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Nitrate and Prussic Acid Poisoning in Cattle[a]

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The Story in Brief

Death loss from nitrate is an occasional problem in ruminants consuming certain annual forages, particularly sorghum hybrids. Nitrate accumulation usually results from plant stress such as drought and is accentuated by excessive soil nitrogen. Most nitrate accumulates in plant stems rather than leaves, and concentration tends to be highest in immature forage. A characteristic symptom of nitrate toxicity is a chocolate-brown color to the blood. Poisoning can be avoided with good management. Fertility programs consistent with plant needs and growing conditions minimize the problem. Potentially dangerous forage should be tested before feeding. Often hay containing excessive nitrate can be fed safely when diluted with other feed, particularly concentrates.

Prussic acid (HCN) causes acute poisoning in ruminants grazing sorghums, especially johnsongrass. Many of the same factors that tend to cause nitrate accumulation -- drought, reduced sunlight, excessive soil nitrogen, young plants -- also increase HCN potential. HCN potential is greater in leaves than stems. Proper curing for hay or ensiling greatly reduces the potential for HCN poisoning. Lush regrowth in sorghums occurs after cutting for hay, grazing or frost. This regrowth is often dangerous. Contrasted to nitrate toxicity, HCN poisoning is characterized by a bright cherry-red color to the blood. As with nitrate, HCN potential can be minimized through proper fertility programs and variety selection and by testing questionable forage.

Introduction

Annual forage crops like sorghums and small grains make valuable contributions to profitable beef production in Oklahoma. They are well adapted, very productive and provide high quality forage. Infrequently, some of these plants accumulate toxins that can result in costly livestock losses.

Nitrate -- The Problem

Nitrate (NO_3) is the primary nutrient form of nitrogen in most soils and is a normal constituent of plants. Normally nitrate is assimilated so rapidly following uptake from soil that its concentration in plant tissues is low. Occasionally, excessive levels occur in plants. The most notorious accumulators of nitrate in Oklahoma are the sorghums. Other annuals that less frequently accumulate nitrate are small grains (wheat, oats, rye and barley) and millet. Some perennial grasses (bermudagrass, fescue and johnsongrass) and certain weeds (pigweed, mustard, nightshade and lamb's quarters) also can contain dangerous levels.

Accumulation is usually triggered by some environmental stress, where plant growth is restricted but absorption of nitrate from soil continues. The most common stress of summer annuals is drought. Lack of moisture, together with excessive soil nitrogen for existing growing conditions, is a frequent cause of toxic levels of nitrate in sorghums. Other stress factors which favor buildup are reduced sunlight from cloudiness or shading, frost, certain herbicides including 2,4-D, acid soils, low growing temperatures, and deficiencies of essential nutrients like phosphorus and sulfur.

When more soil nitrogen is present than needed for maximum growth, some plants tend to accumulate nitrate even without environmental stress. This response is particularly true with hardy soil feeders like sorghums, noted for "luxury consumption" of certain nutrients.

When accumulation occurs, the concentration of nitrate in plant parts is greater in stems than leaves. Seeds seldom contain significant amounts. Rate of uptake diminishes with increasing maturity; thus mature plants usually contain less nitrate than immature ones.

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[a] Adapted from Armbruster, 1979

Differences in potential for accumulation exist among species and varieties.

The level of nitrate that causes toxicity in ruminants varies depending on rate of intake, diet, acclimation to nitrate and nutritional and reproductive status. As a rule, forage containing less than 5,000 ppm NO_3 on a dry matter basis is safe for non-breeding cattle. Forage containing 5,000 to 10,000 ppm NO_3 is considered potentially toxic when provided as the only feed. Forage containing over 10,000 ppm NO_3 is considered dangerous but often can be fed safely after proper dilution with other feeds.

The utilization of high nitrate forages in cow herds and dairies may be more complicated than once believed. Georgia researchers studied reproductive failures in a drought-plagued dairy farm and noted that moderate levels of nitrate in the total diet (> 1200 ppm) were associated with reduced blood progesterone concentrations of cycling and early pregnancy dairy cows. Progesterone is a hormone produced by the corpus luteum on the ovary, and is important in the maintenance of pregnancy. They suggested that the impaired function of the corpus luteum and lowered progesterone concentration was related to the high incidence of early embryonic death and repeat breeder cows found in this herd. Producers must remember that drought may cause many adverse situations that result in reduced reproductive performance. Lowered quality and quantities of feed available can cause a negative energy balance for lactating cows. Any sharp decline in body condition will also impair reproductive performance. Therefore, more research is definitely needed to specifically tie moderate levels of dietary nitrate to reduced reproductive performance.

