What Do I Do If Mycotoxins Are Present?

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All substances are poisons, there is none which is not a poison. The right dose differentiates a poison and a remedy.
Paracelsus (1493-1541)

Introduction

There is a common perception that "human-made" chemicals are more dangerous than "natural" substances. Interestingly, the most toxic compounds known are natural (Cheeke, 1998). Aflatoxin is the most carcinogenic, while the botulism toxin is the most poisonous organic substance ever encountered. Both substances can occur in food (aflatoxin in peanut products and botulism toxin in honey) but are not generally involved in poisoning of humans because of the low concentrations present.

Types of Toxins

Toxins originating from plants are called *phytotoxins*, while ones from microbes are *mycotoxins*, and ones from animals are *zootoxins*. Many plant toxins (e.g., alkaloids, glucosinolates, and saponins) are bitter, while terpenes and essential oils (e.g. sagebrush) have unpleasant odors which reduce palatability. Phenolic compounds (tannins) have an astringent effect because they react with taste receptors in the mouth. Phenolic compounds probably evolved as protection against ultra-violet radiation. Phenolics are excellent antioxidants. Some animals, such as sheep and goats, are tolerant of bitter compounds and may consume unpalatable plants which cattle avoid.

Alkaloids are compounds that contain nitrogen (usually in a ring), are usually bitter, and most are toxic. Other examples of alkaloids are cocaine, LSD, caffeine, and coffee. Glycosides are usually bitter. Examples are prussic acid (e.g., from sorghum-sudan), coumarin (e.g. from sweet clover), and saponins (e.g. causes bloat).

Detoxification may occur in the digestive tract, especially in ruminants. Ruminants are often more resistant to plant toxins than nonruminants because of the degradation or inactivation of toxins by rumen microbes. The liver is a major site of detoxification. It is strategically located for interception of absorbed toxins to prevent entry into the general circulation. The liver contains a number of enzymes, such as the cytochrome P450 system, which are effective in detoxification.

Mycotoxins in Feeds

The major cereal grains are, in order of global importance, wheat, rice, corn, barley, oats, sorghum, millet, and triticale. Cereal grains are the seed of cultivated grasses. Most of the major

food crops (e.g. beans, potatoes, and many vegetables), other than cereal grains, do contain toxins which must be removed or detoxified by cooking prior to consumption.

Based on the previous statements, it might be inferred that cereal grains would be of minor importance in human and animal nutrition. In fact, the opposite is true - by far the most important economic and toxicologic effect of natural toxins are associated with grains. The toxins involved are mycotoxins - toxins produced by fungi during growth or storage of grains. In the past, ergotism caused by ergot alkaloids in grain has been one of the major public health problems. Currently, contamination of corn and other grains with aflatoxin, fumonisins, zeralenone, trichothecenes, ochratoxin, citirin, and many other mycotoxins cause billions of dollars of losses world wide. These losses include direct loss of grain, reduced animal performance, and mortality of animals due to use of mycotoxin-contaminated feed grains. Effects on human health are less easy to document, but several mycotoxins (e.g., aflatoxin and fumonisins) are carcinogenic. Most of the mycotoxins that may occur in cereal grains are also found, but often to a lesser extent, in other feedstuffs, such as soybeans, soybean meal, cottonseed meal, etc. Harvesting whole plants of cereal grains as forage may lead to mycotoxin infestation of the feedstuff, especially if the forage is harvested as silage (e.g., corn silage) at less than optimum moisture. Inadequate moisture will lead to poor compaction and promote mold growth.

Ergot

The general effectors of ergot on livestock can be categorized as follows:

1. Behavioral effects - convulsions, incoordination, lameness, difficulty in breathing, excessive salivation, and diarrhea;

2. Dry gangrene of the extremities;

3. Reproductive effects - abortion, high neonatal mortality, and reduced lactation; and

4. Reduced feed intake and weight gain.

These effects are not seen in all types of livestock; they are fairly species specific and are modified by the ergot source, amount consumed, period of exposure, and age and stage of production of the animal. Two general effects of ergotism are convulsions and gangrene.

Cattle may exhibit both convulsive and gangrenous ergotism. Gangrene of the ear tips and tail may occur, but generally the feet are affected and signs include tenderness of the hind feet. There is little affect on reproduction, with abortion and agalactia (which are seen in swine) not observed.

