

Nitrate and Prussic Acid Toxicity in Forage

Causes, Prevention, and Feeding Management

Nitrate toxicity

Nitrate is a natural product formed from the oxidation of organic compounds. Most of the soil nitrogen absorbed by plant roots is in nitrate form. Normally, nitrate in a plant is rapidly converted to amino acids by the enzyme nitrate reductase. This reduction requires energy from sunlight, adequate water, nutrients, and favorable temperature. When plants are stressed, the nitrate-to-protein conversion is disrupted and nitrates begin to accumulate.

Why nitrates are toxic

Nitrate toxicity is a misnomer because nitrite (NO_2), not nitrate (NO_3), is poisonous to animals. After a plant is eaten, rumen bacteria rapidly reduce nitrates in the forage to nitrites. Normally, the nitrites are converted to ammonia and used by rumen microorganisms as a nitrogen source. If nitrate intake is faster than its breakdown to ammonia, however, nitrites will begin to accumulate in the rumen. Nitrite is rapidly absorbed into the blood system where it oxidizes hemoglobin to methemoglobin. Red blood cells containing methemoglobin cannot transport oxygen, and the animal dies from asphyxiation.

Animals under physiological stress (sick, hungry, lactating, or pregnant) are more susceptible to nitrate toxicity than healthy animals. Toxicity is related to the total amount of forage consumed and how quickly it is eaten, but, generally, if forages contain more than 6,000 ppm nitrate, they should be considered

potentially toxic (Table 1).

Although all livestock are susceptible to nitrate toxicity, cattle and horses are affected most often. Sheep and swine generally do not eat enough high nitrate forage to cause problems.

Symptoms of nitrate toxicity may appear within a few hours after eating or not for several days. Signs of chronic toxicity include reduced appetite, weight loss, diarrhea, and runny eyes. However, these are nonspecific symptoms of numerous disorders and are not a reliable diagnosis of nitrate poisoning. Low nitrate levels can cause abortion without any other noticeable symptoms.

Acute toxicity usually is not apparent until methemoglobin approaches lethal concentrations. Symptoms include cyanosis (bluish color of mucus membranes), labored breathing, muscular tremors, and eventual collapse. Coma and death usually follow within two to three hours. Postmortem confirmation of nitrate toxicity is chocolate-colored blood; however, the color will change to dark red within a few hours after death.

Diagnosis and treatment of nitrate toxicity should be performed by a veterinarian. However, in acute cases where time is limited, an antidote of methylene blue can be injected to convert the methemoglobin back to hemoglobin.

Forages suspected to contain high nitrate levels should be tested by a laboratory before feeding. Unfortunately, different laboratories may report nitrate levels as either nitrate (NO_3), nitrate-nitrogen ($\text{NO}_3\text{-N}$), or potassium nitrate (KNO_3). Potassium nitrate, nitrate-nitrogen, or percent nitrate can be converted to ppm nitrate using the conversion factors in Table 2.

Table 1. Level of nitrate in forage (dry matter basis) and potential effect on animals.

ppm Nitrate	Effect on Animals
0-3,000	Virtually safe.
3,000-6,000	Moderately safe in most situations; limit use for stressed animals to 50% of the total ration.
6,000-9,000	Potentially toxic to cattle depending on the situation; should not be the only source of feed.
9,000 and above	Dangerous to cattle and often will cause death.



Table 2. Conversion factors for expressing nitrate content of forages.

Potassium Nitrate x 0.61	= Nitrate (ppm)
Nitrate-Nitrogen x 4.42	= Nitrate (ppm)
% Nitrate x 10,000	= Nitrate (ppm)

Plant factors

Plant Species. Nearly all plants contain nitrate, but some species are more prone to accumulate nitrate than others. Crops such as forage sorghum, grain sorghum, sudangrass, sudan-sorghum hybrids, and pearl millet are notorious nitrate accumulators. Weed species such as kochia, lambsquarters, sunflower, and pigweed also are routinely high in nitrate.

Under certain environmental and managerial conditions, wheat, corn, alfalfa, soybeans, oats, Johnsongrass, and other plants can accumulate potentially toxic levels of nitrate.

