Chapter 7

Occurrence of Aflatoxins in Food

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Additional information is available at the end of the chapter

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1. Introduction

Mycotoxins are natural contaminants in raw materials, food and feeds [1]. Aflatoxins are toxic metabolites produced by different species of toxigenic fungi, called mycotoxins. The discovery of aflatoxins dates back to the year 1961 following the severe outbreak of turkey "X" disease, in the England, which resulted in the deaths of more than 100.000 turkeys and other farm animals. The cause of the disease was attributed to a feed using thin-layer chromatography (TLC) revealed that a series of fluorescent compounds, later termed aflatoxins, were responsible for the outbreak. The disease was linked to a peanut meal, incorporated in the diet, contaminated with a toxin produced by the filamentous fungus *Aspergillus flavus*. Hence, the name aflatoxins, an acronym, has been formed from the following combination: the first letter, "A" for the genus *Aspergillus*, the next set of three letters, "FLA", for the species *flavus*, and the noun "TOXIN" meaning poison [2].

Aflatoxins (AFs) are difuranocoumarins produced primarily by two species of *Aspergillus* fungus which are especially found in areas with hot, humid climates. *A. flavus* is ubiquitous, favouring the aerial parts of plants (leaves, flowers) and produces B aflatoxins. *A. parasiticus* produces both B and G aflatoxins, is more adapted to a soil environment and has more limited distribution [3]. *A. bombysis*, *A. ochraceoroseus*, *A. nomius*, and *A. pseudotamari* are also AFs-producing species, but are encountered less frequently. From the mycological perspective, there are qualitative and quantitative differences in the toxigenic abilities displayed by different strains within each aflatoxigenic species. For example, only about half of *A. flavus* strains produce AFs-producing species more than 10^6 μg kg^{-1} [4].

Among the 18 different types of aflatoxins identified, the major members are aflatoxin B1 (AFB1), B2 (AFB2), G1 (AFG1), G2 (AFG2), M1 (AFM1) and M2 (AFM2). AFB1 is normally predominant in amount in cultures as well as in food products. Pure AFB1 is pale-white to yellow crystalline, odorless solid. AFs are soluble in methanol, chloroform, acetone, acetoni-

trile. *A. flavus* typically produces AFB1 and AFB2, whereas *A. parasiticus* produce AFG1 and AFG2 as well as AFB1 and AFB2. Four other aflatoxins M1, M2, B2A, G2A which may be produced in minor amounts were subsequently isolated from cultures of *A. flavus* and *A. parasiticus*. A number of closely related compounds namely aflatoxin GM1, parasiticol and aflatoxicol are also produced by *A. flavus.* The order of acute and chronic toxicity is AFB1 > AFG1 > AFB2 > AFG2, reflecting the role played by epoxidation of the 8,9-double bond and also the greater potency associated with the cyclopentenone ring of the B series, when compared with the six-membered lactone ring of the G series. AFM1 and AFM2 are hydroxylated forms of AFB1 and AFB2 [5]. AFM1 and AFM2 are major metabolites of AFB1 and AFB2 in humans and animals and may be present in milk from animals fed on AFB1 and AFB2 contaminated feed [6, 7]. AFM1 may be also present in egg [8], corn [9] and peanut [10, 11].

AFs interact with the basic metabolic pathways of the cell disrupting key enzyme processes including carbohydrate and lipid metabolism and protein synthesis [12]. The health effects of aflatoxins have been reviewed by a number of expert groups. Aflatoxins are among the most potent carcinogenic, teratogenic and mutagenic compounds in nature [13]. The International Agency for Research on Cancer (IARC) has concluded that naturally occurring aflatoxins are carcinogenic to humans (group 1), with a role in etiology of liver cancer, notably among subjects who are carriers of hepatitis B virus surface antigens. In experimental animals there was sufficient evidence for carcinogenicity of naturally occurring mixtures of aflatoxins and of AFB1, AFG1 and AFM1, limited evidence for AFB2 and inadequate evidence for AFG2. The principal tumors were in the liver, although tumors were also found at other sites including the kidney and colon. AFB1 is consistently genotoxic in vitro and in vivo [3].

The Joint Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO) Expert Committee on Food Additives (JECFA) estimated potency values for AFB1 from the epidemiological data. These corresponded to 0.3 cancers/year per 100,000 population per ng aflatoxin/kg body weight (b.w). per day (uncertainty range: 0.05-0.5) in hepatitis B virus antigen positive individuals and 0.01 cancers/year per 100,000 population per ng aflatoxin/kg b.w. per day (uncertainty range: 0.002-0.03) in hepatitis B virus antigen negative individuals. AFM1 has been evaluated separately from AFB1 by the JECFA, because of its potential to be present in milk and milk products of livestock fed on aflatoxins contaminated feed [14]. The JECFA concluded that AFM1 should be presumed to induce liver cancer in rodents by a similar mechanism to AFB1, and that estimates of the potency of AFB1 can be used for determining the risk due to intake of AFM1, including those for populations with a high prevalence of carriers of hepatitis B virus. The carcinogenic potency of AFM1 was estimated to be one-tenth that of AFB1, based on a comparative study in the Fischer rat conducted by Cullen et al. [15].

Humans can be exposed to aflatoxins by the periodic consumption of contaminated food, contributing to an increase in nutritional deficiencies, immunosuppression and hepatocellular carcinoma. AFs have a wide occurrence in different kind of matrices, such as spices, cereals, oils, fruits, vegetables, milk, meat, etc. [5]. About 4.5 billion people, mostly in developing countries, are at risk of chronic exposure to aflatoxins from contaminated food

crops [16]. Therefore, in order to avoid the toxicity, the levels of aflatoxins and similar toxic compounds in foodstuffs have to be monitored closely, and to be kept under control continuously. Otherwise, related health effects like acute and chronic intoxications, and even deaths, will still be an issue [17].

