CONSERVATION AGRONOMY TECHNICAL NOTES

U. S. DEPARTMENT OF AGRICULTURE NEW MEXICO SOIL CONSERVATION SERVICE

NOTE NO. 29

October 28, 1976

RE: LIVESTOCK POISONING FROM FORAGE

The attached bulletins published by the Cooperative Extension Service of New Mexico State University provides information on livestock poisoning from forages.

Bulletins included are: 400 B-807 "Nitrate Poisoning from Forage"; 400 B-808 "Prussic Acid Poisoning"; and 400 B-809 "Control Grass Tetany in Livestock."

Additional copies of these bulletins are available from the County Extension Agent or the Cooperative Extension Service, New Mexico State University.

Attachments

AC
DC
TSC, Portland-2
Adjoining States-1
Director, Plant Science Division, SCS, Washington-2

11-64 4-L-19461

NEW MEXICO STATE UNIVERSITY
Livestock Guide
COOPERATIVE EXTENSION SERVICE

400 B-807

Nitrate Poisoning from Forage

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Nitrate poisoning in man and animals results from ingesting food or water containing high levels of a nitrate, or by inhaling nitrogen gases.

Although the condition is commonly referred to as nitrate poisoning, toxicity is primarily due to nitrite, a chemically reduced form of nitrate. Nitrites may be formed from nitrates before ingestion by animals or within the digestive tract of the animal after nitrate is consumed. The nitrite prevents the blood from carrying oxygen and the animal dies of suffocation.

Small amounts of nitrates are found in most plants and ground water. Under certain conditions nitrates accumulate in these materials to toxic levels.

Poisonous gases can be produced in silos by the reduction of nitrates present in the ensiled material and cause death of animals or people in or near the silo. This phenomenon usually occurs in the first few weeks after forages are put in the silo.

Sensitive Animals

Cattle are most commonly harmed. Sheep are relatively less affected by nitrate poisoning than cattle, either because cattle are better converters or because of the ability of sheep to further breakdown nitrite to ammonia.

Swine are highly susceptible to nitrite poisoning but are not as often affected as cattle or sheep. The different type of digestive tract and the smaller amount of roughages consumed make pigs less likely to ingest toxic amounts of nitrates.

The nitrate hazard in humans is usually associated with nitrates in the water supply or with poisonous gases produced in silos.

Goats, horses, dogs, and birds are also susceptible to nitrate poisoning.

Clinical Signs

In acute poisoning as a result of high concentrations of nitrates (1.5 to 5.0 percent of dry matter) animals are often found dead with no previous history of illness. Poisoned animals have signs of nausea, vomiting, abdominal pain, bloating, fast pulse, and prostration. The blood is usually dark brown but may be cherry red.

Lesions of nitrate poisoning are lung congestion, hemorrhages on the heart, brownish liver, chocolate-brown blood, irritation of the stomach and intestines and sometimes, hemorrhage in the urinary bladder. The brown color of the blood disappears three or four hours after death occurs.

Animal production losses may occur at sub-lethal nitrate levels. Abortion can result even if the parent survives. Milk production loss has also been reported. Vitamin A deficiency has been related to a high nitrogen level.

Testing for Nitrates

The diphenylamine test for nitrates can be used in the field. The test reagent is made by dissolving 500 milligrams diphenylamine in 20 milliliters of water and adding sulfuric acid (carefully) to make 100 milliliters. This stock solution should be stored in a brown bottle. It may be used without dilution to detect small amounts of nitrates or, after dilution with an equal volume of 80 percent sulfuric acid, on plant tissues or rumen contents. The development of a blue colored precipitate within 30 minutes constitutes a positive test.

The steps in conducting a nitrate quick test are:

1. Learn general conditions conducive to high nitrate content in forages.

2. Obtain portion of forage representative of that eaten by the animal. Or, obtain a sample of the rumen contents.

3. Finely crush sample in a low glass container (ash tray, etc.).

4. Add a small amount of distilled water (10-20 drops) and mix well with crushing action.

5. Add 80 drops of indicator (diphenylamine in concentrated sulfuric acid).

6. Formation of a deep blue color or precipitate within 30 minutes is indicative of a high concentration of nitrate in the forage and need for a more quantitative test is indicated.

Treatment

Death usually occurs so suddenly that treatment is not possible. Of animals that are treated there is usually a low recovery rate.

Poisoned animals should be handled quietly, and methylene blue should be administered intravenously. It is usually administered in a 1 to 4 percent solution which contains 5 percent dextrose at the rate of one gram of methylene blue for each 250 pounds of animal weight. Due to the vasodilation effect of the nitrate, vasoconstrictor drugs such as adrenalin should be administered. In chronic poisoning, vitamin A should be given.

