

Understanding and Coping with Effects of Mycotoxins in Livestock Feed and Forage

Safe Levels of Mycotoxins

Even with the best quality-control systems in the world, animal producers often find themselves owning mycotoxin-contaminated grain or feed. 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. The determination as to whether or not a given concentration of mycotoxin is safe will depend on at least the factors which follow (and there may also be other factors).

1. **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, but if just one chemical bond is changed in the structure of the molecule, its toxicity can be reduced dramatically.
2. **Presence of other mycotoxins.** A number of studies have demonstrated that mycotoxins occur simultaneously in field situations. This simultaneous occurrence can profoundly affect the toxicity of the mycotoxins present.
3. **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.
4. **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.
5. **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.

6. **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 percent difference in growth rate. Yet, in integrated-poultry operations a 1 percent difference in growth rate would have a significant economic impact.
7. **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.
8. **Length of time animals are 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, 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.

Table 1. A Partial List of Known Mycotoxins

Aflatoxins	Fusaric Acid	Penicillic Acid
Alternariol	Fusariocin	Penitrem
Citreoviridin	Fusarins	Phomopsin
Citrinin	Islanditoxin	Roridins
Cyclopiazonic Acid	Luteoskyrin	Rubratoxin
Deoxynivalenol	Moniliformin	Slaframine
Diacetoxyscirpenol	Monoacetoxyscirpenol	Sporidesmin
Dicoumarol	Neosolaniol	Stachbotryotoxins
Ergotamine	Ochratoxins	Sterigmatocystin
Ergo Toxins	Oosporein	T-2 Toxin
Fumitremorgen	Paspalitrem	Tremorgens
Fumonisin	Patulin	Zearalenone

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Available Mycotoxin Data

Although between 300 and 400 mycotoxins are known, those mycotoxins of most concern, based on their toxicity and occurrence, are aflatoxin, deoxynivalenol (DON or vomitoxin), zearalenone, fumonisin, T-2 toxin, and T-2-like toxins (trichothecenes). In a recent survey of suspect feed samples, some amount of aflatoxin, deoxynivalenol, or fumonisin was found in over 70 percent of the samples tested. Over a 10-year period, data collected from suspect samples analyzed at the North Carolina State University (NCSU) Mycotoxin Laboratory show: that 20 parts per billion (ppb) or more aflatoxin occurred in 34 percent of corn samples tested; deoxynivalenol was detected in over 60

percent of poultry and dairy feeds tested; zearalenone was present in 15 to 20 percent of feedstuffs tested; and T-2 toxin was present in about 5 percent of the feeds tested. Fumonisin, a mycotoxin often associated with horse deaths, is thought to occur very frequently; however, its discovery is so recent that data on occurrence has not been established. Table 2 contains a summary of mycotoxin analyses obtained from suspect feeds, feed ingredients, and forages collected in North Carolina from 1989 through 1993. While the concentration of aflatoxin in suspect samples has remained fairly constant, the incidence of aflatoxin contamination has decreased. Nonetheless, both the incidence and concentration of aflatoxin can change as suddenly and dramatically as the weather. Thus aflatoxin monitoring should not be neglected. Furthermore, the effects of high levels of aflatoxin on animals are well documented, but exposure of animals to low levels of aflatoxin, especially when combined with other conditions or mycotoxins, can produce confusing symptoms, particularly in field situations.

Deoxynivalenol (DON), zearalenone, T-2 toxin, and fumonisin are all produced by molds of the genus *Fusarium*. Molds in this genus are found in virtually every lot of corn and collectively are capable of producing 70 different mycotoxins. Some strains of *Fusarium* may produce as many as 17 mycotoxins simultaneously. Thus *Fusarium* mycotoxins are the most frequently identified group of mycotoxins in grains and feeds.

Better control of mycotoxins will position producers for greater competitiveness and profitability. In addition, control of mycotoxins in animal diets would reduce the likelihood that mycotoxin residues would appear in animal products destined for human consumption.

Table 2. NCSU Mycotoxin Laboratory Analyses Obtained From Suspect North Carolina Feeds, Feed Ingredients, and Forages Collected From 1989 Through 1993.