In this review, we are presenting a report on the situation of aflatoxin contamination in food and foodstuffs such as oilseeds (peanuts, almonds, pistachios and other tree nuts), cereals (wheat, barley, rice, corn, etc), spices, milk and dairy products and other foods of animal origin (meat, offal, eggs etc) in world. Incidence of contamination will classified as country and type of food and discussed. Also, we are reviewing the scientific literature on aflatoxins in foods and how they affect animal and human health.

2. Occurrence of Aflatoxin in Oilseeds

Oilseed crops are primarily soybeans, sunflower seed, canola, rapeseed, safflower, flaxseed, mustard seed, peanuts and cottonseed, used for the production of cooking oils, protein meals for livestock, and industrial uses. These specific oilseeds are eligible for nonrecourse loans. Other oilseed crops include castor beans and sesame. After extraction of the oil the residue is a valuable source of protein, especially for animal feeding stuffs, as in oil-seed cake or press cake. Oilseeds and their products are mainly consumed as snacks as well as part of the ingredients of certain dishes in human daily diet [18].

According to many reports (Table 1), peanuts are the main susceptible products for aflatoxin contamination [19-21]. Tree nuts such as almonds, walnuts, and pistachios may be contaminated with aflatoxin, though at lower levels than for cottonseed and corn; however, the problem is very significant to producers because: (1) the crop has a high unit value, and (2) much of the crop is sold to European markets that enforce limits significantly lower than those in some countries [22].

For over all sanitary precaution, the European Union has enacted in 1998, very severe aflatoxin tolerance standards of 2 $\mu g\ kg^{-1}$ AFB1 and 4 $\mu g\ kg^{-1}$ total aflatoxins for nuts and cereals for human consumption and this has come into effect from January, 2001. Consumers in the developed world are well aware of the carcinogenic effect of aflatoxins and will thus stay away from a product that has aflatoxin beyond the acceptance level. Exports of agricultural products particularly groundnuts and other oilseeds from developing countries have dropped considerately in recent years resulting in major economic losses to producing countries as a result of this restriction. According to the World Bank estimate, the policy change by the European Union will reduce by 64%, imports of cereals, dried fruits, oil seeds and nuts from nine African countries namely Chad, Egypt, Gambia, Mali, Nigeria, Senegal, South Africa, Sudan and Zimbabwe and this will cost African countries about US $670 million in trade per year. However, the new rule of the EU has been criticized as being too stringent. There is the need for mycotoxin surveillance because of its wide occurrence in contaminated commodities [23].

Aflatoxin contamination of peanut, resulting from invasion by *A. flavus* or *A. parasiticus*, is a major problem in semi-arid tropical regions where plants are primarily rain-fed. Light sandy soils, where peanut is typically cultivated, favor these fungi. While *A. flavus* infection of peanuts does not affect yield, the fungus can produce high levels of aflatoxin in infected nuts, and these toxins can pose serious health risks to humans and animals [24]. The environmental conditions required to induce pre-harvest aflatoxin contamination of peanuts were studied. In the study, peanuts did not become contaminated with aflatoxins in the absence of severe and prolonged drought stress even when the frequency of infection by *A. flavus* and *A. parasiticus* was up to 80%. Also, larger, more mature peanut kernels required considerably more drought stress to become contaminated than did smaller, immature kernels [25]. Peanuts become infected with aflatoxigenic fungus when seed pods come into direct contact with aflatoxigenic fungus in soil. These fungi can invade and produce toxins in peanut kernels before harvest, during drying, and in storage [26].

Dried fruits can be contaminated with aflatoxins. Maximum permitted levels for total aflatoxins in the European Union are 4 μg kg^{-1} in dried fruit intended for direct human consumption. AFB1 was the most common mycotoxin encountered as a natural contaminant in stored samples [25].

Food Type	Country	Contaminated/ Total examined	Aflatoxin	Concentration (ppb)	Method	Reference
Peanut	Argentina	2/50	AFB1 AFG1	435-625 for AFB1 83-625 for AFG1	TLC	[19]
Peanut	Botswana	94/120	Total[a]	12-329	ELISA	[20]
Peanut	China	2/16	AFB1+AFB2	1.96 (mean)	UHPLC	[21]
Peanut	Malaysia	11/13	AFB1	1.47-15.33	ELISA	[27]
Peanut	China	15/65	Total[a]	0.03-28.39	HPLC	[28]
Fresh Peanuts	China	14/35	Total[a]	0.3-7.4	UHPLC-MS/MS	[10]
Musty Peanuts	China	5/5	Total[b]	1.2-1482	UHPLC-MS/MS	[10]
Peanut and products	Trinidad	0/186	AFB1	ND[c]	Charm II	[29]
Peanut and products	Brazil	41/80	Total[a]	43-1099	TLC	[30]
Peanut butter	China	41/50	AFB1	<LOD[d]-68.51	HPLC	[31]

Peanut butter	Sudan	120/120	AFB1	17-170	Fluorometer	[32]
Peanut butters	China	31/33	Total[e]	0.7-96	UHPLC-MS/MS	[10]
Pistachio	Turkey	48/95	Total[a]	0.007-7.72	HPLC	[33]
Pistachio	Iran	3699/10068	AFB1	5.9 ± 41.7 (Mean)	HPLC	[34]
Hazelnut	Egypt	18/20	Total[a]	25-175	TLC	[35]
Walnut	Egypt	15/20	Total[a]	15-25	TLC	[35]
Walnut	China	31/48	Total[a]	0.02-1.20	HPLC	[28]
Hazelnut	Turkey	43/51	Total[a]	<0.625-10	ELISA	[36]
Cacao hazelnut cream	Turkey	38/40	Total[a]	<0.625-10	ELISA	[36]
Unsorted, in-shell almonds	California, USA	10/74	Total[a]	1-107	TLC	[37]
Diced almonds	California, USA	13/27	Total[a]	<LOD-119	TLC	[37]
Sesame	Malaysia	7/8	AFB1	0.54-1.82	ELISA	[27]
Sesame paste	China	37/100	AFB1	<LOD-20.45	HPLC	[31]
Sunflower	Malaysia	6/7	AFB1	1.14-5.33	ELISA	[27]
Nuts	Malaysia	2/7	AFB1	0.66-1.09	ELISA	[27]
Pine nut	China	2/12	Total[a]	0.19-0.25	HPLC	[28]
Dried apricot	Iran	9/30	AFB1	"/0.2	HPLC	[38]
Prune	Iran	2/15	AFB1	"/0.2	HPLC	[38]

