The Growing Problems of Mycotoxins in Animal Feed Industry in West Africa: A Review

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Abstract: This review was developed to help those who might have experienced financial loss to better understand mycotoxin problems and their control. Mycotoxins are secondary metabolites produced by certain toxigenic fungi while growing on organic substances. Scores of compounds produced by fungi have been identified and many are capable of accumulating in certain feed and food commodities. The feed/ingredient contamination with toxins may occur anywhere in the supply chain, starting from the harvest of the feedstuffs, to storage, processing and feeding channels in the farm. Mycotoxin contamination of feed is a recurring problem in livestock feed industry. These mycotoxins when gets into the system of the animals or humans consuming them may cause moderate to severe toxicity symptoms. Low levels of prolonged ingestion of mycotoxins can be more hazardous than what it was thought about. The economic impacts associated with mycotoxins go far beyond the losses incurred by the feedstuff producer. They will be spread throughout the system to handlers, distributors, processors, livestock and dairy operators, consumers, national and state governments. If exports or other markets are lost due to questionable feedstuffs quality, the overall price level for the commodity could fall and result in even greater losses throughout the agricultural sector. Hence, a rational approach has to be adopted for effective control of mycotoxins.

Keywords: Aflatoxin, zearalenone, deoxynivalenol, T-2 toxin, fumonisin, mycotoxins

INTRODUCTION

Livestock industry in West Africa has seen a tremendous growth with the best application of nutritional technologies (John Prabakaran and Dhanapal, 2009). However, feed safety is a concern for achieving productivity (Jimoh and Kolapo, 2008). Even with the best quality control systems in the world, animal producers often find themselves owning mycotoxin contaminated grains or feed (Okoli et al., 2007; Jimoh and Kolapo, 2008). The term mycotoxin was coined in 1962 in the aftermath of an unusual veterinary crisis near London, England, during which approximately 100,000 turkey poults died (Blout, 1961; Forsacs, 1962). When this mysterious turkey X disease was linked to a peanut (groundnut) meal contaminated with secondary metabolites from Aspergillus flavus (aflatoxins), it sensitized scientists to the possibility that other occult mold metabolites might be deadly. Soon, the

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mycotoxin rubric was extended to include a number of previously known fungal toxins (e.g., the ergot alkaloids), some compounds that had originally been isolated as antibiotics (e.g., patulin) and a number of new secondary metabolites revealed in screens targeted at mycotoxin discovery (e.g., ochratoxin A). The period between 1960 and 1975 has been termed the mycotoxin gold rush (Maggon et al., 1977) because so many scientists joined the well-funded search for these toxigenic agents. Depending on the definition used and recognizing that most fungal toxins occur in families of chemically related metabolites, some 300 to 400 compounds are now recognized as mycotoxins, of which approximately a dozen groups regularly receive attention as threats to human and animal health (Cole and Cox, 1981; Latha et al., 2008).

Mycotoxins occur, with varying severity, in agricultural products all around the world (Desjardins, 2006). The estimate usually given is that one quarter of the world's animal feedstuffs (grains and oil pulse) is contaminated to some extent with mycotoxins (Mannan and Johnson, 1985; Fink-Gremmels, 1999; Okoli et al., 2006). Mycotoxins can enter the food chain in the field, during storage, or at later points (Okoli et al., 2007). Mycotoxin problems are exacerbated whenever shipping, handling and storage practices are conducive to mold growth (Ayalew et al., 2006). The end result is that mycotoxins are commonly found in foods. Kuiper-Goodman (1998), a leading figure in the risk assessment field, ranks mycotoxins as the most important chronic dietary risk factor, higher than synthetic contaminants, plant toxins, food additives, or pesticide residues. The economic consequences of mycotoxin contamination are profound. Not surprisingly, many mycotoxins display overlapping toxicities to invertebrates, plants and microorganisms (Bennett, 1987; Alwakeel, 2009).

The question then arises: Is the level of mycotoxin(s) present safe to feed to my animals? Prior to giving specific information, some general concepts regarding the effect of mycotoxins on animals must be understood. Frank et al. (2007) stated that the determination as to whether or not a given concentration of mycotoxin is safe will depend on the factors which are as follow (and there may also be other factors).

