Aflatoxins: An Overview

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Abstract

Aflatoxins are highly toxic and carcinogenic secondary metabolites produced by *Aspergillus flavus*, *A. parasiticus*, and less commonly by other *Aspergillus* species. When ingested, aflatoxins can cause liver necrosis, acute toxicity, cancer, or even death. Aflatoxins are associated with hepatic cell carcinoma, malnutrition, impaired growth and immune suppression, and reproductive health problems. Aflatoxins have been detected in many foods, but the most important contamination has been in maize, peanuts, cottonseed, and tree nuts. Species of aflatoxigenic *Aspergillus* are present and survive in many environments beyond fields. They are found in soil, a wide variety of agricultural equipment, storage areas, processing plants, distribution outlets, and household environments. Maximum allowable levels of aflatoxins in food for humans and feed for animals have been established in most countries. Aflatoxins are detected mostly by analytical methods based on thin layer chromatography, high-performance liquid chromatography, enzyme-linked immunosorbent assay, immunoaffinity with fluorescence, and/or liquid chromatography–mass spectroscopy. Different systems to prevent and control the growth of *Aspergillus* and subsequent aflatoxin production have been developed. Avoiding contamination is most convenient at the preharvest stage. The use of resistant crops; good agricultural practices; use of fungicides, pesticides, and insecticides; harvesting at the proper stage of maturity; and reduced drought stress help to prevent aflatoxin contamination. Unfortunately, these mechanisms to control contamination have not been able to completely eliminate these metabolites from food and feed. Therefore, "last step" approaches to prevent intoxication with these metabolites need to be applied to reduce or prevent their bioavailability.

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Aflatoxins are highly toxic and carcinogenic secondary metabolites produced by the naturally occurring fungi *Aspergillus flavus*, *A. parasiticus,* and less commonly by *A. nomius*, *A. pseudotamarii*, *A. bombysis,* and *A. parvisclerotigenus* in grains, nuts, oil seeds, dried fruits and vegetables, and medicinal herbs (Bathnagar and García, 2001). Aflatoxin contamination significantly reduces the value of foods and feeds and poses health hazards to humans and domestic animals (WHO-IARC, 2002).

The word "aflatoxin" derives from "A" for *Aspergillus*, "fla" for the species *flavus*, and "toxin" for poison. Aflatoxins were first isolated and characterized in 1963 after the consumption of mold-contaminated peanut meal caused the deaths of more than 100,000 young turkeys (turkey X disease; Blout, 1961). Numerous varieties of aflatoxin exist, with the most potent being aflatoxin B_1. Aflatoxins are composed of dihydrofuran or tetrahydrofuran moieties fused to a coumarin ring. The four major aflatoxins are B_1 (AfB_1), B_2 (AfB_2), G_1 (AfG_1), and G_2 (AfG_2), which are each distinguished by the color of their fluorescence (B, blue; G, green) and relative migration distance (1, higher; 2, lower) of the compounds as seen on a thin-layer chromatographic plate under ultraviolet light. *Aspergillus flavus* and *A. pseudotamarii* generally produce only AfB_1 and AfB_2, whereas *A. parasiticus* and *A. nomius* produce aflatoxins AfB_1, AfB_2, and AfG_1 (Bathnagar and García 2001). Not all isolates of *A. flavus* produce aflatoxin, whereas almost all isolates of *A. parasiticus* produce aflatoxins.

AfM_1 and AfM_2, which are hydroxylated metabolites of AfB_1 and AfB_2, are frequently found in the milk of dairy cattle or lactating mothers. These compounds are of concern because of their potential hepatoxic and immunotoxic effects on infants and children. Mammals that ingest AfB-contaminated foods or feeds excrete the aflatoxin metabolite (AfM) into milk. Small quantities of AfM_1 have also been detected in eggs.

We will provide an overview of the important aspects of aflatoxins, from the producing organisms, their characteristics, and factors influencing their growth and mycotoxin production to their effects on humans and animals, including those factors that contaminate food and feed.

Aflatoxigenic *Aspergillus*: The Organism and Its Characteristics

The genus *Aspergillus* belongs to the phylum Ascomycota and the family Trichomaceae. It consists of about 185 species of ubiquitous molds that are widely distributed in the environment, some of which cause human diseases.

