



Cattle Producer's Handbook

Animal Health Section

632

Mycotoxins

*Shanna Hamilton, Extension Educator
University of Idaho*

Certain fungi (molds) produce potentially harmful or toxic substances called mycotoxins. There are thousands of species of fungi, but relatively few of these grow on agricultural products and only a fraction are capable of producing mycotoxins. Toxigenic fungi spores are present almost everywhere, and they can germinate, grow, and elaborate their toxins into a variety of substrates.

The existence of mold is not a dependable sign of toxins nor is the lack of noticeable mold a sign that toxins are nonexistent. Optimal conditions for toxin production are quite variable.

Production of Mycotoxins

Factors influencing mold development are moisture, temperature, aeration, and substrate. Moisture is most important, but the type of mold and whether a toxin is produced will depend on the interplay of all these factors. Molds can grow and mycotoxins can be produced on feedstuffs either pre-harvest or post-harvest, during storage, transport, processing, or even feeding. Most molds do not produce mycotoxins.

The presence of mold spores does not mean that mycotoxins are present, but they can be! Many types of molds do not produce mycotoxins; however, their presence may reduce palatability and energy value.

Physical damage, such as breakage and stress cracks in grain, and environmental conditions, such as heat and water and insect damage, are large factors in mycotoxin contamination. Cool, wet growing seasons as well as drought conditions that delay maturity can result in mycotoxin formation in the field. Recent research indicates moldy feedstuffs cause previously unexplained production and health problems.

High-moisture feedstuffs, especially if ground, are highly susceptible to fungal invasion and toxin formation. To prevent production of the toxin on harvested products, care should be taken to prevent physical damage at harvest and to reduce the moisture level soon after harvesting. Rapid ensiling or the addition of organic acids will aid in preventing the formation of additional mycotoxins. However, these procedures do not reduce the level of contamination that was present before the treatment. Mycotoxins can be present with little or no obvious signs of mold.

Mycotoxin Effects

Since ruminants have high bacterial population, they are able to detoxify most mycotoxins, therefore, cattle are less susceptible generally to mycotoxin toxicity than are non-ruminants (swine, poultry, and humans). However, there is a limit to the amount of a mycotoxin that cattle can detoxify. In addition, classes of cattle that are more vulnerable to mycotoxins would be (1) pre-ruminant calves with limited rumen function, (2) cows having sub-optimal bacterial populaces (i.e., fresh cows with lower intake, cows with high passage rates, and/or producing cows with high intake), and (3) breeding cows or cows in early gestation.

Mycotoxins exploit their effects through three major mechanisms:

1. Mycotoxins reduce the amount of nutrients available for use by the animal; this is a multi-faceted process. An alteration in nutrient content of feed may occur during the molding process. Mold growth can reduce the content of nutrients such as vitamins and amino acids like lysine in feedstuffs. The energy value of feed can decrease due to mold.

2. Mycotoxins can reduce the feed intake, which in turn lowers nutrient intake. Mycotoxin-produced irritation to the digestive systems can decrease absorption of nutrients and inhibit with nutrient metabolism. Reproductive performance can be affected by Zearalenone due to the estrogenic effects. Its derivatives bind with the animal's estrogen receptors causing estrogenic effects.
3. Suppression of the immune system. Certain toxins can reduce immunity by inhibiting protein synthesis. Mycotoxins can increase incidence of disease and reduce production efficiency.

Mycotoxicoses commonly include digestive disorders, reduced feed consumption, un-thriftiness, rough hair coat, abnormal feathering, undernourished appearance, subnormal production, and impaired reproduction. When diagnosing mycotoxin-related disease, signs should be observed carefully as it could be a secondary disease due to the lack of immune system function.

Common Mycotoxins of Cattle

Aflatoxin

Aflatoxins are a group of chemically related mycotoxins and are classified as B₁, B₂, G₁, and G₂. Aflatoxins M₁ and M₂ may appear in the milk of dairy animals that have consumed aflatoxin-contaminated feed or grains. The most common and most toxic of the aflatoxin group is B₁. Aflatoxin was first identified in stored grain, as a product of the fungus *Aspergillus flavus* and *Aspergillus parasiticus*. Aflatoxin mostly affects crops of corn, peanuts, cotton (cottonseed), coconut, and tree nuts. The molds that produce aflatoxin usually do not grow in silage, but aflatoxins already present can survive the acids produced during the ensiling process.