- Chemical class and chemical structure of the mycotoxin in question. A partial list of known mycotoxins is shown in Table 1. The list includes mycotoxins from a number of chemical classes, each of which has different effects on the animals which consume them. However, the exact chemical structure of the mycotoxin involved is also vital. For example, aflatoxin B1 is reported to be the most potent naturally occurring carcinogenic substance known (Al-Mamun et al., 2002), but if just one chemical bond is changed in the structure of the molecule, its toxicity can be reduced dramatically.

<table>
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<tr>
<th>Table 1: A partial list of known mycotoxins</th>
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<tr>
<td><strong>Aflatoxins</strong></td>
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<td><em>Alternariol</em></td>
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<td><em>Citrovinidin</em></td>
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<td><em>Citrinin</em></td>
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<td><em>Fumonisins</em></td>
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Sourced from Frank et al. (2007)
• Presence of other mycotoxins. A number of studies have demonstrated that mycotoxins occur simultaneously in field situations (Ayalew et al., 2006). This simultaneous occurrence can profoundly affect the toxicity of the mycotoxins present.

• Species and strain of the animals involved. Ducklings are 5 to 15 times more sensitive to the effects of aflatoxin than are laying hens, but when laying hen strains are compared, certain strains of hens may be as much as 3 times more sensitive than other strains. This fact, along with the fact that there is continuous genetic improvement of farm animals, can mean that the exact sensitivity of a given animal to one or more mycotoxins is unknown.

• Health status of the animals involved. Stress, physiological state, nutritional standing and disease status will independently and collectively determine the response of a given animal to a specific mycotoxin level or complex of mycotoxins.

• Criteria by which effects are determined. At a given dose, aflatoxin reduces weight gain in growing animals, but disease resistance in the same animal may be reduced by about half that dose.

• Number of animals involved in judging the no effects level. It has been estimated that 400 groups of 10 broilers would be required to detect a 1% difference in growth rate. Yet, in integrated poultry operations a 1% difference in growth rate would have a significant economic impact.

• Sampling and assay procedures. It is imperative that sampling and assay procedures are accurate, since the results are the basis for deciding whether or not to use a given lot of feed or feed ingredient.

• Length of time animals is exposed to the mycotoxin(s). The exact mycotoxin tolerance levels given elsewhere in this publication assume that animals are exposed for a limited period. Obviously, the risks of harm to animals from mycotoxins increase as exposure time increases.

While the question of safe mycotoxin levels is a valid and vital question, it is not an easily answered question for the reasons just outlined. Perhaps the shortest answer to the question is: There is only one safe mycotoxin level and that is zero. However, Marquardt and Frohlich (1992) stated that a risk free environment is never possible and background levels of mycotoxins are commonly found in numerous feed ingredients. In addition, no observable effects levels (NOEL) for mycotoxins do not exist for each animal species. This review was developed to help those who might experience financial loss to better understand mycotoxin problems and their control. Unfortunately one cannot manage all of the factors that may contribute to the development of such problems. This makes it doubly important for as much control as possible to be exercised.

Economic Losses Associated with Mycotoxin Contamination

Estimates suggest that up to 25% of the world’s food crops are affected to some degree by mycotoxin contamination annually (Sinha and Bhatnagar, 1998; Smith and Anderson, 1991). These mycotoxins and their control can result in considerable economic impacts, not only to the producer of the feedstuffs, but also for intermediaries such as elevators, grain buyers, exporters, feed and food processors, users of the commodities such as livestock, poultry and dairy producers, consumers and even national and state governments (Sweeney et al., 2000; Okoli, 2005). Despite the economic importance of mycotoxin contamination of food and feed sources, it is extremely difficult to adequately quantify the economic impact given the wide range of economic effects.
The occurrence and level of mycotoxin contamination in feed and food grains varies among commodities, years and regions (Essien, 2000; Aly Salwa and Anwer, 2009). High levels of aflatoxin contamination and resulting economic losses are more likely to occur in years where environmentally stressful conditions such as extreme drought exist (Sternberg, 1994). However, even in such years, affected feed stuff may be randomly scattered throughout the environmentally stressed (Motalebi et al., 2008) region making estimation of aggregate damage and economic losses difficult at best. Besides environmental factors, mycotoxin levels are also affected by economic factors and their impact on cultural practices such as harvesting, storage technology and the use of insecticides and fungicides (Sternberg, 1994; Essien, 2000). Agricultural price supports and other government farm programs can also have a major impact through their influence on production patterns and cultural practices.

