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Nitrate, Prussic Acid (HCN) and Grass Tetany Problems in Cattle Feeding [1]

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Nitrate

Nitrate toxicity problems may occur when feeding some forages to cattle under certain conditions. Such conditions may prevail when dry weather (drought), follows heavy nitrogen fertilization, when cattle are grazing heavily fertilized and rapidly growing forages (usually with no grain), and when high-producing cows are fed such forages. The problem frequently involves more than the nitrate content of the suspect forage. Other factors usually involved are: 1) previous ration received or adaptation of the animal; 2) other available feedstuffs being fed; 3) total daily intake of nitrate and 4) general health of the animal.

Preventive measures to be taken in order to avoid possible problems include: 1) sampling and testing the feedstuffs; 2) grazing only a couple of animals the first week; 3) feeding a smaller amount the first week, and 4) using the feedstuffs in conjunction with a balanced ration.

Nitrates are simple nitrogen compounds that may accumulate in plants and water. They are normal constituents of feeds and are a source of dietary nitrogen for use by ruminants in normal amino acid synthesis. It is under unusual conditions when the plant stores abnormally high levels of nitrates or when water contains unusually high levels that problems may occur. The amount of toxic nitrate is decreased during fermentation of the crop in the silo. The highest accumulation of nitrates is found in the lower part of the corn and for this reason nitrate toxicity may possibly occur as the corn plants are grazed to the stubble.

The factors which may cause relatively high nitrate concentrations in various crops and weeds are levels of nitrogen in the soil, drought, intensity of light, and species of plants. Heavy applications of barnyard manure as well as nitrogen fertilizer in various forms cause this high level in the soil. Nitrate is found in highest concentration in the plant stem, and in very small quantities in the seed. This probably accounts for the higher levels of nitrates in drought-stricken corn silage, or other silages or hays made from crops where grain growth and development is retarded. Furthermore, during a dry period with resulting slow growth of the plant, apparently the nitrate in the plant is not converted to

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proteins as rapidly or completely as under normal growth conditions.

Toxic symptoms may occur 5 to 7 days after feeding high nitrate forage and death may follow 2 to 24 hours later. Nitrates themselves are relatively non-toxic. When the bacteria in the rumen convert these to nitrites, which are ten times more toxic than the nitrates, then the problem occurs. At lethal levels, the blood becomes chocolate colored instead of red. A brownish discoloration may be noted around white areas of the skin and non-pigmented mucous membrane of the eye, nose, and mouth. Death is actually due to respiratory failure. Other symptoms are staggering, rapid pulse, rapid and labored breathing, frothing at the mouth, frequent urination, diarrhea and incoordination.

The animal's susceptibility to nitrates is apparently affected by many factors. This is why so much controversy exists among various studies. Any change in the feeding program which alters the bacterial population or rumen function decreases the animal's ability to handle nitrates. Diets inadequate in protein and energy also make the animal more susceptible to nitrate poisoning. A deficiency of certain trace minerals and vitamins has also been reported as entering into the effect that nitrates have on the animal's health. Animals are more susceptible to nitrate poisoning when they are in an unhealthy state, nutritionally starved, or are receiving an unbalanced ration. Healthy animals that are receiving adequate quantities of a well balanced ration can tolerate larger quantities of nitrate in their diets than those being underfed.

Table 1 and Table 2 show nitrate levels of concern. Feed and water are considered separately. Safe levels in the feed may cause difficulty when coupled with questionable levels in the water.

Rumen microbes normally convert nitrate (NO_3) to nitrite (NO_2). Nitrite is reduced to ammonia (NH_4) and used by the rumen microbes to make protein. The problem arises when the concentration of nitrate in the rumen is greater than the capacity of the microbes to convert nitrite to ammonia. When this occurs, the nitrite concentration in the blood rises. Nitrite ties up hemoglobin so that the oxygen carrying capacity of blood is reduced and the animal suffocates.

The specific treatment includes the intravenous injection of either a 1% or 2% solution of methylene blue with a suggested dose rate of 20 mg/kg of body weight (1 gram/100 lb. body wt.). Where it is suspected that cattle are being exposed to potentially toxic levels of nitrites or nitrates, it is important that these animals receive adequate carbohydrates in their diet. Although impractical in most situations, contaminated water supplies can be made safe by boiling. Silage suspected of toxic levels of nitrate should be allowed to aerate overnight before feeding.

Prussic Acid

Frequently during late fall or early winter a freeze will occur causing the formation of dangerous glucosides in most varieties of sorghum, Sudan grass and Johnson grass. The glucosides are broken down and the free prussic acid (hydrocyanic acid) is liberated causing poisoning and death of animals consuming the forage. The poisonous properties may develop following a drought as well as a frost. A frost or freeze of young tender growing plants (under knee high) is the most dangerous. Well advanced growth is not likely to be dangerous under most conditions but caution is advised.

A laboratory test may be conducted for the presence of prussic acid. When a problem is suspected, feed forage to only one or two animals or collect a sample of the forage for analysis. If the plants are high in prussic acid, animals will develop symptoms within 1 to 2 hours. (Table 3) Treat affected animals immediately with molasses diluted with water. Contact your veterinarian for other treatments should the problem develop and be caught in time.

Feeding some grain to animals prior to grazing will help prevent the problem since starch from the grain forms glucose in the digestive tract.

Reactions which occur quickly and strongly could support clinical diagnosis of prussic acid poisoning. Clinical signs would include a characteristic and diagnostic "bitter almond" odor on the expired air, convulsions, paralysis, stupor, coma,

and cessation of respiration. Factors influencing the release of HCN from grasses or sorghums would include high concentrations in young, actively growing plants, especially after nitrogen fertilization, or rapid growth after stunting by frost or drought, quantity of plant ingested, previous diet of the animal, pH of rumen, or rate of release of HCN from the glycoside by the enzyme emulsin.