Sources of Nitrates

Nitrates in Soils. Nitrates found in feeds and water result from nitrates produced in or added to the soil. Soil microorganisms reduce nitrogen to ammonium and then to nitrates.

Nitrates in Fertilizers. Commercial nitrogen fertilizers contain nitrogen as nitrate, ammonia, ammonium, or urea. The urea form of nitrogen, when applied to soils, is quickly converted to the ammonium form by enzymatic action. The ammonium form of nitrogen is converted to the nitrate form by microorganisms in the soil.

Nitrates in Forages

There are several factors which may contribute to the accumulation of toxic levels of nitrates in forages.

Environmental Factors:

1. Temperature—The rate of nitrate formation is very slow at soil temperatures below 50°F, but rates of conversion increase in direct proportion to increased soil temperatures up to 80-90°F.

2. Drought—Nitrate concentration in forages increases greatly when drought is experienced.

3. Light intensity—Low light, such as a cloudy day, causes nitrate accumulation in plants, since nitrates accumulate during the night and disperse after sunlight hits them.

Factors Within the Plant:

1. Species of plant—Some plant species convert nitrate to amino acids and protein more slowly than others.

Field crops known to cause nitrate poisoning include:

oats	sorghum	millet
barley	corn	soybean
wheat	Sudan	rape
rye	alfalfa	fescue

Weeds that accumulate nitrates include:

amaranths	witchgrass	smartweed
sunflower	nightshades	burdock
lambsquarter	white cockle	hounds-tongue
Russian thistle	velvetweed	fiddleneck
Canadian thistle	bull thistle	bindweed
sourdock	stinging nettle	elderberry
goldenrod	blue-green algae	ragweeds

Vegetables that accumulate nitrates include:

beets	radishes	squash	spinach
turnip	lettuce	parsnips	mangels
kale	celery	cucumbers	swiss chard

2. Variety of plant—Certain varieties of plants accumulate more nitrates.

3. Part of the plant–Nitrates are taken up through plant roots and move up the stem to the leaves where conversion to protein occurs. In the mature plant, the nitrate content of stems is usually higher than that of leaves. The grain itself is very low in nitrate.

4. Stage of maturity of the plant–The immature plant is usually higher in nitrate content than the mature plant.

Factors related to Toxicity to an Animal:

1. The concentration of nitrate in the forage consumed.

2. The total quantity of nitrate consumed.

3. The speed of intake of the toxic forage.

Management Practices

–Rations high in carbohydrates will reduce and sometimes prevent losses from nitrate poisoning.
–Weed control to remove plants may prevent nitrate poisoning. Plants freshly treated with herbicide may be more susceptible to grazing.
–Poisoned animals should be handled quietly.
–During periods of cold or cloudy weather, avoid grazing the area if possible. Allow animals to eat large quantities of dry forage and then graze suspected areas only during periods of sunlight.
–Test the nitrate concentration of the forage when in doubt.
–Do not graze forage too closely.
–Do not confuse this poisoning with grass tetany or wheat pasture poisoning.

3/71-2M

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NEW MEXICO STATE UNIVERSITY
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400 B-808

Prussic Acid Poisoning

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Some plants are capable, under certain conditions of producing hydrocyanic (HCN), or prussic acid, a highly poisonous substance. The formation of prussic acid is a result of the hydrolysis of glycosides (and is termed *cyanogenetic* or *cyanomorphic*). The violent toxicity to animals is caused solely by the HCN component. Little free HCN is found in healthy, actively growing plants. Free HCN is toxic to plant tissues.

There is little difference between toxic and lethal blood levels. Two milligrams HCN per pound of animal per hour is close to the minimum lethal dose. As a rule of thumb, plants which contain more than 20 milligrams of HCN per 100 grams can be potentially dangerous.

HCN acts by inhibiting the action of an enzyme that serves as a catalyst linking atmospheric oxygen with metabolic respiration. Thus, HCN poisoning causes asphyxiation (or oxygen starvation) at the cellular level.

Animals Affected

All domestic livestock and humans are susceptible to HCN poisoning. Cattle appear to be most susceptible. Ruminants, in general, are more susceptible than animals with simple stomachs.

Symptoms of Poisoning

HCN is one of the most rapid acting poisons. Signs of illness may start within five minutes of the time the animal starts eating the plant. Death may occur within 15 minutes, or the animal may live for several hours. Salivation and labored breathing are usually the first signs, followed by muscular tremors, incoordination, bloating, convulsions, and death due to respiratory failure. The heart may continue to beat for sometime after breathing stops.

Blood appears bright red and there is congestion of lungs and there may be lesions in other internal organs. There may be hemorrhage on the serous surface of the digestive tract.