The effects of feed and water levels are additive, and both should be considered in avoiding or assessing nitrate problems. Common causes of high nitrate levels in water include shallow wells contaminated with surface water, water containing animal wastes, and surface runoff from heavy rain after fertilization with ammonium nitrate. Water containing more than 200 ppm NO_3 is potentially toxic, especially when feed also contains an excessive level.

The nitrate content of feed and water is reported in different forms by laboratories. The form for expressing nitrate levels must be considered to avoid errors in determining the potential for toxicity. The information in Table 1 should aid in interpreting laboratory results.

Although the term "nitrate toxicity" is commonly used, the toxic principle is actually nitrite. Nitrate is converted to nitrite in the rumen. Nitrite is absorbed from the rumen and converts blood hemoglobin to methemoglobin. Methemoglobin cannot transport oxygen to the body tissues, the animals die from oxygen insufficiency.

The first symptom to appear is a grayish, blueish to brownish discoloration of nonpigmented skin and mucous membranes of the mouth, nose, eyes and vulva. This discoloration results from the lack of oxygen of the blood, a distinct characteristic of nitrate toxicity that persists several hours after death. As the syndrome progresses a staggering gait, rapid pulse, labored breathing and frequent urination develop, followed by collapse, coma and death. Symptoms often occur rapidly, within 1/2 to 4 hours after ingestion of a toxic dose. Some animals exhibit symptoms but recover spontaneously and completely. Pregnant animals may abort a few days later. Treatment of nitrate poisoning with methylene blue is effective if administered soon after symptoms appear. The early symptoms of prussic acid and nitrate poisonings are similar. However the treatment for prussic acid inflicted cattle may be lethal to cattle suffering from nitrate poisoning. Call your veterinarian immediately for diagnosis and treatment if any of the above symptoms are present.

Preventing Nitrate Poisoning

Use of true sudans or sudan-sudan hybrids instead of sorghum-sudan or sorgo-sudan hybrids may be warranted to reduce potential for accumulating nitrate. Usually the potential for problems is only reduced, not eliminated. Differences in yield, quality, drought tolerance and insect and disease resistance

Table 1. Equivalent levels of nitrate.

Nitrate (NO_3)		Nitrate-nitrogen (NO_3-N)		Potassium nitrate (KNO_3)	
ppm*	%	ppm	%	ppm	%
200	.02	46	.0046	326	.0326
5000	.5	1150	.115	8150	.8150
10,000	1.0	2300	.23	16,300	1.63

*parts per million

should not be ignored. Considerable information is available from the Oklahoma Agricultural Experiment Station on production characteristics of summer annuals.

Extra caution should be exercised when moisture stress occurs in sorghums before harvest or grazing. Samples of plants from different areas of the field, particularly those showing the severest stress, should be tested for nitrate content. If the level is dangerous, harvest should be delayed until rain comes and the plant increases in maturity. Occasionally forage that is questionable as hay can be grazed safely when forage is abundant, because animals tend to select leaves and refuse stalks. Silage may also be a good alternative since appreciable reduction in nitrate levels occurs during ensiling. Remember, however, forages that contain high concentrations of nitrates at the time of ensiling, although reduced in nitrate content, may still be dangerous when fed.

Poisoning can be avoided by routinely testing any forage -- pasture, hay or silage -- suspected of containing excessive nitrate. A qualitative check called the diphenylamine test (Table 2) can be used to screen forages for potential harm. Positive results indicate more than 5,000 ppm NO_3 and possible danger. Most OSU county Extension offices and many veterinarians have test kits.

If results of the diphenylamine test are positive, forage samples should be sent to a laboratory for quantitative analysis. The Oklahoma Animal Disease Diagnostic Laboratory at OSU analyzes forages for nitrate level at a cost of $6.00 per sample.

Table 2. Diphenylamine Test for excessive nitrate in forage.

1. This is a qualitative test to evaluate forages (hay, pasture, silage) for nitrate levels that are potentially dangerous to ruminants.

2. USE CAUTION: The solution contains a strong acid. Avoid contact with skin, eyes and clothing. Store in a cool dark place. Do not add water or any other material to the solution.

3. Carefully place a drop of the solution at various locations on the inner tissue of the plant stem; repeat for several stems in each sample.