Aflatoxin has a wide range of symptoms including a weakened immune system and reproductive system. The sensitivity varies among species while the liver is the primary aflatoxin target. If the dose is sufficient to produce an acute toxicity, it results in an increased clotting time and hemorrhage, especially in the intestinal lumen. Edema of the gall bladder also occurs.

Acute poisoning causes hepatitis and necrosis of liver cells, resulting in prolonged blood clotting time, with affected animals dying from severe hemorrhages. The changes may be so subtle that they are overlooked, but the poisoning can result in decreased milk production, decreased appetite, poor feed conversion, reduced growth rate, and decreased resistance to disease. It may also cause diarrhea, bloody diarrhea, abortion, or deformities of the fetus.

In cattle, according to Pennsylvania State University Extension, young immature animals are more susceptible and maybe affected when mother's milk contains a diet consisting of over 20 to 40 parts per billion (ppb) in the total ration dry matter. Even lower levels may

have an effect in the farm situation, compared to when a purified toxin is used in the laboratory. This is evidently due to the synergistic effects of other molds and their products in the farm situation.

Human exposure to aflatoxin can result from direct consumption of aflatoxin-contaminated foods, whether processed or unprocessed. Aflatoxin ingested by food-producing animals may also be transferred within the animal's body into meat, milk, or eggs, and these would be potential sources for human exposure. Aflatoxin-contaminated foods are deemed adulterated. Residues of aflatoxin B₁ have been found in the musculature and certain organs of poultry and swine after they were given aflatoxins in their feed.

Aflatoxin B₁ in the dairy ration is transformed to a metabolite, aflatoxin M₁, in the cow's milk. The metabolite is as potent a carcinogen as is the parent toxin; long-term, low-level exposure has been correlated to human cancer. Aflatoxin B₁ present in the cow's feed at a concentration of 100 ug/kg (ppb) can result in aflatoxin M₁ in the milk at a concentration of 1 ug/L. Because the aflatoxins have been shown to be carcinogenic, the FDA implemented feeding guidance regulations.

The FDA has established regulatory guidance levels in animal feed ingredients for aflatoxin in Compliance Policy Guide 7126.33. These levels apply to total aflatoxin—measured in parts per billion (ppb).

Animal feeds:

20 ppb (not corn, peanut products, or cottonseed meal)

Corn and peanut products:

Dairy animals (20 ppb)
Breeding cattle (100 ppb)
Finishing beef cattle (300 ppb)

Cottonseed meal:

Beef cattle (300 ppb)

Piles of cottonseed can develop "hot spots" of aflatoxin levels when left in piles or railcars too long, or when left in transit too long. Cottonseed that may have been certified at only a few parts per billion, 42 initially, may grow to unsafe levels under the proper environmental conditions. Cottonseed initially certified at 15 to 20 ppb stands a good chance of exceeding safe levels by delivery.

- Obtain shipments that are as fresh as possible to avoid or reduce risk
- Ask for a copy of the "Certificate of Analysis" when making purchases
- Store cottonseed and cottonseed seed products out of the weather, if at all possible
- Utilize piles of cottonseed and cottonseed seed products in a timely manner

Fumonisin (B₁ Toxin)

This toxin is measure in parts per million (ppm) and is produced by stains of *Fusarium moniliforme*, a

fungus commonly found in corn. The three types of Fumonisin are B₁, B₂, and B₃, and again, B₁ is the most common. Fumonisin is the cause of equine leukoencephalomania (ELEM) and porcine pulmonary edema syndrome.

Equine are the most susceptible; however, cattle and other species are not apparently seriously affected as they are in other mycotoxins, such as Zearalenone, DON, and DAS, which is also produced by *F. moniliforme*. The American Association of Veterinary Laboratory Diagnosticians Mycotoxin Committee suggested in 1993 that the following guidelines for feeding Fumonisin contaminated corn be observed.

- Beef cattle (<50 ppm non-roughage portion of ration)
- Dairy cattle (insufficient data for recommendation)

Zearalenone (F₂ Toxin)

Main signs are an enlarged vulva, possible irregular heats, and infertility at 4 to 7 ppm in TRDM. No abortions were noted in most cases, and no effects were evident on performance at .5 ppm in corn or about .15 ppm in TRDM.