Stage of Growth. Nitrate content generally is highest in young plant growth and decreases with maturity. Sorghums and sudangrasses, however, are exceptions because concentrations usually remain high in mature plants. If plants are stressed at any stage of growth, they can accumulate nitrate.

Plant Parts. Nitrates normally accumulate in stems and conductive tissues. Highest nitrate levels occur in the lower one-third of the plant stalk. Concentrations tend to be low in leaves because nitrate reductase enzyme levels are high there. Grain does not contain appreciable amounts of nitrate.

Environmental factors

Drought. Nitrates accumulate in plants during periods of moderate drought because the roots continually absorb nitrate, but high daytime temperatures inhibit its conversion to amino acids. During a severe drought, lack of moisture prevents nitrate absorption by plant roots. Following a rain, however, the roots rapidly absorb nitrate and accumulate

high levels. After a drought-ending rain, it requires 7 to 14 days before the nitrates will be metabolized to low levels, provided environmental conditions are optimum.

Sunlight. Nitrate reduction occurs in young leaves and requires light as an energy source. Shaded plants lack sufficient energy to convert nitrate to amino acids. Plants growing in field corners may be shaded and are frequently high in nitrates. Extended periods of cloudy weather increase nitrate content. Dangerously high levels can occur when wet, overcast days follow a severe drought.

Frost, Hail, or Disease. Conditions such as hail, light frost, or plant disease can damage plant leaf area and reduce photosynthetic activity. With less available energy, nitrate reduction is inhibited and nitrates accumulate in the plant.

Temperature. Low temperatures (less than 55°F) in the spring or fall retard photosynthesis of warm-season plants and favor nitrate accumulation. Extremely high temperatures also increase nitrate concentrations by reducing nitrate reductase enzyme activity.

Management factors

Fertilization. Nitrogen fertilization increases soil nitrate levels and the subsequent uptake by plant roots. Nitrogen from decomposing organic matter also can contribute to nitrate accumulation. Applying high amounts of manure or other fertilizer, particularly in the late season, increases concentrations. Split nitrogen applications provide better nutrient distribution and reduce the potential for toxicity.

In addition to excess nitrogen, an imbalance of other soil nutrients can affect forage nitrate levels. Plants growing in soils deficient in phosphorus, potassium, and some trace elements have high nitrate concentrations.

Herbicides. Most broadleaf weeds which accumulate nitrate normally are not eaten by cattle, and weed control is generally unnecessary. However, selectively spraying weeds routinely high in nitrates can reduce the potential hazard

to livestock. Weeds damaged but not killed by a herbicide will have high nitrate levels because of depressed enzyme activity and reduced leaf area.

Harvest Technique. When roughages are made into silage, fermentation normally reduces nitrate levels by 40 to 60 percent. Forages with extremely high nitrate levels at harvest may still be dangerous after ensiling and should be analyzed before feeding. If forages are harvested as hay, nitrate concentrations remain virtually unchanged over time.

High nitrate forages may be grazed, but a dry roughage should be fed first to limit intake. Stocking rate should not be too high because overgrazing forces cattle to eat the stems, which contain the highest nitrate levels. Cattle should be removed from potentially susceptible forage for 7 to 14 days after a drought-ending rain. Lush regrowth of heavily fertilized grasses contains high nitrate levels and should not be grazed.

If plants are fed as green chop, the harvested forage should be fed immediately after cutting, not allowed to heat up. As the plants respire, nitrates are converted to nitrites, which are about 10 times more toxic than nitrates.

Feeding high nitrate forage

Before feeding potentially troublesome plants such as sorghum and sudangrass, analyze the forage for nitrates. Environmental conditions in Kansas create high nitrate concentrations in some forage virtually every year. Consequently, nitrate analysis is necessary to determine if the feed is potentially toxic. It is critical that representative samples be collected. Your Extension agent or laboratory representative can provide information on random sample collection and delivery to the lab. High nitrate forages still can be fed to animals if proper precautions are taken.

