507.9 µg/kg were recovered from the 10 districts, which were far beyond all known acceptable levels. Maxwell *et al*. (2000) also found AF at alarming concentrations of between 3,000-138,000 µg/kg in Nigerian pre-harvest maize samples. In a survey for AF in Ghana which spanned for a decade, the toxin had over 50% prevalence with more than half of the investigated samples having levels exceeding the EU maximum limit (Akrobortu, 2008). Similarly, stored maize from Uganda contain unsafe AF levels (Kaaya and Warren, 2005; Kaaya and Kyamuhangire, 2006). Very high AF level of 46,400µg/kg was found in samples from Kenyan local markets (CDC, 2004).

There is a general paucity of reports on mycotoxin in African indigenous rice. Opadokun (1992) reported AFB in only 13 of the 279 rice samples analyzed in Nigeria with only one having a level above 5μg/kg. One reason why mycotoxicologists are not attentive to local African rice relative to other crops such as maize and peanuts for example is due to the fact that it is entirely consumed within the continent and not available in the European markets. Makun *et al*. (2011) demonstrated a 100% prevalence of AF in Nigerian rice at unsafe levels (range: 28 - 372 μg/kg) (Table 1), and also showed critical contamination by ochratoxin A (OTA) and presence of deoxynivalenol (DON), fumonisins (FB) and zearalenone (ZEA) at trace levels. Although, AF were not found in Ugandan grown rice, however, they have been reported in rice from Cote D'Ivoire (1.5 – 10 μg/kg) and Kenya (294-1050 μg/kg) (Kaaya and Warren, 2005). Data from these few studies underscore the need for more mycotoxin surveys of indigenous rice in Africa.

Wheat does not thrive well in tropical climates so African wheat production is concentrated in the narrow strip along the Mediterranean coast from Morocco to Tunisia, in the Nile valley (Egypt), and in parts of South Africa, Kenya, Ethiopia, Zimbabwe and Sudan. Very little is grown in West Africa however, there is an increase in wheat production in Nigeria and the Democratic Republic of Congo in recent years. The grain is produced mainly for bread making but its bran is increasingly used as component of animal feeds. In spite of the limited production (18.6 million tonnes per year) of the cereal in Africa, 50.4 million tonnes is consumed yearly in the continent (FAOSTAT, 2010). Data on AF in local African wheat (Table 1) reveal moderate contamination across the continent with the highest level of contamination occurring in Nigeria. Over 57% incidence rate of AFB_1 in 53 Algerian pre-harvest and stored wheat samples was established by Riba *et al*. (2010), with only 5 analyzed samples having contents above the EU legal limit. The Tunisian grown wheat had safe levels of AFB_1 (Ghali *et al*. 2008). Of the 51 samples screened from the country, 37% were positive for AFB_1 at levels below 3.4μg/kg. Similarly, with regards to AFB_1, all South African wheat samples analyzed by Mashinini and Dutton (2006) qualified for the EU market, though it occurred simultaneously with DON, ZEA, OT and FB in some of the samples. Although AFB_1 contents in the Kenyan wheat were low (<7 μg/kg), most of the samples were also concurrently contaminated with low but significant levels of DON, T-2 toxin, ZEA and AFB_1 (Muthomi *et al*. 2008). Such co-occurrence of mycotoxins could result in additive or synergistic effects in the host animal (Miller, 1995) as will be discussed in-depth subsequently. Considering the EU and other international and national legal limits, the Nigerian wheat with AFB_1 levels above 17 μg/kg (Odoemelam and Osu, 2009) is of low quality and thus, unfit for human consumption. Makun *et al*. (2010) also found extremely high AFB_1 contaminations of wheat marketed in Minna, Nigeria at unacceptable levels (range: 40-275 μg/kg) in 27 of the 50 tested samples. The severe contamination levels of this crop in Nigeria making it a principal source of mycotoxins raise public health concerns and underscore the need for the regulation of mycotoxins in SSA.

Because it was replaced by maize as a staple food commodity in many rural settlements in Africa (Bandyopadhyay *et al*. 2007), sorghum (also known as guinea corn) is another cereal that has been neglected for some time. However, its rising industrial profile as a suitable raw material for beer brewing has seen to its re-emergence at the world market such that as of 2007, sorghum production in Africa increased significantly even to the detriment of rice and wheat production (FAOSTAT, 2010). The renewed focus on sorghum is also because it is one of the most drought tolerable crops and such high water-use efficient characteristics

makes it the crop of choice to boast food security in drought stricken regions of Africa and for the future against the anticipated water scarcity in the world. Sorghum is a staple grain for over 750 million people in Africa, Asia and Latin America (CODEX, 2011) that is traditionally grown mainly in the semi-arid tropics for human consumption and production of local alcoholic drinks is now a component of animal feeds. Regardless of its inherent resistance to mould infestation due to its high composition of fungicidal principles; phenols and tannin (US Grain Council, 2008), fungal contamination constitutes a major biotic constraint to sorghum improvement and production worldwide. It is estimated that annual economic losses in Asia and Africa due to mould are in excess of US $130 million (Chandrashekar *et al*. 2000). AF is the most investigated mycotoxin in this crop being reported in nine countries at levels of up to 3,282 µg/kg in Brazil (CODEX, 2011). A few reports from some major sorghum producing countries (Table 1) generally reveal a moderate prevalence and low concentrations of AF in the northern part of the continent though many of the contaminated samples from Tunisia (62.50%) had higher contents than the EU limit (Ghali *et al*. 2009). Meanwhile all the samples from Malawi met the EU standard (Matumba *et al*. 2011). The samples analyzed by Makun *et al*. (2009) were mouldy which might explain the observed increased incidence and levels of contamination. The relevance of such biased data cannot be overemphasized as mouldy grains do normally enter the human and animal food chain in Africa. In another unbiased study using representative samples, Odoemelam and Osu (2009) supported the findings of Makun *et al*. (2009) by reporting unacceptable levels of AFB_1 (between 27 to 36 µg/kg with a mean value of 31±3.4) in Nigerian grown sorghum. Twenty six of 69 sorghum samples from Uganda contained the toxins of which 10 of the positive ones had levels over 100µg/kg (Kaaya and Warren, 2005). Besides the entry of AF into human foods via contaminated sorghum, the carryover of substantial amount of these toxins from contaminated sorghum into traditional beer and beverages (Okoye, 1987; Matumba *et al*. 2011) poses additional risk of AF exposure. This should attract much attention to toxic contaminants of the grain but the reality is that there is limited information on mycotoxins in sorghum from Africa which is not commensurate to the escalating economic value of the cereal. Thus, the decision taken by the Codex Committee on Contaminants in Foods at her 5th session held in The Hague, The Netherlands in March, 2011, to prepare a discussion paper on 'mycotoxin in sorghum grain' is a very laudable initiative.

Millet *(Pennisetum* spp.) is the other traditional cereal replaced by maize in Africa in the last three decades. It is resistant to drought and so has been extensively cultivated in arid and semi arid regions. Millet ranks the sixth most important grain in the world, sustaining a third of the world's population and is the fifth most cultivated crop in Africa after maize, sorghum and wheat (Obilana *et al*. 2002). The occurrence of AF in this grain has largely been reported in West Africa particularly in Nigeria, its principal producer in the continent. In a review on chemical safety of traditional grains, Brimer (2011) reported that the average AF content of freshly harvested millet from West Africa was 4.6µg/kg. Odoemelam and Osu (2009) found higher values (range: 34 – 40 µg/kg; mean content: 37.5±2.5 µg/kg) in samples from the forest region of Nigeria. Makun *et al.* (2007) obtained the highest value of 3,495 µg/kg (Table 1) in wet season samples of millet that have been stored for over a year in Niger State, Nigeria. A 1971 report on AF contamination of Ugandan millet as reviewed by Kaaya and Warren (2005) revealed a low incidence (9/55) and AF level of 9 µg/kg. The limited reports on mycotoxins in millet in Africa are understandable as it is one of the 'lost

crops of Africa' and that it is not an export crop. However, the anticipated 4 and 8 times reduction in risks of AF related problems if sorghum and millet, respectively, replace maize as primary staples (Bandyopadhyay *et al*. 2007), should bring these two African traditional crops to the front burner of mycotoxin research worldwide.

There is generally low natural incidence of AF in barley and derived products around the world. The levels reported in Tunisian grain (Table 1) is not an exception to this observation. Very low levels (<3.9 µg/kg) were also observed in barley from South Africa (Maenetje and Dutton, 2007). Because barley is grown in Africa for foods and lager beer production, good agricultural and manufacturing practices along with strict quality control measures are usually put in place in order to gain acceptability in competitive markets and this therefore accounts for the low prevalence of toxic substances in the grain as evident in these few reports. This sort of monitoring should be extended to all other foods and feedstuffs.

