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Aflatoxins in livestock and poultry: A review

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ABSTRACT

Aflatoxin, one of the mycotoxins, contaminates agricultural products and has assumed economic importance because of it influences health of humans and livestock, and marketability of agricultural products particularly in tropical countries. Since there is a risk of exposure of a large cross-section of population, especially children, because of presence of non-permissible levels of aflatoxin in animal feeds, groundnut kernels, confectioneries and in milk, therefore various methods have been described to reduce or alleviate the aflatoxins from them. Consumption of aflatoxin contaminated feed by the animals in India is very high leading to the risk of aflatoxicosis to them and to human beings through consumption of milk and meat. Hence, it is essential to follow sound production, harvesting and storage practices to control the development of aflatoxins in the feeds.

Key words: Aflatoxins, Animal feed

Aflatoxins is a group of approximately 20 related fungal metabolites, however, aflatoxins B_1, B_2, G_1 and G_2 are normally found in foods. Aflatoxins B_2 and G_2 are the dihydro derivatives of the parent compounds. Aflatoxins are produced by at least *Aspergillus flavus*, *A. parasiticus* and *A. nomius*. The optimum temperature for *A. flavus* is 28-30°C and the minimum moisture content is 8-10 %. Aflatoxins are potent carcinogenic, mutagenic, teratogenic and immunosuppressive agents (Fink-Gremmels 1999). The importance of aflatoxins in animal health emerged in 1960, following an incident in the United Kingdom in which 100 000 turkey poults died from acute necrosis of the liver and hyperplasia of the bile duct ("turkey X disease"), attributed to the consumption of groundnuts infected with *Aspergillus flavus*. This event marked a defining point in the history of mycotoxicosis, leading to the discovery of the aflatoxins. Studies showed that aflatoxins are acutely toxic to ducklings, however ruminants are comparatively more resistant. The major impetus arose from epidemiological evidence linking chronic aflatoxin exposure with the incidence of cancer in humans. In 1974, there was an outbreak of acute hepatitis in tribal areas of 200 villages of Anaswala in Rajasthan and Panchmahal district of Gujarat in India and, this disease was caused by the consumption of maize that was heavily contaminated with aflatoxins (Krishnamachari *et al.* 1975a,b).

Groundnut is easily infected with the fungus during storage and leads to production of aflatoxins. Feeding of contaminated groundnuts to humans and groundnut-cake to animals has resulted in aflatoxicosis problem. Concerns over aflatoxin contamination of Indian groundnut in both domestic and international markets restrict the access of produce by marginal farmers to these lucrative markets.

Source of aflatoxins

Many substrates support growth and aflatoxin production by aflatoxigenic molds. Sometimes crops get contaminated with aflatoxin in the field before harvest, mainly because of drought stress (Diener *et al.* 1987, Klich 1987). In storage, moisture content of substrate and relative humidity of the surroundings affect mold growth (Detroy *et al.* 1971, Wilson and Payne 1994). Milk products can also serve as an indirect source of aflatoxin as cows consuming aflatoxin-contaminated feeds, biotransform aflatoxin B_1 metabolically into a hydroxylated form called aflatoxin M_1 (Van Egmond 1989).

Level in feeds

The importance for surveillance of animal feeds for aflatoxins was realized due to their diverse forms of toxicity and also because of legislation in developed countries (D'Mello and Macdonald 1998). In the UK, analysis conducted during 1987–1990 indicated that all imported feedstuffs complied with legislation in force for aflatoxin levels. In China, 85% of maize samples were found contaminated with both aflatoxin B_1 and fumonisin B_1 at levels ranging from 8 to 68 g/kg and 160 to 25 970 g/kg,

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respectively. Feed-grade maize in northern Vietnam had aflatoxin B_1 levels ranging from 9 to 96 g/kg (Placinta *et al.* 1999).

