

Anti-quality Factors in Beef Cattle Diets



Under certain conditions, beef cattle may be exposed to forages and feeds containing toxic or harmful compounds. When consumed, these anti-quality factors can result in reduced cattle growth, depressed reproductive performance, poor health, and even death. Simple management practices often can be implemented to reduce the risk of diet-induced production losses. Identifying potential anti-quality problems in beef cattle diets, using proper treatments, and preventing future occurrences can help protect cattle health and profitability.

Fescue Toxicosis

Cause

Fescue toxicosis is the forage-related livestock disorder that impacts the largest number of cattle in Mississippi and causes the greatest economic losses. Most of the tall fescue acreage in Mississippi is in the northern and central regions of the state. Most tall fescue plants in Mississippi pastures are infected with a wild-type “toxic” endophyte (fungus). The wild-type endophyte produces ergot alkaloids, which are livestock toxins.

Clinical Signs

Consumption of toxic endophyte-infected tall fescue depresses body condition, reproduction, and milk production in cows and weaning weights in calves. Grazing toxic tall fescue pastures or consuming toxic tall fescue hay decreases forage intake, lowers average daily gain, and alters hormone concentrations. Cattle develop rough hair coats (**Figure 1**), exhibit heat stress during warm periods, and suffer losses of ear tips and tail switches during cool periods.

Management Guidelines

Management techniques to lessen the impact of fescue toxicosis include keeping seedheads clipped, reducing late spring nitrogen fertilization, interseeding toxic tall fescue pastures with legumes or other grasses, and



Figure 1. Steer with rough hair coat suffering from fescue toxicosis.

supplementing with nontoxic hay. While there is value to implementing these practices, significant production losses may still occur as long as toxic tall fescue is included in the diet. Storing hay for an extended time before feeding it to cattle is not an effective option for preventing fescue toxicosis, unlike prussic acid poisoning where toxin levels in stored hay decline to safe levels in a relatively short period of time. Use of nontoxic forages instead toxic tall fescue is the most effective form of prevention.

Endophyte-free tall fescue is commercially available and has been marketed as a solution to fescue toxicosis. Although endophyte-free tall fescue does not produce ergot alkaloids and provides good animal performance, removal of the endophyte results in reduced seedling vigor and lower plant persistence. Accelerated forage stand losses in endophyte-free tall fescue pastures, relative to toxic tall fescue pastures, are common without intensive management. Unlike toxic tall fescue, endophyte-free tall fescue can be easily overgrazed and will not tolerate poor management.

“Friendly” endophyte-infected tall fescue contains an endophyte that does not produce ergot alkaloids. It is also

referred to as “novel” or “nontoxic” endophyte-infected tall fescue. Novel endophyte-infected tall fescue combines the plant persistence advantage of toxic tall fescue with the animal performance advantage of endophyte-free tall fescue. Plant persistence is higher in novel endophyte-infected tall fescue than in endophyte-free tall fescue, and cattle performance on novel endophyte-infected tall fescue is similar to performance on endophyte-free and higher than performance on toxic tall fescue.

Part of the success that producers have with maintaining productive stands of toxic tall fescue is attributable to the negative effect of the livestock toxins on forage intake. Forage intake is higher on novel endophyte-infected tall fescue than on toxic tall fescue. Therefore, novel endophyte-infected tall fescue stands require a higher level of grazing management than toxic tall fescue stands and should not be overgrazed. Proper establishment to minimize stand contamination with toxic tall fescue seed and proper management practices to minimize stand losses are also vital considerations when renovating with friendly tall fescue.

Treatment

Although there are several products advertised to alleviate fescue toxicosis, there is currently no drug, feed additive, or supplement proven in university research trials to effectively restore lost cattle growth performance on toxic tall fescue. Cattle should be removed from toxic tall fescue pasture and hay if possible.

Nitrate Poisoning

Cause

Nitrate toxicity can be a lethal problem for all classes of cattle, including stockers. Even when nitrates do not cause death, production losses such as reduced milk yield, lower weight gains, and reproductive problems can occur with moderate levels of nitrates in the diet. Generally, nitrates are present in grazing cattle diets at levels that are not normally toxic, but at high dietary levels, nitrates can cause nitrate poisoning. Nitrates normally found in forages are converted to nitrites, then to ammonia, and then to protein by bacteria in the rumen.

