Forage News
Mississippi State University Extension Service

Forage Related Disorders in Livestock Production

Rocky Lemus
Extension Forage Specialist
Visit us at **http://msucares.com/crops/forages/index.html**

Forages are a major source of nutrients for livestock nutrition and health. Forage toxicity in livestock is caused by the consumption of biomass high in specific compounds found in different forages such as grazing crops, hay, silage, or weeds. The accumulations of these compounds could be related to a plant's physiological responses to biological stress such as endophyte in tall fescue, due to high fertilization rates, or to environmental conditions (drought or frost). Sometimes, the balance of nutrients or presence of some chemical compounds in the forage will have negative effects on animal health. This newsletter will focus on some the forage related disorders including bloat, nitrate poisoning, prussic acid, and tall fescue toxicosis.

Bloat

Bloat is a form of severe indigestion. Bloat is a complex interaction between the animal, the microbes, and particular forage plants, where gases (Methane & CO_2) get trapped in bubbles in the cow's rumen (stomach). During this process, the rumen fills so fast and extends beyond the point where the animal cannot control belching. The extension of the rumen impairs the ability of the abdominal muscles to contract via peristaltic contractions, causing animal death due to suffocation.

Pasture bloat usually occurs when animals are grazing wheat, lush legumes (alfalfa and white and red clovers), or fed green chopped legumes (**Table 1**). The growth stage of maturity of the forage species is the most important factor in preventing bloat. Bloat risk is highest at the vegetative (pre-bud) stage, decreasing progressively as the plant reaches the reproductive stage (full flower or bloom).

Table 1. Levels of bloating caused by different forages.

High Bloat Risk	Low Bloat Risk	Bloat Safe
Alfalfa	Clovers	Lespedeza
	Arrowleaf	Rye
Clovers	*Berseem*	Most perennial grasses (tall
Alsike	*Persian*	fescue, bermudagrass,
Redclover		bahiagrass, dallisgrass)
Sweetclover	Winter annuals	
White	*Oats*	
	Annual ryegrass	
Wheat		

Source: Ball et al., 2002.

To prevent bloat in cattle, a producer should plant pastures consisting of a 30 and 50% percent legume forage mixture. Another approach is to feed cattle dry roughage or on grass pastures

before turning them into legumes pastures. It is also important to provide grass hay when grazing or graze using a rotation with grass pastures. Salt-molasses blocks containing surfactant or detergent type compounds such as poloxalene are an effective tool to use for bloat prevention. A poloxalene block can also be provided to cattle at least three days before grazing a pasture with high bloat risk. Feeding an ionophore can also reduce the risk of bloat. If an advanced stage of bloat is present in the cattle, contact your veterinarian immediately for specific treatments.

Nitrate Poisoning (NO_3)

All forages contain nitrates obtained through nitrogen fertilization or from the soil's nitrogen pool. Nitrates are the precursors to protein formation in plants. Nitrates can accumulate in forage crops (especially in summer annual crops) under stress. The most commonly affected plants include small grains (oats, wheat, and barley), warm-season annual grasses (sorghum, pearl millet, and corn), and broadleaf weeds (pigweed, thistles, goldenrod, and lambsquarter). Usually, perennial forages do not accumulate toxic levels of nitrate. Other factors such as heavy fertilization, drought, heavy rain, long periods of cloudy weather, diseases, soil nutrient deficiencies, chemical injury (herbicide), and frost or hail can also cause nitrate accumulation. Nitrate accumulation occurs when photosynthesis slows down. Concentrations of 1.5 percent or more in plant tissue can be toxic to livestock and concentrations of less than 0.25 percent are considered safe (**Table 2**). Nitrate toxicity in forages does not decrease with time and testing of the forage prior to feeding is critical if possible nitrate toxicity is suspected. Contact your local County Extension office for information on forage nitrate testing.