Ochratoxin

Ochratoxin is produced by a species of *Penicillium* and *Aspergillus* and is a causative agent of kidney disease. The main involvements in the kidneys are reduced feed intakes and performance, hunched stance in calves, and possibly others. Apparently, this is one of the most harmful toxins for poultry and swine.

Vomitoxin (DON or deoxynivalenol)

This toxin is measured in parts per million (ppm) and is produced by several fungi, especially *Fusarium graminearum*, and is mostly found on cereal commodities such as wheat, corn, barley, and ensilages. When this mycotoxin inhabits the animal, symptoms include vomiting, feed refusal, gastroenteritis, diarrhea, immune suppression, and also blood disorders. Other symptoms are off-feed, ketosis, DA's, pronounced milk decrease, and sometimes diarrhea is present. This has un-established impacts on dairy cattle.

In September 1993, the FDA suggested the following guidelines—measured in parts per million:

Ruminating beef and feedlot cattle older than 4 months (10 ppm); grain and grain by-products not to exceed 50 percent of the diet

DAS (Diacetoxysciperol) has similar effects as DON or Vomitoxin.

Ergot

Ergotism is caused by several mycotoxins from molds that invade the seed head of numerous grass and cereal species, especially rye. The ergot sclerotia are often removed from cereal grains during cleaning, but these screenings may be put into livestock feeds. Once

grain has been ground or milled, ergot cannot be recognized without microscopic examination or chemical analysis. The primary clinical signs of ergotism are staggering, nervousness, and motor disorders. More often lameness and tissue necrosis resulting in loss of ears, tail, feet; possible infertility and lactation failure is more common in swine.

Rubratoxin

This toxin also affects the liver and accentuates the effects of aflatoxin when they occur together. It reduces liver function and rate of gain. It may result in hemorrhage and death. There may be a yellow, mottled discoloration of the liver. Suspect feed may show areas of red pigmentation.

Trichothecenes

This family of mycotoxins contains close to 200 to 300 related compounds that apparently exert their toxicity through protein synthesis inhibition at the ribosomal level. Ingestion often results in gastrointestinal effects such as vomiting, diarrhea, and bowel inflammation. Anemia, leukopenia, skin irritation, feed refusal, and abortion are also common. Trichothecenes, as a group, are immunosuppressive.

Slaframine (Slobber Factor)

The major clinical signs from this mycotoxin include profuse slobbering, salivation, increased urine production, and sometimes diarrhea. The fungus and toxin are mostly associated with legume intake and found on forages, especially red clover and red clover hay.

Testing and Diagnosis

The two types of mycotoxin tests are quick tests and confirmatory or quantitative tests. Many testing facilities will do a quick test to confirm the presence of mycotoxins, then a quantitative testing. It is known that fungal growth may occur without the production of mycotoxins. An "at home" test can be done with a "black light" to detect the presence of mold growth on grain and is satisfactory for use as an initial test for aflatoxin. This will check only for the presence of mold growth, and a positive test doesn't mean that a toxin is present. Quick tests are available from many public labs serving veterinarians and the producers (see Appendix A), or even supply stores.

Confirmatory tests should be used when quick tests are strongly positive. Best advice is to consult multiple laboratories to determine what mycotoxins are being tested and that mycotoxin tests are directed toward specific compounds; producers should ask for as many mycotoxin tests as feasible. The basic group should include aflatoxin, zearalenone, deoxynivalenol (DON a.k.a Vomitoxin), T-2, fumonisin, ochratoxin, and diacetoxysciperol (DAS).

Table 1. Mycotoxins commonly affecting cattle.