a. Total: AFB1+ AFB2+ AFG1+ AFG2

b. Total AFs including AFM1 (ND-64.7 ppb) and AFM2 (ND-3.6 ppb)

c. ND: Not detected

d. LOD: Limit of detection

e. Total AFs including AFM1 (ND-4.2 ppb) and AFM2 (ND-1.8)

Table 1. Aflatoxins in oilseeds.

3. Occurrence of Aflatoxin in Cereals

Cereals and its products are the main foods for human consumption throughout the world. The cereal grains belong to corn, rice, barley, wheat and sorghum are found susceptible to AFs accumulation by aflatoxigenic fungus. The problem of aflatoxins occurring naturally in cereals, especially in rice and corn, has become troublesome because of changing agricultural technology. The aflatoxin problem in cereals is not restricted to any geographic or climatic region. Toxins are produced on cereals, both in the field and in storage; they involve both the grain and the whole plant [39].

Our results showed that rice was significantly more heavily colonized by aflatoxin-producing *Aspergillus* spp. than other cereals, with overall aflatoxin levels being correspondingly higher. But this may be caused by the variations in cultivars used. Additionally, corn is the second susceptible after rice for aflatoxin accumulation by *A. fungus* (Table 2).

Rice and sorghum are the most important staple food crops in many countries. In these countries, the majority of rice is grown in the rainy season. During the rainy season, sun drying of rice, which is practiced by most farmers, may not reduce the moisture content of grains sufficiently to prevent growth of fungi. As a result, rice grains with a moisture content higher than the desired level (>14%) may enter the storage system. The harmful effects of such fungal invasion are discoloration of the grain and/ or husk, loss in viability, loss of quality, and toxin contamination. Sorghum is grown in harsh environments where other crops do not grow well. Improvements in production, availability, storage, utilization, and consumption of this food crop will significantly contribute to the household food security and nutrition of the inhabitants of these areas. Sorghum is typically harvested as early as possible so that fields can quickly be planted with another crop. Sometimes the sorghum harvest coincides with heavy rainfall, hurricanes, and floods, all of which promote infection by mycotoxin-producing fungi [25].

As it is known, the consumption of large amounts of AFs contaminated food by starving people can cause toxic hepatitis (jaundice) and death. Aflatoxin epidemics were reported from India, in 1975 among the Bhils (the largest and most widely distributed tribal group in India), who had consumed corn heavily contaminated with *A. flavus*. The epidemic was characterized by jaundice, rapidly developing ascites, and portal hypertension. Approximately 400 persons were affected by the epidemic [40].

Food Type	Country	Contaminated/ Total examined	Aflatoxin	Concentration (ppb)	Method	Reference
Rice	India	814/1200	AFB1	0.1-308	ELISA	[25]
Rice	India	581/1511	AFB1	"/5	HPTLC	[41]
Rice	Iran	59/71	Total[a]	2.097-10.94	HPLC	[42]
Rice	Canada	99/199	AFB1	<0.002-7.1	HPLC	[43]

Rice	Sweden	57/99	Total[a]	0.1-50.7	HPLC	[44]
Rice	China	23/74	Total[a]	0.15-3.88	HPLC	[28]
Rice	Japan	0/48	Total[a]	ND[b]	HPLC	[45]
Rice	Nigeria	21/21	Total[a]	27.7-371.9	HPLC	[46]
Rice	Turkey	56/100	Total[a]	0.05-21.4	ELISA	[47]
Rice	Tunisia	2/16	Total[a]	2.0-7.5	ELISA	[48]
Rice	Tunisia	0/11	Total[a]	ND[b]	HPLC	[49]
Rice	Malaysia	9/13	AFB1	0.68-3.79	ELISA	[27]
Corn	Tunisia	1/17	Total[a]	0.42	HPLC	[49]
Corn	Malaysia	6/8	AFB1	1.75-8.95	ELISA	[27]
Corn	Brazilian	82/214	AFB1	0.2-129	ELISA	[50]
Corn	China	4/18	Total[a]	5.67 (mean)	UHPLC	[21]
Corn	China	52/84	Total[a]	0.02-1098	HPLC	[28]
Corn	Tunisia	9/21	Total[a]	2.9-12.5	ELISA	[48]
Corn flour	Morocco	16/20	AFB1	0.23-11.2	HPLC	[51]
Wheat	Ethiopia	5/120	AFB1	<1.0-12.3	HPLC	[52]
Wheat	Tunisia	15/51	Total[a]	4.0-12.9	ELISA	[48]
Wheat	Tunisia	10/46	Total[a]	0.15-18.6	HPLC	[49]
Wheat	Malaysia	9/14	AFB1	0.55-5.07	ELISA	[27]
Wheat	India	663/1646	AFB1	"/5	ELISA	[41]
Wheat flour	Morocco	3/17	AFB1	0.03-0.15	HPLC	[51]
Sorghum	Tunisia	36/49	Total[a]	0.4-25.8	HPLC	[49]
Sorghum	Ethiopia	5/82	AFB1	<1.0-25.9	HPLC	[52]
Sorghum	India	1173/1606	AFB1	0.01-263.98	ELISA	[53]
Sorghum	Tunisia	13/17	Total[a]	1.7-67.0	ELISA	[48]
Barley	Tunisia	2/46	Total	0.6-0.8	HPLC	[49]
Barley	Ethiopia	13/115	AFB1	<1.0-11.7	HPLC	[52]
Barley	Tunisia	11/25	Total	3.5-11.5	ELISA	[48]
Oat	Malaysia	5/10	AFB1	0.65-2.85	ELISA	[27]
Cereals	Ethiopia	31/352	AFB1	<LOD-26	HPLC	[52]

a. Total: AFB1+ AFB2+ AFG1+ AFG2

b. ND: Not detected

Table 2. Aflatoxins in cereals.