Sheep that consume ergot exhibit difficulty in breathing, excessive salivation, diarrhea, and internal bleeding within the digestive tract. Sheep tend not to graze on grass flowers, and therefore, are less affected than cattle due to a difference in grazing behavior. **Horses** grazing grasses infected with ergot may develop symptoms of convulsion.

Convulsions and gangrene caused by ergotism are usually not seen in **swine**. Abortions may occur, and newborn pigs have a high rate of mortality due to depressed lactation by affected sows. Swine are less sensitive than other livestock to ergot. Growing pigs fed ergotized grain may have reduced feed intake, lowered rate of gain, and gut lesions. Mature **poultry** develop comb gangrene as a major symptom. Ergotism in chicks is characterized by depressed growth, poor feathering, nervousness, incoordination and inability to stand, and the beaks, toenails, and toes become dark and necrotic (Rotter et al., 1985ab). Broiler chicks may be slightly more sensitive to ergot than layer (Leghorn) chicks (Rotter et al., 1985a).

Hyperthermia (elevated body temperature) and susceptibility to heat stress occur in livestock consuming ergot alkaloids (Ross et al., 1989). Pronounced depression in serum prolactin levels also occur, accounting for the agalactia. Ergot alkaloids are not transferred to the milk of cows consuming ergot.

Treatment and Prevention of Ergotism. Removal of animals from ergot-infected pastures or removal of contaminated grain from feed is the only effective treatment. Ergot infestations of grains can be minimized by using clean seed, crop rotation, and deep cultivations. Growing ergot-resistant grains (wheat, barley, and oats) rather than rye or triticale may be advisable in areas where ergot is a problem.

Sclerotia can be removed from grain by standard seed-cleaning techniques. Of course, the screenings from ergotized grain should not be used in feeds. Infected grain can be blended with clean grain to reduce the ergot concentration to a nontoxic level. The tolerance level for ergot in grain in the U.S. is 0.3% crude ergot alkaloid. Levels of 0.1% ergot in complete feeds may have adverse effects on livestock performance.

Aspergillus and Penicillium Toxins

Aflatoxins are a family of compounds produced primarily by toxigenic strains of *Aspergillus flavus* and *Aspergillus paratiticus*. Only about one-half of the known strains of A. *flavus* and A. *parasiticus* produce aflatoxin. Although other fungi, such as *Penicillium* spp., *Rhizopus* spp., *Mucor* spp., and *Streptomyces* spp. are capable of producing aflatoxin, their relevance to livestock production has not been established.

The moisture content of the seed is probably the most important factor. In general, mold growth and aflatoxin formation require a moisture content greater than 14%, a temperature of at least 77°F, and some degree of aeration (O2). When these requirements are met, mold infestation followed by aflatoxin formation are likely to occur.

Three major feedstuffs with high potential for invasion by Aspergillus spp. during growth, harvesting, transportation, or storage are corn, cottonseed, and peanuts. Colonizations on soybeans and small grains generally occur in storage. Storage conditions for soybeans that promote aflatoxin formation, aside from optimal moisture and temperature condition, are lack of aeration systems or their improper use (temperature differential can cause moisture migrations), kernel damage and spore dissemination caused by storage, insects, presence of fines (dust, weed seeds, and broken kernels), and poor sanitary practices in feed areas. In corn, elevated aflatoxin

content in any particular year is usually the result of increased invasion by molds prior to harvest. Drought-stressed corn is susceptible to damage from insects, such as corn earthworms or borers, that feed on the husks or kernel of the ear. Kernels with disrupted seed coats are more accessible to fungal spores that may be present on the sides or in the bodies of insects. Other factors known to be associated with increased aflatoxin contamination of corn include corn left in the field beyond maturity, close planting, competition from weeds, and inadequate fertilization. Stored corn, particularly ground, high-moisture corn has the potential for aflatoxin production. Storage in air-tight silos or incorporation of certain preservatives can effectively retard mold growth. Inadequately dried corn will probably be invaded by various molds. Prepared feeds left for more than a day or two in feeding bins and troughs are also susceptible.