	Aflatoxin			DON			Fumonisin*	
	n	Av	%P	n	Av	%P	n	%P
MIXED FEEDS								
1989-1993	342	116	11.4	572	1169	67.5	222	28.4
1989	38	46	34.2	61	2727	62.3	0	-
1990	40	358	22.5	107	802	73.8	0	-
1991	46	34	8.7	116	511	62.1	0	-
1992	77	24	2.6	94	643	48.9	85	23.5
1993	141	48	7.8	194	1443	77.8	137	31.4
TMR	28	48	17.8	44	1379	61.4	12	25.0
Forages	56	57	8.9	177	1671	66.1	54	26.4
Concentrates	257	138	11.3	350	903	69.1	156	29.5
Corn Grain	52	346	17.3	63	599	63.5	33	60.6
Soybean Meal	11	30	9.1	14	220	57.1	6	0
Cottonseed	39	135	5.1	52	2679	46.2	26	3.8

Corn Silage	36	66	11.1	106	1847	75.5	33	39.4
Grass Hay	4	-	0	26	1110	42.3	9	0
Small Grain & Grass Silage	13	-	0	36	1568	61.1	9	11.1

	T-2 Toxin			Zearalenone		
	n	Av	%P	n	Av	%P
MIXED FEEDS						
1989-1993	430	364	4.2	538	397	18.0
1989	41	151	7.3	62	552	27.4
1990	47	2456	2.1	86	524	18.6
1991	62	-	0	104	375	2.9
1992	86	-	0	91	108	1.1
1993	194	132	7.2	195	325	30.8
TMR	36	-	0	43	237	30.2
Forages	118	290	2.5	169	379	24.8
Concentrates	275	379	5.5	325	464	12.9
Corn Grain	58	1603	3.4	57	294	5.3
Soybean Meal	12	87	16.7	13	113	46.1
Cottonseed	41	274	4.9	50	292	6.0
Corn Silage	69	60	2.9	93	445	32.3
Grass Hay	19	-	0	29	114	13.8
Small Grain & Grass Silage	26	750	3.8	36	283	19.4

NOTE: 586 samples were tested. 2104 individual mycotoxin tests were performed. 73.2% of samples were positive for at least one mycotoxin. 61% of samples were positive for aflatoxin and one other mycotoxin. 28.7% of samples were positive for all individual mycotoxin tested.

n = Number of Assays.

Av = Average concentration (ppb) in positive samples.

%P = Percentage (of samples) Positive. Note: Percentages are based on the following levels--Aflatoxin >20 ppb, DON >40 ppb, Fumonisin >5,000 ppb, T-2 Toxin >50 ppb, Zearalenone >70 ppb.

TMR = Total Mixed Ration for ruminants includes the concentrates and forages which make up the animals' total diet.

*Concentration was not determined on Fumonisin samples.

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Mycotoxins and Animal Health

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. While healthy animals tend to "filter out" or detoxify many of the mycotoxins to which they are exposed, in our consumer-oriented society the issue of mycotoxin residues in milk and animal tissues should not be ignored.

Mycotoxins produce a wide range of harmful effects in animals. 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. In comparison to other animals, poultry species tend to be resistant to the effects of fumonisin, deoxynivalenol, and zearalenone. 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. Since mold activity can generate numerous other mycotoxins as well as reduce the nutritive value and palatability of feeds, the presence of fumonisin, deoxynivalenol, or zearalenone in poultry feeds is cause for concern.

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. Nursing animals may be affected by exposure to aflatoxin metabolites secreted in the milk. 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.

Deoxynivalenol (DON) is, perhaps, the most commonly detected *Fusarium* mycotoxin. DON has been associated with reduced milk production in dairy cattle, 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. In addition, DON levels greater than 500 ppb have been associated with numerous other field problems.

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. At higher doses zearalenone interferes with conception, ovulation, implantation, fetal development, and the viability of newborn animals.

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. Affected animals show signs of weight loss, poor feed utilization, lack of appetite, vomiting, bloody diarrhea, abortion, and (in severe cases) death.

Fumonisin is a mycotoxin which has only recently been discovered. Thus it has not been extensively studied. Nonetheless, it is known that in most animals fumonisin impairs immune function, causes liver and kidney damage, decreases weight gains, and increases mortality rates. Fumonisin also causes leukoencephalomalacia in horses and respiratory difficulties in swine. In some animals fumonisin can also cause tumors. [Click here to return to the Table of Contents.](#)

Effects of Mycotoxins on the Health and Productivity of Specific Production Animals

Swine

Swine are sensitive to mycotoxins, especially nursing or nursery-age swine. In general, mycotoxins cause reductions in feed intake, growth performance, and immune function when levels are relatively low. Producers must be aware that if one toxin is identified in a sample, the chances are high that other toxins are present. Some toxins may not have been identified as of yet, but research on known mycotoxins provides insight into the expected effects in swine and potential methods to reduce those effects. Table 3 contains a summary of the maximum permissible concentrations of mycotoxins in swine feeds.