Adapt Cattle to High Nitrate Feeds Gradually. Nitrate toxicity frequently occurs in animals without prior exposure to nitrates. If nitrate levels in the forage are not excessively high (e.g., over 9,000

ppm) the animal will usually be able to adapt to increasing amounts in the feed. Frequent feeding in limited amounts through the day rather than large amounts once daily will increase the total amount that can be safely fed.

Dilute with Other Feeds. Blend high nitrate forage on a 1:1 basis with other feeds that are low in nitrates. After three to four weeks of feeding, the animals normally become adjusted to nitrates and the proportion of high nitrate forage can be increased.

Supplement Grain. Feeding 2 to 5 pounds of grain dilutes the amount of nitrate in the total ration and provides the energy necessary for bacteria to quickly convert nitrite to ammonia. Molasses also can provide needed energy for nitrite reduction but may be cost prohibitive.

Feed a Balanced Ration. Formulate rations to ensure adequate protein, vitamin A, and other nutrients. Nitrates may increase the requirement for vitamin A, but excessive supplementation is unjustified. Nonprotein nitrogen (urea) may worsen the situation and should not be given with high nitrate forages.

Do not Feed to Stressed Livestock. Animals that are sick, hungry, pregnant, or lactating have a lower tolerance for nitrates than healthy animals.

Provide Clean Drinking Water. Frequent intake of water dilutes nitrate levels. Ponds or ditches which collect runoff from feedlots, heavily fertilized fields, septic tanks, or manure piles are

Table 3. Level of prussic acid in forage (dry matter basis) and potential effect on animals.

ppm HCN	Effect on animals
0-500	Generally safe; should not cause toxicity.
600-1,000	Potentially toxic; should not be the only source of feed.
1,000 and above	Dangerous to cattle and usually will cause death.

likely polluted with nitrates.

Prussic acid poisoning

Prussic acid also is known as hydrocyanic acid or hydrogen cyanide (HCN). Prussic acid poisoning is caused by cyanide production in several types of plants under certain growing conditions. Sorghums and closely related species are the plants most commonly associated with prussic acid poisoning. These plants possess a cyanogenic molecule called dhurrin in their epidermal cells. In healthy, intact leaf tissue dhurrin is nontoxic. However, mesophyll cells located beneath the epidermis have an enzyme that removes HCN from dhurrin. If the leaves become damaged, dhurrin and its hydrolyzing enzyme will intermix and release cyanide.

Why prussic acid is toxic

Once eaten, cyanide is absorbed directly into the bloodstream and binds to enzymes in the cell. This cyanide complex prevents hemoglobin from transferring oxygen to individual cells and the animal dies from asphyxiation.

Cyanide poisoning is related to the

amount of forage consumed and the animal’s physiological condition, but HCN levels exceeding 200 ppm on a wet weight (as is) basis are dangerous. On a dry weight basis, forages with more than 500 ppm HCN should be considered potentially toxic (Table 3).

Prussic acid acts rapidly, frequently killing the animal within minutes. Symptoms include excess salivation, difficult breathing, staggering, convulsions, and collapse. Death from respiratory paralysis follows shortly. The clinical signs of prussic acid poisoning are similar to nitrate toxicity, but animals with cyanide poisoning have bright red blood that clots slowly, whereas animals poisoned with nitrate have dark, chocolate-colored blood. The smell of bitter almonds is often detected in animals poisoned with cyanide.

Because it occurs quickly, the symptoms are usually observed too late for effective treatment. In the absence of a veterinarian, and if there is no doubt about the diagnosis, the animal can be treated with simultaneous injections of sodium nitrate and sodium thiosulfate. Sodium nitrate releases the cyanide from the cell, which then binds with the sodium thiosulfate to form a nontoxic complex that is excreted. Animals alive one to two hours after the onset of visible signs usually recover.

