Aflatoxins are toxic metabolites produced by certain fungi in/on foods and feeds. They are probably the best known and most intensively researched mycotoxins in the world. Aflatoxins have been associated with various diseases, such as aflatoxicosis, in livestock, domestic animals and humans throughout the world. The occurrence of aflatoxins is influenced by certain environmental factors; hence the extent of contamination will vary with geographic location, agricultural and agronomic practices, and the susceptibility of commodities to fungal invasion during preharvest, storage, and/or processing periods. Aflatoxins have received greater attention than any other mycotoxins because of their demonstrated potent carcinogenic effect in susceptible laboratory animals and their acute toxicological effects in humans. As it is realized that absolute safety is never achieved, many countries have attempted to limit exposure to aflatoxins by imposing regulatory limits on commodities intended for use as food and feed. According to the United Nations Food and Agriculture Organization (FAO), 25% of world food crops are affected, and countries that are situated between 40°N and 40°S are most at risk. Over 4.5 billion people in developing countries are at risk of chronic aflatoxin exposure.

Aflatoxins are highly toxic, mutagenic, teratogenic, and carcinogenic compounds that are produced as secondary metabolites by fungi belonging to several Aspergillus species, mainly A. flavus and A. parasiticus (Groopman et al., 1988, Romagnoli et al., 2007; O’Riordan and Wilkinson 2008). Aflatoxins have a high presence in tropical and subtropical regions where humidity and temperature conditions are optimal for toxin production. The name aflatoxin has been derived from the combination of “a” for the Aspergillus genus and “fla” for the species flavus, and toxin meaning poison (Ellis et al., 1991). Discovery of aflatoxins dates back to the 1960s following the severe outbreak of turkey “X” disease (in the other farm animals. The cause was attributed to feed (Brazilian peanuts) contaminated with A. flavus. Aflatoxins are encountered in a wide range of important agricultural commodities, including cereals (maize, sorghum, pearl millet, rice, wheat), spices (chillies, black pepper, coriander, turmeric, ginger), oilseeds (groundnut, soybean, sunflower, cottonseed), tree nuts (almond, pistachio, walnut, coconut), milk (human and animal), and butter. Until now, nearly 18 different types of aflatoxins have been identified wherein the major ones include aflatoxin B1, B2, G1, G2, and M1. Fungal species belonging to Aspergillus flavus typically produce AFB1 and AFB2, whereas A. parasiticus produces AFG1 and AFG2 as well as AFB1 and AFB2. The 4 major aflatoxins (aflatoxin B1, B2, G1, and G2) are based on their fluorescence under blue or green light and their relative mobility during separation by thin-layer chromatography (TLC) Their molecular formulae as
established from elementary analyses and mass spectrometric determinations are:

- B1 : \(\text{C}_{17}\text{H}_{12}\text{O}_{6}\)
- B2 : \(\text{C}_{17}\text{H}_{14}\text{O}_{6}\)
- G1 : \(\text{C}_{17}\text{H}_{12}\text{O}_{7}\)
- G2 : \(\text{C}_{17}\text{H}_{14}\text{O}_{7}\)

Aflatoxins B2 and G2 were established as the dihydroxy derivatives of B1 and G1, respectively. Whereas, aflatoxin M1 is 4-hydroxy aflatoxin B1 and aflatoxin M2 is 4-dihydroxy aflatoxin B2. (Stroka and Anklam, 2002; Bennett and Klich, 2003).