Aflatoxin B1 has been the most extensively studied. Twenty to 200 ppb will cause a decrease in feed intake and growth performance, which can be partially offset by increasing specific dietary nutrients such as lysine or methionine. In severe cases (1,000 to 5,000 ppb) of aflatoxicosis, one can expect acute effects including death. Aflatoxin M1 appears in milk of sows consuming aflatoxin-contaminated diets and may affect piglets nursing those sows.

Feed concentrations of deoxynivalenol (DON) of 300 to 500 ppb are often associated with feed refusal, decreased weight gain, and increased incidence of infectious diseases. DON levels greater than 1000 ppb, will cause feed refusal or decrease in feed intake resulting in severe weight loss. It appears that pigs will often consume a sufficient amount of contaminated feed to induce vomiting. In fact, DON is also called vomitoxin because of its association with swine vomiting.

T-2 toxin has detrimental effects on swine performance, but no effect levels have not been determined for commercial production environments. However, field observations indicated that T-2 and related compounds are associated with decreased productivity at feed concentrations of 200 ppb or less.

Zearalenone will significantly affect the reproductive performance of swine. Prepuberal gilts are the most sensitive to zearalenone. The symptoms commonly observed when feeding diets contaminated with zearalenone include a reddening and increased size of the vulva, and increased size of mammary tissue. Zearalenone will cause embryonic mortality at certain stages of gestation. Fertility problems are often associated with zearalenone concentrations of 100 to 200 ppb in sow feeds.

Table 3. Maximum Mycotoxin Levels for Swine

Maximum Dietary Concentration

Swine Type	Deoxynivalenol ppb	Zearalenone ppb	Aflatoxin ppb
Pigs <75 lb	<300	200	20
Pigs 75 to 125 lb	<300	200	50
Pigs 125 lb to market	<300	200	100
Sow Herd	<300	100	50
Breeding Males	<300	100	50

These levels are based on extensive field observations.

Heat stress, marginal nutrient plane, crowding, disease exposure, the presence of more than one mycotoxin, and drug interactions, as well other factors, increase animals' susceptibility to mycotoxins. Thus these recommendations must be tempered with knowledge of the animals involved.

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Poultry

Aflatoxin affects all poultry species. Although it generally takes relatively high levels to cause mortality, low levels can be detrimental if continually fed. Young poultry, especially ducks and turkeys, are very susceptible. As a general rule, growing poultry should not receive more than 20 ppb aflatoxin in the diet. However, feeding levels lower than 20 ppb may still reduce their resistance to disease, decrease their ability to withstand stress and bruising, and generally make them unthrifty.

Laying hens generally can tolerate higher levels than young birds, but levels should still be less than 50 ppb. Aflatoxin contamination can reduce the birds' ability to withstand stress by inhibiting the immune system. This malfunction can reduce egg size and possibly lower egg production. In addition, one must pay special attention to the use of contaminated corn in layer rations because eggs are promptly used as human food and aflatoxin metabolites have been found in egg yolks.

Mycotoxin levels found in most field situations tend to be low. Yet the combination of low levels of mycotoxins with the stresses associated with commercial production situations and/or exposure to disease organisms can produce effects in poultry which are subtle, indirect, and sometimes ill-defined. Since the effects of mycotoxins on poultry are dependant upon the age, physiological state, and nutritional status of the animals at the time of exposure, and since mold growth at various points within the feed production and distribution system can magnify mycotoxin problems, mycotoxicoses can be difficult to diagnose in field situations.

Mycotoxins produced by the mold genus *Fusarium* include: T-2 toxin and it's chemical relatives (trichothecenes), deoxynivalenol (DON), fumonisin, and zearalenone. Other animals tend to be more sensitive to the effects of fumonisin, deoxynivalenol, and zearalenone when compared to poultry. Nevertheless, detection of these mycotoxins within poultry rations indicates that the ration or the ingredients within the ration have been subjected to mold activity. Since numerous other mycotoxins, as well as reduced nutritive value and palatability of feeds, are generated by mold activity, the presence of fumonisin, deoxynivalenol, or zearalenone in poultry feeds is cause for concern.