Prussic acid concentration factors

Plant Species. Crop species most commonly involved with prussic acid poisoning are sorghums, Johnsongrass, and sudangrass. Potential cyanide production among varieties and hybrids of most summer annual forages varies widely. Grain sorghums are potentially more toxic than forage sorghums or sudangrass, whereas hybrid pearl millet

Summary Guidelines To Reduce Nitrate Toxicity

- Pay close attention to potentially troublesome plants, such as sorghum and sudangrass, which often have high nitrate levels.
- Avoid excessive application of manure or nitrogen fertilizer.
- Raise cutter bar 6 to 12 inches to exclude basal stalks. This also will minimize harvesting many weed species that have accumulated nitrate from shading.
- Delay harvesting any stressed forages. A week of favorable weather generally is required for plants to reduce accumulated nitrate.
- Never feed green chop that has been heated after cutting or held over night.
- Harvest plants containing high levels of nitrate as silage rather than hay.
- Have representative samples of suspect forage analyzed before feeding.

Table 4. Millet and sorghum types and their potential cyanide accumulations.

Millet or Sorghum Types	Cyanide Potential
Pearl and Foxtail millet	very low
Sudangrass varieties	low to intermediate
Sudangrass hybrids	intermediate
Sorghum-sudangrass hybrids	intermediate to high
Forage sorghums	intermediate to high
Shattercane	high
Johnsongrass	high to very high
Grain sorghums	high to very high

and foxtail millet generally have very low cyanide levels (Table 4). Indian-grass, flax, choke cherry, elderberry, and some varieties of birdsfoot trefoil can also cause prussic acid poisoning.

Plant Age and Condition. Young, rapidly growing plants are likely to contain high levels of prussic acid. Cyanide is more concentrated in young leaves than in older leaves or stems. New sorghum growth following drought or frost is dangerously high in cyanide. Pure stands of Indiangrass that are grazed when the plants are less than 8 inches tall can possess lethal concentrations of cyanide.

Generally, any stress condition that retards normal plant growth may increase prussic acid content. Hydrogen cyanide is released when plant leaves are physically damaged by trampling, cutting, crushing, chewing, or wilting.

Drought and Frost. Drought-stunted plants accumulate cyanide and can possess toxic levels at maturity. Freezing ruptures the plant cells and releases

cyanide. After a killing frost, wait at least four days before grazing to allow the released HCN to dissipate.

Prussic acid poisoning is most commonly associated with regrowth following a drought-ending rain or the first autumn frost. New growth from frosted or drought-stressed plants is palatable but dangerously high in cyanide.

Soil Fertility. Plants growing in soils that are high in nitrogen and low in phosphorus and potassium tend to have high cyanide concentrations. Split applications of nitrogen decrease the risk of prussic acid toxicity.

Animals. Most losses occur when hungry or stressed animals graze young sorghum growth. Ruminants are particularly susceptible to prussic acid poisoning because cud chewing and rumen bacteria both contribute to releasing cyanide. The enzyme responsible for hydrolyzing HCN from dhurrin is destroyed in stomach acid, which allows monogastric animals, such as horses and swine, to be more tolerant of cyanide than ruminants.

Feeding grain or hay before turning animals to pasture reduces rapid intake and dilutes the amount of cyanide consumed. Animals do not adapt or become immune to cyanide, but they can detoxify low HCN levels.

Harvest Technique. Prussic acid concentrations are higher in fresh forage than in silage or hay because HCN is volatile and dissipates as the forage dries. However, if the forage had an extremely high cyanide content before cutting, or if the hay was not properly cured, hazardous concentrations of prussic acid could remain. Hay or silage that likely contained high cyanide concentrations at harvest should be analyzed before it is fed.

Summary Guidelines To Avoid Prussic Acid Poisoning

- Do not allow hungry cattle to graze where prussic acid may be a problem.
- Do not allow animals to graze potentially troublesome plants after a light frost or after rain has ended a summer drought.
- Chop or ensile plants high in cyanide to reduce toxin levels.
- Have representative samples of any suspect forage analyzed before feeding.

Dale Fjell
Extension Specialist
Crop Production

Dale Blasi
Extension Specialist
Livestock Production, S.C.

Gene Towne
Extension Assistant
Agronomy



Cooperative Extension Service, Kansas State University, Manhattan

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