3.2 Animal feeds

A majority of animals reared in Africa are free ranged. During the dry season, pastoralists and their flocks travel hundreds of kilometres in search of greener pastures. Invariably, most domesticated African animals are raised on low grade cereals, informal pasture and domestic wastes. Commercially produced animals on the other hand, are essentially fed compounded feeds that are composed primarily of cereals, oilseeds and their by-products including those of animal origin. Even though the main ingredients of the diets of both the roaming and farm animals have been shown in subsection 3.1.1 and 3.1.2 to be heavily loaded with AF in the continent, a review on the occurrence of AF in animals feeds is still appropriate as processing might alter AF levels in the final product. More so, commercial livestock farming is now a major industry in Africa and thus feeds have become a major source of exposure of humans to food borne toxins via consumption of food products obtained from animals fed AF contaminated feeds. In view of the anticipated disparity in toxin content between the feed ingredients used and the compound feed produced, Mngadi *et al*. (2008) analyzed South African animal feeds and the raw ingredients used in manufacturing them for AF, FB, ZEA and OTA. Accordingly, data revealed 17 of the 23 samples tested positive for AF (Table 2) and over 7 of these samples having levels equal or exceeded the legislated limit of 20µg/kg for animal feeds. The raw ingredients namely cottonseed cake, sunflower oil cakes, molasses meal and bagasse were between 4 to 8 times more contaminated than the animal feeds and this disparity was attributed to the heat and other physical and chemical treatments (Rahana and Basappa, 1990) employed during processing that might have eliminated some of the toxins. As shown in Table 1, there is a high prevalence (84.6%) of carcinogenic AF in animal feeds from Kenya with 455 of the tested 830 samples having levels exceeding 5µg/kg, the WHO/FAO limits for feeds destined for diary animals. Poor storage facilities, use of moulded maize for feed production and the absence of monitoring for AF during processing were the reasons for the high frequency and levels of AF in Kenyan feeds (Kang'ethe and Lang'a, 2009). Data on AF in animal feeds from Nigeria (Table 1) clearly points out that while retailing from open bags provokes AF contamination, manufacturing of feeds using materials less susceptible to fungi including wheat offal and palm kernel limits contamination (Adebayo-Tayo and Ettah, 2010). The 6 positive samples among the 13 feeds screened were mainly from retailed shops

by the aforementioned workers had no AF presence in the wheat and palm kernel base feeds. In accordance with EU regulations, poultry feeds from Morocco were safe for animal consumption (Zinedine *et al*. 2007). In light of the significant prevalence of AF in African feeds and feeding stuffs, it is recommended that the use of rapid and sensitive mycotoxin test kits by farmers, manufacturers and consumers to monitor the quality of products will largely lessen the AF burden in the continent.

Commodity	Country	Type of Aflatoxin	Incidence	Range (µg/kg)	Mean level ± SD (µg/kg)	Reference
Animal feeds	South Africa	AF	17/23	0.8-156	38.9	Mngadi *et al*. (2008)
Animal feeds	Kenya	AFB1	703/830	0.9-595	8.9-46.0	Kang'ethe and Lang'a (2009)
Animal feeds	Sudan	AF	36/56	4.1-579.9	130.6	Elzupir *et al.* (2009)
Animal feeds[b]	South Africa	AF	99/108	3.2-950	112.54	Mwanza *et al.* (data unpublished)
Bush mango seeds	Nigeria	AFB1	20/20	0.2-4.0	1.5	Adebayo-Tayo *et al*. (2006)
		AFG1	20/20	0.3-4.2	1.5	
Cottonseed	Nigeria	AFB1	3/8	0.0-271		Gbodi (1986)
		AFB2	3/8	0.0-36.6		
		AFG1	2/8	0.0-183		
		AFG2	1/8	0.0-9.1		
Cottonseed meal[a]	South Africa	AFB1	60/60	13.4-75.7	24.90	Reiter *et al*. (2011)
		AFB2		1.0-5.4	1.84	
		AFG1		6.6-64.3	17.91	
		AFG2		0.3-3.3	0.90	
Pistachio	Morocco	AFB1	9/20	0.04-1430	158±6.3	Juan *et al*. (2008)
Pistachio	Tunisia	AF	21/40	0.24-122.4	21.8±38.0	Ghali *et al*. (2009)
Poultry feeds	Morocco	AFB1	14/21	0.05-5.38	1.26± 0.65	Zinedine *et al*. (2007)
		AFB2		0.03-0.58	0.18±0.18	
		AFG1		ND	ND	
		AFG2		ND	ND	
Poultry/livestock feeds	Nigeria	AFB1	6/13	0.0-67.9	15.5	Adebayo and Etta (2010)

[a]Cottonseed cake was imported from Benin. [b]Animal feeds from rural community of Limpopo province. ND: Not detected

Table 2. Some reports of aflatoxin levels in some animal feeds and fruits from Africa.

3.3 Vegetables

Reports on fungal and mycotoxin contaminations of vegetables are exceptionally uncommon. One of the most recent and novel works in this area is that of Hell *et al*. (2009) who studied 180 dried vegetable samples across three countries in West Africa including Benin, Mali and Togo. AF concentrations were determined in dried okra, hot chilli, tomatoes, melon seed, onion and baobab leaves. The results as seen in Table 1 is one of the very first few reports on AF in dried okra and hot chilli after that of Obidoa and Gugnani (1992). AFs were not detected in the other dried commodities but however, the toxins have been detected in fresh vegetables elsewhere. For instance, Muhammad *et al*. (2004) found the toxins present in fresh tomatoes marketed in Sokoto, Nigeria. Similarly, Sahar *et al*. (2009) also showed the presence of AF in fresh tomatoes, pumpkin, powder chillies and coriander

(dry) grown in Pakistan. Other fruits and vegetables in which the toxins were found in that study include cucumber, persimmon, peanut and peach. Interestingly, Obidoa and Gugnani (1992) had earlier found AF in dried okra, onions, dry pepper and table foods sold at restaurants. The ready-to-eat dishes mainly "gari" (cassava products) and beans served with vegetable soups had AFB1 at levels ranging between 8 and 61 µg/kg with total AF values ranging from 31.21 to 268µg/kg. The data suggest that AFs are common contaminants of most African table foods. Another vegetable that abhor aflatoxigenic fungi and consequently contain AF is oyster mushroom (Jonathan and Esho, 2010). The severe paucity of information on mycotoxins in vegetables necessitates increased research in the area.

3.4 Fruits

In a review of mycotoxins in fruits and fruit-processed products, Fernandez-Cruz *et al*. (2010) revealed that the commonest mycotoxin contaminants of fruits worldwide are patulin (PAT), OTA, AF and *Alternaria* toxins, and that natural AF contamination has been reported in oranges, apple and apple juices, dried apricots, dates, prunes, musts, dried figs and raisins. According to the review, AFs are most frequently reported in dried figs and raisins worldwide at significant levels of up to 550 and 63µg/kg, respectively. Furthermore, their occurrence was reported in apple juice from Egypt and dried raisins and figs from Morocco. Other reviewed literature of interest in this area is that provided by Trucksess and Scott (2008) and Barkai-Golan and Paster (2008). Reports on mycotoxins in fruits are not many in Africa with one reported in pawpaw and pineapple being contaminated by *Aspergillus flavus* in Maiduguri, Nigeria (Akinmusire, 2011). Baiyewu *et al*. (2007) showed the presence of the toxins in pawpaw from South Western Nigeria. Some data on AF incidence in Africa (Table 2) presented low occurrence of the toxins in African fruits. Juan *et al*. (2008) found AFB_1 at levels exceeding the EU limit of 2µg/kg in 5 and 20% of 20 samples each of pistachio and dried raisin, respectively, with the highest value of 1430 µg/kg detected in pistachio. While all dried fig samples were found fit for the EU market with regards to AFB_1 contents, 15% of them had AF levels above the 4 µg/kg, a maximum recommended limit of EU. The use of traditional processing and preservation methods for fruits in rural Morocco that provide optimal conditions for mould growth and mycotoxin formation was adduced as the cause for the increased prevalence of these AF in fruits. AF analysis in Tunisian pistachio performed by Ghali *et al*. (2009) found unacceptable (according to EU standards) concentrations of AF in 17% of the samples. Bush mango (*Irvingia* spp) is a sweet tasting fruit. The pulverized form of the dried seeds is used as condiment and thickeners for soups and stew in West Africa. Investigation into its AF content in Nigeria by Adebayo-Tayo *et al*. (2006) showed a 35% non compliance with the EU AFB_1 standard for fruits. There is need for AF surveys in commonly eaten fruits in Africa namely, oranges, banana, plantain, guava, dates, etc. in order to properly ascertain the extent to which fruits expose the African population to the toxins.