T-2 toxin and trichothecenes can cause mouth and intestinal lesions as well as impair the birds' immune response, causing egg production declines, decreased feed consumption, weight loss, and altered feather patterns. While much is yet to be learned, T-2 toxin and related compounds are currently thought to be the most potent *Fusarium* mycotoxin for poultry.

DON alone has few effects in poultry. However, in field situations the DON level is sometimes associated with reduced feed consumption in layers and broiler breeders. This means that DON may be an indicator that T-2 or other unknown *Fusarium* mycotoxins are present. [Click here to return to the Table of Contents.](#)

Horses

Although the effects of mycotoxins on horses are not well documented in scientific literature, in field situations apparent mycotoxin problems appear to be significant. Mycotoxins have been implicated in a variety of health problems including colic, neurological disorders, paralysis, hypersensitivity, and brain lesions. The cumulative effect of feeding low levels of mycotoxins may also contribute to a gradual deterioration of organ functions. This in turn affects growth rate, feed efficiency, fertility, respiration rate, the ability to perform work, and life span. Cases of mycotoxin-related horse deaths are consistently reported throughout the southeastern United States. Due to the lack of

conclusive scientific research concerning the levels of various mycotoxins tolerated by the horse, emphasis should be placed on feeding mycotoxin-free grain and forage to all horses.

Horses are herbivores with a simple stomach (nonruminant). The large intestine has an active microbial digestive ability to allow digestion of forages. However, in the horse the small intestine, which is the major site of absorption, occurs before the fermentative digestion. As a result, horses are more susceptible to mycotoxins than ruminants, since nutrient absorption occurs prior to fermentative digestion in the horse compared to ruminants in which absorption occurs after fermentative digestion.

Productive or working horses have a high energy requirement and require a higher concentrate intake, and thus would be most susceptible to problems with mycotoxin-contaminated grains. Working horses would include growing horses less than two years of age, brood mares in late gestation and early lactation, and horses at moderate or intense work levels.

Other horses, that are only lightly worked, would be more likely to be exposed to mycotoxin-contaminated hays or forages. Since moldy forages are generally less palatable than normal forage, horses fed moldy forages typically refuse feed before ingesting enough feed to cause severe intestinal tract damage. Mild colic is typically noted in such cases. Unfortunately, most molds associated with grains fed to horses do not readily affect palatability. Consequently, horses are most often exposed to the mycotoxins found in grains. Grain mycotoxins are readily absorbed and should be considered to be potentially lethal for horses.

If mycotoxin-contaminated feeds must be fed, follow these guidelines:

1. Levels in the total diet of mature, nonbreeding horses should not exceed the levels shown in Table 4.
2. Feed mycotoxin-free grains to growing horses (less than two years of age), breeding horses, and working horses.
3. Analyze the feed for mycotoxins often.

Table 4. Maximum Mycotoxin Levels for Mature, Nonbreeding Horses

MYCOTOXIN	LEVEL
Aflatoxin	50 ppb
T-2 Toxin	50 ppb
DON	400 ppb

Zearalenone	100 ppb
Fumonisin	2,000 ppb

Note: The above levels are based on field observations. Controlled scientific studies are needed to clarify specific mycotoxin tolerance and toxicity levels.

Heat stress, marginal nutrient plane, crowding, disease exposure, the presence of more than one mycotoxin, and drug interactions, as well other factors, increase animals' susceptibility to mycotoxins.

Thus these recommendations must be tempered with knowledge of the animals involved.

Additional research is needed to clarify the effects of mycotoxins on horses. Until such data exist, caution should be taken to select and feed mold-free grains and forages. [Click here to return to the Table of Contents.](#)

Dairy Cattle

Aflatoxin-contaminated feed not only reduces animal performance and overall health, but it also creates risks of residues in milk. Aflatoxin is secreted into milk in the form of aflatoxin M1 with residues approximately equal to 1 to 2 percent (1.7 percent average) of the dietary level. This ratio is not influenced greatly by milk production level since higher producing cows consume more feed and have a slightly higher transmission rate. Due to risks of milk residues, dietary **aflatoxin should be kept below 25 ppb**. This level is conservative due to: (1) nonuniform distribution of aflatoxin in grain and feed, (2) uncertainties in sampling and analysis, and (3) the potential for having more than one source of aflatoxin in the diet. Replacement animals may tolerate 50 to 100 ppb aflatoxin.