Nitrate poisoning in cattle results from excessive nitrate consumption from grazed forage, hay, silage, weeds, water, or other sources. Nitrates accumulate in

the rumen when cattle rapidly ingest large amounts of plants containing high levels of nitrates. Although rare, cattle may experience nitrate poisoning from drinking water contaminated with nitrogen-based fertilizer.

Nitrate is absorbed into red blood cells and combines with hemoglobin to produce methemoglobin, a type of hemoglobin that cannot carry oxygen in the blood. Lack of sufficient oxygen transport to tissues results in severe problems, including abortions and possibly death.

Clinical Signs

Signs of nitrate poisoning include bluish discoloration of the skin, bluish-brown mucous membranes, labored or rapid breathing, muscle tremors, lack of muscle control, staggering, weakness, diarrhea, frequent urination, dark- to chocolate-colored blood, rapid pulse, coma, and eventual suffocation. Necropsy results often reveal brown-colored and severely coagulated blood. Pregnant females that survive nitrate poisoning may abort due to lack of oxygen to the fetus. Abortions generally occur 10 to 14 days after exposure to excess nitrates.

Plant Factors

Certain forages and weeds pose higher risks of accumulating potentially dangerous levels of nitrates. Forages known to have the potential for accumulation of toxic nitrate levels include sudangrass, sorghum-sudan hybrids, pearl millet, corn, wheat, oats, soybeans, tall fescue, and bermudagrass. Weeds that pose a threat include pigweed (carelessweed), smartweed, ragweed, lambsquarter, goldenrod, nightshades, bindweed, Canada thistle, and bull or horse (stinging) nettle. Pigweed and the warm-season annual grasses are typically the more likely culprits in most nitrate poisoning cases in Mississippi.

Abnormally high levels of nitrates in plants are caused by various stress factors such as moisture conditions, low temperatures, and soil conditions. Plants will take up very little nitrate from dry soils. Nitrates are often at very high levels in plants for several days following a rain after drought conditions. Frost and low temperatures interfere with normal plant growth and can cause accumulation of nitrates in plants. Frost can cause leaf damage and reduce photosynthetic activity. So, nitrates absorbed by the roots are not converted to plant protein but are accumulated in the stem and stalk.

Deficiencies of essential nutrients such as phosphorus can also lead to plant stress and cause a buildup of nitrates.

Nitrate levels tend to decrease as plants mature. Young plants have higher concentrations of nitrates than more mature plants. Mature plants can still have excess nitrate levels if environmental and soil conditions are favorable for accumulation. Water nitrate levels should also be considered. Nitrate levels (unlike prussic acid levels) in stored forages do not significantly decrease over time, so storing hay containing high nitrate levels is not an effective method of preventing nitrate poisoning. In addition, ensiling is not considered an effective way to reduce nitrate levels in forages.

Management Guidelines

Avoid grazing livestock on heavily nitrogen-fertilized pastures of suspect species during drought or wet conditions through cool, cloudy weather. If animals are grazed on potentially toxic pastures, they should be observed carefully for signs of nitrate poisoning. Forages of concern should be tested for nitrate nitrogen levels. Nitrate nitrogen levels below 1,150 parts per million (ppm) are generally considered safe for cattle. Levels between 1,150 and 2,300 parts per million are potentially hazardous to pregnant or very young animals. However, because nitrate effects on cattle can be variable, these general guidelines may not be applicable in all situations.

The Mississippi State Chemical Laboratory offers two nitrate tests. The qualitative test indicates whether or not nitrates are present in the forage sample. The quantitative test indicates a specific nitrate level present in the forage sample. Nitrate levels should be evaluated according to the guidelines in **Table 1**.

If nitrogen fertilizer has been applied to drought-affected pasture, conduct a nitrate test before grazing or making this forage into hay to make sure nitrate levels are below those considered toxic. Try to limit nitrogen applications during a drought to around 30 pounds per acre or less. This will help reduce the risk of nitrate toxicity and give plants a better chance of using the nitrogen if the weather remains dry.