4. Occurrence of Aflatoxin in Spices

The popularity of hot peppers (*Capsicum annuum* L.), also known as chili peppers, as spices or vegetables and for other uses increases every year. Powdered red pepper is one of the favorite spices in South Asia and is commonly used for flavoring, seasoning, and imparting aroma or coloring to foods. Hot peppers are the principal component of curry and chili powder and can be used to make pepper sauce, red pepper, and paprika [54].

Spices are often contaminated with mycotoxins (Table 3). The climatic conditions prevailing in the tropics are especially favorable for mold contamination and mycotoxin production. Of the different mycotoxins, aflatoxin is the commonest contaminant in spices [55].

Peppers are very susceptible to aflatoxin contamination, which is affected by atmospheric temperature, humidity, insects, and drying and processing conditions. Mold contamination can occur in the field during crop production and during storage when conditions are favorable. Sun drying is a common postharvest practice in some countries, which involves spreading peppers on soil in a single layer. Because of the drying processes are on the soil, some peppers are contaminated with fungus [54, 56].

Substantial aflatoxin contamination of ground red pepper has been reported from Ethiopia, where eight of 60 samples collected from markets, shops and storage facilities were contaminated with AFB1 in concentrations of 250-525 μg kg^{-1} [57]. In Turkey, 24% and 13% of samples of different pepper types contained 7.5-200 [58] and 1.1-97.5 μg kg^{-1} [56] total aflatoxins, respectively. Elshafie et al. [59] were surveyed one hundred and five samples of seven spices (cumin, cinnamon, clove, black pepper, cardamom, ginger, and coriander) for the mycoflora and AFs in the Sultanate of Oman. Twenty fungal species were isolated in which *A. flavus, A. niger. Penicillium, Rhizopus,* and *Syncephalastrum racemosum* were the most dominant. Of the seven spices studied, clove was found to be the least contaminated, while cumin was the most contaminated. None of the 15 selected samples of the spices contaminated by *A. flavus* were found to contain AFs [59]. Cooking revealed that the aflatoxin content of spices did not decrease [60].

Although the mold *A. flavus* grows well on the spices, the production of AFs is lower than in cereals. This indicates that spices are not an ideal substrate for AF formation. It has been shown that essential oils extracted from spices, e.g. cloves, can inhibit mold growth and AF production completely. According to some reports, fungal growth was weak on curcumin, black pepper and white pepper and no AF was detected in black or white pepper after 10 days at 25 °C. According Martins et al. [61], the results of the survey indicate that there is little evidence for significant AF contamination in spices. The majority of samples contained AFs at low levels and others were negative (cardamom, cloves, ginger and mustard). In the meat industry (sausage, dry cured ham, luncheon meat) and in confection of ethnic dishes, AF contamination of spices is probably not relevant as a direct health hazard [61].

Food Type	Country	Contaminated/ Total examined	Aflatoxin	Concentration (ppb)	Method	Reference
Pepper	Turkey	12/90	Total[a]	1.1-97.5	TLC	[56]
Pepper	Turkey	12/49	Total[a]	7.5-200	TLC	[58]
Pepper	Malaysia	4/4	AFB1	0.65-2.1	ELISA	[27]
Ground red pepper	Turkey	33/164	Total[a]	"/5	HPLC	[33]
Ground red pepper	Ethiopia	8/60	AFB1	250-525	ELISA	[57]
Paprika	Spain	8/17	Total[a]	1.8-50.4	HPLC	[62]
Smoked Paprika	Spain	4/4	Total[a]	22.3-83.7	HPLC	[62]
Chilli	Malaysia	8/8	AFB1	0.58-3.5	ELISA	[27]
Chilli	Spain	6/11	Total[a]	1.9-65.7	HPLC	[62]
Cumin	Malaysia	2/3	AFB1	1.89-4.64	ELISA	[27]
Spices	Hungary	23/91	AFB1	0.14-15.7	HPLC	[55]
Spices	Portuqual	34/79	AFB1	1.0-59.0	HPLC	[61]

a. Total: AFB1+ AFB2+ AFG1+ AFG2

Table 3. Aflatoxins in spices.

5. Occurrence of Aflatoxin in Milk and Dairy Products

Most of AFB1 and AFB2 ingested by mammals are eliminated through urine and faeces, however a fraction is biotransformed in the liver and excreted together with milk in the form of AFM1 and AFM2, respectively. AFM1 could be detected in milk 12-24 h after the first AFB1 ingestion, reaching a high level after a few days. The ratio between AFB1 ingested and AFM1 excreted has been estimated to be 1-3% [5].

The system responsible for the biotransformation of AFB1 basically has five mechanisms, represented by reactions of reduction, hydration, epoxidation, hydroxylation and ortho-demethylation. The aflatoxicol is produced by reduction of AFB1 by an NADPH-dependent cytoplasmic enzyme present in the soluble fraction of the liver. The toxicity of aflatoxicol is apparently much smaller than AFB1, but the conversion is reversible and the aflatoxicol can serve as a reservoir toxicity of AFB1 in the intracellular space, it can be converted in this mycotoxin by microsomal dehydrogenase. The aflatoxicol can also be metabolized to AFM1 and AFH1. The hydration process results in a metabolite AFB2a. This compound has the main action the inhibition of enzymes, in the liver and other tissues, causing a reduction in proteic synthesis AFM1 and AFQ1 are results of hydroxylation reaction

of AFB1. These compounds have a hydroxyl group, allowing their conjugation with glucuronic acid, sulfate and glutathione, making them very water-soluble substances that can be excreted in the bile, urine and milk. Most of the aflatoxins are excreted between 72 to 96 h after the exposure, with the liver and the kidney retaining the waste for a longer period compare to other tissues [63].