Aspergillus flavus is the name used to describe both a species and a group of closely related species. *A. flavus* is the aflatoxigenic fungus that is most widely distributed in tropical and subtropical zones of the world. This fungus is primarily a saprophytic organism with the ability to grow on a variety of substrates or other organisms, a result of the numerous enzymes the fungus contains (Bathnagar and García, 2001). Proteinases degrade protein substrates, such as elastin in mammals and insects; endo-polygalacturonase has been associated with plant pathogenesis;α-amylase digests starch and may play a critical role in the induction of aflatoxin biosynthesis; and several others, such as lipases, cutinases, and xylanases, are involved in fungi metabolism. *Aspergillus flavus* can grow on either complex protein substrates or complex carbohydrate substrates such as elastin, mucin, cellulose, chitin, pectin, or xylan. This allows *A. flavus* to have a wide range of hosts, including humans, animals, and plants.

Aspergillus flavus and *A. parasiticus* are ubiquitous in hot, humid environments where mean temperatures are about 27°C and the relative humidity ranges between 80 and 90%. *A. flavus* survives at higher temperatures than *A. parasiticus* and is the predominant pathogen in arid region, including the Middle East, Africa, and Southeast Asia. The optimal temperature for *A. flavus* is 37°C, but it can grow at temperatures ranging from 12 to 48°C. Such a broad temperature range contributes to its pathogenicity in humans.

Species of *Aspergillus* are found in soils of various regions of the world. In soil, *Aspergillus* can exist as fungal hyphae, spores (conidia or ascospores), or sclerotia. Species of *Aspergillus* are differentiated by morphological characteristics and color. Colonies are flat, granular, downy to powdery in texture, and often have radial grooves (Fig. 1–1). The colony surface is yellow initially but turns dark yellowish green with age. Conidia are asexual reproductive structures. The conidiophores (also known as the stalk) are specialized fungal hypha that produce conidia. These originate perpendicular to the foot cell (in the supporting hyphae) and terminate in vesicles at the apex (Fig. 1–2). Generally, conidiophores are unbranched and composed of three parts: the foot cell, stipe, and vesicle. The outer surface of the conidiophore can vary from smooth to rough. The conidiophores of *A. flavus* (up to 800 μm long and 15–20 μm wide) are rough and uncolored. The vesicle (20–45 μm) is elongated when young and becomes globose as the culture ages (Bathnagar and García, 2001). The phialides are conidiogenous cells and may be organized as a single layer of phialides or sterigmata (uniseriate) attached to the vesicle directly or as a double layer of cells, in which the phialides are supported by metulae (biseriate). The production of phialides and foot cells is a hallmark of the *Aspergillus* species.

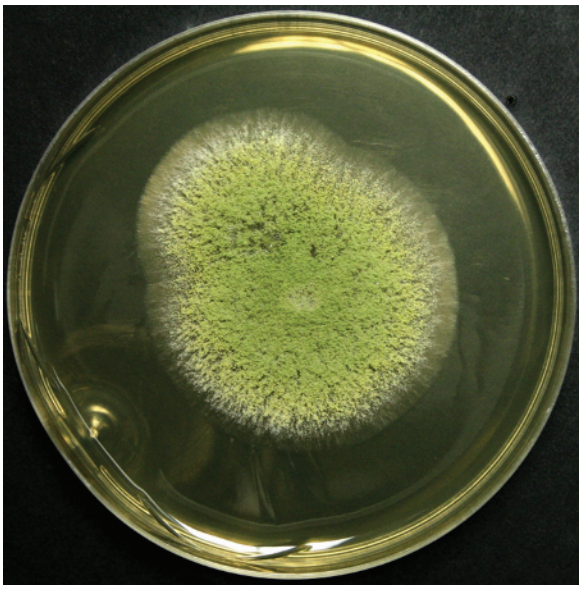

Fig. 1–1. Colony of *Aspergillus flavus* in agar.