When aflatoxin-containing cottonseed is processed for oil, most of the toxin is concentrated in the meal. Feeding dairy cattle cottonseed meal contaminated with aflatoxin is a problem because of possible translocation of the metabolite aflatoxin M1 into milk.

Acute intoxication of farm animals is less likely to occur than chronic aflatoxicosis. The principal target organ in all species is the liver. Numerous liver functions are affected, and the cumulative impact can be fatal to animals. As the liver loses function, other effects appear, such as impaired blood clotting, jaundice, and reduction in serum protein. Rabbits and ducks are highly sensitive to aflatoxin, whereas sheep are less sensitive. The acute lethal dose (LD50) is not very useful in predicting actual field contamination problems.

Chronic intoxications (chronic poisoning or aflatoxicosis) can result when low levels of toxin are ingested over a prolonged period of time. In general, affected livestock exhibit decreased growth rate, lowered productivity (milk or eggs), and immunosuppression. Reduced growth rate is considered the most common effect associated with chronic aflatoxicosis in farm animals. In young animals fed low levels of toxin, this may be the only detectable abnormality. The lack of other clinical signs frequently cause aflatoxicosis to remain undiagnosed, resulting in serious economic loss. Liver damage is also prevalent in chronic aflatoxicosis in all species. At necropsy, the liver is usually pale to yellow, and the gall bladder may be enlarged.

Swine. Feed containing 0.4 ppm or greater aflatoxin B1 fed from weaning to market weight can adversely affect pigs. Among the mildest effects are decreased feed efficiency and poor rate of gain. More severe effects may result in death, acute hepatitis, systemic hemorrhage, or nephrosis.

Although baby pigs are more sensitive than older pigs, exposure during gestation or during suckling is generally not great enough to cause adverse effects. However, stunted growth has been observed in piglets that nursed on sows fed contaminated feed; aflatoxin M1, which is a toxic metabolite of aflatoxin B1, produced in the liver, is transferred into milk. There is no evidence that aflatoxin is detrimental to the reproductive performance of either sows or boars. However, there are indications that aflatoxin may suppress the immune system of pigs, causing decreased resistance to infectious diseases in the field.

Stress from handling can suddenly trigger widespread hemorrhaging; pressure in the ham can result in ataxia, exemplified by the pig assuming a dog like sitting position. Administration of vitamin K is an effective treatment. Moreover, supplementation of the diet with additional

vitamin K or menadione has been shown to improve weight gain during this recovery period. Also, supplementation with additional protein affords some protection during exposure.

Poultry. Avian species are quite variable in sensitivity to chronic aflatoxicosis. Turkey poults and ducklings are the most sensitive; a dietary level of 0.25 ppm impairs their growth, whereas levels of 1.5 ppm in broiler and 4 ppm in Japanese quail are required to reduce growth rate. Age is another factor causing variable toxicity. Poults and ducklings are more sensitive than their adult counterparts, whereas no age effect is apparent in chickens.

While growth retardation is usually the first adverse effect to appear in mammals, this is not the case in birds. Impaired blood coagulation, increasing susceptibility to carcass bruising, and decreased resistance to infections result from dosages below those affecting growth. Dietary concentrations of aflatoxin greater than 2 ppm can significantly diminish egg production in layers, with production decreased to 50% with 10 ppm and 0% at 20 ppm.

Increasing dietary protein content counteracts growth impairments in broilers. Increasing the fat content of the diet reduces mortality in broilers but does not overcome other toxic effects associated with aflatoxicosis. In contrast, a deficiency of thiamin or vitamin A afford some protection against aflatoxicosis. Rickets and decreased bone strength are symptoms of aflatoxicosis. In chickens, impaired lipid transport occurs at dosages considerably lower than that required for reduction of growth rate (may cause "fatty livers").

Ruminants. Early signs of poisoning include reduction in feed intake, followed by weight loses or decreased rate of gain. Other effects associated with chronic intake of aflatoxin-containing feed in dairy and beef cattle are decreased feed efficiency, immunosuppression, increased susceptibility to stress, and decreased reproductive performance. Calves are more sensitive than adult cattle. A dosage level of about 0.2 mg/kg of body weight per day causes reduced rate of gain and impaired blood coagulation in calves. In young beef cattle (400 to 500 lb) and in yearling steers, diets containing 7 to10 ppm of aflatoxin cause decreased growth and feed efficiency. Recovery of normal rates of feed intake and growth after exposure cease is very slow.