3.5 Roots and tubers

Roots and tubers are a major source of nutrition in the world after cereals. They are basic diets for about a billion people in the developing countries, providing 10% of world's food

energy and protein intake (Shewry, 2007). They account for 40% of food eaten by half the population of SSA, contributing 20% of total energy intake in the continent (FAOSTAT, 2010). Cassava, potatoes, yam and taro form the bulk of roots and tubers consumed worldwide. Cassava and yam are not vulnerable to AF contamination (Bankole *et al*. 2006) and even the processed products such as cassava and yam chips and their flour have low contamination rates. Analysis of cassava and yam chips from Benin showed no contamination by AF (Gnonlonfin *et al*. 2008) neither was the toxins found in cassava products from Tanzania (Muzanila *et al*. (2000), Nigeria (Jimoh and Kolapo, 2008) and Ivory Coast (Kastner *et al*. 2010). However, data obtained from Cameroon (Table 1) show low levels of the toxins in stored cassava chips at levels greatly dependent on processing practices, storage facilities and duration of storage (Essono *et al*. 2009). Higher prevalence and contents of AF are observed in yam based products than in cassava products. Apart from the 100% frequency (Table 1) shown in Yam chips from Benin (Bassa *et al*. 2009), 23% of the 107 samples analyzed by Mestres *et al*. (2004) from the same country had AF levels over the 15 µg/kg CODEX standard value for total AF. AFB_1 and G_1 were detected in yam chips from Nigeria at levels ranging from 5-27µg/kg (Jimoh and Kolapo, 2008). AFB_1 was also found in 22% of samples from Nigeria (Bankole and Mabekoje, 2003) and a larger survey conducted later in same country found the toxin at a prevalence rate of 54.2% in the same products at toxicologically significant levels (range: 4–186 µg/kg; mean: 23 µg/kg), 32.3% with AFB_2 (range: 2-55 µg/kg), 5.2% were positive for AFG_1 (range: 4-18 µg/kg), while 2 samples contained AFG_2) (Bankole and Adebanjo, 2003). The present unwholesome practice of storing and marketing high moisture cassava and yam products is responsible for the contamination in Africa (Essono *et al*. 2009) and should be discouraged.

3.6 Animal products

3.6.1 Milk and milk products

Animals fed AFB_1 and B_2 contaminated feeds excrete into their milk the less toxic AFM_1 and M_2, respectively. AFM_1 is of particular interest being the hydroxylated metabolite of AFB_1 and is known to have 2-10% of the carcinogenic potency of the parent compound (Zinedine *et al*. 2007). The carryover of this carcinogen in cow at a transfer ratio (consumed AFB_1 to excreted AFM_1) of 200:1 (Smith and Moss, 1985) which could be as high as 40:0.05 (JECFA, 2001) into human and animal milk that are the main sources of nutrition for infants whose vulnerability due to undeveloped immune system is obvious, poses serious health concern. Its stability to heat, cold storage, freezing and drying (Yousef and Marth, 1985) during processing makes dairy products another important source of AFM_1 exposure. Milk and milk products are traditionally staple food commodities for the nomadic population of Africa. They are recognized by the elites as natural balanced diet and so are increasingly consumed by the urban populace in the continent. Therefore, they can no longer be ignored as they are among the main entry routes of AFM_1 into the human dietary system in Africa.

The natural occurrence of AFM_1 in raw cow milk has been reported in quite a number of African countries as reflected in Table 1. A regional variation has been demonstrated in the data with lower concentrations occurring in the drier North Africa (Egypt and Morocco) than in the more humid SSA (Nigeria, Sudan, Kenya, Cameroon and South Africa). Many of the milk samples from the region (7.4, 18.7, 40, 52.6, 61.9 and 83.3 % of samples from Morocco, Kenya, Cameroon, Egypt, South Africa and Sudan, respectively, had AFM_1

contents above the legislated levels (0.05µg/L) of several countries including those in the EU. Similarly, their recorded mean levels according to JECFA (2001) are higher than those reported for European (0.023 µg/L), Latin America (0.022 µg/L), Far Eastern (0.36 µg/L), Middle Eastern (0.005 µg/L) and African diets (0.0018 µg/L). Since AFM_1 content in milk is a good indicator of AFB_1 contamination in feeds, hence at a transfer rate of 200:1 for cows it can be estimated that diary animals in Africa are exposed to between 3.6 – 414 µg/kg of AFB_1 in their rations since the observed range of AFM_1 averages (Table 1) for Africa is 0.018 to 2.07 µg/L. Such high levels of AF in feeds in the continent have been shown in sections 3.1 and 3.2. The presence of AFM_1 in milk from breastfeeding mothers (Atanda *et al*. 2007) and other body fluids of diseased patients (Tchana *et al*. 2010) sometimes at above regulated levels, is confirmatory that humans are exposed to high AF levels in Africa and because milk is primary to infant nutrition it gives cause for considerable concern.

Raw milk is usually processed into dehydrated dairy products such as cream, butter, cheese and milk powder in order to extend its shelf-life. Yoghurt, another product of milk which has become part of human dietary system has a similar processing method as cheese making, only that the process is arrested before the curd forms. The separation of the components of milk during processing leads to distribution of AFM_1 into dairy products with consequent lower levels of the toxins in the individual products than in milk. In spite of this anticipated loss, AFM_1 has been found in African dairy products at unacceptable levels. High prevalence (75%) of AFM_1 with all positive samples having levels exceeding the legislated limit of 0.05 µg/L was seen in Libyan cheese (Elgerbi *et al*. (2004). Amer and Ibrahim (2010) found AFM_1 in 50/150 Egyptian cheese samples at levels between 0.051 to 0.182 µg/L and all the contaminated samples had levels beyond the EU regulated limit. Similarly, the toxin contaminates cheese, ice cream and yoghurt at intolerable amounts in Nigeria (Atanda *et al*. 2007). In order to determine how safe imported powdered milk samples are for human consumption, Makun *et al*. (2010) analyzed AFM_1 in 100 samples sold in the Lagos metropolis. As seen, 6 of the 19 positive samples had levels above the EU limit, however, all the tested samples were considered safe in Nigeria as levels of the toxin were below the country's maximum tolerable limit of 1 µg/kg. Clearly, the main strategy to reduce incidence of milk toxins is by feeding animals with AF free feeds.

3.6.2 Animal tissues

Relative to such mycotoxins as OTA, a much smaller proportion of AF is absorbed into animal tissues. The transfer ratios into tissues for AFB_1 can range between 1,000 and 14,000 (Smith and Moss, 1985). Any animal exposed to such high amount of mycotoxins would have shown some toxicity signs or even death and would likely not enter the human food chain in the developed world. Conversely, diseased animals are still eaten in Africa and as such, human exposure to AF via consumption of animal tissues can be a reality in the continent. There is a disparity between the AF content of beef and those of other edible organs with lower values obtained for the muscle tissue. This was shown by Oyero and Oyefolu (2010) when analysing fresh and sun-dried beef and edible organs (liver, kidney and heart). While, the dried animal tissues had lower levels of AFB (beef: 2.9, liver: 3.1, heart: 27.9, kidney: 75.8 ng/kg) and AFG (beef: 4.4, liver: 3.1, heart: 55, kidney: 141.3 ng/kg) than for fresh samples whereby AFB levels were for beef liver, heart, kidney were 21.7, 33.9, 55.9 and 85.2 ng/kg, respectively, and AFG levels in these organs recorded were

respectively, 27, 41.4, 74.1 and 70.7 ng/kg. Accordingly, the levels of the toxins in the edible organs were consistently higher than those in beef with kidney being the most vulnerable. It is therefore, suggested that withdrawal of animals from contaminated feeds onto mycotoxin free diets for 3-4 weeks could have allowed for sufficient withholding period to clear the muscles and organs from the toxins. Despite the low carryover rates of the toxins into animal tissues, AF contaminate fresh and processed meat (especially liver and kidney) at toxicologically significant levels of up to 325µg/kg in Egypt (Aziz and Youssef, 1991; Abdelhamid, 2008). It is only proper to indicate further herein that consumption of animal visceral organs (*kayan chiki*) may constitute a major source of AF exposure than muscle.

The garnishing of beef with peanut paste to produce dried beef product (*kilishi*) and roasted beef (*Suya*) is a common practice in West Africa that elevates the AF content of these processed meat products to over 194µg/kg (Jones *et al*. 2001; Chukwu and Imodiboh, 2009) which is beyond any known accepted maximum. Smoke-drying is the commonest method of preservation of fish, another major source of protein in the African continent. The inadequacy of the method with regards to preservation from contamination of smoke-dried fish by aflatoxigenic fungi and AF (Table 1) has been demonstrated (Adebayo-Tayo *et al*. 2009).