Four other types of aflatoxins, M1, M2, B2A, G2A, that are produced in minor amounts, have been isolated from cultures of \(A. \text{flavus}\) and \(A. \text{parasiticus}\). A number of closely related compounds, aflatoxin GM1, parasiticol, and aflatoxicol are produced by \(A. \text{flavus}\). Aflatoxin-producing fungi show wide variations in their growth requirements. For example, the minimum temperature range for growth of \(A. \text{parasiticus}\) is 6 to 8\(\degree\)C and maximum is 44 to 66\(\degree\)C, optimum being 25 to 35\(\degree\)C (Diener et al., 1982), while \(A. \text{flavus}\) can produce toxin between 12 and 42\(\degree\)C and its optimum is 28 to 30\(\degree\)C (Brackett, 1989). Presently, it is estimated that human consumption of aflatoxins ranges between 0 and 30,000 ng/kg/d with an average intake of 10 to 200 ng/kg/d (Revankar, 2003). The maximum acceptable levels of AFB1 in cereals, peanuts, and dried fruits, either for direct human consumption or as an ingredient in foods, has been set by the European Committee Regulations (ECR) as 4 ppb for total aflatoxins (AFB1, AFG1, AFB2, and AFG2) and 2 ppb for AFB1 alone (Moss, 2002; Stroka and Anklam, 2002). Out of the nearly 18 different types of aflatoxins identified to date, the Intl. Agency for Research on Cancer (IARC) has classified 4 aflatoxins (AFB1, AFG1, AFB2, and AFG2) as Group 1 carcinogens (Chiavaro et al., 2001).

Imwidthaya et al. (1987) has reported percentage of aflatoxin contamination in various food products in Thailand.

**Percentage of aflatoxin contamination in various food products in Thailand:**

<table>
<thead>
<tr>
<th>Food and food products</th>
<th>Contaminated samples (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rice</td>
<td>10</td>
</tr>
<tr>
<td>Peanuts</td>
<td>43</td>
</tr>
<tr>
<td>Corn</td>
<td>20</td>
</tr>
<tr>
<td>Soybean</td>
<td>25</td>
</tr>
</tbody>
</table>

Aflatoxin contamination has been detected in various foods, such as fermented food and beverages (Sripathomswat et al., 1981), milk and dairy products (Galvano et al., 2001; Srivastava et al., 2001), melon seeds (DiProssimo and Malek, 1996), and chillies (Reddy et al., 2001), and spices (Vrabcheva, 2000).

**Health risks**

**Aflatoxin poisoning (aflatoxicosis):** Consumption of foods/feeds contaminated with high levels of aflatoxins may lead to acute aflatoxicosis and regular intake, even at low levels (ppb), is reported to be responsible for stunting and loss of weight among children, and in some cases has led to the development of hepatocellular cancer (Bhat and Vasanthi, 2003; Hall and Wild, 2003). Aflatoxins have also been linked with kwashiorkor, a protein-energy malnutrition disease (Adhikari et al., 1994). Reports are available wherein AFB1 and aflatoxicol (a metabolic product of AFB1) were detected.
more frequently in the serum, liver, urine, and stools of children suffering from kwashiorkor (Apeagyei et al., 1986; Hendrickse and Maxwell 1989; De Vries et al., 1990).

The role of aflatoxins in the development of Reye’s syndrome (encephalopathy with severe lesions in kidney and liver following influenza or varicella) has never been proved, regardless of the frequent detection of aflatoxins in the liver of children who have died of this disease (Dvorackova et al., 1977; Hogan et al., 1978; Casteels-van Daele and Eggermont, 1994). Egal et al. (2005) have reported that 90% of children in West Africa (Benin and Togo) are exposed to aflatoxins due to consumption of contaminated maize and groundnuts, which leads to a measurable impairment of child growth. Severe liver lesions in malnourished adults during the 1970s, with fatal outcome have been reported after severe cases of acute aflatoxicosis in parts of Asia and Africa (Krishnamachari et al., 1975; Bhat and Krishnamachari, 1977). Aflatoxins are perceived to be co-factors in the higher incidence of liver cancer (hepatocellular carcinoma) along with hepatitis-B virus in tropical Africa (FAO, 1997). Hepatitis-B virus (HBV) interferes with the ability of hepatocytes to metabolize aflatoxins, and an aflatoxin M1-DNA conjugate exists for a longer time in the liver, increasing the probability of damage to tumor suppressor genes. This effect is synergistic with the resulting damage far greater than just the sum of aflatoxins or HBV individually (Williams et al., 2004).

The FDA tolerance level for aflatoxin in human food is 20 μg/kg; for breeding livestock feed 100 μg/kg; breeding cattle feed 20 μg/kg; and poultry feed 300 μg/kg. According to the FAO/WHO expert committee recommendations (1990) the tolerance limit for AFB1 is 5 μg/kg food products, for AFM1 it is 0.05 μg/kg milk products, and for B1+G1+B2+G2 it is 15 μg/k, as for example in raw peanuts.