Identify areas of the farm that have better water-holding capacity and apply fertilizer to these areas only. While this may not always be an option, most producers have a mixture of soil types on their farms. It is often very easy to see these in a drought, as the ridges become brown and the valleys or bottoms stay green. As these different areas become apparent, it is better to put nitrogen fertilizer on the ground with better water-holding capacity to avoid wasting fertilizer by applying it to the more drought-prone soils.

Cattle in poor health and condition are more susceptible to nitrate poisoning. Feeding nonprotein nitrogen (urea) with hay containing high nitrate levels can also increase the risk of nitrate poisoning. Yet high energy levels in the diet can help to increase the conversion rate of nitrate to ammonia in the rumen. Therefore, cattle in good condition and with higher energy levels in the diet are at lower risk of suffering from nitrate toxicity.

Do not allow hungry cattle to graze high-nitrate forages or consume high-nitrate hay. Starved cattle may consume excessive amounts of high-nitrate forages, putting them at increased risk of developing nitrate poisoning. Supplement cattle with feed grains or byproducts before grazing high-nitrate forages. Dilution with low-nitrate forage can also be a good option. Overstocking and strip grazing increase the

Nitrate Concentration ^{1,2}		Recommended Management
0.0 to 0.5%	0 to 5,000 ppm	Safe to feed
0.5 to 1.0%	5,000 to 10,000 ppm	Risk to pregnant animals and to cattle that are not accustomed to high-nitrate containing forage
1.0 to 2.0%	10,000 to 20,000 ppm	Not more than half of the diet
>2.0%	>20,000 ppm	Do not feed
¹ If nitrate-nitrogen values are needed, multiply the nitrate concentration values by 0.23. ² If potassium-nitrate values are needed, multiply the nitrate concentration values by 0.14.		

amount of high-nitrate plant parts (stalks and stems) that cattle consume. Avoid these grazing practices if there is a chance for nitrate toxicity. If possible, limit grazing cattle on high-nitrate pastures during the daytime for the first week. This will reduce consumption of high-nitrate forage and help acclimate cattle to high nitrate levels.

Treatment

A veterinarian can provide advice and assistance in treating nitrate poisoning cases. For acute cases, contact a veterinarian immediately. Prompt intravenous injection of a 4 percent solution of methylene blue at a rate of 100 cc per 1,000 pounds of body weight is often the recommended treatment. This treatment may be repeated in 20 to 30 minutes if the initial dose is not effective. Methylene blue is a reducing agent that converts methemoglobin to oxyhemoglobin, reversing the effect of the nitrates by restoring oxygen-carrying capacity in the blood. To reduce nitrate intake in chronic cases, forage containing high levels of nitrates should be eliminated or at least diluted with other forage and feeds.

Prussic Acid Poisoning

Cause

Prussic acid (hydrocyanic acid or HCN) can accumulate to toxic levels in the leaves of johnsongrass, sorghum, sudangrass, sorghum-sudan hybrids, and wild cherry. Pearl millet does not produce prussic acid. Dangerous levels of prussic acid are most likely to occur immediately after a frost. Young forage growth can be potentially toxic following a severe drought. Prussic acid levels may temporarily increase with herbicide application.

Clinical Signs

Prussic acid interferes with the oxygen-transferring ability of the red blood cells, causing eventual suffocation and death. Excessive salivation, rapid breathing, and muscle spasms may be evident within 10 to 15 minutes of consumption of prussic acid-containing forage. Cattle may stagger, collapse, and die.

Management Guidelines

Prussic acid levels in forages deteriorate over time unlike nitrate levels. Forage containing high levels of prussic acid is usually safe to feed three weeks after ensiling. Hay that is dry enough to bale (less than 18 to 20 percent

moisture) is typically safe to feed. Standing plants may be safe for cattle at least 1 week after the last green material has been frosted. However, there may be danger zones in portions of pastures, particularly in low-lying areas. Hungry cattle should not be turned out onto potentially toxic pastures or have access to wild cherry trees.

Treatment

Animals affected by prussic acid poisoning may be treated with intravenous injection of 30 percent sodium thiosulfate (Cya-dote) solution. Proper dosage and administration of these injections is critical. Producers are advised to consult with a veterinarian for treatment recommendations.