**Producer Costs**

The most obvious economic impacts will occur at the feed stuff producer level where losses will include yield losses, increased production cost, increased marketing risks and costs and increased post-harvest costs (Anonymous, 1989; Opara and Okoli, 2005). Increased production costs could include increased expenses for irrigation to reduce drought stress and increased pesticide use to reduce plant pathogens that produce mycotoxins (Essien, 2000). Increased post-harvest costs could include sampling and testing, increased storage and drying costs, detoxification, increased transportation involved with limited markets and the disposal of unusable feed stuff (Essien, 2000). Marketing may provide some of the greatest challenges to the producer. Above certain contamination levels, as set by the FDA, use of the commodity will be restricted to certain markets. Those restricted markets may be more difficult for producers to identify and with few potential outlets, producers may find themselves at the buyer’s mercy in price negotiations. Price discounts could be substantial depending on the level of contamination and the supply of and demand for the contaminated commodity.

**Livestock and Dairy**

Economic losses to livestock and dairy producers from mycotoxin contamination may be less obvious and more difficult to quantify (Bhuiyan et al., 2003). While mortality loss or milk contamination losses can be estimated, more subtle chronic effects such as losses in feeding efficiency, infertility, immunological problems and overall quality losses are much more difficult to estimate (Wilson et al., 1998; Bhuiyan et al., 2003). The various health problems that could arise depend on the contamination level of feed being used and the susceptibility of the species being fed (Shimoda, 1979; Bhuiyan et al., 2003). Monitoring and testing increase the cost of production, but are of vital importance to livestock and dairy producers who could suffer severe losses if grain contaminated beyond safe levels were used in feeding rations (Wilson et al., 1998). Producers feeding their own heavily contaminated crops to their own livestock, rather than taking price discounts or destroying or treating them, could compound losses to the farm operation.

**Handlers, Distributors, Processors**

Handlers, distributors and processors who knowingly or unknowingly purchase mycotoxin contaminated feed stuff face many of the same difficulties as producers (Opara and Okoli, 2005). They are faced with extra costs and care in drying and storage, restricted markets, price discounts and product loss and disposal of contaminated products,
as well as the extra costs associated with testing and monitoring (Jimoh and Kolapo, 2008). They can also suffer the loss of a customer or an entire market if the quality and dependability of their product comes into question (Ayejuyo et al., 2008).

Social and Consumer Costs

Social costs associated with mycotoxin contamination although difficult to estimate, can be substantial, especially when outbreaks occur (Van Egmond and Speijers, 1994). These costs include regulatory costs such as establishing standards and tolerance levels, monitoring and testing, enforcement of regulations and Research and Extension education efforts (Wogan, 1965; Weidenborner, 2001). Consumer costs could include higher product prices as some increased costs faced by handlers and processors are passed on to consumers or as supplies are restricted (Okoli, 2005). While unlikely in the U. S., with its extensive testing and regulatory safe-guards, consumers could also be faced with a less nutritious food supply and debilitating effects of mycotoxicosis if the contaminated feed stuff were to enter the food system (Sweeney et al., 2000).