**Guidelines for Grain Use**

Owing to the carcinogenic properties of this mycotoxin, the Federal Drug Administration (FDA) has established the following guidelines. The maximum allowable level of aflatoxin in feed grains for interstate commerce is 20 ppb. The maximum level of aflatoxin in a feed should not exceed 100 ppb for within state use.

**Concentration at which regulatory agencies will consider action**

<table>
<thead>
<tr>
<th>Aflatoxin</th>
<th>A</th>
</tr>
</thead>
<tbody>
<tr>
<td>Milk</td>
<td>0.5 ppb</td>
</tr>
<tr>
<td>Food for human consumption</td>
<td>20 ppb</td>
</tr>
<tr>
<td>Corn grain intended for finishing beef</td>
<td>300 ppb</td>
</tr>
<tr>
<td>Corn grain intended for finishing cattle</td>
<td>200 ppb</td>
</tr>
<tr>
<td>Corn grain intended for breeding cattle</td>
<td>100 ppb</td>
</tr>
<tr>
<td>Corn grain intended for breeding swine and mature poultry</td>
<td>50 ppb</td>
</tr>
<tr>
<td>Corn grain and other animal feeds</td>
<td>20 ppb</td>
</tr>
<tr>
<td>Corn and other animal feed ingredients</td>
<td>20 ppb</td>
</tr>
</tbody>
</table>

**Presence of aflatoxins in raw agricultural products:**

Aflatoxins often occur in crops in the field prior to harvest. Postharvest contamination can occur if crop drying is delayed and during storage of the crop if water is allowed to exceed critical values for the mold growth. Insect or rodent infestations facilitate mold invasion of some stored commodities.
Aflatoxins are detected occasionally in milk, cheese, corn, peanuts, cottonseed, nuts, almonds, figs, spices, and a variety of other foods and feeds. Milk, eggs, and meat products are sometimes contaminated because of the animal consumption of aflatoxin-contaminated feed. However, the commodities with the highest risk of aflatoxin contamination are corn, peanuts, and cottonseed.

Corn is probably the commodity of greatest worldwide concern, because it is grown in climates that are likely to have perennial contamination with aflatoxins and corn is the staple food of many countries. However, procedures used in the processing of corn help to reduce contamination of the resulting food product. This is because although aflatoxins are stable to moderately stable in most food processes, they are unstable in processes such as those used in making tortillas that employ alkaline conditions or oxidizing steps. Aflatoxin-contaminated corn and cottonseed meal in dairy rations have resulted in aflatoxin M1 contaminated milk and milk products, including non-fat dry milk, cheese, and yogurt.

**Aflatoxins in milk:** Aflatoxin contamination in milk and its products is of extreme importance and is a serious problem, as most of the human species as well as animals, particularly the young nurturing ones, are dependent on milk as a part of complete basal nutrition. Infants are particularly more sensitive to toxins than adults. The IARC (1993a) categorizes AFM1 as a possible human carcinogen. The European Communities and the Codex Alimentarius have fixed the limit of AFM1 intake to a maximum of 50 ng/kg (Anonymous 2001). Compared to AFB1, AFM1 is rather less carcinogenic and mutagenic; however, it has been reported to exhibit a high level of genotoxic activity in animals (JECFA 2001). Several reports are available wherein AFM1 has been found in milk. It has been detected in breast milk and in cord blood and maternal blood in African countries (like in Sudan, Ghana, and Kenya), the Guangi Xi region of China. Contamination of milk by AM1 might occur in 2 ways, directly due to intake of contaminated feeds by animals that might pass into the milk, or indirectly following contamination of milk and milk products with fungi (Applebaum et al., 1982; Sugiyama et al., 2008). However, it should be noted that aflatoxin M1 is a metabolite of aflatoxin B1, and therefore the possibilities of any direct carryover of AFM1 from feed to milk could be ruled out. It is generally recognized that contamination of milk and milk products with AFM1 varies according to geographical location (dry or wet) and season (hot or cold).