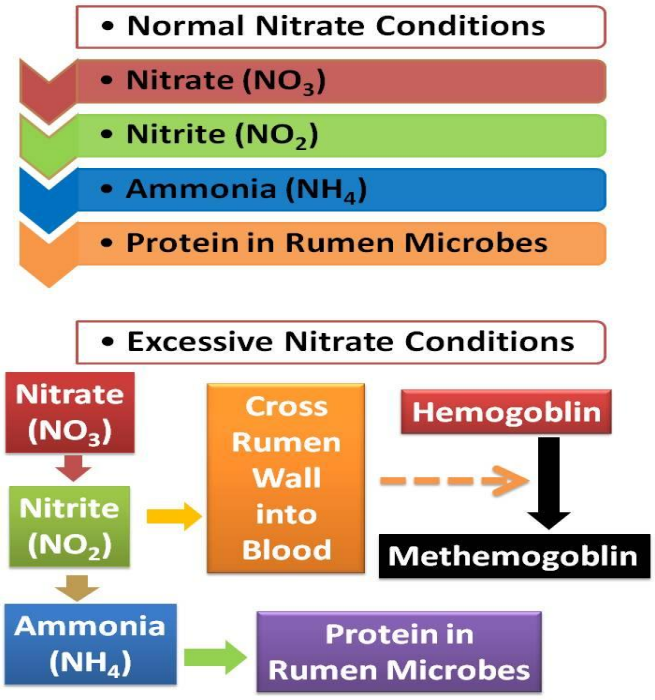

Figure 1. Representation of nitrate toxicity in cattle.
Source: Thomas and Norman, 2001.

Nitrates themselves are relatively non-toxic. Microbes in the rumen usually convert nitrate to nitrite and then reduce to ammonia. The ammonia is used to make protein. Nitrate poisoning occurs when the nitrate concentration in the rumen is higher than the capacity of the microbes to convert nitrite to ammonia. Nitrite can then escape into the bloodstream where it ties up hemoglobin to produce methemoglobin, reducing the oxygen carrying capacity in the blood and causing the animal to suffocate and die (**Fig. 1**). Sick, hungry, lactating, or pregnant cattle are more susceptible to nitrate toxicity.

Table 2. Nitrate concentration in forages on dry matter basis.

Nitrate Concentration			
%	Parts per million (ppm)		Recommendations
0.0 to 0.25	0 to 2500	SAFE	Forage or feed is considered safe. Be cautious with pregnant and young animals at the upper level range.
0.25 to 0.50	2500 to 5000	CAUTION	Considered safe when fed in a balanced ration. Forage with this level of toxicity should be limited to 50% of the total ration. Be aware that long term feeding of this type of forage can cause Vitamin A deficiency. It is recommended not to feed with liquid feed or other protein nitrogen supplementation. Cautious feeding is recommended with young and pregnant animals.
0.50 to 1.50	5000 to 15000	DANGER	This type of forage or feed should be supplemented with minerals, energy (high TDN), and Vitamin A. Feed should be limited to 25% percent of the total ration.
>1.5	>15000	TOXIC	Considered very toxic. Do not use this type of forage in free-choice feeding. This type of forage might be used if grounded, mixed, and limited to 15% of the total ration.

Source: Scharko, 2000; Ball et al., 2002; Olson et al., 2002.

Excess nitrates usually accumulate in the lower stems of some plants when plants become stressed. Minimizing high fertilization rates is a cautionary measure to utilize under stressful

conditions. To prevent nitrate poisoning, harvesting or grazing forages suspected of nitrate toxicity should be done 7 to 10 days after the end of the drought. Grazing or harvesting suspected forages in the afternoon is also a preventive measure since photosynthetic processes metabolize nitrates. Another approach will be chopping and diluting suspected hay with hay that is known to have low nitrate levels. Be cautious about this type of management. If it is suspected that cattle have been exposed to potentially toxic levels of nitrates or nitrites, it is important to make sure that the animal receives adequate levels of carbohydrates in the diet. If silage is suspected or confirmed with toxic nitrate levels, forage should be allowed to aerate overnight before feeding.