MYCOTOXINS AND ANIMAL HEALTH

Mycotoxins produce a wide range of harmful effects in animals (Aly Salwa and Anwer, 2009). The economic impact of reduced animal productivity, increased incidence of disease due to immunosuppression, damage to vital organs and interferences with reproductive capacity is many times greater than the impact caused by death due to mycotoxin poisoning (Robens, 1990; Van Egmond and Speijers, 1994). In comparison to other animals, poultry species tend to be resistant to the effects of fumonisin, deoxynivalenol and zearalenone (Marquardt and Frohlich, 1992; Aly Salwa and Anwer, 2009). However, the presence of these mycotoxins within poultry rations is an indication that mold activity has occurred in the ration or in the ingredients within the ration (Van Egmond and Speijers, 1994; Ayalew et al., 2006). Since, mold activity can generate numerous other mycotoxins as well as reduce the nutritive value and palatability of feeds (Essien, 2000) the presence of fumonisin, deoxynivalenol, or zearalenone in poultry feeds is cause for concern (Blaney and Dodman, 2002; Isakeit et al., 2008). While young animals are most susceptible to the effects of aflatoxin, all ages are affected and clinical signs include gastrointestinal dysfunction, reduced productivity, decreased feed utilization and efficiency, anemia and jaundice (Opara and Okoli, 2005). Nursing animals may be affected by exposure to aflatoxin metabolites secreted in the milk (Van Egmond, 1989). Aflatoxin causes a variety of symptoms depending on the animal species. However, in all animals, aflatoxin can cause liver damage, decreased reproductive performance, reduced milk or egg production, embryonic death, teratogenicity (birth defects), tumors and suppressed immune system function, even when low levels are consumed (Van Egmond, 1989).

Deoxynivalenol (DON) is, perhaps, the most commonly detected Fusarium mycotoxin. DON has been associated with reduced milk production in dairy cattle, (Bankole and Adebanjo, 2003) vomiting by swine consuming contaminated feed or their refusal to eat feed containing the toxin and inhibiting reproductive performance and immune function in several animal species (Van Egmond, 1991). In addition, DON levels greater than 500 ppb have been associated with numerous other field problems (Desjardins, 2006).

Zearalenone mimics the effect of the female hormone estrogen and at low doses, increases the size or early maturity of mammary glands and reproductive organs (Blaney and Dodman, 2002). At higher doses zearalenone interferes with conception, ovulation, implantation, fetal development and the viability of newborn animals (Urly et al., 1966).
The T-2 toxin and its chemical relatives cause irritation, hemorrhage and necrosis throughout the digestive tract, depress the regenerative process in the bone marrow and spleen, impair immune system function and cause changes in reproductive organs (Fink-Gremmels, 1999). Affected animals show signs of weight loss, poor feed utilization, loss of appetite, vomiting, bloody diarrhea, abortion and (in severe cases) death (Fink-Gremmels, 1999).

Fumonisins are mycotoxins which have only recently been discovered (Hamilton et al., 1982). Thus, it has not been extensively studied. Nonetheless, it is known that in animals fumonisins impair immune function, cause liver and kidney damage, decreases weight gains and increases mortality rates (Van Egmond, 1991). Fumonisins also cause leukoencephalomalacia in horses and respiratory difficulties in swine. In some animals fumonisins can also cause tumors.

Although, the following section emphasizes the effects mycotoxins exert on animal health and performance, mycotoxins may also be a hazard to human health since animal products consumed by people may contain mycotoxin residues (Pier et al., 1980). While healthy animals tend to filter out or detoxify many of the mycotoxins to which they are exposed (Coelho, 1990; Bray and Ryan, 1991). In our consumer-oriented society the issue of mycotoxin residues in milk and animal tissues should not be ignored.

**Prevention and Management of Mycotoxins**

**Prevention of Feed Contamination**

Controlling mold growth and mycotoxin production is very important to the feed manufacturer and livestock producer (Essien, 2000). Control of mold growth in feeds can be accomplished by keeping moisture low, keeping feed fresh, keeping equipment clean and using mold inhibitors (Wilson et al., 2002). Grains and other dry feed such as hay should be stored at a moisture level 14% or less to discourage mold growth. Aeration of grain bins is important to reduce moisture migration and to keep the feedstuffs dry.

**MOISTURE CONTROL**

Moisture is the single most important factor in determining if and how rapidly molds will grow in feeds (Young and Cousin, 2001). Moisture in feeds comes from three sources: (1) feed ingredients, (2) feed manufacturing processes and (3) the environment in which the feed is held or stored (Sydenham et al., 1991). To control the moisture content of feeds successfully, moisture from all three sources must be controlled.