The carry-over of AF from feed to poultry by-products including meat and eggs has been investigated and found to be quite low varying with the product. In a feeding trial, Hussain *et al*. (2010) demonstrated AF transfer ratios of 1: 914 and 1: 1939 for the liver and muscle of broilers respectively. In hen, the transfer ratios are 1: 1103 for edible organs (gizzard, kidneys and liver) and 1: 33,100 for breast muscle (Wolzak *et al*. 1986). The carryover into eggs occurs at obviously high ratio of 1:6633 (Wolzak *et al*. 1985). The corresponding ratio of AFB_1 in feeds to residual levels in egg yolk and albumen were shown to be 1:4615 and 1:3846, respectively, in chicken hen (Bintvihok *et al*. 2002). In light of these high ratios, the presence of AF in eggs from Egypt (Abdallah *et al*., undated) and Cameroon (Tchana *et al*. 2010) at levels of up to 7.68 µg/kg (Table 1) indicates that poultry animals consume AF in feeds at alarming concentrations. Although the reported AF levels in both meat and meat products, and eggs from Africa seem insignificant, with chronic intake of such amount simultaneously occurring with other food borne toxicants can have deleterious health impact (Speijers and Speijers, 2004).

3.7 Fermented products

Although fermentation reduces mycotoxins in contaminated food products (Hell and Mutegi, 2011), there is ample evidence to suggest that fermented products in Africa contain significant levels of AF. Kpodo *et al*. (1996) detected AF at levels as high as 289µg/kg in fermented maize dough in Ghana. The presence of AF was observed in all samples of fermented yams and plantains analyzed from the Southern region of Nigeria (Jonathan *et al*. 2011) at levels ranging between 37.67 – 96.34 µg/kg. Detectable (5.2 – 14.5 µg/kg) amounts of the toxins were also found in fermented cassava products from Cameroon (Essono *et al*. 2009). Sorghum based traditional opaque beer from Malawi contained AF at levels above the CODEX permissible limit of 10 µg/kg (Table 1) (Matumba *et al*. 2011). In their review on mycotoxin problem in Africa, Wagacha and Muthomi (2008) reported incriminating levels of AF (200, 000 – 400, 000 µg/l) in 33% of traditionally brewed beer in South Africa. Levels of up to 50µg/kg were found in sorghum based local beer from Lesotho (Sibanda *et al*. 1997).

With these unsafe levels in our fermented products, it will only be proper to adhere to the advice of Pietri *et al*. (2010) that if raw materials comply with the legislated limits, contribution of a moderate daily consumption of beer to AFB_1 intake will not contribute significantly to exposure of the consumer.

3.8 Other foods

Plants and plant products used as medicinal herbs, tea and spices may be commonly contaminated by AF at significant levels of up 2,230µg/kg especially in the case of liver curative herbal medicine sold in India (Trucksess and Scott, 2007). According to these authors, contamination of the toxins has been observed in ginger, garlic and capsicum. A survey conducted for aflatoxigenic fungi and AF in spices from Egypt (Aziz and Youssef, 1991) found black and white pepper contaminated with AF at unhealthy levels (range: 22-35 µg/kg). Zinedine *et al*. (2006) found natural presence of AFB_1 in black pepper, ginger, red paprika and cumin from Morocco at average levels of 0.09, 0.63, 2.88 and 0.03µg/kg, respectively, with the highest level of contamination found in red paprika (9.68µg/kg). Although moulds are frequently isolated from African herbal plants (Bankole and Adebanjo, 2003), it seems the herbs are not prone to AF contamination (Katerere *et al*. 2008). Equally, a search on the literature failed to provide data on the incidence of AF in African tea.

An attempt is made in this chapter to provide an extensive review on AF contamination of commodities in Africa and further information on the subject is referred to the review of Sibanda *et al*. (1997), Shephard (2003), Bankole and Adebanjo (2003) Bankole *et al*. (2006) and Wagacha and Muthomi (2008).

4. Human AF exposure

An important part in elucidating the health effects of AF in humans is the estimation of exposure to the toxin that they receive. A tedious and rather inaccurate estimate of exposure level can be established by determining mean toxin level in the food people eat/amount eaten, either in the basic commodity, e.g. maize kernels and rice as in the case for Nigeria, or in its ready-to-eat form, e.g. porridge for those leaving in the rural area of Limpopo in South Africa. In order to approximate the level of exposure of Africans to AF, let's assume a Nigerian that eats 138kg of cereals annually (Bandyopadhyay *et al*. 2007) as a representative of Africa and whose main staples are maize and rice consumed in a ratio of about 3:1. From Table 1, the AFB_1 mean values for indigenous maize and rice destined for human consumption in the continent are approximately 258 and 83µg/kg, respectively. It can therefore be estimated that 29,500 µg/kg of AF is consumed yearly or let's say 81µg/kg daily by the subject from the two cereals. Even though food processing such as sorting, cooking and others factors will reduce the content (Hell and Mutegi, 2011), what might be left will still be incriminating causing certain chronic intoxications which are discussed subsequently in this chapter. Again, if 350 µg/kg is the mean level of AF in foods that can elicit acute symptoms (Azziz-Baumgartner *et al*., 2005), it can rationally be deduced that Africans are exposed to sub acute doses of AF in maize and rice amongst other food crops. While, the inferences made herein are generalized for Africa with regards to AF intake, it is not without flaws and exceptions, as it can also be reasonably inferred that Africans are exposed to the toxins from virtually all the foods they consume as seen in Table 1. This corroborates with high incidence of the toxin and its biomarkers in their body tissues and

fluids (Gong *et al*. 2003, Tchana *et al*. 2010). This is also in agreement with the fact that in many regions of the continent, the estimate on the frequency of human exposure to AF is about 98% (Wild, 1996).

More precise estimates of AF exposure among humans are best achieved by estimating the toxins and associated metabolites in bile, urine, faeces and hair as well as their distribution in blood, milk, liver, kidney and semen (Njobeh *et al*. 2010). Probably, the easiest one to use is the urine, which can conveniently be sampled and if a 24 hour sample is taken, it can give a fairly accurate content of AFB_1 ingested over a period from the measurement of levels of AFM_1 (Nyathi *et al.* 1987) or adducts (Groopman *et al.* 1992). Another useful method is to assay the amount in the blood and that bound to blood albumin. The latter can be regarded as a good biomarker for AFB_1 exposure (Wild *et al.* 1990) and in effect, removes AFB_1 from circulation. If one assumes that the half-life of albumin is about 20 days (Wild *et al.* 1990), then correlating the amount of conjugate with intake, an estimate of exposure that took place the previous week can be established. A similar case may be made for liver AFB_1-DNA adducts and this has been demonstrated in animals and by extrapolation, a relationship between the level of AFB_1 albumin and AFB_1-DNA conjugates is established (Wild *et al.* 1996).

5. Human health implications to AF exposure with reference to Africa

It is seen how there is extremely high degree of human exposure to AF in many parts of the African continent as previously stated in Section 4.0 of this chapter. Such level of exposure has enormous health and socioeconomic implications. Aspects of the effects of AF contamination of food and feed commodities in terms of the economy will also be highlighted in this review. In general, a disease caused by a mycotoxin is termed "mycotoxicosis" (aflatoxicosis in the case of AF). Aflatoxicosis seems a commoner in developing countries (Williams *et al.* 2004). Furthermore, for mycotoxins to exhibit disease symptoms of a chronic nature, they occur in feed or food at one part per billion (ppb)(μg/kg), the exception being FB_1 which is present at three parts per million (ppm) (mg/kg) mark before symptoms in farm animals begin to be manifest (Bucci and Howard 1996). The difficulty when considering levels of mycotoxins for intoxication in human is that, except for "natural" cases of mycotoxicoses, disease causing levels of mycotoxins cannot be derived and hence, extrapolations from animal experiments have to be made. From known cases of human aflatoxicosis (Azziz-Baumgartner *et al.* 2005), it would seem that acute symptoms are found when levels in food ingested were at a mean value of 350ppb. Again the difficulty with such estimates, however, is gauging the exact amount of the toxin ingested per kg body weight, because this will depend not only on how much is ingested at a specific time, but for how long. Acute doses of mycotoxins may be ingested at one time or over a short period, whilst chronic levels may be consumed over long periods. An example in point of the latter is AFB_1 in groundnut, a staple where rural people ingest this mycotoxin most of their life time and as a consequence, this may increase the incidence of liver cancer (hepatocarcinoma) proportionately (Peers and Linsell, 1973) in the region. A similar situation is found where FB_1 from maize as a staple is ingested but the consequence of a lifetime of exposure to this toxin is not as clearly defined as that of AFB_1, because FB_1 has a much lower toxicity but there are indications that it has a role in various diseases conditions (Dutton 2009). This is discussed subsequently in detail.