A tolerable daily intake of 0.2 ng kg^{-1} b.w. for AFM1 was calculated by Kuiper-Goodman [64] and this toxin has been categorized by the International Agency for Research on Cancer (IARC) as a class 2B toxin, a possible human carcinogen. In the assessment of cancer risk, the infants are more exposed to the risk because the milk is a major constituent of their diet. It must be also considered that young animals have been found to be more susceptible to AFB1 (and so probably AFM1) than adults. Therefore the presence of AFM1 in milk and milk products is considered to be undesirable [65].

The carcinogenicity of AFM1 may be influenced by the duration and level of exposure. Exposure is most likely to occur through the frequent consumption of milk and milk by-products (infant milk, cheese, butter, yoghurt). Several studies in different countries have reported high or low contamination levels of AFM1 in different categories of milk and dairy samples. These significantly variable AFM1 levels may be due to several influencing factors such as cheese manufacturing procedures and storage, types of cheese, conditions of cheese ripening, analytical methods and finally the geographical and seasonal effects [6].

The concentration of AFM1 is relatively increased in cheese samples because of its affinity to proteins. During cheese making, AFM1 can be decreased in cheese by increasing renneting temperature from 30 to 40°C, decreasing cutting size of curd and increasing press time from 1 to 2 h, which causes more loss of AFM1 in the whey [66]. On a weight basis, however, AFM1 concentration in cheese actually increases. In soft cheese, it becomes 2.5 to 3.3 times higher and in hard cheese, 3.9 to 5.8 times higher than in the milk from which the cheeses were made. Converting milk that may contain aflatoxin into a cheese, such as feta cheese, reduces the exposure of the consumer to this toxin. During pasteurization of milk, about 90% or more of the AFM1 is retained in the milk but during cheese manufacturing, there is a partitioning of AFM1 between the cheese, whey, and brine. During cheese manufacturing, results on the distribution of AFM1 between curd and whey can be variable. This variability has been associated with the type of cheese, the particular cheese-making process applied, the type and degree of milk contamination, and the analytical method employed. Lopez et al. [67] manufactured cheese using artificially AFM1 contaminated milk and found that the greatest proportion of toxin (60%) was in whey, while 40% AFM1 remained in cheese. Some researchers also reported that the greatest proportion of AFM1 was in the curd ranging between 66-80% [68]. About 37% of the AFM1 in milk is lost from the cheese into the whey, and another 30% diffuses from the cheese into brining solution during storage. Thus, the amount that would be ingested in a 30 g serving of cheese made from milk containing 500 ng AFM1/L would be only 35 ng AFM1 compared to 125 ng AFM1 from a 250 g serving of fluid milk. Thus, consumers in a region where there are high aflatoxin levels in milk would be at less health risk if the milk is pasteurized and converted into a cheese such as feta or other white pickled cheese before it is delivered to the consumer [69]. Applebaum et al. [70]

reported that AFM1 concentration in cheese was about four times higher than the cheese milk. The increase in AFM1 concentration in cheese has been explained by the affinity of AFM1 for casein.

The Commission of the European Communities established a limit for AFM1 of 50 ng kg^{-1} for milk and a variable limit for cheese, depending on concentration caused by drying process or processing. Milk containing AFM1 concentrations above the action level must be discarded, causing significant economic loss for the dairy producer. Similar regulations exist in most developed countries.

In this Regulation the Commission stated that ''even if AFM1 is regarded as a less dangerous genotoxic carcinogenic substance than AFB1, it is necessary to prevent the presence in milk, and consequently in milk products, intended for human consumption and for young children in particular''. The Commission has also set a limit for AFB1 of 5 μg kg^{-1} for supplementary feedstuffs for lactating dairy cattle. However this tolerance level is difficult to observe because the average daily individual intake in a herd should be limited to 40 μg AFB1 per cow, in order to produce milk with less than 50 ng AFM1 per kg [65].

Many factors may affect the formation of aflatoxins in animal feeds. Geographic and climate changes can affect the farm management practices and feed quality. These effects can lead to the wide variations in AFM1 levels in milk (Table 4). The preserved fodder such as silage and hay might have been contaminated by aflatoxin producing fungi and the improper storage led to aflatoxin production. The level of AFM1 in feed in rainy seasons is more than in dry seasons. It can be also probable to use higher amounts of contaminated concentrates in the cold months [71].

Food Type	Country	Contaminated/ Total examined	Aflatoxin	Concentration (ppb)	Method	Reference
Raw Milk[a]	Italy	125/161	AFM1	<0.023	HPLC	[72]
Raw Milk[a]	Greece	40/58	AFM1	0.005-0.055	HPLC	[73]
Raw Milk[a]	North African	35/49	AFM1	0.03-3.13	HPLC	[74]
Raw Milk[a]	Italy	?/310	AFM1	0.002-0.09	HPLC	[75]
Raw Milk[a]	Trinidad	13/212	AFM1	NM[a]	Charm II	[29]
Raw Milk[a]	Slovenia	0/60	AFM1	ND[b]	HPLC	[76]
Raw Milk[a]	Indonesia	65/113	AFM1	5-25	ELISA	[77]
Raw Milk[a]	China	12/12	AFM1	0.16-0.5	ELISA	[78]
Raw Milk[a]	Croatia	NM[a]/61 (one sample exceeded limit EU)	AFM1	0.0006-0.059	ELISA	[79]
Raw Milk[a]	Turkey	43/50	AFM1	<0.03	ELISA	[80]