In India, 42% of feed samples in Gujarat were found contaminated with aflatoxin (Fulsounder and Shukla 1977); 70% of groundnut-cake samples sold in Hapur market in Uttar Pradesh, were reported to be contaminated with aflatoxin B_1 (Table 1) and the levels ranged from 113-2250 ppb (Rampal *et al.* 1979). Reddy *et al.* (1984) recorded aflatoxin levels of 578, 71, 38, 43 and 23 ppb in groundnut-cake, maize, pearl millet, broken rice and rice polish, respectively. Very high levels of aflatoxin were recorded in all the feed samples by Balasubramanian (1985). Balaraman and Gupta (1990) estimated that concentration of aflatoxin was 626±240 and 881±340 µg/kg in 25.93% of feeds and 67.39% of feed mixtures, respectively. Among the feeds, 50% maize, 30% rice bran, 25% millet wastes and 13% mustard oilcake samples were positive for aflatoxin; none of the samples of finger millet and barley showed any contamination. Among the feed mixtures the incidence and concentration of aflatoxin were higher in poultry feed mixtures than that in cattle and pigs feeds. Paddy straw was not found contaminated.

A survey on the aflatoxin B_1 content in groundnut-cake and cattle feeds revealed the range of 200-2000 and 50 µg/kg levels, respectively (Sharma *et al.* 1994). All the 272 poultry feed samples collected during 1993-95 from different parts of Haryana were positive for aflatoxins. Aflatoxin levels were higher than the permissible limit in 36.7 and 33.6% of the samples collected during 1993-94 and 1994-95, respectively (Mahipal *et al.* 1996). Various cattle feed samples (82%) collected from Karnal (Haryana) were positive for aflatoxin (Prasad *et al.* 1997). Aflatoxin levels in decreasing order were groundnut-cake, wheat bran, maize grain, cottonseed-cake, mustard-cake and barley. Chicken feed ingredients including groundnut-cake, maize, millets, rice bran, sorghum, soybean, sunflower and mixed feeds, were assayed for aflatoxins and 38% samples were found contaminated with aflatoxins (Thirumala *et al.* 2002). The incidence scores of aflatoxin contamination in excess of 10 µg/kg were 43% for maize, 60% for mixed feeds, 27% for groundnut, 20% for sorghum, 50% for sunflower, 21% for rice bran, and 1 of 8 for millet. Gowda *et al.* (2003) reported that groundnut-cake, maize and compounded feeds contained very high levels of aflatoxin.

Table 1. Aflatoxin levels (µg/kg) in animal feeds

Feed samples tested	Positive samples	AFB_1 level	Reference
36	18	-	Mishra and Singh (1978)
10	7	113-2250	Rampla *et al.* (1979)
530	230	649-2009	Pater *et al.* (1981)
127	45	1-900	Singh *et al.* (1983)
101	101	350-7749	Balasubramanium (1985)
137	97	200-2000	Sharma *et al.* (1994)
56	46	20-4200	Prasad *et al.* (1997)
216	83	-	Thirumala *et al.* (2002)

Aflatoxicosis is not restricted to India, but such reports are emerging from other countries as well. In Germany, Bluthgen and Ubben (2000) reported that the single feeds contained <0.3-3.4 µg/kg and the mixed dairy concentrates contained 0.1-1.4 µg/kg of aflatoxin B_1. In Portugal 45% animal feed samples of different origins had aflatoxin B_1 levels varying between 0.1-16 µg/kg (Martins and Martins 1999).

Metabolism

Aflatoxin B_1 is the most toxic, both for humans and animals (Eaton and Groopman 1994) and is a potent carcinogen. The principal target organ is the liver where the reactive aflatoxin 8, 9-epoxides induce hepatocellular damage. Cytochrome P450 enzymes convert aflatoxins to the reactive 8,9-epoxide form, which is capable of binding to both DNA and proteins. A reactive glutathione *S*-transferase system in the cytosol and microsomes catalyzes the conjugation of activated aflatoxins with reduced glutathione, leading to the excretion of aflatoxin. Variation in the level of the glutathione transferase system and in the cytochrome P450 system are thought to contribute to the differences observed in inter-species aflatoxin susceptibility (Alexandros and Jouany 2002). Bovine species are generally less sensitive compared to non-ruminants because aflatoxins are partly degraded by the forestomach flora.