5.1 Toxicology of AFB_1

AFB_1 created great interest amongst the medical profession once it was established as a powerful carcinogen (Wogan, 1973). In fact it is claimed to be the most powerful naturally occurring carcinogen known. The toxin is also mutagenic and teratogenic (Raisuddin *et al.* 1993). Because of these toxic properties, several investigations have been carried out over the years since its discovery both *in vitro* and *in vivo* to elucidate its mode of action as a carcinogen, perhaps to the detriment of studying other toxic properties it exhibits as well as those of its congeners AFB_2, AFG_1 and AFG_2. To justify this obsession with AFB_1, it is fair to point out that it is the most commonly occurring and at the highest levels as well as being the most potent when compared with the other AF. However, it might be worthwhile investigating the toxic properties of AFB_2 more closely, as this has all the molecular attributes of AFB_1 apart from the bishydro-furano double bond that confers the carcinogenic property. This double bond makes all the difference, as it allows detoxifying systems (cytochrome P_{450}) in the body to convert it to an epoxide by cytochrome P_{450}s, specifically CYP3A4, CYP3A5, and/or CYP1A2 (Gallagher *et al.*, 1994; Wang *et al.*, 1999) to the exo-8, 9-epoxide, which ironically is the "activated" form of the molecule that can form adducts with deoxyribonucleic acid (DNA) leading to guanine nucleotide substitutions (Lilleberg *et al.*, 1992). In passing it is also important to note that cytochrome P450 types also convert AFB_1 to other derivatives e.g., AFM_1, AFP_1 and Q_1 (Campbell and Hayes 1976). The epoxide is a good alkylating agent and can react with bases such as those in DNA and RNA to form the AF– alkylated form. Obviously other factors are involved, as not all alkylating agents are carcinogens and further, AFB_1 has a tendency to specifically attack guanine, one of the four DNA bases (Taylor 1992). Because AFB_1 is somewhat non-polar, it passes though membranes and other lipid barriers easily and also has a slight water solubility so it passes from the aqueous phase at low concentrations and accumulates in fat soluble phases such as adipose tissues. Furthermore, the molecule itself, because it is primarily aromatic in nature, the main core is a coumarin structure, which is rather flat and therefore, can intercalate into DNA (Jones *et al.* 1998). The epoxide of course can react with other nucleophiles, including those acting as part of the detoxification system, e.g., glutathione (Gopalan *et al.* 1992) and proteins such as blood albumin.

Because of the interaction of AFB_1 with DNA, it is reasonable to ascribe it to be its main toxicological action, certainly at low chronic level of exposure. AFB_1 can form AFB_1-DNA adducts, DNA strand breaks, DNA base damage and oxidative damage that can lead to cancer (Wang and Groopman, 1999). This damage can be repaired by various mechanisms, e.g. base excision repair (Wood, 1999). However, certain mutations that occur due to AFB_1's action may interfere in these repair mechanisms, in particular, the xeroderma pigmentosum complementation gene group D (XPD) which is one of the groups encoding for groups in the nucleotide excision repair pathways. Recent findings suggest that two loci in this group are of particular importance in modulating the AFB_1 related development of liver cancer (Long *et al.* 2009). Probably of more direct importance is the action of AFB_1 on the p53 gene where it causes an AGG to AGT transverse mutation at codon 249 (Bressac *et al.* 1991). Gene p53 is responsible for producing the p53 protein which has an important role in the regulation of cell cycle and in suppressing genome mutation (May E. and May P., 1999).

5.2 Disease conditions in Africa linked to AF exposure

When considering mycotoxins and their effects in Africa there is a tendency to concentrate on those countries in SSA. The assumption being that those countries constituting the Sahara desert and border areas of the Mediterranean Sea are either more like European countries in terms of commercial food production and consumption; or the countries are so dry and unsuited to mass agrarian exploitation that they do not have the same problems of human mycotoxicoses as the rest within the continent. This is an erroneous supposition, as all foodstuffs at some stage may be contaminated with fungi and mycotoxins in these countries (Mokhles *et al,.* 2007, Ghali *et al.,* 2010) but it is fair to say that in SSA, apart from the major cities, the African population is rural, mainly relying on subsistence agriculture. Because this type of activity is unregulated and often is insufficient to maintain a proper nutritional supply to these populations, it is not unreasonable to suppose that they are exposed routinely to mycotoxins and often have little natural resilience to their effects. Consequently, several diseases, which can be correlated to exposure to mycotoxins or can be in part attributed to them, are known. As AF is ubiquitous in African commodities (Nyathi *et al.* 1989), it is not surprising that several of such conditions are caused or exacerbated by these toxins. However, as indicated above, the main problem is AFB_1 and these diseases will be discussed in terms of this toxin.

5.2.1 Acute toxicity

Although most naturally occurring cases of acute toxicity caused by AFB_1 are observed mainly in animals such as one that lead to the discovery of AF in turkey poults (Blount, 1961), there are several cases of human acute aflatoxicoses (most of which often go unnoticed). Since 1982, deaths caused by AF-contaminated maize have repeatedly occurred in the Eastern Province of Kenya (Probst *et al.* 2007). Similar cases have also previously been recorded at various times in India (Krishnamachari *et al.* 1975) and Malaysia (Lye *et al.* 1995). A recent case reported in Kenya is as a result of consuming contaminated maize (Nyikal *et al.* 2004) with 125 deaths out of 317 cases (Azziz-Baumgartner *et al.,* 2005) being recorded. The symptoms are anorexia, malaise and low grade fever leading to acute jaundice and lethal hepatitis. This outbreak was associated with high levels of AF in maize for human consumption (mean concentration of 355ppb) that led to increased levels of AF B_1 albumin adduct and higher hepatitis B titres in the patients than controls (Azziz-Baumgartner *et al.,* 2005). There seems to be little in the way of treatments for these cases, apart from straight forward strategies of antimicrobials and support for the damaged organs (Mwanza *et al.,* 2005) as the toxin acts rapidly and can be lethal.

5.2.2 Conditions of the liver

As the liver is the main organ of detoxification and the first major organ to be exposed to dietary intake of xenobiotics, it is not surprising to find that several liver conditions have been associated with AF, particularly in under developed countries, especially in Africa. The fact that many African crops, in particular, the staples such as ground nut and maize, can be routinely contaminated with AF, leads to an intuitive feeling that many diseases, including those of the liver, can be linked directly or indirectly, to AF, especially AFB_1. As already intimated, this is a dangerous supposition, as many chronic diseases are multi-factorial in nature, and as in any scientific hypothesis, needs strong evidence of support. This is not

easily gathered in the rural areas of Africa where infrastructure is poor, health services varying from none to basic clinics and health centres with little time or inclination to gather usefully directed statistics. Even the analysis and quality control of staples is dependent upon external scientific studies, which often are one of investigations that merely provide a snapshot of a true situation. Consequently, the information available to investigators attempting to correlate disease conditions to mycotoxin exposure is sparse and patchy with respect to various African countries. In addition, the practise of comparing these conditions in Africa to those in developed countries may highlight interesting differences that can give clues to the aetiology of African disease but may also lead to western scientists dismissing explanations of such chronic diseases in Africa, because of well researched conditions in their own countries, which have explanations other than dietary ones. Clearly, people living in African rural areas have an environment that is completely different to those living in western cities and hence, elevated incidence of certain medical conditions may be explained by factors other than those appertaining let's say for example, in Europe or North America. In any case, there are readily available systems in developing countries particularly in Africa that could provide natural environments for human experimentations as it is in theory, possible to compare the chronic diseases in rural populations against those of urbanized populations of the same race group (Njobeh *et al*., 2010) elsewhere.

5.2.2.1 Hepatitis

Hendrickse (1991) lists five possible roles for AFB_1 in human disease including fatal AFB_1 poisonings that can "masquerade as hepatitis". This condition is an inflammation of the liver cells caused by various agents including viruses e.g., hepatitis B virus, (HBV) and may be self healing or in extended chronic cases, lead to cirrhosis. Several cases of hepatitis have been reported in the literature including one in India that was attended by high mortality (Krishnamachari *et al.* 1975). The outbreak was associated with maize contaminated by AF and it was concluded that it was as a result of aflatoxicosis.

5.2.2.2 Cirrhosis

Cirrhosis of the liver is a well known disease condition usually related to alcohol consumption. However, it may be caused by exposure of the organ to toxic principles other than ethanol and can often be found in children because of their higher susceptibility to toxin exposure. In certain countries in Africa (e.g. Ethiopia) (Tsega, 1977) and India (Yadgiri *et al.,* 1970), the condition has been described and efforts made to correlate it with AF intake (Tandon *et al.,* 1978). The whole issue is somewhat clouded because of the commoner occurrence of hepato-cellular carcinoma (HCC) together with cirrhosis, in areas of high AF exposure and hence, a relationship has been suggested by the two conditions; simply this would be that the onset of childhood and other cirrhoses leading to liver cancer (Lata, 2010). There is no real evidence, however, that there is a link (Kew and Popper 1984) and further, that the roles of HBV, hepatitis C (HVC) or AFB_1 is by no means clear. In some cases of cirrhosis, there is an over expression of the p53 gene and this has been related to simple cellular stress or mutation of the gene, which occurs in many of the subjects exposed to HBV and/or AFB_1 (Livni *et al.,* 1995). More recent work on cirrhosis in The Gambia (Kuniholm *et al*. 2008) tentatively concludes that health effects due to exposure to AFB_1 could include this disease. It would seem that, because of various factors that African populations face, including alcohol abuse, it is difficult to tease out any particular one and ascribe a role to it, other than it does make a contribution to the overall condition.