Although adult cattle are not as sensitive and the signs are not as evident as in growing animals, chronic aflatoxicosis is characterized by anorexia, a drying and peeling of skin or the muzzle, prolapsed rectum, liver damage, elevated levels of blood constituents, such as cholesterol and bilirubin, and edema in the abdominal cavity. There is growing evidence of anti-reproductive effects of aflatoxin in ruminants, including decreased fertility in sheep and abortion and birth of underweight calves in cattle. Milk production may be dramatically decreased in dairy cows fed aflatoxin-containing feed. However, it is not clear to what degree this effect is the direct result of decreased feed intake. No effect on milk fat has been observed. In cows given aflatoxin and then challenged with mastitis-causing microbes, there were pronounced teat inflammations and higher bacteria counts in milk than for mastitic cows receiving no aflatoxin (Brown et al., 1981). Numerous countries, including the Unites States, have legislation on limits of aflatoxin in feedstuffs for dairy cattle and aflatoxin M1 concentration in milk and milk products. The U.S. Food and Drug Administration prohibits interstate movement of feed containing more than 20 ppb of aflatoxin and prohibit the sale of milk with more than 0.5 ppb aflatoxin. Approximately 1.0% of the aflatoxin consumed will be excreted in the milk. Milk products, especially cheeses,

will be more highly concentrated with aflatoxin than the milk from which they were made. Ingested aflatoxin is rapidly eliminated from the body, so tissue residues in meat are not significant.

Carcinogenesis. Aflatoxin is carcinogenic in several species, including rats, ducks, mice, trout, and subhuman primates. Dietary levels of aflatoxin B1 as low as 15 ppb fed chronically to rats caused a high rate of hepatic carcinomas (tumors). Carcinogenesis in livestock and poultry appear to be a rarity but has been reported in swine fed contaminated feed for long periods (> 2 years). Pigs that survived the acute toxicity phase after being fed highly contaminated cottonseed and peanut meal all developed hepatic carcinoma much later (Carnaghan and Crawford, 1964). The lack of observed carcinogenesis in livestock could also be because they are usually marketed long before tumors would become clinically apparent. Rainbow trout are extremely susceptible to aflatoxin-induced liver cancer.

There has been much speculation linking aflatoxin consumption and liver cancer in humans based on numerous epidemiological studies which suggest such an association (Hall and Wild, 1994). These interactions are particularly important in tropical countries because of widespread contamination of food with mycotoxins and a high incidence of viral hepatitis.

Feed Processing to Reduce Aflatoxin Exposure. Aflatoxin production in corn and other commodities can occur both pre- and post-harvest. Drought and insect damage are the two most frequent causes of fungal infestation of corn. Prevention of mold growth and aflatoxin formation in stored grains is accomplished by proper storage conditions of low humidity and protection against exposure to moisture. Preservatives and anti-fungal agents, such as propionic acid, may be somewhat effective in reducing aflatoxin formation but should not be used as a substitute for proper drying and good post-harvest management. The most effective method of detoxification of aflatoxin-contaminated grain is ammoniation with anhydrous ammonia (Phillips et al., 1994; Park et al., 1988). Ammoniation can reduce aflatoxin levels by more than 99% (Phillips et al., 1994).

Another approach to reducing aflatoxicosis in livestock is the use of dietary additives that bind aflatoxin and prevent their absorption from the gut. These include various clay minerals, such as bentonites and hydrated sodium calcium aluminosilicates (Lindemann et al., 1993.). At a concentration of 0.5% of the diet, aluminosilicates are effective at binding aflatoxin and preventing their absorption in both ruminants and nonruminants. Some other commercial products are sold as "mycotoxin binders", with effectiveness possibly variable and unknown by the authors. Binders are not effective with other mycotoxins, one cannot make claims that binding agents reduce all toxins, and these absorbent materials are not specifically approved by FDA for the prevention or treatment of mycotoxicoses. There is evidence that binders are nonselective and may bind minerals and vitamins, thus reducing their absorption. Therefore, adequate mineral and vitamin supplementation must occur when using these binders.