4. If an intense blue color appears in a few seconds, the forage contains potentially dangerous levels of nitrate.

5. If the results are positive (blue color), send the forage to a laboratory for quantitative analysis before feeding.

6. Occasionally, false positive reactions occur; however, any sample resulting in a positive reaction should be tested in the laboratory.

7. Avoid contaminating the solution in the bottle with plant tissue or other material; discard any solution that is not clear.

When forage is collected for analysis, it is essential that representative samples be taken. Samples should contain all parts of the plant. Just sending leaves of a forage could result in an underestimation of the potential nitrate problem. Although samples are often pooled for other laboratory analyses like moisture and protein, nitrate tests are often required on individual bales or from specific areas of a field to accurately assess the potential for toxicity. To illustrate this point, an evaluation of 15 large round bales of sorghum hybrid hay from one cutting showed considerable variation from bale to bale, with nitrate levels ranging from 17,500 to 39,000 ppm.

In most instances, hay high in nitrate can be fed safely with adequate laboratory testing and good management. The best alternative is to dilute dangerous forage with feeds low in nitrate, preferably concentrates. Unfortunately for many producers, proper dilution makes it necessary to grind and mix. Gradual acclimation to questionable feed is a good practice to minimize risk. Animals should be healthy, on a good plane of nutrition and filled with low nitrate feed before access to nitrate-containing forage.

Table 3 gives guidelines for using forages that contain nitrates.

Feeding of corn for 10 days prior to ingestion of high nitrate feeds has been shown to alleviate some of the symptoms noted in cows that were fed the high nitrates but had no access to the grain. The cows that had been fed 7 lbs. of grain prior to and during the challenge with high nitrate had only moderate changes (25% - 35%) in the percentage of hemoglobin that was changed to methemoglobin. The cows fed no grain but challenged with high nitrate ingestion had 60% - 70% of the hemoglobin converted to methemoglobin. The amount of oxygen carrying capacity of the blood was greatly decreased and several cows had to be treated with methylene blue because of nitrate toxicity symptoms. In the same experiment, it was noted that 3.5 lbs of corn fed per day was not enough to slow the conversion of hemoglobin to methemoglobin to safe proportions.

Prussic Acid -- The Problem

Prussic acid, also called hydrocyanic (HCN), is normally not present in plants. However, several common plants can accumulate large quantities of cyanogenic glycoside. When plant cells are damaged by wilting, frosting or stunting, the glycoside degrades to form free HCN. Conditions in the rumen also favor degradation of the glycoside to free HCN. Thus plants which contain the glycoside have the potential to cause HCN toxicity when consumed by ruminants.

In Oklahoma, plants most likely to cause HCN poisoning are sorghums. The potential is greatest for johnsongrass and least for true sudans. Other materials with HCN potential include white clover, vetch seed and chokecherry.

As with nitrate buildup, some stress usually triggers accumulation of cyanogenic glycoside in plant tissue. The potential for accumulation and HCN toxicity increases during drought. Occasionally, poisoning occurs when hot, dry winds induce temporary

Table 3. Nitrate levels in forages for cattle.

(Guidelines adapted from Guthrie, 1986 Georgia Extension Service)

Level of NO_3 (Dry Basis)		
Parts per million	Percent	Comment
0-1000	0.0 - .10%	This level is considered safe to feed under conditions where excessive nitrates from other sources (i.e., water) are not a problem.
1000 - 5000	.10 - .50%	This level could be fed to non-breeding cattle. Forages containing over 2500 ppm should be limited to 50% of total ration on a dry matter basis for pregnant cows and lactating dairy cows. Expect erratic and lowered milk production when nitrates exceed 2500 ppm in forage.
5000 - 10000	.5 - 1.0%	Feed may be fed to non-breeding animals if limited to 50% of the total dry matter in the ration. Expect potential abortions, drop in milk production and nitrate symptoms if feed is not diluted more.
10000+	1.0% or more	Expect abortions, acute symptoms and death.

moisture stress in plants. The potential for poisoning is greater with excessive soil nitrogen and young plants. Toxicity is also more likely when periods of rapid growth are followed by cool, cloudy weather. Lush regrowth after cutting for hay, grazing or frost is particularly dangerous.

Unfortunately for the livestock producer, often the only indication of prussic acid poisoning is dead animals. HCN is one of the most potent, rapid-acting poisons known. It interferes with oxygen use at the cellular level. When a lethal dose is consumed, animals die from asphyxiation in a few minutes.