5.2.2.3 Hepatocellular Carcinoma (HCC)

This chronic disease is a major global health problem, causing over 600,000 cases per annum (Ferenci *et al.* 2010) and accounts for over 70% of all liver carcinomas (Lata, 2010). In Africa, the most likely cause or promoter of such a cancer is AFB_1 (it has been estimated that AFB_1 may play a causative role in 4.6-28.2% of all global HCC cases) (Liu and Wu, 2010), which is a primary carcinoma in the case of areas where factors such as hepatitis and AF are found. In other situations, the cancer may be of secondary type, i.e., an infiltration of a metastasised type from other parts of the body. Thus the situation is, not as clear cut as might have been thought. Areas in Africa where groundnut is consumed regularly, which is one of the principal sources of the mycotoxin, such as Sudan (Omer *et al.* 1998); Mozambique and Kenya, have high levels of liver cancer which have been shown to be correlated to AFB_1 levels (van Rensburg *et al.*, 1985). These areas also have a high incidence of viral diseases that can also affect the liver, e.g., the highly contagious HBV plus HIV infection (Kew, 2010a); HCV (Ashfaq *et al.* 2011); and iron over load (Kew and Asare, 2007) which tends to cause the issue. Hence it has been argued that these viruses are the cause of the high liver cancer incidence with other factors such as alcoholism and mycotoxins being of secondary importance (Stoloff, 1969; Perz *et al.*, 2006). Given the multi-factorial nature of cancer, these claims are dangerous ones to make and also apply to the school of thought that says HCC is mainly due to AFB_1. Nevertheless, hepatitis viruses do seem to play a major role in the development of HCC in African countries (Kirk *et al.*, 2004; Ocama *et al.*, 2009). It is, however, known that the risk posed by AFB_1 is independent of that of HBV (Blondski *et al.*2010) although where both factors occur together, the risk is increased. It has been suggested, for example that cirrhosis of the liver, as previously discussed, may be a precursor to liver carcinoma (Kew, 2010a; Lata, 2010). It seems, therefore that HCC is multi-factorial, although some factors may contribute more than others, depending upon circumstances, e.g., in Africa where AFB_1 is commonly found in food, it may play a bigger role here than elsewhere, where it is not found to any extent. Further, in simple terms one thinks of a cancer initiator and a cancer promoter. It may well be that the highest liver cancer areas have a combination of both cancer forming factors plus others such as genetic ones. This certainly is the case for AFB_1 as it has been seen that AFB_1 needs to be activated by the cytochrome P_{450} system in particular the CYP 3A4 version plus CYP3A5 and 3A7 (Kamdem *et al.*, 2006). Hence those persons with genetics dictating less expression of these forms of cytochrome P_{450} would, in theory, have less chance of developing the cancer and vice versa. As earlier mentioned, AFB_1 may cause a missense mutation, where a guanine residue is converted to a thymine in the 249 codon of the p53 gene and this is considered to be an important marker in the promotion of HCC by AFB_1 (Bressac *et al.*, 1991). In a study that was conducted on Chinese subjects, this mutation was detected in the blood and liver of HCC patients (Jackson *et al.*, 2001). However, not all the patients had this mutation in their livers, indicating yet again a multi-factorial situation, although some of the liver negative subjects did have the mutation in their plasma samples. It was concluded that the detection of the codon 247 mutation may present a method of an early diagnosis of HCC.

5.2.3 Kwashiorkor

Kwashiorkor is a malnutrition condition which is in essence a protein deficiency of the young like marasmus but unlike it, the patient has sufficient calories. It is characterized by oedema, anorexia, dermatitis and an enlarged liver with fatty infiltrates (Bhattacharyya,

1986). Because of the liver's involvement, it was suggested that AFB_1 may be involved (Hendrickse *et al.,* 1983). This appeared to be supported by other observation, e.g., relating peak prevalence for kwashiorkor with climatic conditions such as high humidity (de Vries and Hendrickse, 1988); general occurrence of AFB_1 in children's excreta (de Vries *et al.* 1987, 1990); liver (Lamplugh and Hendrickes, 1982); serum (Coulter *et al.,* 1986; Hatem *et al.,* 2005); and its reduced product, aflatoxicol, in livers of Ghanaian children (Apeagyei *et al.,* 1986).

Other work, however, does not support the hypothesis that AF have a role in Kwashiorkor (Househam and Hundt, 1991). What AFB_1 exposure seems to influence in kwashiorkor patients is that their recovery time in hospital is lengthened and this is linked to a difference in the way AFB_1 is metabolised as compared to control non-kwashiorkor infants (Ramjee *et al.,* 1992). It could, therefore, be argued that kwashiorkor is protein deficiency in children who are receiving sufficient carbohydrate such as diet on maize gruel given to weaned children. Maize is not a balanced source of protein but has plenty of starch and hence its use as a staple would fit this hypothesis. Oedema would arise due to lack of sufficient blood proteins, i.e., hypoalbuminemia (Waterlow, 1984). This view has been questioned as it has been observed that the oedema symptoms can disappear before the blood albumin levels return to normal (Golden *et al.,* 1980). An alternative mechanism suggested was that the condition was caused by oxidative species not being regulated by anti-oxidants, e.g., vitamin E, which were depleted because of the poor diet (Golden and Ramdath, 1987). In order to test this, children in Malawi were treated with antioxidants such as riboflavin, vitamin E and selenium (Ciliberto *et al.,* 2005) but both the treated and control group given placebos showed similar levels as kwashiorkor as evidenced by oedema. More recent work seems to suggest that the incidence of kwashiorkor in Africa may be dropping, as evidenced by a Nigerian study (Oyelami and Ogunlesi, 2007). This was put down to better management of diarrhoeal diseases. An interesting point that was made in an earlier publication (Enwonwu, 1984) is that protein deficiency affects the cytochrome P_{450} system, which would allow for the accumulation of AFB_1 in kwashiorkor children due to lack of conversion products. Because of the lack of the AFB_1 epoxide production, its carcinogenic property would be lost, which, ironically, would be re-instituted if protein supplementation was given. It would seem that the underlying causes of kwashiorkor still remains a mystery, is multi-factorial (Oyelami *et al.,* 1995) and unrelated to the lack of any particular food or nutrient (Lin *et al.,* 2007) although potassium supplement appeared to help (Manary and Brewster, 1997). However, it is still tempting to evoke some toxic factor in the diet of children that interacts with other factors to produce the condition. Kwashiorkor has been referred to at a certain time as a "maize disease" (Adhikari *et al.,* 1994). This label has been used in other circumstances with reference to *Fusarium* mycotoxins, in particular the FB, i.e., FB_1 (Dutton, 2009). It is not impossible that these mycotoxins rather than AFB_1 have some role in the development of kwashiorkor.

5.2.4 Miscellaneous

5.2.4.1 Reye's syndrome

This can be a fatal disease that affects mainly children and is typified by fatty liver, swelling of the brain (encephalopathy) and hypoglycemia. The liver becomes enlarged and firm but with no jaundice. There is also evidence of kidney damage. It is not particularly associated with Africa and indeed many cases were reported in first world countries (Harwig *et al.,*

1975). When first described (Reye *et al.,* 1963), the cause was unknown but it often occurred after a mild respiratory tract infection and, because of involvement of the liver, as in kwashiorkor and similar cases in Thailand, it was suggested that AFB_1 may be involved (Becroft, 1966; Olson *et al.,* 1971). This was supported by further studies where materials resembling AF were isolated from the liver of a girl of 8 months of age (Becroft and Webster, 1972) and AFB_1 in the livers of 5 children (Stora *et al.* 1983) but other investigations showed that the presence of AF in Reye's syndrome livers was variable and probably, the syndrome was the result of multiple interrelated factors (Ryan *et al.*, 1979; Rogan *et al.*, 1985). Nevertheless, concern was raised with respect to the presence of AF in the serum and urine of children with Reye's syndrome and it was concluded that this was of general public health importance (Nelson *et al.*, 1980). Because there was no clear cut evidence that AF played a central role in Reye's syndrome, other factors were considered. One of these *inter alia*, was the use of salicylates (Aspirin) in treating children with various infections, e.g., respiratory viral infections (Trauner, 1984). It has been claimed that since aspirin was abolished as a medication for influenza and similar diseases for patients under 18 years old, Reye's syndrome has become very rare (Kimura, 2011) and is considered a secondary mitochondrial disease.