Ochratoxin

The ochratoxins are produced by several species of *Aspergillus* and *Penicillium*. Kidney problems are a common symptom of ochratoxicosis. Ochratoxicosis, particularly kidney disease

in swine, is quite prevalent in certain regions and is usually associated with the feeding of contaminated barley. Ochratoxin A (OA) has also been detected in wheat, oats, barley, corn, beans, peanuts, hay, green coffee beans, and mixed feeds.

In general, optimal conditions for OA production are a moisture content of 19 to 22% and a temperature of about 75°F. Toxin production can still occur at temperatures as low as 39°F. Surveys of feedstuffs for OA generally reveal frequent contamination in the cooler climates of the world, such as North America and Europe. However, usual concentrations of OA are below toxic levels. Ammoniations of OA-containing grain is effective in eliminating it's toxicity (Marquardt and Frohlich, 1992). Binding agents have not generally been effective or economical. Supplemental ascorbic acid (vitamin C) counteracts the toxicity of OA to some degree (Haazele et al., 1993).

Biological effects. At necropsy, the kidneys appear gray in color, have a granular surface, and are usually enlarged. Edema occurs in swine. Although the liver is not a major target organ, some toxic effects occur there.

Effects in different species. Among farm animals, monogastric species (horses, swine, and poultry) are much more sensitive to ochratoxins than ruminants. As with other mycotoxins, impaired growth in young animals, including calves, is the first observable sign of intoxication. Young animals are clearly more sensitive than adults. Ochratoxicosis is generally not diagnosable until postmortem examination of kidneys is conducted.

Adult ruminants are afforded protection by rumen microorganisms, particularly by protozoa. Hydrolysis of OA is greater in ruminants fed hay instead of grain, suggesting that OA-contaminated grain would be most effectively detoxified if considerable hay were included in the diet.

In swine, the primary syndrome is called porcine nephropathy, which generally occurs after chronic ingestion of diets with concentrations of 0.2 to 4 ppm OA. The effects on the kidneys are comparable to those seen in a variety of other species (e.g. poultry, rats, fish, and monkeys) and are similar to the human syndrome called Balkan nephropathy.

Avian ochratoxicosis has been documented in broilers, layers, and turkeys. As in other species, the principal effect is nephropathy. The oral LD50 varies among different species (e.g. broilers, 3.4 mg/kg; poults, 5.0 mg/kg; and quail, 16.5 mg/kg). Other clinical signs are neurological effects, which include tremors, flailing, and loss of righting reflex. Another unique effect in poultry is visceral gout, characterized by white urate deposits throughout the body cavity and internal organs. The OA may decrease bone strength in young broiler chicks. In layers, concentrations of OA as low as 0.5 ppm can decrease egg production and feed consumption. In turkeys, ochratoxicosis is characterized by mortality, decreased growth rate and feed efficiency, nephropathy, and decreased carcass pigmentation. The latter effect may result in an increase in the number of carcass condemnations.

Swine known to have OA exposure should be fed OA-free feed for at least four weeks before slaughter to allow OA elimination from tissues. Compared to other species, the pig eliminates

OA residues quite slowly. The residue problem in chickens is less than for swine; concentrations up to 30 ppb have been found in chickens with avian nephropathy. In ruminants, OA hydrolysis in the rumen decreases the possibility of residue as well as toxicosis.

Citrinin

Citrinin-induced kidney damage is similar to that caused by other mycotoxins. Citrinin and OA frequently coexist in moldy feed. The present consensus of opinion is that citrinin is a contributor rather than a primary cause of kidney problems in swine and poultry.

Fusarium Toxins

Zearalenone. Zeralenone can cause estrogenic effects and infertility in animals. Swine are very sensitive to zeralenone, but the presence of refusal toxins (trichothecenes) may limit intake. The syndrome is characterized by swelling of the vulva and mammary glands, anal prolapse, and vaginal prolapse. Poultry are extremely resistant to zeralenone toxicosis. Cattle and sheep are less sensitive than pigs to the estrogenic effects.