Name	Source and classifications	Clinical signs
MAJOR TOXICITY		
<i>Aflatoxin</i>	B ₁ , B ₂ , G ₁ , G ₂ , M ₁ , M ₂ Most common B ₁ Derived from <i>Aspergillus Flavus</i> and <i>Aspergillus parasiticus</i> Commonly found on corn, peanuts, cottonseed, coconut, and tree nuts.	Weakened immune system Weakened reproductive system Liver is the primary target. Edema of gall bladder. Hepatitis, necrosis of liver cells. Decreased appetite, poor feed conversion, reduced growth rate and decreased resistance to disease. It may also cause diarrhea, bloody diarrhea, abortion, or deformities of the fetus.
<i>Fumonisin (B₁ toxin)</i>	B ₁ , B ₂ , B ₃ Most common B ₁ Commonly found on corn. Produced by strains of <i>Fusarium moniliforme</i>	Pulmonary edema syndrome
<i>Vomitoxin (DON or deoxynivalenol)</i>	Produced by <i>Fusarium graminearum</i> . Found on most cereal commodities: wheat, corn, barley, and ensilages.	Vomiting, feed refusal, gastroenteritis, diarrhea, immune suppression, and blood disorders. Pronounced milk decrease.
<i>Zearalenone (F₂ Toxin)</i>		Enlarged vulva, irregular heats, and infertility at 4 to 7 ppm in TRDM.
<i>Ergot</i>	Several mycotoxins that invade seed head, common with rye. Common in quack grass, Dallisgrass, and grains infected with a hard seed head.	Staggering, nervousness, and motor disorders. Often lameness, tissue necrosis, resulting in loss of ears, tail, possible infertility, and lactation failure.
LESSER TOXICITY		
<i>Rubratoxin</i>	Suspect feeds could show red pigmentation.	Reduces liver function and rate of gain. Hemor- rhage and death can occur. Yellow, mottled discoloration of the liver.
<i>Trichothecenes</i>	200 to 300 related compounds As a group are immunosuppressive.	Gastrointestinal effects; vomiting, diarrhea, bowel inflammation. Anemia, leukopenia, skin irritation, feed refusal, and abortion.
<i>Ochratoxin</i>	From <i>Penicillium</i> and <i>Aspergillus</i>	Reduced feed intakes and performance. Hunched stance.
<i>Slaframine (Slobber factor)</i>	Associated with legume intake; found in forages, red clover, and red clover hay.	Profuse slobbering, increased urine production, diarrhea.

Testing for mycotoxins is indicated when there is a fall in health or production (especially in large numbers) that cannot be explained. A sample of all feed-stuffs being fed should be tested. It is recommended to start by testing total mixed rations (TMR) when feeding ruminants. If the sample of the TMR is found to have mycotoxins, the individual ingredients should be tested separately. If supplements and forages are fed separately, tests should be separately. Culture of mold or even isolation of specific molds from feeds means nothing. The specific toxin must be isolated and/or its toxicity must be demonstrated.

Toxins that are not specifically looked for in the testing will be missed. The diagnostic problem is further aggravated by wide variations among samples of the same feed and sensitivities of animals. Interactions between the mycotoxins and other stresses on the animals are possible factors. These factors make it extremely

difficult to recommend safe concentrations in the feed. Some mycotoxins can be identified in the rumen contents or urine.

Treatment

Basic treatment is to remove the contaminated feed and provide good nursing care of the animals. All surgical procedures should be delayed until the liver function and blood clotting mechanisms have returned to near normal. Increases in production, performance, and health often transpire within 3 to 7 days to several weeks after contaminated feeds are removed from the ration or their intakes severely reduced. Suggested feed additives are used to bind (Aluminosilicates or bentonites may bind mycotoxins in digestive tract and reduce absorption) mycotoxins that may be existent. Continue to check feedstuffs for mycotoxins if the problems are not completely alleviated.

Prevention

While inevitable, problems arise, but don't feed products that are obviously contaminated with mold until they have been tested. Pre-harvest control is the best prevention; certain measures can be taken to reduce risk of mycotoxins and serious effects on production, performance, and health. Produce or purchase varieties of crops that are local to the area in which your farm is located in respect to days to maturity and growing season.

Follow recommended harvesting practices when maintaining your own crops; take special care in storing at proper moisture levels. Avoid damaged grain if it is to be stored in a dry form and avoid fallen stalks. Clean and properly maintain storage facilities; especially silos. Check your stored crops for signs of heating, molding, and other decline. Use silage preservatives or additives when ensiling process is at risk due to moisture levels that are below or above recommendations. Handle your crops wisely; clean equipment used in harvesting, storing, and feeding frequently. Prevent mycotoxins and mold problems in ensiled or other wet feeds by removing them from storage just before feeding.