Alfatoxin metabolite M_1 appears in milk and milk products because of direct intake of aflatoxin B_1-contaminated feed. Besides M_1, other aflatoxin metabolites, viz. M_2, M_4, and B_1 (Cheeke and Shull 1985) are excreted with milk.

Clinical symptoms

Anorexia, icterus, depression, weight loss, nasal discharge, gastrointestinal affections, haemorrhages, ascitis and pulmonary oedema are clinical signs of aflatoxin exposure in animals (Raisbeek *et al.* 1991). Aflatoxins cause liver damage, decreased milk and egg production (Jones *et al.* 1982) and suppression of immunity in animals (Balaraman 1986,Arora *et al.* 1993). Though acute clinical intoxications due to aflatoxin exposure are rarely seen, but, sub-optimal weight gain, lower milk and egg production, and an increased susceptibility towards infectious diseases may lead to considerable economic losses in animal production due to aflatoxin exposure (Raisbeek *et al.* 1991, Shane 1994).

Nursing animals may be affected by exposure to aflatoxin metabolites secreted in the milk. Clinical signs include gastrointestinal dysfunction, decreased feed consumption and efficiency, anaemia and jaundice. Acute aflatoxin toxicity was demonstrated in mammals, fish, birds, rabbits, dogs and primates. Ducks, turkeys and trout are all highly susceptible.

The LD_{50} (lethal dose) is between 0.5 and 10mg/kg body weight. The liver is the principal target organ, although the site of the hepatic effect varies with species. Effects on the lungs, myocardium and kidneys were observed and aflatoxin can accumulate in the brain. Teratogenic effects following administration of high doses of aflatoxin were reported in some species.

Aflatoxin M_1 is a hepatotoxic and hepato-carcinogenic. The acute toxicity of aflatoxin M_1 seems to be similar or slightly less than that of aflatoxin B_1 but its carcinogenic potency is probably one or even two orders of magnitude lower than that of aflatoxin B_1 (Henry *et al.* 2001). Aflatoxin M_1 is not only detected in dairy milk, but also in breast milk of nursing mothers. Using aflatoxin M_1 as possible marker of exposure to aflatoxin B_1, El-Sayed *et al.* (2002) reported a mean level of 0.3 ± 0.5 µg/litre in breast milk of nursing mothers, and a corresponding mean blood level of 1.2 µg/litre.

Ruminants: Ruminants are more resistant to aflatoxins than non-ruminants. Balaraman (1986) concluded that the safe dietary level of aflatoxins for growing calves was 0.26 mg/kg. Chronic exposure of ruminants to aflatoxins decreased breeding efficiency, birth of smaller and unhealthy calves, diarrhoea, acute mastitis, respiratory disorder, prolapsed rectum, hair loss, and reduced feed consumption (Alexandros and Jouany 2002). The liver and kidney are mainly damaged by aflatoxins (Suliman *et al.* 1987, Edrington *et al.* 1994). In cattle and small ruminants, clinical symptoms occurred after their exposure to concentrations of 1.5 – 2.23 and > 50 mg/kg feed, respectively (Miller and Wilson 1994). Postmortem examination of exposed animals revealed liver cell damage (centrolobular necrosis) bile duct proliferation and kidney lesions. Blood biochemical parameters altered reflecting the degree of liver damage. Decreasing milk production of exposed animals and a photosensitizing effect can precede gross clinical signs of intoxication (Miller and Wilson 1994). Lactating animals, fed on aflatoxin contaminated feed, excreted aflatoxin M_1 into the milk within 24 hr, which may cause aflatoxicosis among its consumers. This is why government regulations specify that milk must be free of aflatoxins. However, action is not taken until the aflatoxin level exceeds 0.5 µg/litre in market milk, the level below which there is no hazard for the consumers. In ruminants, a considerable part of ingested aflatoxin B_1 is degraded in rumen, and does not reach systemic circulation. The absorbed fraction of aflatoxin B_1 is extensively metabolized in liver, resulting predominantly in aflatoxin M_1, which enters the systemic circulation or is conjugated to glucuronic acid, and subsequently excreted via bile (Allcroft and Carnagham 1963). Circulating aflatoxin M_1 can be excreted through kidneys or mammary glands to appear in the milk.