Corn is the most frequently contaminated crop. Production of zeralenone usually occurs in storage when moisture and temperature conditions are not optimal. However in the field, infected ear corn may develop a rot on the crown or cob (Gibberella rot). The ear is most susceptible to Gibberella rot during silking. It is believed that ideal conditions for development of Gibberella rot are chronic rainfall combined with mean temperature of > 70°F during silking. The equivalent of Gibberella rot in wheat, barley, and oats is called scab, which is characterized by a dark discoloration of the kernel. The amounts of zeralenone produced by either Gibberella rot or scab in the field are usually less than caused by storage conditions.

Trichothecenes. Greater trichothecence production is usually found in years when the autumn is cool and wet and harvest is delayed. Frost-damaged corn is more susceptible to fungal invasion. Vomiting and feed refusal are associated with trichothecenes, particularly deoxynivalenol (DON; vomotoxin). Swine are especially sensitive to these toxic effects. As little as 5% infected kernels or about 10 ppm of DON in feed causes feed refusal in swine. It is not advisable to reverse the refusal by masking moldy feed (e.g., with molasses) because of the possible development of toxin reactions from other trichothecenes that are probably present in the feed. The DON has been very prevalent in the Midwest, and if it is found in feed, it is regarded by many feed industry personnel as the identifier that other mycotoxins may be present in the feed. Skin rashes can occur on animals and farm workers handling moldy hay or straw.

Fumonisins. Equine leucoencephalomalacia (ELM) is a lethal disease of horses characterized by neurological effects. Clinical signs include hypersensitivity, ataxia, posterior weakness, convulsions, and inability to swallow. The liver may also be damaged. The liver lesions are associated with short-term, high dose exposure to fumonisins, while brain lesions are associated with longer-term, lower-dose exposure. In pigs, the typical syndrome observed with fumonisin toxicosis is pulmonary edema. Consumption of moldy corn or corn screenings is the usual cause. Concentrations of fumonisins in corn screenings can be about 10 times higher than for intact

corn, indicating that corn screenings should be used with caution in animal and poultry feeds. Ammoniation is not an effective tool to detoxify fumonisin-contaminated corn.

Poultry are relatively resistant to some fumonisins. However, caution is needed because interactions with other toxic compounds may exist. Ruminants are less susceptible to fumonisin than are horses, swine, and poultry. Signs in ruminants are associated with liver and kidney function. Esophageal cancer in humans has been linked to consumption of fumonisin-contaminated corn.

General Management for Feeding Moldy Feed

Moldy Feeds

Mold growth, accompanied by heat and aerobic conditions, takes place in most feeds when their moisture content is above 15 to16%. The presence of mold does not necessarily mean that the feeds can not be used. Many molds do not produce toxic mycotoxins, thus may cause minimal problems when fed to livestock. However, some molds produce toxic mycotoxins that can be very harmful. Although mycotoxins may not be present, high mold counts can cause reduced feed intake and indigestion.

Symptoms vary with the particular mold and toxin. However, it helps to remember that cattle are more resistant, although not immune, to mold toxins than either swine or poultry. Young animals are more susceptible than mature animals, and pregnant or lactating animals are especially susceptible. High producing dairy cows and rapidly growing feedlot cattle are more susceptible to the effects of mycotoxins than low producing animals (e.g., yearlings on a growing ration).

The ruminal fermentation helps to reduce the harmful effects of some mycotoxins. Mycotoxins may be detoxified or altered in the rumen, but the rate of detoxification can differ for the different types of mycotoxins. The extent of detoxification of any particular mycotoxin partially depends on the rate of passage of feed from the rumen. Rumen turnover rates are about eight times longer in a beef cow than in a lactating dairy cow, caused by level of dry matter intake. Ruminants are nevertheless susceptible to the deleterious effects of molds and mycotoxins.

Mycotoxins can have a very pervasive, yet subclinical, effect on both performance and health in animals that can easily go unnoticed. If you wait until clinical symptoms of mycotoxin problems are obvious, you no doubt have waited too long.

Don't wait for a necropsy - monitor feed intake and conduct standard feed tests. Typically, moldy feeds are less palatable and may reduce dry matter intake (Figure 1). This will lead to a reduction of nutrient intake, reducing weight gain or milk production. Performance losses of 5 to10% may occur with moldy feeds, even in the absence of mycotoxins. Moldy feed may reduce digestibility and energy content. Molds grow and propagate, deriving energy from the feed's protein, fat, and carbohydrates. Dietary fat, in particular, is reduced in mold infected feeds. Suggested book values for energy should be multiplied by 0.95 in the presence of suspected mold (i.e. 95% of energy value). Some research suggests reducing energy values by as much as 10% (i.e., 90% of energy value).