Raw Milk[a]	Iran	60/60	AFM1	2.0-64.0	HPLC	[81]
Raw Milk[a]	Pakistan	177/232	AFM1	0.002-1.9	ELISA	[82]
Raw Milk[a]	Pakistan	63/120	AFM1	0.004-0.174	HPLC	[83]
Raw Milk[a]	Syria	70/74	AFM1	0.02-0.69	ELISA	[84]
Raw Milk[a]	South Korea	48/100	AFM1	0.002-0.08	HPLC	[85]
Raw Milk[a]	Portugal	25/31	AFM1	<0.005-0.05	HPLC	[86]
Raw Milk[a]	Iran	128/128	AFM1	0.031-0.113	ELISA	[87]
Raw Milk[a]	Iran	117/140	AFM1	<0.01-0.10	ELISA	[88]
Raw Milk[a]	Spain	3/92	AFM1	0.014-0.019	HPLC	[89]
Buffalo raw milk	Pakistan	153/360	AFM1	0.002-0.087	HPLC	[83]
Pasteurized milk[a]	Greece	113/136	AFM1	0.005-0.05	HPLC	[73]
Pasteurized milk[a]	Morrocco	47/54	AFM1	0.001-0.117	HPLC	[51]
Pasteurized milk[a]	Brazil	7/10	AFM1	0.01-0.02	HPLC	[90]
Pasteurized milk[a]	Iran	83/116	AFM1	0.006-0528	ELISA	[91]
Pasteurized milk[a]	Iran	624/624	AFM1	0.045-0.08	ELISA	[92]
Pasteurized milk[a]	Syria	10/10	AFM1	0.008-0.765	ELISA	[84]
Pasteurized milk[a]	Iran	48/48	AFM1	0.01-0.10	ELISA	[88]
Pasteurized milk[a]	Brazil	58/79	AFM1	0.05-0.24	HPLC	[93]
Milk[a] (Raw, pasteurized and powder)	Argentina	18/77	AFM1	0.01-0.03	ELISA	[94]
UHT Milk[a]	Greece	14/17	AFM1	0.005-0.05	HPLC	[73]
UHT Milk[a]	Turkey	75/129	AFM1	Max.0.54	ELISA	[95]
UHT Milk[a]	Turkey	67/100	AFM1	0.01-0.63	ELISA	[96]
UHT Milk[a]	Brazil	40/40	AFM1	0.010-0.5	HPLC	[90]

UHT Milk[a]	Turkey	50/50	AFM1	0.01-0.244	ELISA	[7]
UHT Milk[a]	Iran	116/210	AFM1	0.012-0.249	ELISA	[97]
UHT Milk[a]	Iran	68/109	AFM1	0.006-0.516	ELISA	[91]
UHT Milk[a]	Brazil	53/60	AFM1	0.015-0.5	HPLC	[93]
UHT Milk[a]	Iran	48/48	AFM1	0.01-0.10	ELISA	[88]
UHT Milk[a]	Turkey	14/24	AFM1	<0.01-0.05	HPLC	[98]
UHT-whole milk	Portugal	17/18	AFM1	<0.005-0.059	HPLC	[86]
UHT-semi skimmed milk	Portugal	20/22	AFM1	<0.005-0.061	HPLC	[86]
UHT-skimmed milk	Portugal	23/30	AFM1	<0.005-0.02	HPLC	[86]
UHT-Pasteurized milk	Japan	207/208	AFM1	0.001-0.029	HPLC	[99]
Ewe's milk	Greece	19/27	AFM1	0.005-0.055	HPLC	[73]
Ewe's milk	Greece	27/54	AFM1	<0.005-0.182	ELISA	[100]
Ewe's milk	Syria	13/23	AFM1	0.006-0.634	ELISA	[84]
Goat milk	Greece	12/20	AFM1	0.005-0.05	HPLC	[73]
Goat milk	Syria	7/11	AFM1	0.008-0.054	ELISA	[84]
Milk (ewe, goat and buffalo mix)	Italy	85/102	AFM1	0.05-0.25	ELISA	[101]
Infant milk food, Milk based cereal, weaning food, infant formula and liquid milk	India	76/87	AFM1	0.063-1.012	ELISA	[102]
Milk powder	Brazil	72/75	AFM1	0.01-0.5	HPLC	[90]
Milk powder	China	15/15	AFM1	Max 0.54	ELISA	[78]
Milk powder	Syria	1/8	AFM1	0.012	ELISA	[84]

Milk powder	South Korea	17/24	AFM1	0.083-0.342	HPLC	[103]
Cheese[b]	Iran	66/80	AFM1	0.15-2.41	TLC	[104]
Cheese[b]	China	4/4	AFM1	0.16-0.32	ELISA	[78]
Cheese[b]	Lebanon	75/111	AFM1	0.056-0.315	ELISA	[105]
Cheese[b]	Iran	30/50	AFM1	0.041-0.374	ELISA	[106]
Cheese[b]	Brazil	39/58	AFM1	0.01-0.304	IAC/LC	[107]
Cheese[b]	North African	15/20	AFM1	0.11-0.52	HPLC	[74]
Cheese[b]	Turkey	14/20	AFM1	<0.155	ELISA	[80]
Cheese[b]	Turkey	10/200	AFM1	0.1-0.6	ELISA	[108]
Cheese[b]	Iran	93/116	AFM1	0.052-0.745	ELISA	[109]
Cheese[b]	Turkey	82/100	AFM1	<0.05-0.8	ELISA	[110]
Cheese[b]	Turkey	36/127	AFM1	0.07-0.77	ELISA	[111]
White brined cheese[b]	Turkey	31/50	AFM1	0.1-5.2	Fluorometri	[112]
White brined cheese[b]	Turkey	159/193	AFM1	0.052-0.86	ELISA	[113]
Herby cheese[b]	Turkey	52/60	AFM1	0.16-7.26	Fluorometri	[112]
Cream cheese[b]	Turkey	44/49	AFM1	Max 0.25	ELISA	[36]
Cream cheese[b]	Turkey	8/200	AFM1	0.1-0.7	ELISA	[108]
Cream cheese[b]	Turkey	99/100	AFM1	0.01-4.1	ELISA	[114]
Cream cheese[b]	Iran	68/94	AFM1	58.3-785.4	ELISA	[109]
Kashar cheese[b]	Turkey	47/53	AFM1	"/0.25	ELISA	[36]
Kashar cheese[b]	Turkey	12/200	AFM1	0.12-0.8	ELISA	[108]
Kashar cheese	Turkey	8/28	AFM1	<0.37	ELISA	[80]
Kashar cheese[b]	Turkey	109/132	AFM1	0.05-0.69	ELISA	[96]