Ergot Poisoning

Cause

Ergot poisoning is the result of toxin production by a parasitic fungus that grows in the seed heads of bahiagrass, annual ryegrass, small grains, and especially dallisgrass. The condition is also known as dallisgrass staggers. Wet growing conditions favor ergot development in grasses. Ergot poisoning is most common in warm-season grasses in late summer or early fall as seed heads reach maturity. Entire grains in grass seed heads are replaced by dark purple to black, hardened bodies of the fungus. Seeds appear swelled and have a sticky sap material on them. It is not uncommon for only a few animals instead of the entire herd in a pasture to consume infected seed heads and develop ergot poisoning.

Clinical Signs

The toxins interfere with circulation in cattle. This reduces blood flow to the extremities (tail, hooves, ears). An early sign of ergot poisoning is lameness. Continued exposure to ergot toxins may cause sloughing of the tail switch and hooves. Stimulation and depression of the central nervous system, elevated body temperature, increased respiratory rate, and increased heart rate are other signs that may be observed.

Management Guidelines

Clipping of pastures may reduce the risk of ergot poisoning by limiting seed head development and ergot growth. Avoid harvesting fields with large quantities of potentially infected seed heads for hay.

Treatment

Cattle should be immediately removed from toxic pastures and placed on an ergot-free diet.

Bermudagrass Staggers

Cause

Bermudagrass staggers is also called bermudagrass tremors. It is a rare nervous system disorder resulting from ingestion of alkaloids produced by fungal-infected bermudagrass. The condition may occur when cattle graze mature bermudagrass pastures during autumn or winter following a period of damp, cloudy weather. Hay harvested from infected pastures can remain toxic for up to 2 years.

Clinical Signs

Signs of bermudagrass staggers are similar to ergot poisoning. Cattle twitch, shake, and show poor coordination. Front legs become weak while rear legs are stiff. Excited cattle may fall to their knees.

Management Guidelines

Bermudagrass staggers can be prevented by maintaining pastures in a vegetative stage. Avoid grazing overgrown, mature, matted bermudagrass.

Treatment

Cattle should be immediately removed from toxic pastures and given alternative forage or feed sources.

Mycotoxins

Producers are sometimes faced with decisions on feeding moldy feeds or forages. With feed prices relatively high, it may be tempting to use moldy feed or forage supplies in beef cattle nutrition programs. It is important to recognize risky feeding situations and to be familiar with the potential impacts of feeding moldy feedstuffs.

Certain species of fungi (molds) produce toxic substances called mycotoxins. These fungi may be found growing on feed, silage, or hay in the field or in storage. Most mycotoxin production occurs in the field before harvest, but poor storage practices can increase already-existing mycotoxin levels. Mycotoxins can cause cattle health and productivity problems at very low dosages, parts per million (ppm) or parts per billion (ppb). Mycotoxins are not necessarily produced whenever

feed or forage becomes moldy, but evidence of mold indicates a risk of toxins. Fungi growth may be present but undetectable upon casual observation.

Hundreds of mycotoxins have been identified. Mycotoxins of greatest importance worldwide include aflatoxins, trichothecenes, fumonisins, zearalenone, ochratoxin A, and ergot alkaloids. Mycotoxins are regulated by the Food and Drug Administration. The FDA Center for Veterinary Medicine focuses on five major mycotoxins in the United States: aflatoxins, fumonisins, vomitoxin, ochratoxin A, and zearalenone.

Toxin-producing fungi include molds from the *Aspergillus*, *Fusarium*, and *Penicillium* genera. Mississippi weather can be conducive to growth of molds in feedstuffs that are of concern for use in beef cattle diets. Generally, cool, wet conditions favor *Fusarium* species growth, while hot, dry conditions favor *Aspergillus* species growth.

Aspergillus Toxins

Aflatoxin

The *Aspergillus* species molds can produce aflatoxin and ochratoxin under stress conditions. Aflatoxin is likely the most commonly produced mycotoxin in the southern United States. In fact, aflatoxins occur most commonly in the warm, humid regions of the nation. Both aflatoxin and ochratoxin cause livestock health problems. Aflatoxins are produced by the *Aspergillus* species, *A. flavus* and *A. parasiticus*. Corn, cottonseed, peanuts, and sorghum are potential feed substrates for aflatoxin production. High levels of aflatoxins are associated with above-average temperature and below-average rainfall. For instance, aflatoxin production may occur with maturing corn undergoing drought and insect stress during prolonged periods of hot weather and drought. However, weather conditions do not guarantee mycotoxin production.