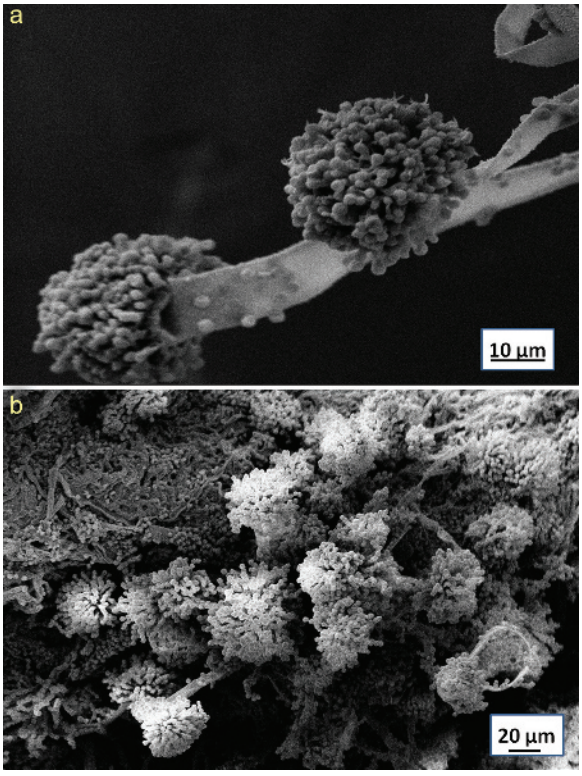

Fig. 1–2 (a) Scanning electron micrograph of conidiophores of *A. flavus* conidia. (b) Scanning electron micrograph of growth of *A. parasiticus* (Lozano-Muñiz et al., 2011). Reproduced with permission from the Journal of Food Agriculture and Environment.

The conidia are 2 to 5 μm in diameter and are arranged in a radial chain over the distal end of the phialides. The color of the conidial head depends on the color of the conidia. In *A. flavus*, it is light to deep yellow green or olive green.

Microscopic survival structures called sclerotia can be produced by *A. flavus* to overwinter in the soil. These are compact, hard masses of mycelia that vary in size and shape. The color of sclerotia varies from yellow to brown or black. Sclerotia are key to the identification of *A. flavus*.

Aflatoxins and Human and Animal Health

When ingested, aflatoxins can cause liver necrosis, acute toxicity, cancer, or even death. Aflatoxins are associated with hepatic cell carcinoma, malnutrition, impaired growth and immune suppression, and reproductive health problems in both humans and animals (Hedayati et al., 2007).

Aflatoxicosis is the major syndrome associated with the consumption of food highly contaminated with aflatoxins. The severity of the disease depends on the amount consumed and the frequency. Acute aflatoxicosis, which is relatively infrequent, results after high to moderate consumption; it causes fatty, pale, and decolorized livers, derangement of normal blood-clotting mechanisms resulting in hemorrhages, a reduction in the total serum proteins of the liver, accumulation of blood in the gastrointestinal canal, glomerular nephritis, and lung congestion. Chronic aflatoxicosis appears when moderate to low concentrations are consumed for a long period (Bathnagar and García, 2001; WHO-IARC, 2002). In this case, symptoms include a congested liver with hemorrhagic and necrotic zones, proliferation of the hepatic parenchyma and epithelial cells of the bile duct, congested kidneys, and occasional hemorrhagic enteritis. When low concentrations are consumed, native resistance and immune systems could be impaired (Kensler et al., 2011).

Hepatotoxicity results when aflatoxin is degraded by hepatic tissue and damages the cells. AfB_1 is degraded by transformation to its reactive form and then to a less toxic and easily excretable metabolite. Reactive intermediates interact with cell macromolecules, resulting in fatty, pale livers, moderate to extensive necrosis, and hemorrhage (Hedayati, et al 2007; Kensler, et al 2011).

AfB_1 is the most potent naturally formed known hepatocarcinogen and is classified as class 1 (carcinogenic to humans) by the International Agency for Research on Cancer (IARC). Carcinogenicity has been demonstrated in many animal species, including some rodents, nonhuman primates, and fish (Wild and Montesano, 2009; WHO-IARC, 2002).