In dairy cattle DON is associated with reduced feed intake, lower milk production, elevated milk somatic cell counts, and reduced reproductive efficiency. Milk production loss appears to occur when diets contain more than 300 ppb DON. Although controlled research has shown no cause and effect relationship between DON levels and reduced milk production, field observations have shown that reductions in milk output of 25 pounds per cow were seen when DON was 500 ppb or more. This suggests that DON may serve as a marker for feed that was exposed to a situation conducive to mold growth and mycotoxin formation. The possible presence of other mycotoxins, or factors more toxic than DON, seems likely. Dietary levels of 300 to 500 ppb DON in dairy feeds indicate mycotoxin problems and warrant attention.

Zearalenone causes estrogenic responses in dairy cattle, and large doses of this toxin are associated with abortions. Other responses of dairy animals to zearalenone may include reduced feed intake, decreased milk production, vaginitis, vaginal secretions, poor reproductive performance, and mammary gland enlargement in virgin heifers. Establishment of a tolerable level of zearalenone for dairy cattle is difficult, and is at best

only a guess based on a meager amount of data and field observations. As with DON, zearalenone may serve as a marker for toxic feed. It is suggested that zearalenone not exceed 250 ppb in the total diet.

In dairy cattle T-2 toxin has been associated with feed refusal, production losses, gastroenteritis, intestinal hemorrhages, and death. T-2 has also been associated with reduced immune response in calves. Data with dairy cattle are not sufficient to establish a tolerable level of T-2 in the diet. Therefore, a practical recommendation may be to avoid T-2 in excess of 100 ppb in the total diet for growing or lactating dairy animals.

Fumonisin is another commonly isolated mycotoxin. However, fumonisin has only recently been isolated and only enough data exist to know that levels in excess of 20,000 ppb are potentially toxic to ruminants. [Click here to return to the Table of Contents.](#)

Beef Cattle

Aflatoxin and other mycotoxins can have considerable effects on beef cattle although the problems are usually less critical than for swine and poultry. Consumption of feeds highly contaminated with aflatoxin may reduce growth rate and increase the amount of feed required per pound of gain. Calves are generally more sensitive to feed contamination than adult cattle. In affected calves, some cases have revealed severe rectal straining and a prolapsed rectum. Lactating cows show a significant reduction in milk yield. Research has shown that high levels of aflatoxin can also cause liver damage in adult cattle. Feeding a high level of aflatoxin may also depress immune function, resulting in disease outbreaks.

Based on the feeds available, those contaminated with aflatoxin should be fed at the lowest level possible and for the shortest period of time practical. The effects of aflatoxin fed to cattle depend on the level of aflatoxin in the ration, the length of the feeding period, and the age of the animal. If aflatoxin-contaminated feeds must be fed to beef cattle, follow these guidelines (on a dry matter basis):

1. Creep feeds and diets for gestating and lactating beef cows should contain less than 20 ppb of aflatoxin.
2. Unstressed, growing-finishing cattle in excess of 400 pounds may be fed diets containing up to 100 ppb of aflatoxin.
3. Diets for stressed feeder cattle should contain no more than 20 ppb of aflatoxin. Stressful conditions include weaning, shipping, extreme heat or cold, diseases, and parasites.
4. Animals destined for slaughter should receive aflatoxin-free diets for at least 3 weeks before slaughter.

Since cattle in the southeast are typically fed high forage diets, they are usually fed grain only as a supplement. Thus a relatively high level of aflatoxin can occur in the grain before it exceeds the tolerable dietary level. In general, cattle will eat about 2.5 percent of their body weight as dry matter. This can be used to calculate the contribution of grain to

their total ration, and the tolerable level of aflatoxin in the grain. For example, growing calves weighing 600 pounds will consume about 15 pounds of total feed (600 lb multiplied by 2.5% equals 15 lb). If they are fed 3 pounds of grain plus forage-to-appetite, the grain will make up about 20 percent of their total diet (3 lb divided by 15 lb equals 20%). In this case the grain may contain up to 500 ppb of aflatoxin (100 ppb divided by 20% equals 500 ppb). Aflatoxin levels allowable in the grain, given different rates of inclusion in the beef ration, are illustrated (Table 5).