Table 2 lists FDA action levels for total aflatoxins in livestock feed. Action levels are established by the FDA to control levels of contaminants in human food and animal feed. The blending of a food or feed containing a substance in excess of an action level or tolerance with another food or feed is not permitted. The final product resulting from blending is unlawful, regardless of the level of the contaminant.

Aflatoxin is a potent carcinogen, protein synthesis inhibitor, and immunosuppressant. Aflatoxin-contaminated

Class of Animals	Feed	Aflatoxin Action Level
Finishing beef cattle	Corn and peanut products	300 ppb
Beef cattle, swine, or poultry	Cottonseed meal	300 ppb
Breeding cattle	Corn and peanut products	100 ppb
Immature animals	Animal feeds and ingredients, excluding cottonseed meal	20 ppb
Dairy animals or unknown use	Animal feeds and ingredients	20 ppb

grain should not be fed to lactating dairy cattle, as it will appear in the milk. Cattle fed aflatoxin-contaminated feedstuffs may exhibit dry muzzles and decreased body temperature. Aflatoxins have tremendous antibiotic activity and disrupt normal rumen function. Young animals are more susceptible to aflatoxicosis than adult livestock. Likewise, monogastric animals (swine, poultry) are more susceptible than ruminants (cattle, goats, deer).

Ochratoxin

Ochratoxin is a relatively uncommon mycotoxin of cereal grains (corn, barley, wheat, and rye) that is produced by *Aspergillus* species (*A. ochraceus*) and *Penicillium* species (*P. viridicatum*). Ochratoxin, a suspected carcinogen, causes increased water consumption and urination and can lead to permanent scarring of the kidneys. At least nine ochratoxins have been identified, but ochratoxin A is the most common and has the greatest toxicological significance. No FDA action, advisory, or guidance levels are currently established for ochratoxin A in U.S. feed.

***Fusarium* Mycotoxins**

Cool, wet conditions favor the growth of *Fusarium* mold species, which can produce several mycotoxins detrimental to livestock. *Fusarium* mold species can produce fumonisins, vomitoxin, and zearalenone.

Fumonisin

Fumonisin, a group of toxins produced mainly in corn and particularly corn screenings, are believed to be most prevalent when cool weather and high humidity at crop maturity follow hot and dry weather. Fumonisin are thought to be carcinogens and are produced by *Fusarium* species (*F. verticillioides*). Liver damage and elevated serum liver enzymes occur when livestock consume fumonisins. Cattle can develop mild liver lesions at fumonisin

concentrations above 100 parts per million. However, this liver damage is temporary, and liver function returns to normal when fumonisin exposure ends. Toxin concentration is typically highest in broken grain.

For breeding cattle, FDA guidance levels for total fumonisins in animal feeds indicate that contaminated corn and corn by-products should not exceed 50 percent of dietary dry matter. Fumonisin levels in corn and corn by-products should not exceed 30 parts per million. In finished feeds, fumonisin should not exceed 15 parts per million for breeding cattle. In calves 3 months of age and older being raised for harvest, the fumonisin levels in corn and corn by-products should not exceed 60 parts per million. In finished feeds, fumonisin should not exceed 30 parts per million for calves 3 months of age and older being raised for harvest.

Vomitoxin

Vomitoxin (also known as deoxynivalenol, DON, or refusal factor) gets its name from the resulting vomiting and feed refusal that it induces in swine. It is produced by *Fusarium* species (*F. graminearum*) and is commonly found on wheat, barley, rye, and oats. Vomitoxin occurrence is most frequently reported in cool, temperate regions of the northern United States and Canada.

Vomitoxin is a protein synthesis inhibitor, affecting the digestive tract and immune system. Cattle are quite tolerant to vomitoxin, apparently due to rumen microbial activity. Cattle have consumed up to 10 parts per million vomitoxin with no adverse effects. Dietary dry matter concentrations of up to 21 parts per million vomitoxin in growing cattle diets were demonstrated to have no adverse effects on health or production performance. In cows fed 6.4 parts per million vomitoxin (diet dry matter) for 70 days, no vomitoxin residue was found in their milk. Advisory levels from FDA regarding vomitoxin in livestock feeds indicate that, in ruminating beef and feedlot

cattle older than 4 months, vomitoxin contaminated grain and grain by-products should not exceed 50 percent of the diet with maximum vomitoxin levels of 10 parts per million in grains and grain by-products and 5 parts per million in finished feed.