Aflatoxin exposure through food is a significant risk factor for hepatocellular carcinoma. This disease is more prevalent in people with chronic hepatitis B virus infection (Wild and Montesano, 2009). In fact, the risk of liver cancer in humans exposed to chronic hepatitis B virus infection in combination with aflatoxin is up to 30 times greater than the risk in individuals exposed only to aflatoxin (Hedayati et al., 2007; Kensler et al., 2011; Wild and Montesano, 2009).

AfB_1 is predominantly metabolized in the liver by specific cytochrome P450 enzymes to produce an AfB_1-8,9-exo-epoxide. This compound can then bind to proteins and cause acute toxicity (aflatoxicosis) or to DNA forming a promutagenic AfB_1-N_7-guanine DNA adduct that results in G to T transversion mutations, increasing the risk of hepatocellular cancer. In addition to alkylating DNA, AfB_1 can induce the

formation of reactive oxygen species, which leads to the oxidation of DNA bases. Other toxic compounds produced by *A. flavus* are sterigmatocystin, cyclopiazonic acid, kojic acid, β-nitropropionic acid, aspertoxin, aflatrem, gliotoxin, and aspergillic acid (Hedayati et al., 2007).

Contamination of Food and Feed

Although aflatoxins are an important problem in warm regions of the world, the international trade distribution of different agricultural commodities means that every region could be affected by contaminated products. Aflatoxins have been detected in many foods of animal and plant origin, including corn meal, rice, sorghum, barley, rye, wheat, peanut, soya, peanut, cottonseed, oilseeds, pumpkin seeds, cassava, pistachio nuts, Brazil nuts, spices, cocoa, medicinal plants, bread, macaroni, copra, milk, cheese, sausage, cooked meat, and meat pies (Gourama and Bullerman, 1995). However, the most important contamination has been encountered in maize, peanuts, cottonseed, and tree nuts (Bathnagar and García, 2001).

Aspergillus flavus is frequently found in fields of maize and cottonseed, and in the case of *A. parasiticus,* with peanuts. This distribution of the fungus facilitates its invasion of plants and developing seeds or nuts before harvest, often resulting in contaminated products unfit for consumption. This close association is not seen with other crops and usually no contamination is detected at preharvest in them (Do and Choi, 2007).

Maize

Insect damage is associated with *A. flavus* infection of maize (*Zea mays* L.) grain. Because *A. flavus* is a wound pathogen, it is not surprising that insect damage often correlates with the subsequent production of mycotoxins in corn (García and Heredia, 2006). Invasion via the silks is also possible. Studies have found that drought stress often leads to cracks in the seed and provides an easy entry site for insects and fungi, thereby increasing insect activity and *A. flavus* colonization of the kernels. *Aspergillus flavus* usually colonizes the embryo tissue and the aleurone layer first, before spreading into the endosperm. This suggests that the embryo is the point of entry for *A. flavus.* The field contamination process is known to be influenced by numerous plant-stress factors, such as temperature, humidity, evapotranspiration, water availability to the crop and fungus, and many other factors that can be difficult to control .

Peanut

Peanuts (*Arachis hypogaea* L.) are susceptible to infection by both *A. flavus* and *A. parasiticus*. Although *Aspergillus* is present in peanut fields during growth of the crop, extensive invasion by *A. flavus* and contamination with the aflatoxins rarely occurs unless the fruit is damaged or the plant is subjected to environmental stresses, such as drought, soil temperatures around 30°C during the last 30 to 50 d before harvest, or insect damage.

Contamination of peanuts can occur in several ways: for example, in the field during the latter part of the growing season if considerable numbers of organisms exist in the soil; after the peanuts are dug up but before they are harvested, usually as a result of humidity caused by rain; during the transport of peanuts from the field, or during improper storage. The main source of contamination is the soil, where the fungus can grow in unharvested peanuts. Uncultivated soils usually contain very low amounts of *A. flavus*, but soils from peanut fields usually contain higher levels of conidia. Under drought stress, the number of conidia increases.

Aspergillus mainly infects developing peanuts in the soil by direct entry through the shell. This

is usually preceded by physical damage caused by insects, other microorganisms, or cracks. Although penetration through the pod is slow, once the pods have been damaged, invasion of the kernels is much more rapid. Infection is also possible through the pegs and flowers. The optimum conditions for kernel invasion are temperatures from 25 to 35°C, humidity from 85 to 99%, and kernel moisture from 20 to 30%. Kernel moisture, which is reduced during curing, is maintained below 10% for safe storage. Other factors involved in infection are overmaturity or immaturity of pods, pod injury, invasion of other fungi, and unfavorable weather conditions during the growing season.