**Aflatoxins in raw drugs:** Even though considerable advances have been achieved in modern medicine, there has been a renewed interest in the use of traditional plant-based products for a variety of therapeutic purposes (Rates 2001). Currently, a large share in the health care market has been taken over by products based on the popularity of health foods (nutraceuticals=functional foods) of plant origin. Contamination of crude drugs of plant origin (as in Ayurvedic and Chinese medicine, and others) incurs heavy economic losses in the tropics and subtropics. The conventional methods of collection, storage, and marketing usually promote the association with several toxigenic molds. Several reports are available on aflatoxins contaminating raw drugs of plant origin, who reported levels ranging between 0.09 and 0.88 µg/mL of the culture filtrate. The researchers also reported that out of 158 isolates of A. flavus from the drug-yielding plants 49 were toxigenic in
nature and the amount of AFB1 produced by the toxigenic isolates ranged between 0.86 and 5.24 µg/mL. They examined a total of 84 medicinal plants and spices and reported 17 samples to be contaminated by AFB1 which ranged between 10 and 160 µg/kg. Ali et al., (2005) evaluated 23 commercial samples of traditional herbal medicines from Malaysia and Indonesia and found the presence of aflatoxin in most of the samples. The mean levels of AFB1, AFB2, and AFG1 in positive samples were 0.26 (70%), 0.07 (61%), and 0.10 (30%) µg/kg, respectively, and one of the samples was positive for AFG2 at a level of 0.03 (4%) µg/kg. Though these are just a few of the examples to cite, an alarming increase among consumers relying on food of plant origin, renders it a necessity to undertake safety measures against fungal contamination and mycotoxins that might be present in raw materials possessing nutraceutical value.

Aflatoxins in eggs: Consumption of egg as a rich source of protein is well known. It has been reported that AFB1 bio-transformation in the liver of hens could generate a variety of toxic hydroxylated metabolites that can be transferred to eggs. Hens that are fed with contaminated feeds with more than 3300 mg/kg of AFB1 over a period of 28 d were reported to produce contaminated eggs. Also, reports are available on the presence of aflatoxin residues transmitted into eggs. However, since 1974 the EC has set a limit of 20 µg AFB1/kg of layer feed.

Factors favoring aflatoxin production

Fungal growth and aflatoxin contamination are the consequence of interactions among the fungus, the host and the environment. The appropriate combination of these factors determines the infestation and colonization of the substrate, and the type and amount of aflatoxin produced. However, a suitable substrate is required for fungal growth and subsequent toxin production, although the precise factor(s) that initiates toxin formation is not well understood. Water stress, high-temperature stress, and insect damage of the host plant are major determining factors in mold infestation and toxin production. Similarly, specific crop growth stages, poor fertility, high crop densities, and weed competition have been associated with increased mold growth and toxin production. Aflatoxin formation is also affected by associated growth of other molds or microbes. For example, preharvest aflatoxin contamination of peanuts and corn is favored by high temperatures, prolonged drought conditions, and high insect activity; while postharvest production of aflatoxins on corn and peanuts is favored by warm temperatures and high humidity.

Recent methods of analysis for aflatoxins in foods and feeds

Sampling and sample preparation:

Sampling and sample preparation remain a considerable source of error in the analytical identification of aflatoxins. Thus, systematic approaches to sampling, sample preparation, and analysis are absolutely necessary to determine aflatoxins at the parts-per-billion level. In this regard, specific plans have been developed and tested rigorously for some commodities such as corn, peanuts, and tree nuts; sampling plans for some other commodities have been modeled after them. A common feature of all sampling plans is that the entire primary sample must be ground and mixed so that the analytical test portion has the same concentration of toxin as the original sample.

Solid-phase extraction:

All analytical procedures include three steps: extraction, purification, and
determination. The most significant recent improvement in the purification step is the use of solid-phase extraction. Test extracts are cleaned up before instrumental analysis (thin layer or liquid chromatography) to remove coextracted materials that often interfere with the determination of target analytes.