Tests for HCN

The picrate test for cyanide has been adapted for field use on plant or stomach-content samples. Filter-paper strips dipped in an aqueous solution containing five percent Na_2CO_3 and 0.5 percent picric acid (shelf life not over four months) are allowed to almost dry. The strips are then suspended in a test tube over a sample of rumen contents, minced liver, or shredded plant material, to which has been added a few drops of chloroform or dilute acetic acid, and incubated at 30-35°C (equivalent to under-arm temperature). A positive result is indicated by a color change from yellow to red or maroon. The speed and intensity of this reaction may be used as an approximation of the amount of cyanide present. In some cases a color change may be detected almost immediately, but a negative test may not be assumed until after three hours have passed without change. It must be remembered that this test is sufficiently delicate to detect cyanide concentrations well below the toxic level.

Steps in Conducting Prussic Acid Quick Test

1. Learn general conditions and forage types known to lead to prussic acid buildup.
2. Place finely divided or crushed sample in test tube.
3. Moisten with water if dry.
4. Add 3 to 5 drops of chloroform to the sample. (Ten drops of vinegar can be used in an emergency)
5. Place moistened test paper in the test tube so it does not touch the sample.
6. Stopper the test tube tightly.
7. A color change from the original yellow to orange or brick red indicates HCN. The amount of HCN is related to the rapidity of color change.

Treatment

Sodium nitrite and sodium thiosulfate are specific antidotes for hydrocyanic poisoning. Solutions should be given intravenously because the poison acts rapidly. A typical preparation contains 1.2 percent sodium nitrite and 7.4 percent sodium thiosulfate given in a dose of 125 to 250 ml. by intravenous injection and repeated if necessary. Care must be given to distinguish between nitrate and HCN poisoning since this treatment would cause death if the animal suffered from nitrate poisoning. Methylene blue is sometimes used as a universal treatment for HCN and nitrate poisoning. Consult a veterinarian for rates.

Factors that influence HCN poisoning

Plant Factors

–Species of plants. Many species are valuable forage plants during much of the year but accumulate high levels of HCN under specific situations. Some of these plants include:

Annual goldeneye	Cocklebur
Sudangrass	Lima beans
Johnsongrass	Choke Cherry
Catclaw	Cherry
Bahia	Apples
Mountain Mahogany	Poison suckleya
Flax	White Clover
Vetch seed	Arrowgrass
Corn	Milo
Plums	

–Stage of growth. Young plants and sprouts contain more HCN than mature plants.
–Sunlight. Plants growing in the shade have more HCN than those grown in sunlight.

Animal Factors

–Speed of ingestion and release of HCN during digestion.
–Rumen microflora provoke and ruminal pH encourages HCN formation.
–Amount of moisture in the rumen. The animals may be found dead near water as moisture increases HCN formation.
–Size of animal.

Environmental Factors

–Freezing, wilting, or crushing of the plant causes the formation of a large concentration of HCN in the plant.

Management Factors

–When any of the cyanogenetic plants have been subject to drought, freezing, or other damage, they should not be grazed unless tested for hydrocyanic acid.
–Second growth sorghums should be grazed with caution.
–Remove all animals from the source when an animal is found poisoned on hydrocyanic acid.

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400 B—809

Control Grass Tetany in Livestock

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Grass tetany is a metabolic non-infectious disease of livestock. It resembles milk fever though distinctly different in many aspects. Grass tetany is also called grass staggers, wheat-pasture poisoning, lactation tetany, hypomagnesemia, and green oat tetany.

The exact mechanism of poisoning is not well understood. A magnesium deficiency in the animal serum is associated with tetany. Normal levels of blood magnesium are around 2 mg. per 100 milliliters of plasma. If the level drops down to almost 1, it is referrred to as hypomagnesia; and if it drops below 1, tetany can be expected to occur. A test of serum magnesium in affected animals is not a good indicator of grass tetany. Immediately preceding the occurrence of visual symptoms, plasma magnesium is low. As tetany begins, plasma magnesium usually increases to normal and is no longer a diagnostic symptom.

This problem is usually associated with animals grazing lush pastures in the spring, but it can occur in the fall and winter.

It is a world-wide problem. Its occurrence is sporadic and unpredictable for any given area. Animals on one farm or ranch may be affected while those on neighboring areas may have no problems.

An animal is unable to eat enough forage during periods of lush growth to provide adequate nutrients. For example, a 1000 pound lactating cow should consume 30 lbs. of dry matter per day. If the forage contains only 20 percent dry matter, the animal must eat 150 pounds of feed per day to meet these requirements.