Kashar cheese[b]	Turkey	85/100	AFM1	0.05-0.80	ELISA	[110]
Tulum cheese[b]	Turkey	16/20	AFM1	<0.378	ELISA	[80]
Tulum cheese[b]	Turkey	81/100	AFM1	0.05-0.80	ELISA	[110]
Ewe's cheese	Turkey	14/50	AFM1	0.02-2.0	TLC	[6]
Dairy drinks	Brazil	10/12	AFM1	0.01-0.05	IAC/LC	[107]
Milk products	China	66/104	AFM1	Max 0.5	ELISA	[78]
Butter	Turkey	92/92	AFM1	0.01-7.0	ELISA	[114]
Butter	Turkey	25/27	AFM1	Max 0.1	ELISA	[36]
Butter	Turkey	66/80	AFM1	0.01-0.12	ELISA	[115]
Yoghurt	Brazil	49/65	AFM1	0.01-0.529	IAC/LC	[107]
Yoghurt	Italy	73/120	AFM1	<0.032	HPLC	[72]
Yoghurt	Turkey	68/104	AFM1	<0.1	ELISA	[116]
Yoghurt	South Korea	31/60	AFM1	0.017-0.124	HPLC	[103]
Yoghurt	Portugal	2/48	AFM1	0.043-0.045	HPLC	[117]
Fruit yoghurt	Portugal	16/48	AFM1	0.019-0.098	HPLC	[117]
Fruit yogurt	Turkey	7/21	AFM1	<0.1	ELISA	[115]
Strained yoghurt	Turkey	29/52	AFM1	<0.15	ELISA	[116]
Yogurt (whole fat)	Turkey	18/25	AFM1	<0.069	ELISA	[80]
Yoghurt (Semi fat)	Turkey	10/25	AFM1	<0.078	ELISA	[80]
Infant formula	South Korea	18/26	AFM1	0.032-0.132	HPLC	[103]
Infant formula	Iran	116/120	AFM1	0.001-0.014	ELISA	[87]
Dairy dessert	Turkey	26/50	AFM1	<0.08	ELISA	[80]

a.Cow milk, b. Cow cheese

Table 4. Aflatoxins in milk and dairy products.

6. Occurrence of Aflatoxin in Other Foods of Animal Origin

Meat refers to meat flesh, skeletal muscles, connective tissue or fat and others than meat flesh, including brain, heart, kidney, liver, pancreas, spleen, thymus, tongue and tripe that is used as food, excluding the bone and bone marrow and it contains high biological value protein and important micronutrients that are needed for good health throughout life. Residues of aflatoxins and their metabolites could be present in the meat, offal and eggs of animals receiving aflatoxin contaminated feeds (Table 5). In addition to the economic losses, aflatoxin in feeds could pose a risk to human health because of ingestion of aflatoxin containing foods derived from the animals fed the toxin-contaminated diet [118].

Cytochrome P450 enzymes (CYP) (including CYP1A2, CYP3A4 and CYP2A6) in the liver and other tissues convert AFB1 to epoxides (AFB1-8,9-exo-epoxide, and AFB1-8,9-endo-epoxide), and to AFM1, AFP1, AFQ1, and its reduced form aflatoxicol. Of the epoxides, the AFB1-8,9-exo-epoxide can form covalent bonds with DNA and serum albumin resulting in AFB1-N7-guanine and lysine adducts, respectively. Like AFB1, AFM1 can also be activated to form AFM1-8,9-epoxide that binds to DNA resulting in AFM1-N7-guanine adducts. These guanine and lysine adducts have been noted to appear in urine. The metabolites AFP1, AFQ1, and aflatoxicol are thought to be inactive and are excreted as such in urine, or in the form of glucuronyl conjugates from bile in feces [119].

When chicken exposed to AFB1 with contaminated rations, AFB1, AFM1, and aflatoxicol have been detected in liver, kidneys, and thigh muscles. Besides these, AFB2a has also been detected in livers of both broilers and layers on a ration contaminated with a mixture of aflatoxins [120]. In laying hens the effects of exposure to AF are a dose-dependent decrease in egg production and egg quality with increased susceptibility to salmonellosis, candidiasis, and coccidiosis. AFs and some of their metabolites can be carried over from feed to eggs in ratios ranging from 5,000:1 to 66,200:1 and even to 125,000:1, whereas in other trials no measurable residual AFB1 or its metabolites were found in eggs. These contrasting results may be ascribed to the administration of naturally contaminated feeds containing different AF with different levels of toxicity [8].