5.2.4.2 Lung cancer

Oyelami and co-workers (1997) found that children with various diseases, including pneumonia in Nigeria had high levels of AF in their lungs. The route of acquisition of this toxin was not clear but there is evidence to suggest that the lung can be exposed to AFB_1 by inhalation (Hayes *et al.,* 1984) and that this may give rise to lung cancer (Dvorackova *et al.* 1981). Human lung microsomes cytochrome P_{450} does not seem to be well expressed, the CYP1A2 form is not expressed at all (Wheeler *et al.* 1990) although some CYP1A2 activity is (Kelly *et al.* 1997). The exposure of lung cells, however, to polycyclic aromatic hydrocarbons induces cytochrome P_{450}s that may activate AFB_1 (van Vleet *et al.,* 2001). A study (Donnelly *et al.,* 1996) was conducted on human lung tissue fractions to investigate the possible role of other oxidative systems in the activation of AFB_1 and its deactivation by glutathione S-transferase. The study concluded that AFB_1 activation in human lung was primarily due to lipoxygenase and prostaglandin H synthase activity and that low conjugation activity contributed to human pulmonary susceptibility to AFB_1. Similar studies (van Vleet *et al.,* 2002a) showed that cytochrome P_{450} in lung was capable of activating AFB_1 (van Vleet *et al.,* 2002b) and CYP1A2 has greater importance in lung tissue in activating AFB_1. Later work has indicated that cytochrome P_{450} CYP2A13 is highly expressed in human bronchial cells, indicating that this cytochrome may be responsible for AFB_1 activation in the lung (Zhu *et al.,* 2006). Irrespective of what activates AFB_1, it seems that exposure of humans to AFB_1 inhalation may result in an increased risk of lung cancer (van Vleet *et al.,* 2002b). In the case of rural Africans, this is then a possibility as they process their maize manually (partly by winnowing), often in enclosed conditions where they may be exposed to dust from contaminated cobs.

5.2.4.3 Immuno-suppression and reproductive problems

An often neglected aspect of chronic mycotoxin exposure in humans is the effect of these on the immune system. With the arrival of infections such as HIV, which also attacks this system and is a major problem in SSA, the extra burden of immuno-suppressors may affect

the course of the immune deficiency syndrome (Bondy and Pestka, 2000; Dutton, 2004; Jiang *et al.,* 2008). There is a strong evidence to show that mycotoxins can promote "secondary infections" of organisms that are generally commensal in an animal due to immuno suppression, e.g. OTA in pigs (Stoev *et al.,* 2000). Several mycotoxins have been shown to be immuno-suppressive or have a potential to be so (Sharma, 1993) and these include AFB_1 (Cusumano *et al.,* 1996). These effects may be compounded by the interaction of more than one mycotoxin, which may occur in substrates (Hussein and Brasel, 2001) that may cause synergism, although doubts have been expressed on assessing these effects (Speijers and Speijers, 2004). There is, however, evidence from *in vitro* experiments that this may occur (Creppy *et al.,* 2004, Luongo *et al.,* 2006; Del Rio Garcia *et al.,* 2007; Orsi *et al.,* 2007; Smith *et al.,* 1997) and it is reasonable to suppose that these effects may be extrapolated to humans.

The action of AFB_1 on the human reproductive system and gestation and birth defects is experienced by animals (Ibeh and Saxena, 1997) so it not unreasonable to suppose that these may occur in humans and, from a recent review on the subject, it would seem that AFB_1 exposure in humans has several effects on reproduction (Shuaib *et al.,* 2010a). Several studies have measured the blood albumin-AFB_1 conjugate levels in pregnant African women and have found substantial levels as might be expected (Turner *et al.,* 2007). In one study, there was a correlation between these levels and anaemia in pregnant women in Ghana, if iron deficiency anaemia was excluded (Shuaib *et al.,* 2010b). In the case of men, sperm abnormalities have been associated with AFB_1 in their semen (Ibeh *et al.,* 1994). Other effects are low birth weight (de Vries *et al.,* 1989) and jaundice in neonates (Abulu *et al.,* 1998) which was correlated with AFB_1 in cord blood and effects on their immune system (Turner *et al.,* 2003). AFM_1 is a cytochrome P_{450} mediated hydroxylated product of AFB_1 found in most mammal secreted milks, previously exposed to AFB_1 (Motawee *et al.,* 2009). It does have toxic and carcinogenic properties, although not as marked as AFB_1 (Hsieh *et al.,* 1984). It has been found in breast milk of women from several African countries (Coulter *et al.,* 1984; Zarba *et al.,* 1992). Its presence in cow's milk, particularly in that produced in African rural areas (Mwanza, 2007; Tchana *et al.,* 2010) is of great concern, considering its use in the nutrition of children.

To summarize on the health implications associated with human exposure to AF from the African viewpoint, it is but normal to state that of the AF, AFB_1 is considered to be the most important and much likely to be involved in human diseases. Although most work has concentrated on its role, sight must not be lost of the fact that its congeners, AFG_1 and M_1, which are also present in the environment, are capable of being converted to the active epoxide derivatives by the cytochrome P_{450} system. AFM_1 is of particular importance, because of its occurrence in milk from dairy cattle fed feeds contaminated with AFB_1. With AFB_1 itself, as discussed herein, it is very difficult to tease out its exact role in various human disease conditions. In animal trials, it has been shown to be a powerful carcinogen and there is no reason to suspect that it does not have a carcinogenic effect in humans. A strong claim that the role of mycotoxins in human disease has been ignored was recently made (Wild and Gong, 2010) and interestingly, AFB_1 and FB_1 were cited as major culprits, because of their common occurrence in staple foods, often in combination. However, because of the complicating issues in the human environment and lack of direct experimentation on humans, it is difficult to assign a definitive role in any disease condition. In the case of HCC for example, at best we can claim that AFB_1 is responsible for 5-28% of all

cases in Africa, although the detection of specific point base mutations in liver cancer cells may allow for an estimation of the contribution of AFB_1 to human liver cancer. Whatever the precise medical role of mycotoxins in human diseases is , one cannot help but have a strong empathy with the pleas of Wild and Gong (2010) that they should be taken very seriously in Africa and other developing parts of the world.

6. Possible intervention control strategies for AF in Africa

Considering the action and toxic nature of AF, it may have been thought that attempting to completely eliminate their actions in humans or perhaps, animals would be rather futile. However, several approaches have been or can be taken, either during pre or post-exposure that can assist in alleviating or moderating the actions of AF, particularly those of AFB_1. Whatever actions are taken or have been adopted, do require financial considerations and because the current global financial situation is not in a good stead, make this even much more difficult, but of great importance. In order to assess "overall disease burden" (ODB), the concept of "disability-adjusted life year" (DALY) is used (Murray, 1994). It is defined as the number of years lost due to early death, ill health or disability and this is deduced by the addition of two variables, i.e. "years of life lost" (YLL) to "years lived with disability" (YLD). It follows that one DALY is equal to one year of healthy life lost. In order, therefore, to calculate the cost effectiveness of a programme to prevent HCC for example, the DALYs for this disease must be determined followed by computing the likely lowering of this figure. Against this, is the cost of reducing mycotoxins like AFB_1 and FB_1 in a food staple such as maize. This in turn depends upon regulations governing the health risk factors, which in order to reduce risk to health, must become more stringent resulting in economic losses in the crop due to it not meeting the recommended standard. For example, it is claimed that export losses from AF in groundnut may exceed US$ 450 million, if the level were 4ppb AF (European Union regulation) as opposed to the level imposed in USA of 20ppb which would result to about $100 million loss (Wu, 2004). An important aspect of this approach is the fact that WHO has in the past not recognised AF as a high priority problem within the top 6 health problems but it has been argued that AF not only has an impact on HCC, but probably modulates several of the other top 6 problems (Williams *et al.*, 2004). In order to determine the cost effectiveness of various interventions, it is necessary to establish the cost effectiveness ratio (CER), which is the gross domestic product (GDP) multiplied by DALY saved per unit cost. In a study involving AF in maize in Nigeria and groundnut in Guinea, two strategies were compared viz: pre-harvest control; and post harvest interventions (Wu and Khlangwiset, 2010a). Accordingly, it was shown that the cost of both interventions exceeded the monetised values of lives saved and quality of life gained by reducing HCC, if applied nationwide. Furthermore on this study of Wu and Khlangwiset (2010a), CER for biocontrol in Nigeria ranged from 5.1 to 24.8 and for post harvest intervention for groundnut in Guinea from 0.21- to 2.08. Any intervention with a CER <1.0 is considered very cost effective and more that >0.33 as just simply cost effective (Wu and Khlangwiset, 2010a). The implications of such calculations cannot be under estimated, as they do not only indicate to governments and world bodies their value, but also have an ethical dimension in terms of human and animal sufferings.

When considering interventions, several routes may be taken (Wu and Khlangwiset, 2010b). The best approach is that of prevention which is always better than cure. One such intervention is that of releasing non-aflatoxigenic strains of *Aspergillus flavus* into the agricultural environment and such a commercial product called Afla-Guard® is available commercially. This results in suppression of naturally occurring aflatoxigenic strains (Abbas *et al.,* 2011). Another is the introduction of genetically modified variety of crops, e.g., genetically modified (GM) Bt maize which inhibits insect damage and hence fungal infection (Wu, 2006). Another preventive measure is feeding of animals with amino acids and vitamins particularly lysine and vitamin C that have protective actions against mycotoxins (Obidoa and Gugnani, 1992; Smith *et al*., 2000). A more traditional way is the use of fungicides and pesticides, although current preference is not in favour of this. The use of natural predators (cats and dogs) at fields and storage sites to deter rodents, birds and monkeys is a very practicable preventive control strategy for Africa.