Cottonseed

Cottonseed meal is a co-product of cottonseed oil extraction and can be used as livestock feed. The whole cottonseed and the meal provide a good supply of protein, fat, and fiber. However, aflatoxin contamination of cottonseed has long been a concern for the cottonseed industry because aflatoxins in the contaminated product can be transformed by dairy cows to aflatoxin M_1 and secreted in their milk. When cottonseed is crushed for oil, the toxin can be concentrated about twofold in the meal.

Aflatoxin contamination of cottonseed is frequently associated with *A. flavus,* which is a commensal in cotton (*Gossypium hirsutum* L.) plant infection, which occurs through the exit holes of the pink bollworm. Infection of developing bolls and the production of aflatoxins occurs following damage to the developing bolls and partial suture opening, followed by exposure to high humidity and warm temperature, either before or after harvest. Late rain, late irrigation, dew or high relative humidity, and delayed harvest are factors that can increase aflatoxin contamination of cottonseed. Daily minimum temperatures above 24°C, in combination with precipitation exceeding 2 to 3 cm, can lead to extensive aflatoxin formation.

Aspergillus flavus generally invades the boll and the seed before the boll is fully opened, and invasion is highest at an optimum temperature range of 30 to 35°C. Aflatoxin production within invaded seeds is maximal at 25 to 30°C. Accumulation of aflatoxins occurs in the seed meat before harvest, and there is little evidence to indicate that the toxin is formed in stored cottonseed.

Insect damage by the pink bollworm (*Pectinophora gossypiella* Saunder) is recognized as an important cause of infection. Lygus (*Lygus hesperus* Knight) and stink bug (*Chlorachroa sayi* Stal) have been implicated in the dissemination of *A. flavus* propagules, although insects are often well controlled in cotton crops.

Factors Affecting Pathogen Growth and Mycotoxin Production

Species of aflatoxigenic *Aspergillus* are present and survive in many environments beyond fields. They are found in soil, a wide variety of agricultural equipment, storage areas, processing plants, distribution outlets, and household environments. *Aspergillus flavus* has been isolated at all latitudes but has been found at higher frequencies in tropical, subtropical, and warm, temperate climates. Its optimal growth range is 28 to 37°C at humidities higher than 85% (Klich, 2007a,b). However, the fungus can grow at a wide range of temperatures, and some strains grow well at temperatures of 12 to 48°C or above and water potentials of –35 MPa or less (Bathnagarand García, 2001). Unlike most fungi, aflatoxigenic *Aspergillus* can survive or even multiply under conditions of high temperature and very low water. The ability of *A. flavus* to survive in these extreme conditions

allows it to outcompete other organisms for substrates in the soil or plants.

Aflatoxin formation is affected by a number of biotic and abiotic factors in the environment. The optimum temperature for aflatoxin production is between 24 and 30°C, with some variation due to the strain and substrate. On substrates such as cottonseed, shelled peanuts, rice, and maize grain, *A. flavus* and *A. parasiticus* have an optimal temperature range for aflatoxin production of 20 to 35°C. Only small amounts of toxin are produced at 10 or 40°C. *A. flavus* populations in the soil are genetically diverse and, therefore, aflatoxin-producing fungi vary widely in their aflatoxin-producing capacity. Almost all isolates of *A. parasiticus* produce aflatoxins, while the toxigenic ability of *A. flavus* varies with strain, substrate, and geographic origin (Do and Choi, 2007; Klich, 2007a,b).

Spores (conidia) of *Aspergillus* germinate across a broad range of temperatures (12–37°C). Germination can occur under low water potentials (–27 MPa), but optimal germination usually occurs at about –3 MPa. Within 3 d of germination, a conidium can form a colony capable of producing millions of conidia. Thus, under optimal conditions, *Aspergillus* can colonize substrates very quickly.