Nitrate poisoning symptoms could manifest 5 to 7 days after feeding high nitrate forage rations. Death can occur 2 to 24 hours after the manifestation of symptoms. When high nitrite levels are present in the blood, it becomes chocolate colored instead of the normal red color. Depletion of oxygen in the blood will cause respiratory failure. A brownish discoloration may be noted around white areas of the skin and non-pigmented mucous membrane of the eye, nose, and mouth. Other symptoms include staggering, rapid pulse, rapid and labored breathing, frothing at the mouth, frequent urination, diarrhea and uncoordination. Treating nitrate toxicity includes an intravenous injection of either a 1% or 2% solution of methylene blue. Consult your veterinarian for suggested dose rates or other alternative treatments.

Prussic Acid (HCN)

Prussic acid toxicity is also known as hydrocyanic acid or hydrogen cyanide (HCN). Prussic acid poisoning is caused by cyanide production in several forages under certain growing conditions. Grain sorghums, sorghum-sudan hybrids, sudangrass, Johnsongrass, pearl millet, and hybrid foxtail millet are the most commonly associated forages with prussic acid toxicity (**Table 3**). Cattle, sheep and goats appear to be the most susceptible livestock to prussic acid toxicity. Prussic acid is not usually a problem in horses, but sorghum type forages including Johnson grass and sorghum-sudan hybrids have been associated with causing urinary tract infections in horses (crystitis syndrome). The symptoms are similar to colic, but include blood in the urine, urine dribbling, and abortion in pregnant mares. The condition can be fatal and it is recommended not allowing horse to graze on these plants.

Table 3. Potential prussic acid toxicity of different forage species.

Forage Species	Potential HCN Toxicity
Forage sorghums	Intermediate to high
Foxtail millet	Very low
Grain sorghums	High to very high
Johnsongrass	High to very high
Pearl millet	Very low
Sorghum-sudan hybrids	Intermediate to high
Sudangrass common varieties	Low to intermediate
Sudangrass hybrids	Intermediate

Source: Sulc, 2000.

Environmental conditions such as drought or frost are usually associated with high levels of HCN in forages. Concentrations of HCN are usually at the highest level in younger plants

(under knee high) versus more mature plants and on regrowth following haying or grazing. Toxic levels are higher in fresh forage than in cured forage. If the hay is not properly cured, toxic levels of HCN can be present (**Table 4**). The HCN concentration in the leaves is higher than the stems with the upper leaves containing higher concentration than the lower leaves. Prussic acid concentration decreases as the plants become taller and more mature. Usually plants higher than 18 inches are less likely to have high HCN. Prussic acid toxicity might not be dangerous under mature conditions, but caution is advised when grazing. Forage plants have a higher potential for HCN formation when soil is high in nitrogen and deficient in phosphorous and potassium. Plants growing under drought conditions and receiving more than 75 lb N/ac in one application could contain high HCN concentrations. An increase in HCN concentrations can also result when forages are treated with broadleaf herbicides such as 2,4-D at rates that can stunt plant growth.

Table 4. Prussic acid (HCN) concentration in forages

HCN Concentration (ppm)			
Dry Matter	**Fresh Harvested**		**Potential Effect on Livestock**
0 – 500	0 – 100	**SAFE**	Forage is generally safe and should not cause toxicity.
500 – 1000	100 – 200	**DANGEROUS**	Potentially toxic and forage should be fed at a restricted rate in the diet.
>1000	>200	**TOXIC**	Very dangerous to livestock and will usually cause death. Drying, ensiling, or allowing the forage to mature cold will reduce prussic acid concentration. Retest before feeding.

Source: Stoltenow and Lardy, 1998; Harris and Shearer, 2003; Strickland et al., 2004.