The aflatoxin M_1 in milk of dairy cows was 1–2 % of the ingested aflatoxin B_1 (Van Egmond 1989). This carry-over rate, however, can vary in individual cow, from day to day and from one milking to the next milking as it is influenced by feeding regime, health status, individual capacity for biotransformation, and finally by the actual milk production. For high yielding dairy cows (40 litres of milk/day) carry-over percentages was as high as 6.2 % (Veldman *et al.* 1992). Lembhe (1977) revealed the presence of varying amounts of aflatoxin M_1 in milk. M_1 in milk was observed in 40, 55 and 70 % samples in buffaloes fed aflatoxin B_1 in their diet @ 1, 2 and 3 mg, respectively, and the milk toxin retains the toxicity and carcinogenicity of the original toxin consumed by the animals (Yadgiri and Tulpule 1974). Vandana and Chauhan (1991) reported that 40 % milk samples were positive for aflatoxin M_1. In general, the ratio of dietary aflatoxin B_1 to that of milk was 300: 1 (Chhabra and Wadhwa 2004). Chopra *et al.* (1999) reported that excretion rates of aflatoxin M_1 in milk was 0.634, 0.391, 0.424 and 0.586% following the dietary aflatoxin B_1 at 0, 25, 50 and 100 µg/kg, respectively. The milk aflatoxin content remained below 0.05 µg/litre in cows fed on a diet containing aflatoxin B_1 up to 50 µg/kg, which is the acceptable level. A steady state of excretion of aflatoxin M_1 in the milk is achieved after 3-6 days of constant feeding of aflatoxin B_1. This toxin becomes undetected within 2-8 days of withdrawal of contaminated feed (Prasad and Chopra 1995, Rao and Chopra 2001).

Poultry: Aflatoxin toxicity (aflatoxicosis) causes serious health hazard to poultry and other avian species (Raina and Singh 1991). High dietary levels cause mortality, but very low levels are detrimental, if fed continuously. Clinical symptoms of aflatoxicosis are anorexia, decreased weight gains, decreased egg production, haemorrhage, embryo toxicity, and increased susceptibility to environmental and microbial stressors (Chaudhary 1991).

Aflatoxins decreased the activity of several enzymes needed for the digestion of starches, proteins, lipids, and nucleic acids (Osborne and Hamilton 1981), which contribute to the malabsorption of nutrients associated with aflatoxicosis. This toxin is primarily a hepatotoxin in young broiler chicken. One effect that is used as a diagnosis of aflatoxicosis in poultry is an enlarged, fatty, yellow and friable liver in broilers consuming aflatoxins contaminated feed (Verma *et al.* 1991). Panda (1991) and Rao and Reddy (1994) found beneficial effect of a herbal preparation, in reducing hepatic damage caused by aflatoxins in poultry.

Aflatoxin level of 0.8 and 1.6 mg/kg diet depressed body weight gain, feed intake, retention of protein, calcium and phosphorus and livability (Beura *et al.* 1993), however, due to increased dietary protein intake (22 to 25 or 27%) through contaminated diet, the toxicity was inhibited even at these high doses of aflatoxin. Sensitivity or resistance to aflatoxins is inherited as distinctive characteristics of breed and strain (Table 2). The adverse effect of aflatoxins on performance of chicken is also dose and time related.

Arora *et al.* (1988) did not observe any effect of feeding

aflatoxin up to 1.5 mg/kg in the diet of cobb chicks for 7 weeks, which revealed this broiler strain could tolerate dietary aflatoxin level up to 1.71 mg/kg.

Swine: Aflatoxin toxicity has been reported in suckling pigs, growing and finishing swine, and breeding stock. Clinical and pathological signs include decreased growth rate, poor efficiency of feed utilization, toxic hepatitis, nephrosis, and systemic hemorrhages. Aflatoxins can be transferred from sows to piglets and can affect the biological and immunological response of the neonatal pig. The effects of aflatoxins in pigs vary depending on the age of animal, diet, level of toxin, and length of exposure. Its effect appear to be the most severe in young pigs. Aflatoxins are absorbed rapidly from gastro-intestinal tract and concentrate in liver and kidney. Chronic aflatoxin intake impairs resistance and immune system of pigs resulting in more infectious disease problems.