**Moisture in Feed Ingredients**

Since, corn and other grains are a primary source of the moisture and molds found in feed, the first important step in controlling moisture in feed is to control it in the grains from which the feed is prepared (Opara and Okoli, 2005). Since, all feed ingredients contain moisture, they should be monitored and their moisture content controlled.

It is commonly believed that the amount of moisture in grain is too small to permit mold growth except in rare and unusual circumstances. However, moisture is not evenly distributed in grain kernels. A batch of grain containing an average of 15. 5% moisture may, for example, contain some kernels with 10% moisture and others with 20% moisture. The moisture content of individual grain kernels is directly related to the amount of mold growth that occurs: that is, kernels with higher moisture contents were more susceptible to mold growth. In addition to moisture, the amount of mold growth is about five times greater for
broken kernels than for intact kernels. Thus the fraction of commercial grain, known as broken kernels and foreign matter, can be expected to have a higher mold and mycotoxin content than the portion composed of whole kernels.

**Moisture in Feed Manufacturing Processes**

Grains are commonly ground with a hammer mill to aid in mixing and handling, to improve digestibility and to improve the pelleting process. This grinding process creates friction, which causes heat to build up (Rheeder et al., 2002). If unchecked, temperature increases greater than 10 degrees Fahrenheit will cause significant migration of grain moisture encouraging mold growth. This is particularly true in cold weather when temperature differences cause moisture to condense on the inside walls of bins. Air assisted hammer mill systems reduce heat buildup in the product and in turn, reduce moisture problems.

The pelleting process involves mixing steam with the feed, pressing the mixture through a die and then cooling the pellets to remove heat and moisture. Generally, heat and 3 to 5% moisture are added to the feed during the pelleting process in the form of steam. If the pelleting process is done correctly, this excess moisture is removed from the feed before shipment. If, however, this excess moisture is not removed when the pellets are cooled, mold growth will be encouraged. Since feeds containing moisture are warmer than normal, storing hot or warm pellets in a cool bin will cause moisture to condense on the inside of the bin.

Although, pelleting of feed has been shown to reduce mold counts by a factor of 100 to 10,000, many mold spores remain in the feed after it has been pelleted (Opara and Okoli, 2005). After pelleting, the remaining spores can grow if conditions are right. Thus the pelleting process delays, but does not prevent, the onset of mold growth and plays only a minor role in efforts to control molds. In addition, pelleted feeds may be more easily attacked by molds than nonpelleted feeds.

**Moisture and Feed Storage Environment**

To control mold growth, obvious sources of moisture in the feed handling and storage equipment must be eliminated. These sources may include leaks in feed storage tanks, augers, roofs (either at the barn or at the feed mill) and compartments in feed trucks. A fact about feed moisture often overlooked is that it changes in relation to the feed's environment (Smith and Anderson, 1991). Since animals kept in confinement housing add moisture to their environment by respiration and defecation, the air in these houses can be very humid. Feed that was initially very low in moisture content will gain moisture when placed in a humid environment. The humidity in confinement housing should therefore be controlled by providing adequate ventilation.

**Keeping Feed Fresh**

Time is required for both mold growth and mycotoxin production to occur. It is therefore important to have feeds delivered often so that they will be fresh when used (Etuk et al., 2005). Feeds should generally be consumed within 10 days of delivery. It is equally important to manage the feed delivery system to ensure that feeds are uniform in freshness. Field surveys have shown that poultry farms producing birds with the poorest performance were those with the most feed in their feeder pans. On these farms, the feeds contained the greatest amount of moisture and had the highest number of molds (Smith and Anderson, 1991). If the feeder system is allowed to keep the feed pans full at all times, the feed in the pans will be significantly older than that in the storage tank. The animal will tend to eat
primarily the feed in the top layer and the feed at the bottom of the pans will age, providing greater opportunities for molds to grow. The animals’ performance may suffer as a result. To prevent this problem, the feeder system should be turned off weekly. The animals will then be forced to clean out all of the feed in the feeders before it becomes excessively old.