Table 5. Allowable Aflatoxin in Grain for Beef Cattle

Percentage of Grain in Diet	Aflatoxin Level in Total Diet		
	20 ppb	50 ppb	100 ppb
20%	100 ppb	250 ppb	500 ppb
40%	50 ppb	125 ppb	250 ppb
60%	33 ppb	83 ppb	167 ppb
80%	25 ppb	63 ppb	125 ppb

This table assumes that aflatoxin is contained only in grains. This assumption is not always correct. Each dietary component should be tested for aflatoxin prior to use of any contaminated grains.

Heat stress, marginal nutrient plane, crowding, disease exposure, the presence of more than one mycotoxin, and drug interactions, as well other factors, increase animals' susceptibility to mycotoxins.

Thus these recommendations must be tempered with knowledge of the animals involved.

Other mycotoxins (DON, T-2, and zearalenone) present in grains, silages, and hays may cause problems with performance and immune status of beef cattle. However, little research is available on the levels of the individual toxins that may be tolerated by animals. In cases of disease outbreaks and reproductive problems, the feed should be tested for a full range of mycotoxins. Large producers should consider routinely screening feeds for mycotoxins.

Until further information is available, the producer should limit dietary mycotoxins to the levels listed (Table 6).

Table 6. Maximum Mycotoxin Levels for Beef Cattle

MYCOTOXIN	LEVEL
DON	500 ppb
T-2	100 ppb
Zearalenone	250 ppb
Fumonisin	50,000 ppb

Heat stress, marginal nutrient plane, crowding, disease exposure, the presence of more than one mycotoxin, and drug interactions, as well other factors, increase animals' susceptibility to mycotoxins.

Thus these recommendations must be tempered with knowledge of the animals involved.

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Prevention and Management of Mycotoxins

Prevention in Silages

Prevention of mycotoxins in silages includes following accepted ensiling practices aimed at inhibiting deterioration primarily through elimination of oxygen. 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 bunks 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. [Click here to return to the Table of Contents.](#)

Prevention of Feed Contamination

Controlling mold growth and mycotoxin production is very important to the feed manufacturer and livestock producer. Control of mold growth in feeds can be accomplished by keeping moisture low, keeping feed fresh, keeping equipment clean, and using mold inhibitors. Grains and other dry feed such as hay should be stored at a moisture level 14 percent or less to discourage mold growth. Aeration of grain bins is

important to reduce moisture migration and to keep the feedstuffs dry. [Click here to return to the Table of Contents.](#)

Moisture Control

Moisture is the single most important factor in determining if and how rapidly molds will grow in feeds. 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. 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. 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 percent moisture may, for example, contain some kernels with 10 percent moisture and others with 20 percent 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. 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 percent 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. 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. 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. [Click here to return to the Table of Contents.](#)

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. 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. 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 animals 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. [Click here to return to the Table of Contents.](#)

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, 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.

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Use of Mold Inhibitors

Types of Mold Inhibitors

The use of chemical mold inhibitors is a well-established practice in the feed industry. 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. 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.

Dispersion

Many factors influence the effectiveness of mold inhibitors, and proper attention to these factors can enhance the benefits they provide. 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. 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.

Copper Sulfate

The practice of recommending copper sulfate as a treatment for fungal diseases in animals goes back many decades. The effectiveness of copper as a mold inhibitor is difficult to document. Although copper sulfate in the diet has been shown to improve body weight and feed conversion efficiency in broilers, excessive levels of copper may be toxic to young animals and will accumulate in the environment. In addition, recent research has indicated that feeding copper sulfate to poultry causes the formation of mouth lesions similar to those formed by some mycotoxins. Similar mouth lesions might be formed in other animal species. [Click here to return to the Table of Contents.](#)

Animal Management

If unacceptable mycotoxin levels occur, removal of the contaminated feed is preferable. 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. The addition of antioxidants to the animal 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. 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 protecting animals 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. [Click here to return to the Table of Contents.](#)

Mycotoxin Sampling, Testing, and Test Kits

Since mycotoxins are not evenly distributed in grain or in mixed feeds, taking a feed or grain sample which will give a meaningful result in mycotoxin analyses is difficult. Grab samples generally give very low estimates of mycotoxin content. In fact, nearly 90 percent of the error associated with mycotoxin assays can be attributed to how the original sample was collected. This is due to only 1 to 3 percent of the kernels in a contaminated lot containing mycotoxin, and these contaminated kernels are usually not evenly distributed within the lot of grains.