**Thin-layer chromatography:**

Thin layer chromatography (TLC), also known as flat bed chromatography or planar chromatography is one of the most widely used separation techniques in aflatoxin analysis. Since 1990, it has been considered the AOAC official method and the method of choice to identify and quantify aflatoxins at levels as low as 1 ng/g. The TLC method is also used to verify findings by newer, more rapid techniques.

**Liquid chromatography:**

Liquid chromatography (LC) is similar to TLC in many respects, including analytic application, stationary phase, and mobile phase. Liquid chromatography and TLC complement each other. For an analyst to use TLC for preliminary work to optimize LC separation conditions is not unusual. Liquid chromatography methods for the determination of aflatoxins in foods include normal-phase LC (NPLC), reversed-phase LC (RPLC) with pre- or before-column derivatization (BCD), RPLC followed by postcolumn derivatization (PCD), and RPLC with electrochemical detection.

**Immunochemical methods:**

Thin layer chromatography and LC methods for determining aflatoxins in food are laborious and time consuming. Often, these techniques require knowledge and experience of chromatographic techniques to solve separation and interference problems. Through advances in biotechnology, highly specific antibody-based tests are now commercially available that can identify and measure aflatoxins in food in less than 10 minutes. These tests are based on the affinities of the monoclonal or polyclonal antibodies for aflatoxins. The three types of immunochemical methods are radioimmunoassay (RIA), enzyme-linked immunosorbent assay (ELISA), and immunoaffinity column assay (ICA).

**Confirmation of identities of the aflatoxins:**

Although analytical methods might consist of different extraction, clean-up, and quantitation steps, the results of the analyses by such methods should be similar when the methods are applied properly. Since the reliability of the quantitative data is not in question, the problem still to be solved is the confirmation of identity of the aflatoxins. The confirmation techniques used involve either chemical derivatization or mass spectrometry (MS).

**Safety issues in handling moldy grains and aflatoxins:**

Safety is a key issue for scientists working in the aflatoxin area. Steps must be taken to minimize exposure to the toxins as well as to the toxin producing microorganisms, *Aspergillus flavus* and *Aspergillus parasiticus*. A safety program should be established that meets the requirements of the Laboratory Standard of the Occupational Safety and Health Administration (1990) and the guidelines of the National Institutes of Health (1981) covering use of chemical carcinogens.

**Monitoring techniques for assessing human exposure to aflatoxins**

In the last few years, new technologies have been developed that more
accurately monitor individual exposures to aflatoxins. Particular attention has been paid to the analysis of aflatoxin DNA adducts and albumin adducts as surrogates for genotoxicity in people. Urine samples collected after exposure to aflatoxins were found to contain 2, 3-dihydroxy-2-(N7-guanyl)-3-hydroxyaflatoxin B1, trivially known as AFB-Gual. Wild et al. (1986) used highly sensitive immunoassays to quantitate aflatoxins in human body fluids. An enzyme linked immunosorbent assay (ELISA) was used to quantitate aflatoxin B1 over the range of 0.01 ng /ml to 10 ng/ml, and was validated in human urine samples. Using this method, aflatoxin-DNA adduct excretion into urine was found to be positively correlated with dietary intake, and the major aflatoxin B1-DNA adduct excreted in urine was shown to be an appropriate dosimeter for monitoring aflatoxin dietary exposure.

CONTROL AND MANAGEMENT OF AFLATOXINS

Regulatory control:

Aflatoxins are considered unavoidable contaminants of food and feed, even where good manufacturing practices have been followed. The FDA has established specific guidelines on acceptable levels of aflatoxins in human food and animal feed by establishing action levels that allow for the removal of violative lots from commerce. The action level for human food is 20 ppb total aflatoxins, with the exception of milk which has an action level of 0.5 ppb for aflatoxin M1. The action level for most feeds is also 20 ppb. However, it is very difficult to accurately estimate aflatoxins concentration in a large quantity of material because of the variability associated with testing procedures; hence, the true aflatoxin concentration in a lot cannot be determined with 100% certainty.