Analyses to date on several strains of African star grass have yielded HCN levels from 27 to 108 ppm. It is apparent that these pastures would be safe. Work will continue to establish the variation in African star grasses in early, rapidly growing plants, those recently fertilized or in young, rapidly regrowing plants.

Research data has repeatedly confirmed the value of sodium nitrite and sodium thiosulfate in effectively controlling animals experimentally and clinically poisoned on cyanogenic plant material. In fact, dosage with these two antidotes in combination will effectively protect against 40 times the lethal dose of cyanides. Giving cattle 30 to 125 ml of a 2.0% sodium nitrite and 30% sodium thiosulfate solution intravenously, produces methemoglobin which competes with the cytochrome oxidase enzyme necessary for oxygen transportation and release to the tissues. Cyanmethemoglobin is formed. Further detoxification is achieved by the sodium thiosulfate solution intravenously, which reacts to form thiocyanate (SCN-), a relatively non-toxic substance readily excreted in the urine.

Therefore, although the grass may be potentially hazardous, the incidence of losses reported have been relatively rare. It is important that the antidotes, which are commonly handled by the local veterinarian, are quickly available and that he be immediately called should toxicity occur.

Grass Tetany

Grass tetany is a metabolic disorder caused by a magnesium (Mg) deficiency. It is known by various names including hypomagnesemic tetany, grass tetany, lactation tetany, grass staggers, wheat pasture poisoning, or winter tetany. As the name implies, it is most likely to occur after animals are turned out on pasture. It is more common during cool, cloudy, and rainy weather and often occurs when cool weather is followed by a warm period. Animals get grass tetany most often when grazing cool-season grasses or small grain pastures in spring and fall. Rapidly growing lush pastures are the most dangerous. It has occurred on perennial ryegrass, crested wheatgrass and all small grain pastures such as oats, wheat and rye. It is not common on legume pastures.

Grass tetany occurs most frequently on pastures grown on soils low in available magnesium and high in available potassium. Heavy applications of broiler house litter or other high-nitrogen and potassium manures may increase the hazard of grass tetany. Forages containing less than 0.2% magnesium and over 3% potassium and 4% nitrogen (25% protein) are likely to cause grass tetany if the right conditions prevail. Forages that are high in potassium and nitrogen should also contain at least 0.25% magnesium on a dry matter basis. Many cows demonstrate clinical symptoms almost indistinguishable from milk fever.

Grass tetany leads quickly to death if not treated. The symptoms, given in order in which they occur are excitement and nervousness, incoordination, muscle twitching, grinding of teeth, frequent urination, viciousness, staggering and falling, labored breathing, tetanic contraction of muscles, convulsions and death. The complete sequence of visible symptoms often occurs in only 6 to 10 hours.

Preventive programs sometimes useful include the application of magnesium fertilizer to the soil to help increase the level of magnesium in the plants. Also, high magnesium mineral mixtures or supplements may be made available to the cows a few weeks prior to placing animals on questionable pastures. Studies conducted at Tennessee showed an improvement in the problem when supplemental magnesium was fed in the form of a magnesium oxide-salt-dry molasses- cottonseed meal mixture. Dairymen using a supplemental grain feeding program are encouraged to add 5 to 10 lb. of MgO per ton when grazing such pastures.

Several commercial preparations are available for the treatment of hypomagnesemia. Treatment can be successful if given early and without excessive handling of the affected animals. USDA leaflet No.

561 reported that 200 ml of a 50% solution of magnesium sulfate injected under the skin increased the level of magnesium in the blood in 15 minutes. Also, an intravenous injection of chloral hydrate or magnesium sulfate to calm excited animals before treating with a calcium-magnesium gluconate solution is re-commended. Such intravenous injections must be administered slowly since a danger of heart failure is always possible if given too rapidly.

Heart and respiratory rates should be monitored, and if they increase rapidly treatment should be suspended or continued at a slower rate. Failure to do so may end in death due to cardiac failure. Animals on such pastures where problems are encountered should be immediately removed and provided rations containing higher levels of magnesium.

Table 1.

Table 1. Levels of nitrate in feed and expected response.	
Nitrate (KNO_3) in Feed	Comment or Animal Response
0.0-0.5	Normal if on an adequate diet.
0.6-1.0	Milk production drop, slow at first, increasing after 6th or 8th week. Typical vitamin A deficiency.
1.0-1.5	Milk production loss within 4 or 5 days. Reproduction difficulty.
1.5-up	Sudden death, abortions, severe depression, difficult respiration.
Nitrate Seminar. Dr. A. A. Case and Dr. G.B. Garner. Univ. Of Missouri, Columbia, MO.	

Table 2.

Table 2. Levels of nitrate in water and expected response.*	
Nitrate (KNO_3) in Feed	Comment
(p.p.m.)	
0-74	Not harmful. Safe.
74-220	Safe if feed is low in nitrates and nutritionally balanced. Doubtful.
220-370	Could be harmful over long periods of time. Risky.
370-740	Possible acute losses. Do not use.
740 +	Increased possibility of losses. Toxic.
*How Real is the Nitrate Problem? Dr. K.L. Dolge, Agway, Inc., Syracuse, NY (Feedstuffs, July, 1967:40)	

Table 3.

Table 3. Levels of prussic acid in feshly harvested plants.	
Level	Response
0-100 ppm	Safe to pasture
100-150 ppm	Possibly hazardous
150-200ppm	Dangerous to pasture
200 ppm	Very dangerous to pasture