Horses: Clinical signs of aflatoxicosis in horses include reduced feed intake, rapid weight loss, lethargy, lack of muscular control, circling, tetany and even death. Target organs appear to be the liver and kidneys. Horses on a high plane of nutrition seem to tolerate aflatoxin toxicity in corn.

FDA action levels

Aflatoxins are the only mycotoxins currently regulated by the US Food and Drug Administration (FDA). Action levels for livestock represent the level of contamination at which the feed may be injurious to their health or result in contamination of milk, meat or eggs. Acceptable levels set for animal feed for different categories of livestock is shown in Table 3. European Union have recommended the maximum permission level in feed as 5 µg/kg against maximum level 50 and 20 µg/kg in cattle and poultry feeds recommended by Bureau of Indian Standards in India. Indian levels appear to be on the higher side due to the kind of available feed ingredients and lack of scientific data. However, situation in near future will improve due to the awakening of poultry and dairy farmers about the detrimental effect of aflatoxin and positive effect of incorporation of feed processing technologies on the aflatoxin content of the feed.

Detoxification of aflatoxins

Physical, chemical and biological approaches were used for detoxification of aflatoxins present in feeds (Sundararasu and Muruganandam 1992, Devegowda *et al.* 1994,Mani *et al.* 1997). The lactone ring of aflatoxins makes them susceptible to alkaline hydrolysis, and processes involving ammonia or hypochlorite were investigated as means for their removal from feeds. Various physico-chemical treatments for detoxification of dietary aflatoxins are listed in Table 4.

Drying: Exposing feeds to sunlight is a simple and cheap mean for their decontamination (Sundararasu and Muruganandam 1992). Drying at 120°C for 2–3 hr resulted in reduction of aflatoxin content by 60-90 % (Gowda *et al.* 2004).

Roasting: Roasting corn at 290-330° F can reduce the aflatoxin content from 40 to 80%, with higher temperatures resulting in greater reductions. However, the temperature required to reduce aflatoxin are higher than those used in a normal corn roasting process. Some loss in feed value can be expected while roasting at these temperatures.

Ammoniation: Ammonia, applied either as gas (anhydrous ammonia) or liquid (aqua-ammonia), reacts with the aflatoxin molecule and destroys its toxicity. Proper treatment can reduce aflatoxin concentrations by 95% or more. Swine and poultry may be reluctant to eat the treated grain if ammonia smell is present, however, no such problem was reported on feeding treated corn to livestock. Concentrated anhydrous ammonia is hazardous to humans and livestock, explosive, and corrosive to equipment and storage bins, therefore, it should be done only by trained operators. Casual attempts to ammoniate corn may produce poor results and serious injuries. Ammoniation discolors grains, turning it to a light caramel color, which buyers may object.

Ghosh *et al.* (1996) reported that propionic acid @ 0.5%, at 10 % moisture level was most effective in preventing aflatoxin biosynthesis in groundnut-cake followed by sodium bisulfite and sodium hydroxide.

However, none of these methods seem to fulfill the efficacy, safety, safeguarding of adsorbant nutritional elements and cost requisites of a detoxification process (Piva *et al.* 1995).

Adsorbents: Inert adsorbents are used for sequestering the aflatoxins present in the feed and reduce their absorption from the gastro-intestinal tract (Ramos *et al.* 1996). The

Table 2. Tolerance levels (µg/kg) of dietary aflatoxin in different poultry birds

Poultry	Tolerance level
Crossbred broilers	400
Pure-bred broiler chicks	200
White Leghorn chicks	150
Quail chicks	300
Quail layers	300
Guinea fowl keets	1500
Layers	600

Table 3. Acceptable levels of total aflatoxins in feeds for livestock

Class of animals	Action level (ppb)
Dairy	20
Immature animals	
Immature poultry	
Breeding cattle	100
Breeding swine	
Mature poultry	
Finishing swine	200
Finishing cattle	300

addition of non-nutritive binding agents, such as the zeolite clays and aluminosilicates gave effective protection against aflatoxin toxicity (Ivan *et al.* 1992, Ramos *et al.* 1996). The basic mechanism for their action appears to involve aflatoxin chemisorption in the gastro-intestinal tract of animals, resulting in a major reduction in aflatoxin bioavailability.