Environmental conditions in the field that favor disease development include high soil or air temperatures and drought stress. Other factors, including nitrogen stress, plant density, and insect attack, may influence aflatoxin contamination in the field (Klich, 2007a,b).

Prevention and Control

Different systems to prevent and control the growth of *Aspergillus* and subsequent aflatoxin production have been developed. Some of them are applied preharvest or postharvest. Avoiding contamination is most convenient at the preharvest stage. The use of resistant crops, good agricultural practices, use of fungicides, pesticides, and insecticides, harvesting at the proper stage of maturity, and reduced drought stress help to prevent aflatoxin contamination.

The use of crops with resistance to fungal infection can help reduce aflatoxin contamination. In maize, several resistance mechanisms have been identified: for example, the presence of resistance-associated proteins, both constitutive (but highly expressed) and induced, in resistant lines. The constitutive proteins delay fungal infection until the induced proteins that are synthetized as a result of the infection can control the disease.

The use of chemical insecticides to reduce aflatoxin contamination is another alternative, although it has disadvantages, such as the cost of multiple insecticide applications and a possible impact on beneficial insects. On the other hand, several studies indicate that maize expressing *Bacillus thuringiensis* crystal proteins exhibit lower aflatoxin levels in many cases.

In many parts of the world, maize is grown without irrigation, as a result predisposing plants to drought stress and subsequent *Aspergillus* infection. Under drought stress, the plant is not able to maintain its physiological systems, and dry-matter accumulation is reduced in seeds, often resulting in cracks that facilitate the invasion of microbes and insects. Proper irrigation reduces drought stress and the soil temperature.

After harvesting, the formation of aflatoxins can be prevented by rapid drying, proper storage conditions, and chemicals. However, these approaches are difficult to achieve, particularly for small farms, because they can greatly increase the cost of the product.

Cooking processes, such as nixtamalization and extrusion, are two procedures that

decrease the level of aflatoxins in corn. Several studies demonstrate the efficacy of the nixtamalization process in aflatoxin decontamination. This is a treatment in which Maize is cooked for 45 to 60 min in an alkaline solution 1%$Ca(OH)_2$ (slaked or hydrated lime), followed by steeping for 14 h. The cooking liquid is discarded, and the cooked kernels (nixtamal) are washed to remove the pericarp and excess lime. Typically, the nixtamal is then ground to yield masa, which is baked or fried to produce tortillas, chips, or other products (García and Heredia, 2006). Extrusion is a technology that has been adapted in many procedures in the food industry in which mixing, shearing, cooking, puffing, and drying take place in a single continuous process that has the advantages of lower energy consumption and less liquid waste.

Despite the many pre- and postharvest approaches that have been developed for the prevention and control of *Aspergillus* infection and aflatoxin contamination of food and feeds, aflatoxins are continually present at the tables of consumers and in the feed of animals. Thus, "last step" approaches to avoid intoxication have been developed, such as the use of clays to absorb aflatoxins from food and feed and to reduce or prevent its bioavailability or other simple methods, including sorting and disposal of visibly moldy or damaged seeds.

Sampling and Detection

Maximum allowable levels of aflatoxins in food for humans and feed for animals have been established in most countries. The number of countries with mycotoxin regulations rose by 30% from 1995 to 2003, significantly so in Africa and Asia/Oceania. These countries have at the very least regulatory limits for AfB_1 or the sum of AfB_1, AfB_2, AfG_1, and AfG_2 in foods and/or feeds. Unfortunately, in many developing countries the imposed regulatory limits have little effect on aflatoxin exposure, since most of the crops are grown by small farmers for their own consumption (FAO, 2004).

The upper bound for the sum of aflatoxins ranges from 0 to 35 $\mu g\ kg^{-1}$, with a median limit of 10 $\mu g\ kg^{-1}$. Twenty-nine countries have a limit of 4 $\mu g\ kg^{-1}$, which is the most common. The U.S. FDA cap is 20 $\mu g\ kg^{-1}$ for human food. In the European Union, the level is 4 $\mu g\ kg^{-1}$, but more recently, ceilings of 0.10 $\mu g\ kg^{-1}$ for AB_1 and 0.025 $\mu g\ kg^{-1}$ for AM_1 have been set for infant foods. In India, the regulatory limit is 30 $\mu g\ kg^{-1}$ for all foods.