Zearalenone

Zearalenone (also known as F-2 toxin or giberella toxin) is an estrogenic mycotoxin that occurs in corn, wheat, barley, and sometimes oats. Zearalenone is produced by *Fusarium* species (primarily *F. graminearum*) with high humidity and low temperatures favoring its production. Zearalenone and vomitoxin sometimes occur together. Cattle consuming more than 10 parts per million zearalenone may exhibit infertility and estrous cycle disruptions. Heifers are sensitive to zearalenone effects on reproduction when concentration exceeds 5 parts per million of the dietary dry matter intake. At present, no FDA action, advisory, or guidance levels are established for zearalenone in United States feed supplies.

Producer Actions To Combat Mycotoxin Problems

In cases of disease outbreaks and reproductive problems where feed is a potential culprit, the feed in question should be removed from cattle diets and tested for a full range of mycotoxins. Large operations should consider routinely screening feeds for mycotoxins. Proper sampling techniques for mycotoxin analysis is critical as mycotoxin formation is not uniform throughout a feedstuff.

Mycotoxin formation risk can be minimized by frequent drying and cleaning (scraping) feed storage bins. Caked material and moisture should be removed during routine feed storage facility cleaning to reduce mold and mycotoxin contamination. Storage of commodities under low (less than 14 percent) moisture conditions will minimize fungal growth and mycotoxin production. Facility maintenance must be sufficient to protect feed supplies from moisture. Adequately drying grains prior

to storage and keeping grains free of insect damage may decrease mycotoxin occurrence. Silage and baleage will not typically undergo molding in the ensiling container but may mold when exposed to oxygen out of storage.

Badly molded feedstuffs, soured feedstuffs, or moldy legume hays should not be fed to livestock. Avoid feeding mold-contaminated feedstuffs to pregnant cattle altogether. In situations where mycotoxins are detected, there are often as many or more clean lots of feedstuffs as contaminated ones. Testing each lot is the only way to confirm mycotoxin presence or absence and levels. Environmental stress, poor nutrition, disease exposure, multiple mycotoxin presence, and other factors increase cattle susceptibility to mycotoxins. If moldy, wet, or flood-damaged feed must be fed, proceed with extreme caution, particularly when feeding moldy protein concentrates.

Once mycoses or mycotoxicosis is confirmed in forages or grains, one of the few practical strategies appears to be blending to an acceptable nontoxic concentration at feeding (except for aflatoxin-contaminated feedstuffs). Consider these factors when attempting to dilute mycotoxin levels in contaminated feedstuffs. Initially mix moldy feedstuffs with uncontaminated feedstuffs at a level not to exceed 10 percent of the total dietary dry matter of moldy feedstuffs to significantly reduce potential mycotoxin intake. Watch cattle carefully for reduced feed intake and signs of illness including respiratory or nervous disorders (resulting from inhalation of mold spores). Contact a veterinarian and completely remove potentially contaminated feedstuffs from cattle diets if signs of illness are observed.

In addition to the potential for toxins produced by molds, heated or spoiled feedstuffs may also have reduced nutritive value for livestock. Molds remove nutrients from feedstuffs. Consider also that other species of livestock respond differently to mycotoxin exposure. Horses or swine, for example, may be highly susceptible to specific mycotoxins or levels of mycotoxins that would not be problematic in cattle, or vice versa.

Conclusions

Anti-quality problems in beef cattle diets may not be a concern until animals in the herd are affected. That can be too late. It is important to be alert for “red flags” in animal behavior and appearance to catch a problem early and minimize losses. A local veterinarian should be familiar with nutritional disorders that are common in the area and can assist with prevention and treatment programs.

It is important to recognize potential causes of anti-quality problems in beef cattle diets. Producers can implement proper forage, feeding, and animal management practices to minimize the risk of conditions that result in production, animal, and economic losses. For more information on anti-quality factors in beef cattle diets, contact your local MSU Extension office.

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