Rao *et al.* (2004a) reported that activated charcoal @ 2% in concentrate mixture containing 300 µg/kg aflatoxin prevented its adverse effects on feed intake and growth performance of animals. On the basis of changes in the concentration of blood enzymes and protein fractions, Rao *et al.* (2004 b) concluded that activated charcoal was better in protecting the toxic effects of aflatoxin than sodium bentonite. Kumar *et al.* (2004) found ultrasil as the best binding agent among activated charcoal, ultrasil, sodium bentonite, yeast extract and esterified glucomannan. Charcoal addition in the diet on reducing toxic effects of aflatoxins in poultry was also reported (Anjaneyulu and Rao 1993, Jindal *et al.* 1994). The levels of various dietary additives to be included in the ration for protection against aflatoxins are given in Table 5.

Table 4. The physico-chemical treatments for inactivation of preformed aflatoxins in maize and groundnut-cake

1	Raising the moisture level up to 20%. Autoclaving at 5 PSI for 1 hr followed by drying in an oven at 80°C
2	Adding sodium hydroxide (15 g/kg) and mixing. Raising the moisture content up to 20%, autoclaving at 5 PSI for 1 hr and drying in an oven
3	Agitation of 1 kg feedstuff with 20 g $Ca(OH)_2$ followed by addition and mixing of formaldehyde to raise the moisture content up to 15%. Autoclaving at 15 PSI for 1 hr and drying
4	Addition of liquor ammonia to yield 6% concentration. Raising of moisture content up to 20%. Storing airtight for 20 days. Heating at 35°C and drying in an oven

Microbiological methods: Certain strains of lactic acid producing bacteria, propionibacteria and bifidobacteria have cell wall structures that can bind mycotoxins (Ahokas *et al.* 1998, El-Nemazi *et al.* 1998, Yoon and Baeck 1999) and restrict their bioavailability in animal body. Mycotoxins are then eliminated in the faeces without significant detrimental effects on the animals or any risk for toxic residues in edible animal products. Glucomannans extracted from external part of cell wall of yeast *Saccharomyces cerevisiae* are able to bind certain mycotoxins because of large area available for exchange. Thus, 500 g of glucomannans from yeast cell-wall have the same adsorption capacity as 8 kg of clay (Devegowda 2000). This binder reduces the aflatoxin M_1 content of milk by 58% in cows given a diet contaminated with aflatoxin B_1 @ 0.05% of dry matter. Certain micro-organisms are also able to metabolize mycotoxins (*Corynebacterium rubrum*) in contaminated feed or to biotransform them (*Rhizopius*, *Aspergillus*, *Eurotium*) (Nakazato *et al.* 1990). These biological processes are generally slow and have a low efficiency. Cotty and Bhatnagar (1994) proposed that isolates of *A. flavus* and *A. parasiticus* strains, which do not produce aflatoxins may be used to out-compete natural toxin-producing strains. These strains occupy the same ecological niche as toxin-producing strains, so they decrease the level of contamination with toxin-producing moulds.

Analytical methods for aflatoxin detection: Detection methods range from simple and inexpensive fluorescence techniques to expensive and time consuming chemical procedures such as GC, TLC or HPLC. Black light method is used as a crude method of detecting aflatoxins, but, it is not reliable because the compound that produces the greenish-yellow fluorescence is kojic acid and not aflatoxin. More advanced fluorescent procedures such as minicolumn and fluorometric-iodine methods can also be used for detection of aflatoxin. HPLC has replaced TLC as the officially approved method of testing for mycotoxins by the Association of Official Analytical Chemists. Methods that use direct competitive ELISA technology and commercial kits are available and the cost of analysis per sample is about Rs 200.