Integrated aflatoxin management:

Strategies to address the food safety and economic issues employ both preharvest and post harvest measures to reduce the risk of aflatoxin contamination in food and feed. Post harvest measures, such as adequate storage, detection and decontamination or disposal as well as continuous monitoring of potential contamination during processing and marketing of agricultural commodities, have proved to be critical and indispensable in ensuring food and feed safety. However, the post harvest contamination is usually the result of preharvest presence of fungal contamination. Preharvest control includes good cultural practices, biocontrol and development of resistant varieties of crops through new biotechnologies. Processed food cannot be safe if prevention, control, good manufacturing practices and quality control are not used at all stages of production. The Hazard Analysis and Critical Control Point (HACCP) approach to processing aflatoxin contaminated commodities should be considered.

Preharvest control:

Preharvest control includes good cultural practices such as insect control, irrigation during drought conditions, planting and harvesting dates, cropping patterns etc. Pre harvest prevention especially through host resistance is probably the best and widely explored strategy for control aflatoxins. Currently new biotechnological approaches are employed for preharvest control of aflatoxin. Host resistance enhancement can be achieved through identification of germplasm resistance to aflatoxin and also identification of natural resistance
mechanisms and traits (Brown et al., 2003; Cleveland et al., 2003).

Chromosomal regions with resistance to *A. flavus* and inhibition of aflatoxin production in corn have been identified through Restriction Fragment Length Polymorphism (RFLP) (White et al., 1998). However, limiting the growth of aflatoxigenic fungi might at times not be enough to maintain aflatoxins “at acceptable” levels in corn crops. Studies of the genetics of aflatoxin biosynthetic pathway for understanding how and why this fungus makes aflatoxin have enabled scientists to examine strategies to interrupt aflatoxin synthesis, thereby, preventing aflatoxin contamination of crops. The fungal genome of *A. flavus* has been sequenced to understand the regulation of aflatoxin formation by environmental factors (Bhatnagar, 2010).

**Biocompetitive agents:**

Microbes have been suggested as an agent of control of mycotoxin contamination. *Aspergillus niger* when cultured with *A. flavus* on maize substrates suppressed aflatoxin production by lowering the pH of the substrates. Several hydrolases of *Trichoderma* were recently identified and purified. Some of the genes coding them were cloned and sequenced and transformant were obtained, which confirmed that over production of single protease or chitinase resulted in better biocontrol agent. *T. viride* was found to inhibit the production of aflatoxin B1 (73.5%) and aflatoxin G1 (100%) when cultured with *A. flavus* (Bilgrami and Choudhary, 1998). *Bacillus pumilus* is also reported to inhibit the growth and aflatoxin production by *A. flavus* to the extent of 99.2%. An active compound being produced by *B. pumilus* was identified (Sinha and Choudhary, 2008).

Aqueous plant extracts of cinnamon, peppermint, basil, origanum, epizote, clove and thyme caused total inhibition of fungal development on maize kernels and optimal dosage varies from 3 to 8 per cent. Bankole and Joda (2004) observed efficacy of lemongrass (*Cymbopogon citrates*) powder and essential oil on *A. flavus* growth and aflatoxin contamination.

**Post harvest control:**

Once crop becomes infected under field conditions, the fungal growth continues usually with increasing vigour at post harvest stage and in storage. If corn is dried below this level no additional growth of fungus or production of aflatoxin will occur if proper storage practices are followed. In addition, fungus can survive in residues left in storage and feeding facilities and thereby, produce mycotoxins under such conditions. Food and feed residues should be discarded soon and storage and feeding facilities should be decontaminated. Periodical evaluation of storage suitability be monitored with the help of CO2 - sensor. However, in most of the cases preharvest and post harvest preventive strategies have to be supplemented with control strategies viz Elimination and/or Detoxification.

CO2 sensors can be effectively used to monitor early detection of spoilage during storage (Bortosik et al., 2008; Maier et al., 2010).

**Physical separation:**

The principle of this method is based on the identification of damaged kernels in the seed lots because of variations in size, shape, colour and also visible mould growth.
Aflatoxin contaminated kernels are usually damaged, shriveled or discoloured (Natrajan et al., 1975). Another approach is through floating and density segregation. It was observed that 95% of the aflatoxin in 21 of 29 samples of peanuts was contained in kernels that floated in tap water (Kirskey et al., 1989).