To prevent HCN toxicity, it is important to graze only when plants (sorghum, sorghum-sudan hybrid, or johnsongrass) have reached a height of 18 to 24 inches. Do not allow animals to graze fields with succulent, young, short growth which might be toxic. Regardless of the height, it is not advised to feed any form of drought-damage forage within 5-7 days following a good rain. During this rapid period of growth and recovery, accumulation of HCN and nitrates in young tissues is more likely to occur. Avoid grazing wilted plants or plants with young regrowth and do not depend on drought damaged forages as the only feed source. Keeping dry hay or

green chop for other forage crops available is a good strategy. Suspected mildly toxic forages can be fed to livestock receiving dry hay or grain feed. Uneven forage growth in sorghum hybrids could be utilized as silage. Silage could have toxic levels of prussic acid, but it usually escapes as a gaseous form while being moved and fed. If frosted forage is ensiled, allow fermentation to take place for at least six to eight weeks before feeding. Although HCN usually dissipates within 48 hrs after a killing frost, do not use frost-damaged forages as pasture or green chop during the first 7 to 10 days or until the biomass has dried out and turned brown. Avoid grazing at night when a frost is likely to occur.

Prussic acid is a potent and rapidly acting toxic compound. Animals feeding on forages high in prussic acid will develop symptoms within few hours after consuming to toxic forage. Animals are often found dead. Some of the symptoms include voiding of urine and feces, convulsions, paralysis, coma, staggering, drooling (salivation), runny eyes (lacrimation), and breathing cessation. The mucous membranes are usually bright pink, and the blood will be a characteristic bright cherry red. In the early onset of toxicity, treat the animal with molasses that has been diluted with water. Feeding small or moderate amounts of grain prior to grazing on summer annual forages will help prevent possible toxicity. Starch from the grain is broken into glucose in the digestive tract preventing the rate of release of HCN from the glycoside by the enzyme emulsin. Livestock affected by prussic acid could be treated with a combined intravenous dose of sodium nitrite and sodium thiosulfate. The dosage and method of administration are very critical. Contact your veterinarian for specific doses and other treatments if prussic acid toxicity is suspected and can be caught in time.

Tall Fescue Toxicosis

Kentucky-31 (K-31) tall fescue dominates the majority of the tall fescue pastures in Mississippi. It contains an endophyte, a fungus that lives symbiotically inside of a plant and produces toxins that can affect animal health and performance. The enodphyte produces toxins known as ergot alkaloids and some of the compounds include clavinet alkaloids, lysergic acid amides, and ergopeptimes. Ergovaline and lolitrem B are the most prevalent alkaloids proven to be responsible for fescue toxicosis (**Table 5**). These ergot alkaloids are usually highly concentrated in the seed although their toxicity can also be measured in the leaf and stem of the fescue plant. Concentrations of the ergot alkaloid usually peak in late spring when seed heads are present, decreasing during the summer and then increasing again in the fall. Producers should be aware that high concentrations of ergot alkaloids not always coincide with the visible symptom of toxicosis in the livestock due to the residual effects. The residual effect is due to fat deposits in the animal that can serve as a reservoir for toxic alkaloids, allowing the alkaloids to be released gradually during the grazing season and after the animals have been removed from infected tall fescue pastures.

A fungal endophyte in tall fescue is thought to be responsible for causing three types of disorders: fescue foot, bovine fat necrosis and fescue toxicity. The occurrence of tall fescue toxicity is determined by the percentage of infected tall fescue plants in the pasture, the length of time the animals spend grazing infected tall fescue, the weather, and pasture fertility management.

Fescue foot usually occurs when cattle graze infected fescue in cool weather. Cattle suffering from fescue foot have a rough hair coat, signs of weight loss, difficulty regulating body temperature (fever), an increase respiration rate, tissue death in the legs, lameness and swelling of the legs, sloughing of hooves, and loss of tips of tails and ears.

Table 5. Ergovaline and Lolitrem B threshold levels for different livestock classes.

Livestock	Ergovaline	Lolitrem B
	---------- parts per billion (ppb) ----------	
Horses[1]	300 – 500	Not determined
Cattle	400 – 750	1800 – 2000
Sheep	500 – 800	1800 – 2000

[1]Except for mares in the last 60 to 90 days of pregnancy when the threshold is zero.
Source: Aldrich-Markham et al., 2003.

Bovine fat necrosis is a condition caused by hard fat deposits in the abdominal cavity. This condition can cause digestive problems and problems with foaling in horses that could lead to death for the foal. It is recommended to remove pregnant mares from infected fields 90 days prior to the expected foaling date. There is a high association of bovine fat necrosis and high fertilization of K-31 tall fescue pastures with broiler litter or high nitrogen rates.