A similar principle applies to feed storage tanks. The feed next to the wall is last to exit the tank and therefore stays in the tank the longest. The feed in contact with the wall is also the only portion of the feed that changes appreciably due to temperature. These factors make feed in contact with the wall susceptible to moisture migration and mold growth. It is best to maintain two feed tanks so that one tank can be completely emptied and cleaned before it is refilled with new feed.

**Equipment Cleanliness**

When feed is manufactured and delivered to farms, it may come in contact with old feed that has lodged or caked in various areas of the feed storage and delivery systems. This old feed is often very moldy and may seed the fresher feed it contacts, (Rheeder *et al.*, 2002) increasing the chances of mold growth and mycotoxin formation. To prevent this problem, caked, moldy feed should be removed from all feed manufacturing and handling equipment.

**Use of Mold Inhibitors**

**Types of Mold Inhibitors**

The use of chemical mold inhibitors is a well established practice in the feed industry (Coelho, 1990). However, mold inhibitors are only one of several tools useful in the complex process of controlling the growth of molds and they should not be relied upon exclusively. The main types of mold inhibitors are (1) individual or combinations of organic acids (for example, propionic, sorbic, benzoic and acetic acids), (2) salts of organic acids (for example, calcium propionate and potassium sorbate) and (3) copper sulfate (Coelho, 1990). Solid or liquid forms work equally well if the inhibitor is evenly dispersed through the feed. Generally, the acid form of a mold inhibitor is more active than its corresponding salt.

**Factors Influencing the Effectiveness of Mold Inhibitors**

**Dispersion**

Many factors influence the effectiveness of mold inhibitors and proper attention to these factors can enhance the benefits they provide (Frank *et al.*, 2007). Mold inhibitors cannot be effective unless they are completely and thoroughly distributed throughout the feed. Ideally, this means that the entire surface of each feed particle should come in contact with the inhibitor and that the inhibitor should also penetrate feed particles so that interior molds will be inhibited.

The particle size of the carriers for mold inhibiting chemicals should be small so that as many particles of feed as possible are contacted. In general, the smaller the inhibitor particles the greater the effectiveness. Some propionic acid inhibitors rely on the liberation of the chemical in the form of a gas or vapor from fairly large particle carriers. Presumably, the inhibitor then penetrates the air spaces between particles of feed to achieve even dispersion.

**Effect of Feed Ingredients**

Certain feed ingredients may also affect mold inhibitor performance. Protein or mineral supplements (for example, soybean meal, fish meal, poultry by-product meal and limestone) tend to reduce the effectiveness of propionic acid (Wilson *et al.*, 1998). These materials can neutralize free acids and convert them to their corresponding salts, which are less active as
inhibitors. Dietary fat tends to enhance the activity of organic acids, probably by increasing their penetration into feed particles. Certain unknown factors in corn also alter the effectiveness of organic acid inhibitors.

**Time Dependence**

When mold inhibitors are used at the concentrations typically recommended, they in essence produce a period of freedom from mold activity. If a longer mold-free period is desired, a higher concentration of inhibitor should be used. The concentration of the inhibitor begins to decrease almost immediately after it is applied as a result of chemical binding, mold activity, or both. When the concentration of the inhibitor is reduced until it is incapable of inhibiting mold growth, the mold begins to use the inhibitor as a food source and grows. In addition, feeds that are heavily contaminated with molds will require additional amounts of inhibitor to achieve the desired level of protection.

**Influence of Pelleting**

The widespread use of pelleted feeds in the feed industry is beneficial to the use of mold inhibitors. The heat that the feed undergoes during pelleting enhances the effectiveness of organic acids. Generally, the higher the pelleting temperature, the more effective the inhibitor. Once mold activity commences in pellets, however, it proceeds at a faster rate than in nonpelleted feed because the pelleting process that makes feed more readily digestible by animals also makes it more easily digested by molds.