Other studies have shown that the magnesium may be in the feed but unavailable to the animal. This may occur as a result of precipitation or chelation of the magnesium before absorption from the intestine. High levels of nitrogen or ammonia and potassium tend to decrease the availability of magnesium for the animal.

ANIMALS AFFECTED

This occurs primarily in cattle, sometimes in sheep and rarely in horses. Ruminants are more susceptible than nonruminants; older animals are more susceptible than younger ones; lactating animals more than dry animals; animals consuming young, immature forage more than those consuming mature or cured forages, and animals consuming roughages are more susceptible than those fed concentrates.

SYMPTOMS OF POISONING

In general, symptoms of acute toxicity may appear within a week or less or may not be developed for as long as six months. Animals affected may suddenly stop grazing and exhibit a feeling of discomfort and unusual alertness associated with muscular twitching. Startling reactions, such as continuous bellowing or running, may result when the animal is excited. The animal eventually starts staggering, collapses, followed by stiffening of muscles and violent jerking

convulsions with the head pulled back. The eyes and ears twitch, and chewing motions produce froth around the mouth.

A reflex response will occur if the animal is touched or if it hears a sudden noise. Labored breathing and a pounding audible heart develop followed by coma. Between convulsions, the animal may appear relaxed. Death occurs within an hour or up to six hours from the appearance of first signs unless treatment is given.

Chronic deficiencies are reported in many areas during the winter period when forage quality is low. This type is usually not accompanied by gross symptoms. It appears that animals may have considerable powers of adaptation to gradually reduced magnesium levels. Animals so affected develop a clumsy walk, or exaggerated motions and convulsions may develop if adequately stimulated.

Both acute and chronic grass tetany are characterized by loss of appetite, suppressed milk flow and dullness.

Milk fever of lactating cows is quite similar except animals become paralyzed rather than show violent muscular response. Serum calcium is low when milk fever is encountered, which may or may not be the case with grass tetany.

Livestock loss from nitrate poisoning may occur on areas similar to where grass tetany is experienced. Symptoms of nitrate poisoning include signs of nausea, vomiting, abdominal pain, bloating, fast pulse, prostration and dark brown or cherry red color of the blood. In both nitrate and grass tetany, animals may be found dead with no previous history of illness.

TREATMENT

Treatment of affected animals consists of an intravenous injection of commercially prepared calcium and magnesium salts in solution, at the rate of 300 to 500 ml. for an adult animal, supported by a subcutaneous injection of magnesium sulfate at the rate of 200 to 300 ml. of a 50 percent solution. A relaxant may be necessary before starting the intravenous medication. Initial response to this treatment is usually good, but relapses are common. Animals that recover enough to stay on their feet for 24 hours usually do not have the disease again. Treatment should be within the first two or three hours from the onset of symptoms.

A preventive treatment by feeding magnesium oxide at the level of two ounces or more per head daily during the danger period is recommended. There appears to be little or no buildup of magnesium reserves in the tissues by prior high magnesium diets. Magnesium levels may drop to the danger point as abruptly as within 48 hours.

The palatability of magnesium is very low. Therefore, a grain cube or other palatable carrier for the mineral may be necessary. Adequate salt is also necessary to reduce the incidence of this problem.

Heavy fertilization with magnesium of ranges or pastures may reduce the problem, but is generally not as effective as feeding the mineral.

PLANTS CONTRIBUTING TO THE PROBLEM

During periods of lush growth on predominately grass pastures, both natural and cultivated, the problem seems to be the worst. Numerous species of native range grasses have been reported to cause this problem. Introduced, seeded species such as crested wheatgrass appear to have problems more commonly. Cultivated species such as wheat, oats, rye, and barley commonly contribute to the problem.

WHAT CAUSES TOXIC CONDITIONS?

Climate—Cool moist and often cloudy conditions are associated with poisoning.

Plant—Rapidly growing, lush conditions appear to promote toxicity.

Fertilization—Heavy fertilization of plants with nitrogen and potassium correspond to toxicity.

Rumen action—An increased ruminal production of ammonia is associated with a decreased urinary excretion of magnesium. Under these conditions, dietary magnesium may be converted to the relatively insoluable hydroxide.

Class of Animal—Lactating animals appear to be more susceptible than dry animals.

Kind of forage—Roughages contribute to tetany more than concentrates.

MANAGEMENT PRACTICES

- If tetany develops in animals within the herd, change feed immediately. Any change should help.
- The addition of higher magnesium feeds would be of benefit.
- Be prepared to treat animals if tetany is noticed. This may not be possible due to speed of death.
- Graze pastures that might produce tetany with steers or dry cows where possible.
- Be on guard when the season is warm to initiate early growth and then becomes wet and cool.

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