The FDA has established certain guidelines listed below on the Idaho State Department of Agriculture website (<http://www.agri.idaho.gov/>). Check with your local state department of agriculture for specific

guidelines in your area. For example, Idaho state law requires that any load of cottonseed or cottonseed seed products destined for Idaho be accompanied by a "Certificate of Analysis" certifying the aflatoxin level in the shipment to 20 ppb or less if being fed to dairy cattle. Several incidents of cottonseed shipments exceeding this tolerance have been recorded in Idaho. Cottonseed and cottonseed meal may be imported into Idaho certified at aflatoxin levels of up to 300 ppb to be fed to finishing beef cattle under a special permit from the Idaho State Department of Agriculture.

Additional Resources

- Adams, Richard S., Kenneth B. Kephart, Virginia A. Ishler, Lawrence J. Hutchingson, and Gregory W. Roth. *Mold and Mycotoxin Problems in Livestock Feeding*. Dairy and Animal Science. Penn State Coop. Ext. 1, 4, 5, 7-14.
- Robinson, P. H. 2012. Did the Wet Fall Weather Increase Mycotoxin Levels of Your Silage? *California Dairy Newsletter*. CE Specialist, Department of Animal Science, UC Davis. Vol. 4 Issue 1 (February).
- Takasugi, Patrick A. 2003. *Mycotoxins and Animal Feeds*. Idaho State Department of Agriculture; Commercial Feedstuffs Annual Report. Division of Plant Industries, Bureau of Feeds and Plant Services, Commercial Feed & Fertilizer Section. 41-43.
- Whitlow, L. W., and W. M. Hagler. *Mycotoxin Contamination of Feedstuff—An Additional Stress Factor for Dairy Cattle*. North Carolina State University. 1, 3, 6-16

Appendix A: AAVLD Accredited Laboratories to Test for Mycotoxins

ARIZONA

Arizona Veterinary Diagnostic Lab
2831 N. Freeway
Tucson, AZ 85705
Phone: 520-621-2356
Fax: 520-626-8696
<http://microvet.arizona.edu>

CALIFORNIA

CA Animal Health & Food Safety Lab System
University of California, Davis
West Health Science Drive
PO Box 1770
Davis, CA 95617-1770
Phone: 530-752-8709
Fax: 530-752-5680
<http://cahfs.ucdavis.edu>

COLORADO

Colorado State University
Veterinary Diagnostic Lab
CSU D Lab
Fort Collins, CO 80523
Phone: 970-297-1281
Fax: 970-297-0320
<http://www.dlab.colostate.edu>

MONTANA

Montana Department of Livestock
Montana Veterinary Diagnostic Laboratory
South 19th and Lincoln
PO Box 997
Bozeman, MT 59718
Phone: 406-994-4885
Fax: 406-994-6344
<http://www.discoveringmontana.com/liv/lab/index.asp>

OREGON

Veterinary Diagnostic Laboratory
Oregon State University
Magruder Hall, Room 134
30th and Washington Way
PO Box 429
Corvallis, OR 97331
Phone: 541-737-3261
Fax: 541-737-6817
<http://www.vet.orst.edu>

UTAH

Utah Veterinary Diagnostic Laboratory
950 East 1400 North
Logan, UT 84341
Phone: 435-797-1895
Fax: 435-797-2805
<http://www.usu.edu/uvidl>

WASHINGTON

Washington Animal Disease Diagnostic Laboratory
Washington State University
155N Bustad Hall
PO Box 647034
Pullman, WA 99164-7034
Phone: 509-335-9696
Fax: 509-335-7424
http://www.vetmed.wsu.edu/depts_waddl

WYOMING

Wyoming State Veterinary Laboratory
1174 Snowy Range Road
Laramie, WY 82070
Phone: 519-824-4120 ext. 54502
Fax: 519-821-8072
<http://ahl.uoguelph.ca>



©2016

Issued in furtherance of cooperative extension work in agriculture and home economics, Acts of May 8 and June 30, 1914, by the Cooperative Extension Systems at the University of Arizona, University of California, Colorado State University, University of Hawaii, University of Idaho, Montana State University, University of Nevada/Reno, New Mexico State University, Oregon State University, Utah State University, Washington State University and University of Wyoming, and the U.S. Department of Agriculture cooperating. The Cooperative Extension System provides equal opportunity in education and employment on the basis of race, color, religion, national origin, gender, age, disability, or status as a Vietnam-era veteran, as required by state and federal laws.

Fourth edition; December 2016 Reprint