At the present time, there is no cure for tall fescue toxicosis. Fescue toxicity occurs during hot weather. Some of the symptoms are low animal daily gains (**Fig. 2**), reduced forage intake, lowered conception rates, intolerance to heat, failure to shed the winter hair coat, fever, excessive salivation, vasoconstriction (narrowing of blood vessels and restricting blood flow), nervousness, low serum prolactin, agalactia (milk production failure), thick and retained placenta, and dystocia (calving difficulty). These symptoms usually appear within 10 to 20 days of feeding on endophyte infected fescue.

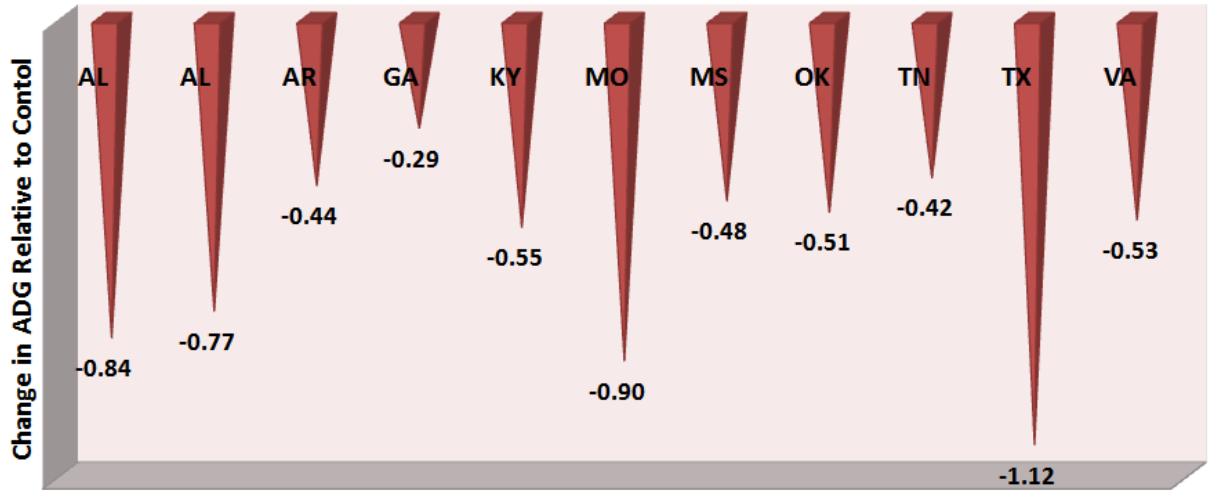

Figure 2. Change in animal daily gains of steers grazed on pastures with a high-endophyte (16 to 100%) or a low-endophyte (0 to 8%) tall fescue. **Source:** Roberts and Andre, 2004.

There are several management strategies designed to limit the amount of toxins that are ingested by the livestock. One strategy to reduce toxicity is the replacement of K-31 with

cultivars that are endophyte-free or contain novel endophytes. Another approach to reduce the toxicity effect includes interseeding legumes to dilute the toxins and reduce tall fescue consumption. A grazing management approach includes rotating the cattle to non-toxic pastures and reducing seed development by early close grazing of tall fescue. Other strategies include supplementing the diet and limiting the rate of nitrogen fertilization in the summer. The only known means of spread of the endophyte is by infected seed. It is possible to prevent tall fescue toxicity by using certified endophyte-free seed or by just using a small percentage of tall fescue in the pasture.

Summary

When producers rely on pastures to supply the nutrient requirements of the livestock, it is important to pay close attention to how forages are managed along with weather conditions and fertility strategies. Forage testing becomes a very important part of determining nitrate and prussic acid toxicity. Toxic substances in forages and weed consumed by livestock could affect animal performance and health by reducing productivity, causing symptoms of illness and in some cases death. This document should be used by cattle producers as a guide when evaluating and treating either individual animals or groups of animals experiencing disorders caused by different forage species.