When seen, clinical signs occur in rapid succession. Initially there is excitement and muscle tremors. Rapid and difficult breathing follow. The animal goes down, gasps for breath and may convulse. The pupils are dilated and mucous membranes are bright pink. A characteristic sign of HCN toxicity is bright cherry-red color to the blood, a symptom that persists several hours after death. Although blood is oxygenated, HCN interferes with the release of oxygen from oxyhemoglobin to other tissues. This situation contrasts with nitrate toxicity where oxygenation of blood is restricted.

Treatment of HCN poisoning, with a mixture of sodium nitrate and sodium thiosulfate or with methylene blue, can be successful if administered soon after symptoms appear. A veterinarian should be consulted for diagnosis and use of treatment drugs, because HCN toxicity is often confused with nitrate poisoning and other toxins of plant origin. A veterinarian can also assist in collecting plant and animal tissues for analysis and in interpreting laboratory results.

Preventing Prussic Acid Poisoning

As with nitrate, most problems with prussic acid can be avoided with proper management of forage and animals. Any forage crop though to contain HCN potential should be tested before animals are grazed or fed. Sorghums fertilized heavily with nitrogen and stunted by drought or cool, cloudy weather should be suspected. Risk of poisoning from sorghums can be reduced by using a maximum of about 50 pounds of nitrogen per application.

Young plants have a higher HCN potential than more mature ones, so grazing of sorghums should not begin until plants are 18 to 24 inches in height. This practice also applies to regrowth that occurs after cutting for hay or grazing. If regrowth occurs following frost, grazing should be delayed until a hard freeze kills the entire plant. Sorghums should not be pastured following killing frost until plants have thawed and wilted for a few days.

Pastures should be grazed to a uniform height, then animals should be removed to prevent selective consumption of lush regrowth. Rotation grazing and heavy stocking rates help in this regard. To acclimate cattle to new pasture, it is advisable to fill animals on native grass or hay during the day, then graze sorghums in late afternoon and evening.

Proper field curing or ensiling results in considerable loss of HCN. If growing forage is questionable as pasture, harvesting for hay or silage reduces the potential for HCN toxicity. However, if hay is poorly cured before baling, extremely high in HCN potential at cutting or contains johnsongrass, it still may cause problems.

Plant varieties differ in their potential for prussic acid poisoning. As with nitrate, chances for HCN toxicity are somewhat lower with true sudans and sudan-sudan hybrids than with sorghum-sudan or sorgo-sudan hybrids. Information on HCN potential of summer annuals, together with data on yield and other characteristics, is available from the Oklahoma Agricultural Experiment Station.

A quick qualitative test for HCN potential in plant tissue can be performed. Most veterinarians have the chemicals available to conduct the test. This test also can be used to confirm the presence of HCN in rumen contents of animals that die from prussic acid poisoning. Leaves are higher in HCN potential than stems. Glycoside levels increase during the morning, then level off and begin declining in the afternoon and evening. Therefore, samples for prussic acid analysis must include leaf tissue and should be collected in late morning or early afternoon.

A quantitative analysis indicating the level of HCN in plant tissue can be performed by the Oklahoma Animal Disease Diagnostic Laboratory in Stillwater at a cost of $6.00 per sample. Proper preservation of samples for chemical analysis is a must; otherwise HCN can be liberated and lost, and results are of little value. Because of the volatility of hydrocyanic acid, false negative test results can occur.

Fresh forage should be randomly sampled from several locations in the field. If hay is sampled, cores should be taken from several bales. Two or three handfuls per sample should be sealed in a plastic bag, stored in the dark, refrigerated unfrozen, and delivered to the laboratory without delay.

The level of HCN required to cause toxicity varies, depending on rate of intake and individual animal tolerance. Generally speaking, any forage analyzing more than 750 ppm HCN on a dry matter basis should be viewed as dangerous.

Summary

Nitrate and prussic acid poisonings are both potentially lethal to cattle. Nitrates will remain in the forage for months, therefore the potential problem in summer forages may not be apparent until the winter feeding begins. Prussic acid poisoning most often occurs when cattle ingest standing forages in pastures and fields and is much less likely to be present next winter in harvested hays. Testing forages for nitrate content can allow Extension specialists and veterinarians to help the producer manage the feeding of potentially dangerous forage. If poisoning symptoms such as those listed above occur, call your veterinarian immediately. Quick diagnosis and treatment is vitally important.

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