Other alternatives: Recently, organic farming is being emphasized to reduce the risk of contamination and heavy metals in foods/feeds and to improve its nutritional quality. Giangacomo Lorenzini (2004) investigated that aflatoxin M_1 contamination occurred in milk from both organic and conventional dairy herds in Tuscany between summer 2003 and summer 2004. It is believed that the risk may be higher in organic farming because preservatives and crop protection chemicals are not used, but the problem is also related to the climate, because the warm climate increases the risk of

Table 5. Dietary inclusion levels of various additives for protection to broilers against dietary aflatoxins

Detoxifying agents	Quantity (g/100 kg feed)
Activated charcoal	100–200
Hydrated sodium calcium aluminosilicate (HSCAS)	100–200
Esterified glucomannan (EGM)	50–100
Herbal mixture (*Acacia catechu*, 25%, *Phyllanthus niruri*, 400%, *Andrographis paniculata*, 25%, base 10%)	50–75
Butylated hydroxyanisole	50–100
DL-methionine	100–200
Selenium	0.200–0.300
Butylated hydroxy toluene	50–150
L-lysine HCl	150
Water soluble vitamins	Double of the requirements
Increase the dietary protein level	Up to 26 to 28%

aflatoxin contamination in maize. The aflatoxin M_1 levels fell more quickly in the organic than that in the conventional milk, probably because the few organic farms involved quickly switched to the use of aflatoxin free maize grain. Clearly, this is not a problem for organic farming. Possible solutions include the use of early maturing maize varieties, and more effective drying of maize grain.

Prevention

Establishing a "safe" level of aflatoxins contamination is not possible because the response to mycotoxins is influenced by age and species of the animal, environmental and disease challenges and nutrition. Controlling the adverse effects of aflatoxins requires a multifaceted approach.

Hazard Analysis Critical Control Point (HACCP) is a preventive, not reactive, tool that places the protection of the food supply from microbial, chemical and physical hazards in the hands of the food management system. This system is designed to minimize the risk of food by identifying the hazards, establishing control points and monitoring these controls. To design an effective HACCP-based mycotoxin management programme, climate, farming systems, pre-harvest and post-harvest technologies etc. should be considered. In an ideal HACCP-based system, mycotoxins would be minimized at every phase of production, harvesting, processing and distribution (Park *et al.* 1999). Such systems are increasingly being used in human food production and should be employed for animal feeds as well.

Different countries have established different safety levels of aflatoxins in feeds and foods. Codex Alimentarius Commission has proposed very low levels of 0.05 μg aflatoxins /kg milk. The consumption of aflatoxin contaminated feeds by the animals is very high in India, and it is very difficult to meet these standards. Hence, proper storage conditions of grains and other feed ingredients have to be followed to produce milk with low aflatoxin levels so as to compete the international market. Safe production, harvest, and storage practices can reduce the risk of aflatoxin contamination. The following points should be considered while making a decision on using aflatoxin contaminated grain:

1. Determine the aflatoxin concentration in the grain.
2. Determine the age and species of livestock to be fed. Young, fast growing animals are most adversely affected within a species. Breeding stock may not be affected but the developing fetuses are very susceptible to low levels of aflatoxins.
3. Ducklings, turkeys, chickens, swine, other simple stomach animals, ruminants and sheep are affected in decreasing order.
4. Consider the willingness to assume risks associated with feeding contaminated grain.

CONCLUSION

The biological implications of the presence of aflatoxins in human and animal foods have been investigated. The teratogenicity, carcinogenicity and general toxicity of this mycotoxin poses a risk to animal and human health. As interest of consumers is growing in food safety, the animal and human food industries and farmers must be made aware of possible mycotoxicological risks. Improvement in seed production, cultivation, harvest and storage of forages and cereals, is essential to reduce the level of contamination of foods and feeds. The total elimination of moulds and their toxins is impossible, hence, there is a need for development of additional preventive measures with the use of toxin-binding agents that limit the bioavailability of toxin in animals and further transfer to humans.

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