Ideally, we do not want to use moldy feed with our livestock. Moldy feeds may mean trouble, but sometimes we may have to consider its use (Figure 2). If moldy hay, grain, or concentrates are to be fed, observe the following precautions:

1. Send a representative sample of feed to a laboratory.

2. Introduce moldy feeds into the ration gradually. It takes cattle a few days to adjust to the poor taste and dust; some cattle never adjust.

3. Minimize the use of moldy feeds by mixing it with good quality feed.

4. If problems are encountered, stop using the moldy feed.

5. Do not cover the taste with molasses. Do not force them to clean-up moldy feed.

6. If possible, avoid feeding moldy feed to young, lactating, or gestating animals. These classes of animals are all particularly susceptible to problems caused by mold.

7. Feed moldy feed outside so as to reduce human and animal exposure to the effects of dust and spores on the respiratory system.

8. Producers should be aware of the health hazards involved in working with moldy feeds and take every precaution to decrease personal exposure.

9. Screenings from moldy grain can be even higher in contamination due to concentrating the mold (Don't feed screenings from moldy feed).

Why We are Concerned

While problems with many plant toxicants are less now than in the past because of recognition of the cause and changes in feed processing, problems caused by mycotoxins are in many situations becoming of increasing importance. Factors responsible for this include continuous cropping with one crop (monoculture) which allows fungal populations to build up, increased corn and soybean production in the southern and southeastern sections of the U.S. where the climate is favorable to fungal growth, and additional knowledge of the effects of mycotoxins. Higher energy cost may prompt farmers to alter corn drying procedures and store corn at higher moisture content, allowing fungal growth during storage. Mycotoxin problems also show a marked seasonal variation. Epidemic years can be due to climatic factors, such as drought, predisposing the stressed plant to fungal infection. Flooding can also cause extensive problems with mycotoxins.

Warm humid conditions favor fungal growth. In many "tropical" areas, storage conditions are frequently inadequate. Epidemiological studies in Africa and Asia have shown a strong positive association between liver cancer rates in humans and dietary aflatoxin intake (Eaton and Groopman, 1994).

Most poisonous plants are broad-leaved forbs or shrubs and can be killed by phenoxy herbicides without damage to grasses; therefore, chemical spraying and reseeding with desirable vegetation may be warranted. Caution should be used when pastures are sprayed for the control of poisonous plants. There is often an increase in palatability of plants following spraying associated with the wilted condition or perhaps increase in sugars in the sprayed material. Livestock may consume poisonous plants after spraying, whereas previously they would not. It is advisable to remove animals from pastures for one to two weeks after spraying for weed control.

Poisonous plants are often localized in their distribution, and sometimes a relatively small stand of poisonous plants can have a major effect on livestock. Choke-cherry poisoning often occurs where the plant is located near water sources. Animals may consume choke cherry leaves as they are congregated for getting a drink, and when they drink, the water in the rumen speeds up the hydrolysis to yield free cyanide.

Animal management practices to avoid losses from toxins requires knowledge of the general characteristics of poisonous plants (Kruegar and Sharp, 1978): They are usually unpalatable, frequently become less toxic as they mature, usually make a greater proportion of the available forage in the early spring, and become proportionately less abundant as forage species begin growth. Hence, animal management on early spring forage is important. Schuster (1978) listed the following suggestions for management of livestock to minimize losses from poisonous plants:

1. Learn identification and toxic principles of poisonous plants.

2. Use good grazing management to maintain forage in a condition that is not conducive to the development of high densities of poisonous plants.

3. Adjust stocking rates so that animals have ample availability of forage relative to the amount of poisonous plants present.

4. Supplement with salt, minerals, and other nutrients as needed.

5. Avoid grazing livestock in areas where toxic plants are abundant by herding or fencing off the infected area.

6. Use a class of livestock not generally poisoned by the plants present.

7. Avoid turning hungry animals onto pasture containing poisonous plants. This is especially important when sheep or cattle are released after being transported or worked in a corral.