Wolzak et al. [121] have reported that tissue residues of aflatoxins were highest in kidney, gizzard, and liver (average concentration 3 μg kg^{-1} mass) when broilers were exposed for 4 weeks to a mixture of AFB1 and AFB2. After 7 days of removal of the contaminated feed, aflatoxin residues could not be detected in above tissues. Hussain et al. [120] also indicated that the elimination of AFB1 in chicken increased during longer exposure to AFB1. They fed broiler chicks on rations containing 0, 1.6, 3.2, and 6.4 μg AFB1/kg for 7, 14, or 28 day's age. After 2 to 3 days of exposure, AFB1 could be detected in livers of the birds exposed to 1.6 μg AFB1/kg and higher dietary levels of the toxin. After cessation of toxin feeding, AFB1 residues decreased in livers and muscles of all the chicks, with lower levels at 10 days post-cessation in the chicks exposed to higher toxin levels. They concluded that the residues of AFB1 in tissues increase with increase in dietary concentration of the toxin but decrease with increase in age (or after longer exposure) of broiler chicks. The elimination of AFB1 from tissues was rapid in older birds than in younger birds [120].

Poultry birds fed AF contaminated rations under experimental conditions resulted in the presence of AF residues in their edible tissues like liver and muscles. Residues of AFB1 in liver of broiler and layer birds have been reported to vary from no detection to 3.0 μg kg^{-1} by feeding 250-3310 μg kg^{-1} AFB1 for variable periods [118, 119]. The wide variations in the tissue AF residue concentration suggested that these levels might be influenced by different factors including dietary AF levels, duration of administration, age, type of the birds etc. However, effect of such factors upon concentration of AFB1 residues in poultry meat (liver and muscles) and clearance of AFB1 from the body tissues after withdrawal of dietary AF have not been adequately studied [120].

Dietary contamination of aflatoxins pose a big risk to human health including acute aflatoxicosis, Hepatocellular carcinoma, hepatitis B virus infection, growth impairment in different regions of the World particularly Asian and African countries [122]. European community and many other countries have imposed 2 μg kg^{-1} AFB1 as maximum tolerance level in human food products. Birds fed Afs, following ingestion are rapidly metabolized into nontoxic substances in the body. A rapid decrease in AFB1 residues below the tolerance limits from the muscles and liver within 3 and 7 days of withdrawal of dietary AFB1 and that it may not become a significant human health risk. However, in areas with no regulatory limits on AFB1 levels of poultry feed, the secondary exposure to aflatoxins through consumption of chicken liver and meat derived from the poultry fed AF contaminated feed may pose a risk to consumers health [123].

Food Type	Country	Contaminated/ Total examined	Aflatoxin	Concentration (ppb)	Method	Reference
Chicken Liver	Thailand	248/450	AFB1	Mean 0.6092	HPLC	[118]
Chicken Muscle	Thailand	96/450	AFB1	Mean 0.0451	HPLC	[118]
Meat	Jordan	12/50	Total[a]	0.15-8.32	HPLC	[124]
Fresh Fish	Egypt	10/30	Total[a]	22-70.5	Florometric	[125]
Salted fish	Egypt	12/30	Total[a]	18.5-50	Florometric	[125]
Smoked Fish	Egypt	8/30	Total[a]	32-96	Florometric	[125]
Egg	Jordan	5/40	Total[a]	0.01-6.15	HPLC	[124]
Processed egg	Saudi Arabia	0/25	Total[a]	ND[b]	ELISA	[126]
Unwashed egg	Saudi Arabia	3/25	Total[a]	0.61-1.19	ELISA	[126]

a. Total: AFB1+ AFB2+ AFG1+ AFG2

b. ND: Not detected

Table 5. Aflatoxins in other foods of animal origin.

7. Conclusion

AFs are toxic secondary metabolites produced by *Aspergillus* fungus growing in susceptible agricultural commodities. They can result in major economic losses and can negatively affect animal and human health. This review has sought to summarize the possible AFs contamination in a wide array of agricultural commodities worldwide. AFs contamination can occur both in temperate and tropical regions of the World. Major food commodities affected are cereals, nuts, dried fruit, spices, oil seeds, dried peas and beans and fruit. Regulations for major mycotoxins in commodities and food exist in at least 100 countries, most of which are for aflatoxins, maximum tolerated levels differ greatly among countries [27].

Frequent analytical surveillance program by food control agencies is highly recommended to control the incidence of aflatoxins contamination in food grains to ensure food safety and to protect consumer's health [27]. Some analytical techniques such as thin-layer chromatography (TLC), high performance liquid chromatography (HPLC), two-dimensional thin layer chromatography and enzyme-linked immunosorbent assay (ELISA) have been available for the qualitative and quantitative analysis of AFs. Poor separation, unsatisfied accuracy and low sensitivity limit the application of TLC. Although ELISA is a fast and sensitive method for AFs analysis is liquid chromatography combined with fluorescence detection, which has been extensively studied in various food matrices. However, conventional approach by HPLC in a gradient reversed phase mode typically using columns with 6 μm particles often costs a lot of time to get a complete separation of the target compounds and additionally, in order to improve detection limits of AFB1 and AFG1 a tedious pre- or post- column derivatization must be done [10].

The inability to control and at times even predict AF production makes it a unique challenge to food safety. To avoid aflatoxin problem in food grains, farmers should improve the practice of drying seeds to the required moisture content immediately after harvest. They must also develop proper storage structures by spraying fungicides or some other chemicals to reduce Aspergilli and subsequent toxin accumulation on food grains under storage conditions. Although prevention is the best control strategy, it is not always possible to prevent all mycotoxin contamination. Optimal postharvest storage conditions will minimize consumer exposure to AFs, but decontamination procedures may be needed in some cases. One approach to managing the risks associated with AF contamination is use of an integrated system based on the Hazard Analysis and Critical Control Point (HACCP) approach. This approach involves strategies for prevention, control, good manufacturing practices, and quality control at all stages of production, from the field to the final consumer [25]. Cheap and environmentally sustainable methods that can be applied pre or post-harvest to reduce the contamination of AFs are available. These methods include proper irrigation, choice of genetically resistant crop strains and bio-pesticide management which involves using a non-aflatoxigenic strain of *Aspergillus* that competitively excludes toxic strains. Other methods include sorting and disposal of visibly moldy or damaged seeds, reducing the bioavailability of aflatoxins using clay and chemo-protection [16].

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