**Filtration**

Aflatoxin was removed up to 90% through single filtration, but in recirculation of oil, they could achieve even up to 100% removal since aflatoxin in crude peanut oil remains in finely suspended form and can easily be separated by filtration. Basappa and Sreenivasamurthy (1979) at CFTRI, India have developed a special filter pad system which can easily be adopted in oil mills to remove aflatoxin from crude oil. This filter pad can be prepared by impregnating fuller’s earth salt slurry in between two filter cloth layers and dried completely at 100°C for eight hours.

**Milling**

In the processed products the levels of aflatoxin vary with the nature of processing, food materials and the affinity or solubility of toxin in the products. Laboratory studies have reported that during wet milling of inoculated corn, aflatoxin B1 was distributed in the milling fractions viz primarily in steep water (39% to 42%), fibre (30% to 38%), with the remainder found in gluten (13% to 17%), germ (6% to 10%) and starch (only 1%). In a good risk management plan, individual fraction should be considered for further utilization. In another investigation (CFTRI, India) it was found that 85% of the aflatoxins present in groundnut seeds goes into the cake after crushing in expeller oil mill or hydraulic press, and only 15% remained in oil (Basappa and Sreenivasamurthy, 1974).

**Solvent extraction**

Aflatoxin is soluble in polar solvent such as methanol and is insoluble in water and petroleum hydrocarbons. The extraction methods used in analysis of aflatoxins employ chloroform/water and acetone/water to remove the mycotoxins. Aqueous isopropanol has been found to be an effective solvent for removal of aflatoxins from both contaminated cottonseed and groundnuts. Six extractions with 80% aqueous isopropanol at 60°C resulted in complete removal of aflatoxin in both meals. A binary solvent system of 90% acetone and 10% water (by weight) reduced the aflatoxin content of pre pressed cottonseed and groundnut meal to less than 10µg/kg in small scale batch extractions and less than 40 µg/kg in continuous plant extractions. Removal of all aflatoxins from peanut meal by an aqueous solution of calcium chloride has also been reported (Sreenivasamurthy et al., 1971).

**Detoxification strategies:**

Because aflatoxin contamination is unavoidable, numerous strategies for their detoxification have been proposed. These include physical methods of separation, thermal inactivation, irradiation, and solvent extraction, adsorption from solution, microbial inactivation, and fermentation. Chemical methods of detoxification are also practiced as a major strategy for effective detoxification:

**Structural degradation following chemical treatment:**

A diverse group of chemicals has been tested for the ability to degrade and
inactivate aflatoxins. A number of these chemicals can react to destroy (or degrade) aflatoxins effectively but most are impractical or potentially unsafe because of the formation of toxic residues or the perturbation of nutrient content and the organoleptic properties of the product. Two chemical approaches to the detoxification of aflatoxins that have received considerable attention are ammoniation and reaction with sodium bisulfite. Many studies provide evidence that chemical treatment via ammoniation may provide an effective method to detoxify aflatoxin-contaminated corn and other commodities. The mechanism for this action appears to involve hydrolysis of the lactone ring and chemical conversion of the parent compound aflatoxin B1 to numerous products that exhibit greatly decreased toxicity. On the other hand, sodium bisulfite has been shown to react with aflatoxins (B1, G1, and M1) under various conditions of temperature, concentration, and time to form water-soluble products.

Modification of toxicity by dietary chemicals:

The toxicity of mycotoxins may be strongly influenced by dietary chemicals that alter the normal responses of mammalian systems to these substances. A variable array of chemical factors, including nutritional components (e.g. dietary protein and fat, vitamins, and trace elements), food and feed additives (e.g. antibiotics and preservatives), as well as other chemical factors may interact with the effects of aflatoxins in animals.

Alteration of bioavailability by aflatoxin chemisorbents:

A new approach to the detoxification of aflatoxins is the addition of inorganic sorbent materials, known as chemisorbents, such as hydrated sodium calcium aluminosilicate (HSCAS) to the diet of animals. HSCAS possesses the ability to tightly bind and immobilize aflatoxins in the gastrointestinal tract of animals, resulting in a major reduction in aflatoxin bioavailability.

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