For whole kernel grains, a properly taken composite sample of at least ten pounds is required for a reasonably accurate, mycotoxin analysis. Trucks can usually be sampled with a grain probe, but bins must often be sampled as grain is being withdrawn.

Analytical techniques for the detection of mycotoxins continue to improve. Several commercial laboratories now test for a variety of mycotoxins. Although analytical costs can be a constraint, these costs may be insignificant compared with the economic consequences of production and health losses associated with mycotoxin contamination.

Commercial antibody test kits for screening or quantitation are currently available for aflatoxins, zearalenone, deoxynivalenol (DON), T-2 toxin, ochratoxin A, and fumonisins. These antibody methods, while they are still being improved, are good if used properly. The mycotoxin test kits in Table 7 have been tested and found to perform in a wide variety of laboratories.

Screening of corn for possible aflatoxin contamination using a "black light" was a popular technique 15 to 20 years ago. In spite of the widespread use of black lighting to screen for aflatoxin and other mycotoxins, research has shown that the technique detects

materials which are not mycotoxins, and is, therefore, inappropriate. **The black light test should never be used for any kind of mycotoxin screening.**

The minicolumn is a small column containing silica gel and Florisil (or other adsorbents) to which sample extracts are applied for detection of aflatoxin. Minicolumns were also very popular for aflatoxin screening until antibody-based test kits became widely available over the last few years. If properly used, the minicolumn test is capable of giving good results for aflatoxin under certain conditions. However, like the black light, it has often been mishandled and misused. **The minicolumn is no longer recommended.**

Better yet, because little is known about the above mycotoxins, and because many unidentified mycotoxins exist, cattle producers should avoid feeding moldy feeds if at all possible.

Table 7. Some of the Commercially Available Mycotoxin Test Kits

MANUFACTURER	MYCOTOXINS DETECTED	TEST KIT NAME
Editek P. O. Box 908 1238 Anthony Rd. Burlington, NC 27215 Phone: (910) 226-6311 Fax: (910) 229-4471	Aflatoxin Ochratoxin T-2 Toxin Zearalenone	EZ-Screen
International Diagnostic System Corp. 2620 S. Cleveland Ave. Suite 100 St. Joseph, MI 49085 Phone: (616) 428-8400 Fax: (616) 428-0093	Aflatoxin (4 Kits)	1. Afla 20 Cup 2. Afla 10 Cup 3. Afla 5 Cup 4. Afla B1, ELISA Test
	Zearalenone (2 Kits)	1. One Step ELISA, Quantitative Test 2. I. D. Block, ELISA Antibody
Neogen Corporation 620 Leshner Place Lansing, MI 48912 Phone: (517) 372-9200 800) 234-5333 Fax: (517) 372-2006	Aflatoxin T-2 Toxin Vomitoxin (DON) Zearalenone Fumonisin Aflatoxin M1 Ochratoxin	AgriScreen Veratox

VICAM
313 Pleasant St
Watertown, MA 02172
Phone: (800) 338-4381
(617) 926-7045
Fax: (617) 923-8055

Aflatoxin
Fumonisin
Ochratoxin
Zearalenone

Aflatest-P
Fumonitest
Ochratest
Zearalatest

NOTE: Absolute detection limits of mycotoxin test kits will vary and should be examined in relation to the needs of the user.

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Suggested Further Reading

Anonymous. 1979. *Aflatoxin and other mycotoxins: An agricultural perspective*. Council for Agricultural Science and Technology, 250 Memorial Union, Ames, IA 50011, Report No. 80, pp 56.

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Bray, G. A. and D. H. Ryan, eds. 1991. *Mycotoxins, cancer, and health*. Pennington Center Nutrition Series, Vol. 1, Louisiana State University Press, Baton Rouge, LA, pp 331.

Coelho, M. B. 1990. *Molds, mycotoxins and feed preservatives in the feed industry*. BASF Corporation, 100 Cherry Hill Road, Parsippany, NJ 07054, pp 159.

Robens, J. F. ed. 1990. *A perspective on aflatoxins in field crops and animal food products in the United States: A symposium*. U.S. Department of Agriculture, Agricultural Research Service publication number ARS-83, pp 157.

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