Post harvest treatments are a little more difficult due to the persistence of AF in commodities even after processing. Early harvesting, effective drying (to moisture level of less than 14%), cleaning, removal of damaged produce (sorting), e.g., small and discoloured groundnuts (Chiou *et al.,* 1994); good storage facilities with controlled humidity (Kew, 2010b) and packaging can all contribute to lowering the level of the mycotoxin in the final product. While these remain the most effective post harvest control measures for Africa, other alternative but less effective measures include reduction of storage time, use of chemical and botanical preservatives, and detoxification of contaminated produce. The most commonly used chemical preservatives are the organic acids; formic, acetic, propionic, sorbic and benzoic acids. Nonetheless, they are ineffective in foods that contain basic components that neutralize these acids (Smith and Moss, 1985). Alkalis, strong acids and oxidizing agents are quite effective in detoxifying AF but because they could drastically change the properties of the products, ammoniation is still the most preferred and developed detoxification procedure. But the changes in chemical compositions and organoleptic properties of ammoniated meals makes them unfit for human consumption nevertheless good enough for animals. Commercialization of the ammoniation procedure in Africa by governments and private companies as has been successfully done in the USA, could help provide livestock farmers in developing countries with relatively safer feeds in the face of highly contaminated feedstuffs and shortage of feeds.

The toxic and 'off flavour' products of chemical preservation and detoxification processes has led scientists to search for natural, safer and environment-friendly fungicidal products. Among such African based studies, *Lippia multiflora* leaf extract has been shown to have fungistatic effect on *A. flavus* (Anjorin *et al*., 2008). More intense field trials of such promising plant products and their subsequent formulation into botanical fungicide would be impressive for the continent. Gamma irradiation of AF contaminated foods lowers both the toxicity (Ogbadu and Bassir, 1979) and production (Ogbadu, 1979; Ogbadu, 1980a; Ogbadu, 1981; Ogbadu, 1988) of the toxin in irradiated foods and so this could be a good post harvest, processing and packaging treatment option for African countries if suitable infrastructures are put in place. Hazard Analysis Critical Control Point (HACCP), a proactive management system in which food safety is maintained through the analysis and control of biological, chemical, and physical hazards from raw material production, procurement and handling, to manufacturing, distribution and consumption of the finished product has become a priceless tool for mycotoxin control (FAO, 2003).

Clinical applications in the control of conditions such as HCC have been applied with varying degree of success. This range from preventative measures such as the use of Novasil clay being added to the diet to bind AF (Afriyie-Gyawu *et al.,* 2008); and HVB immunisation (Kew 2005). The use of drugs can be considered in two parts, one that blocks cytochrome P_{450}s responsible for the activation of AFB_1 to an active (its epoxide) form, e.g. oltipraz (Langouet *et al.,* 1995; Wang *et al.,* 1999) and natural foods, e.g. Brassicas (Manson *et al.,* 1997); and those which may have some other non-clear cut effect such as the use of plant extracts as protective agents (Kotan *et al.,* 2011); boric acid (Turkez and Geyikoglu, 2010); sorafenib, a blocker for signalling pathways involved in HCC (Dank, 2010).

7. Legislation

In order to protect consumers against the hazards of mycotoxins, many countries including 15 of those from Africa (Sibanda *et al*., 1997; Fellinger, 2006; Njobeh *et al*., 2010) have instituted legislation against some mycotoxins notably AF. According to these authors, the maximum tolerable limits for AF in human foods in Africa is between 5-20 ppb, while for animal feeds is from 5 to 300 ppb with infant foods having the least regulated levels (0.05-10 ppb) (0.05 ppb for AFM_1 in the case of South Africa). While these maximum allowable limits would protect citizens from the dangers of AF, the biggest challenge in regulating mycotoxins in the continent is the lack of enforcement of legislation partly attributed to the presence of informal food market systems operating in most countries. Under this market structure, raw agricultural produce from farms and storage barns are sold directly to consumers without being screened for mycotoxins neither are they subjected to inspection for spoilage. Furthermore, government agencies charged with the responsibility to regulate mycotoxins are non-existent in many of these countries and even when available, they are dysfunctional as they are composed of deplorable infrastructures and logistics. An effective mycotoxin surveillance and food quality control unit which ensures that all foods and feeds destined for human and animal consumption are devoid of mycotoxins at harmful levels must be in place to implement mycotoxin legislation in the continent.

8. Prevention by surveillance and awareness campaign

Assessing the levels of mycotoxins and indeed other food toxicants is paramount to evaluating food safety. In line with the recommendation for effective mycotoxin survey and food and feed inspection for implementation of legislation for food safety, African governments must build or strengthen already existing regional laboratories to monitor mycotoxins in foods and feeds on regular basis. And to ensure that they are in compliance with set standards. Invariably, Africa must reinforce its food quality control agencies and this can only be achievable if professionals working in such establishments possess the academic as well as technical capacity for mycotoxin management which calls for the inclusion of courses on mycotoxins in the curricula for training of agriculturists, medical personnel and laboratory based scientists. Awareness on the adverse impact of mycotoxins should not be limited to professionals in the food and feed related industries, but to the entire consumers. Public awareness campaign on impact and prevention of mycotoxins especially the notorious AF via electronic and print media and other information dissemination modes is therefore an imperative. Such scientific and public enlightenment interventions require concerted national and international multidisciplinary strategies (WHO, 2006). It is but imperative to engage both national and

international bodies to partner with one another to effectively manage mycotoxins. For example in Nigeria, experts in devised but related fields from academia and research institutions formed the Nigerian Mycotoxin Awareness and Study Network in 2005 (NMASN-www.ngmycotoxin.org) with a common goal of offering scientific and technical support towards managing mycotoxins in the country. In doing so, the network organizes annual workshops for stakeholders. Similarly this year (2011), the International Society of Mycotoxicology organised a world conference in Cape Town, South Africa on mycotoxin reduction (www.mycoredafrica2011.co.za) which brought scientists from all over the globe to not only share knowledge and expertise but to establish research collaborations towards strengthening the capacity of the African mycotoxicologists and laboratories. European Union (Leslie *et al*., 2008) and World Health Organisation (WHO, 2006) had earlier organized such international conferences in 2005 and 2006 in Ghana and Congo Brazzaville, respectively. It is only pertinent now to encourage scientists and institutions involved in mycotoxin research in Africa to collaboratively seek accessible research grants from the AU, EU and other foreign funding agencies for more effective investigations and control of mycotoxins.

9. Summary

Aflatoxins are toxic secondary metabolites produced notably by *Aspergillus flavus* and *A. parasiticus* that frequently invade foods and feedstuffs before and after harvest. The four major aflatoxins include aflatoxin B_1, B_2, G_1 and G_2 with aflatoxin B_1 recognized as the most prevalent and toxic of all aflatoxins. Their presence in foods and feeds is inevitable and as such, humans and animals are exposed to them on a continuous basis leading to a wide array of health complications. Particularly aflatoxins B_1, they have been directly linked to hepatocarcinoma and deaths among humans and animals. Although this may be the case worldwide, the situation in sub-Saharan Africa is very severe as increased levels of exposure to this group of mycotoxins is a common phenomenon since it presents suitable environmental conditions for aflatoxin concentration in various food and feed materials. Again, the problem is further exacerbated by increased prevalence in the continent, of such endemic diseases as malaria, hepatitis and HIV/AIDS. In Africa recently, we have experienced the most fatal aflatoxin-poisoning outbreaks including two episodes in Kenya and one in Nigeria. In view of the significance therefore, of aflatoxin exposure, this chapter reviews the disparity in aflatoxin contamination of food and feeds worldwide with particular emphasis on Africa. It has also expounded briefly on those factors that influence the distribution of aflatoxins in various food and feeds. Additionally, an in-depth review is provided on the negative public health problems and the impact in the economy associated with this notorious group of secondary metabolites with particular reference made from the African context, while also discussing those control strategies possible within the continent's technological capacity.

10. Keywords

Aflatoxins, Occurrence, Agricultural commodities, Health, Control, Africa.

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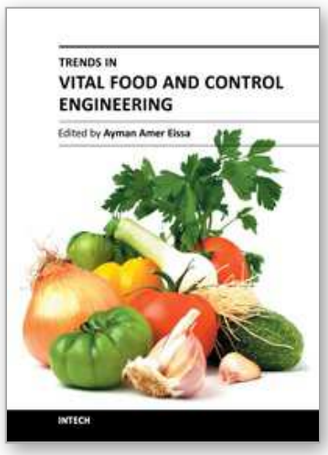

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