8. Provide adequate watering facilities to prevent nonselective grazing following water deprivation and subsequent watering.

9. Reduce poisonous plant populations by use of mechanical, chemical, biological, or other control methods.

In some cases, providing dietary supplements to livestock may help to control losses from a toxicant. It is often believed, although definitive proof seems to be lacking, that lack of salt and minerals may increase the likelihood that animals will consume poisonous plants. Livestock deficient in phosphorus may develop a depraved appetite (pica), which could result in consuming toxic plants. Another explanation of this finding is that farmers who provide mineral supplements probably also provide superior pasture management.

<u>Human Concerns.</u> Mold spores are of concern. When inhaled, the spores can cause the lungs to become abnormally sensitive to these particular spores. Chronic respiratory disease and even death can occur if exposure to the moldy feedstuff is continued. The spores are doubly dangerous because farmers can develop sensitivity known as "Farmers Lung." Symptoms appear four to eight hours after exposure to spores and can include headache, loss of appetite, fever, and chills. Body temperature may rise to 105oF. There are varying degrees of shortness of breath along with a dry, hacking cough. Although other symptoms subside quickly, breathlessness may persist for several weeks and improve slowly. Continued exposure can lead to irreversible lung damage, but people vary in susceptibility. At present, the only real cure for Farmers Lung is to keep away from the molds responsible for the disease.

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Figure 1. Action chart for defining strategies for dealing with mold and mycotoxin-contaminated feeds.[1]

When to Test	
Field conditions	• Stalk rot or ear rot in corn • Scab in wheat or barley
Grain conditions	• Low bushel weight • Visible mold, musty odor
Animal conditions	• Reduced dry matter intake • Respiratory distress, diarrhea • Reduced production or growth

What to test for:	**Purpose of test:**

Moisture/dry matter	Indicator of mold growth conditions
Crude protein, crude fat	Indicator of mold growth activity
Starch	To evaluate effects of mold activity when formulating diets
NDF, ADF, ash	May increase when mold utilizes energy in grain
Mold count	Discount energy and protein values when >1,000,000 cfu/g
Mycotoxins	Test when any of the �when to test� conditions exist
Management	
Mycoses	• Use feed commodities with low spore aerosilation potential • Blend with better quality feedstuffs at feeding
Mycotoxicoses	• Blend to acceptable nontoxic concentration at feeding, except for aflatoxin-contaminated commodities • Use of clay, bentonite, or aluminosilicates can reduce effects of aflatoxin in some animals

[1]Taken from DiCostanzo et al. (1995).

Figure 2. Management to minimize problems with molds and mycotoxins.

Strategy	Time Period	Management Item	Guidelines
PREVENTION	Harvest	Proper moisture	• Less than 15% moisture for dry grains • No less than 22% moisture for high-moisture grains for ensiling • If proper moisture not achieved, but only marginally high (grain) or low (silage), application of propionic acid may be helpful • If aflatoxin is known to be present, ammoniation can be helpful
		Equipment	• Equipment should be operating properly to minimize broken kernels of dry grains
	Storage	Grain	• Bins should be leak-proof • Remove old-grain, damaged kernels, and any foreign matter from bottom of bin • Grain with elevated moisture should be aerated and/or dried with heat • Check grain periodically for temperature, moisture, and insect

			damage
		Silage	• Structure should be well-sealed • Discard moldy areas when silo is first opened or when moldy pockets are found during feeding • Feed at sufficient rate per day to minimize spoilage (must feed more in summer)
MINIMIZING PROBLEMS WITH THE MOLDS AND MYCOTOXINS PRESENT	Feeding	Ration formulation	• If molds and mycotoxins are suspected, see Figure 1 for �What to test for� • Based on test results, formulate ration to account for quality of contaminated feed • Dilute contaminated feed with good quality feed and feed the mixture to the following animals (ranked in descending order of preference): non-lactating, non-gestating adult animals; growing animals; gestating animals; lactating animals (especially high yielding); and neonate animals
		Feed additives	• Consider the feeding of binder materials, especially if alfatoxin is present • Adding a feed stablizer at the time of mixing may increase bunk life when high-moisture feed is fed
		Last alternative	• Discard feed