**Prevention of Mycotoxin in Silages**

Prevention of mycotoxins in silages includes following accepted ensiling practices aimed at inhibiting deterioration primarily through elimination of oxygen (Opara and Okoli, 2005). Some silage additives (such as ammonia, propionic acid, microbial cultures, or enzymatic silage) may be beneficial in preventing mycotoxins because they are effective at reducing mold growth. Silo size should be matched to herd size to ensure daily removal of silage at a rate faster than deterioration. Feed bunk should be cleaned regularly. Care should be taken to ensure that high moisture grains are stored at proper moisture content and in a well-maintained structure.

**ANIMAL MANAGEMENT**

If unacceptable mycotoxin levels occur, removal of the contaminated feed is preferable (Van Egmond and Speijers, 1994). While it is often not possible to completely replace the ration, particularly the forage ingredients, obviously, moldy feeds should be removed. Acidic diets may intensify the effects of mycotoxins and should be avoided in these situations. Increasing nutrients such as protein, energy (fats and carbohydrates) and vitamins in the diet may also be advisable (Etuk et al., 2005). The addition of antioxidants to the animal feeds assists in dealing with the effects of mycotoxins.

The possible use of inorganic binders (mineral clays) to bind mycotoxins and prevent them from being absorbed by the animal's gut, has received a lot of research attention recently (Smith and Anderson, 1991; Subramanya, 2005). These clay products (which include zeolites, bentonite, bleaching clays from refining of canola oil and hydrated sodium calcium aluminosilicates (HSCAS) have been shown to change the responses of rats to zearalenone and T-2 toxin. However, it should be clearly understood that binding of some mycotoxins may be weak or nonexistent and that clay products differ in their ability to bind mycotoxins.
While one HSCAS product called NovaSil has been shown to bind aflatoxin protectively against aflatoxicosis, under FDA regulations these clay products cannot be sold as mycotoxin binders. Nonetheless, many clay products are GRAS (Generally Recognized As Safe) and are used as anticaking or free flow additives for feeds.

**CONCLUSION**

Mycotoxins occur, with varying severity, in agricultural products all around the world. Mycotoxins can enter the food chain in the field, during storage, or at later points. Mycotoxin problems are exacerbated whenever shipping, handling and storage practices are conducive to mold growth. The end result is that mycotoxins are commonly found in foods. The economic consequences of mycotoxin contamination are profound. Feedstuffs with large amounts of mycotoxins often have to be destroyed. Alternatively, contaminated grains are sometimes diverted into animal feed. Giving contaminated feeds to susceptible animals can lead to reduced growth rates, illness and death. Moreover, animals consuming mycotoxin-contaminated feeds can produce meat and milk that contain toxic residues and biotransformation products. Court actions between grain farmers, livestock owners and feed companies can involve considerable amounts of money. The ability to diagnose and verify mycotoxicoses is an important forensic aspect of the mycotoxin problem. Nevertheless, many mycotoxins survive processing into flours and meals. When mold-damaged materials are processed into foods and feeds, they may not be detectable without special assay equipment. It is important to have policies in place that ensure that such hidden mycotoxins do not pose a significant hazard to human and animal health. Considerable research has been devoted to developing analytical methods for identifying and quantifying mycotoxins in food and feeds. The chemical diversity of mycotoxins and the equally diverse substrates in which they occur pose challenges for analytical chemistry. Each group of compounds and each substrate have different chemical and physical properties, so the methods for the separation of toxins from substrates must be developed on a case-by-case basis.

Since it is normally impractical to prevent the formation of mycotoxins, the food industry has to established internal monitoring methods. Similarly, several national and international organizations and agencies have special committees and commissions that set recommended guidelines, develop standardized assay protocols and maintain up-to-date information on regulatory statutes. There are also several mycotoxin associations and consumer regulatory agencies survey for the occurrence of mycotoxins in foods and feeds and establish regulatory limits. Guidelines for establishing these limits are based on epidemiological data and extrapolations from animal models, taking into account the inherent uncertainties associated with both types of analysis. Therefore, there is a need for worldwide harmonization of mycotoxin regulations. Estimations of an appropriate safe dose are usually stated as a tolerable daily intake.

**REFERENCES**


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