Regulations for AM_1 are in place in more than 60 countries, most of which (including many in the European Union, Africa, Asia, and Latin America) have a limit of 0.05 $\mu g\ kg^{-1}$, although many countries, including the United States, have a limit of 0.5 $\mu g\ kg^{-1}$ (FAO, 2004; van Egmond and Jonker, 2004). Since the amount of AM_1 in milk is directly related to the that of AfB_1 in animal feed, it is important to control the quantity of the latter in feeds. In the United States, upper limits of 20 to 300 $\mu g\ kg^{-1}$ of aflatoxins are set for feeds, depending on the animal and maturity status (FAO, 2004; van Egmond and Jonker, 2004). (Table 1–1).

Aspergillus and aflatoxins are not evenly distributed throughout food and feed. For example, in peanuts, aflatoxin may be concentrated in only a few kernels. Therefore, an appropriate sampling procedure is necessary to get an estimate of toxin contamination.

Aflatoxins are detected mostly by several analytical methods based on thin layer chromatography (TLC, one of the earliest methods used to detect aflatoxins), high-performance liquid chromatography (HPLC), enzyme-linked immunosorbent assay (ELISA), immunoaffinity with fluorescence, and/or liquid chromatography–mass spectroscopy (LC/MS), although other

Table 1–1. U.S. upper limits of aflatoxins set for feeds for various animals and their maturity status.†

Animal feeds	Action level (ppb)
Corn and peanut products intended for finishing (i.e., feedlot) beef cattle	300
Cottonseed meal intended for beef, cattle, swine, or poultry (regardless of age or breeding status)	300
Corn and peanut products intended for finishing swine of 100 pounds or greater	200
Corn and peanut products intended for breeding beef cattle, breeding swine, or mature poultry	100
Corn, peanut products, and other animal feeds and feed ingredients, but excluding cottonseed meal, intended for immature animals	20
Corn, peanut products, cottonseed meal, and other animal feed ingredients intended for dairy animals, for animal species or uses not specified above, or when the intended use is not known	20

† Modified from http://www.fda.gov/ICECI/ComplianceManuals/CompliancePolicyGuidanceManual/ucm074703.htm (accessed 2013).

methods, such as capillary electrophoresis, may be used, but primarily for research purposes. These methodologies vary from time-consuming use of toxic solvents to commercially rapid, simple assays. Most of these methods are based on the fluorescence properties of these compounds, and most require the extraction of aflatoxin from food or feed, followed by cleaning to avoid interference from other compounds, and a concentration step (Do and Choi, 2007).

The detection of aflatoxin by HPLC is one of the most commonly used and practical methods. It requires prior extraction of aflatoxins by aqueous mixtures of organic solvents such as methanol, dichloromethane, acetone, or acetonitrile. Commercial ELISA methods are available from various companies. Aflatoxin in cereals and feed is detected and quantified using microtiter plates after extraction, filtration, and dilution. Other simple commercial methods involve monoclonal antibody-based affinity chromatography to isolate AfB_1, AfB_2, AfG_1, and AfG_2 from feed, food, grains, and nuts and AM_1 from dairy products, and their content is determined with a fluorometer. These columns are also used as a cleanup step for many HPLC analyses. Other commercial, lateral-flow-based strip tests detect and quantify aflatoxin in feed and grain very simply, which avoids the need for a highly trained technician.

Conclusion

Aflatoxigenic *Aspergillus* is a common contaminant of soils and crops. It is well known that aflatoxins can cause liver necrosis and acute toxicity and are associated with hepatic cell carcinoma. Other effects include malnutrition, impaired growth and immune suppression, and reproductive problems. In spite of the known toxicity of aflatoxins, the consumption of contaminated food and feed is still an important cause of significant health problems throughout the world. Because many factors influence the growth of pathogens and mycotoxin production pre- and post-harvest, mechanisms to control contamination by aflatoxins have not been able to completely eliminate these metabolites from food and feed. Thus, aflatoxins are continually present at the tables of consumers and in animal feed; therefore, “last step” approaches to prevent poisoning with these metabolites need to be applied to reduce or prevent their bioavailability.

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