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Prussic acid poisoning in livestock

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Quick Facts

- Prussic acid poisoning can be a lethal problem for cattle grazing sorghums.
- A characteristic sign of prussic acid toxicity is bright cherry-red color to the blood, a symptom that persists several hours after death.
- Treatment of prussic acid poisoning, with a mixture of sodium nitrate and sodium thiosulfate or with methylene blue, can be successful if administered by a veterinarian soon after symptoms appear.
- A qualitative test for prussic acid potential in forages is presented.

Prussic acid, also called hydrocyanic (HCN), normally is not present in plants. However, several common plants can accumulate large quantities of cyanogenetic 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 that contain the glycoside have the potential to cause HCN toxicity when consumed by ruminants.

In Colorado, 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 cyanogenetic glycoside in plant tissue. The potential for accumulation and HCN toxicity increases during drought. Occasionally, poisoning occurs when hot, dry winds induce temporary moisture stress in plants. The potential for poisoning is greater with excessive soil nitrogen and young plants. Toxicity also is 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 follows. 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. The rumen may be distended with gas, and the odor of "bitter almonds" may be detected when the body cavity is opened.

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 also can 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 thought to contain HCN 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 (23 kilograms) 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

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(46 to 61 centimeters) 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. Spraying of cyanogenetic plants with a herbicide may increase the toxic hazard.

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.

A quick qualitative test for HCN potential in plant tissue is outlined in Table 1. 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.

Table 1: Test for prussic acid potential in forage.

1. This is a qualitative test to evaluate forages (hay, pasture, silage) for prussic acid poisoning potential in ruminants.

2. Prepare picrate paper by wetting filter paper with a solution of 5.0 grams of sodium bicarbonate and 0.5 gram picric acid in 100 ml water.

3. Dry the paper and cut into strips about ¼" x 1½". Store dried strips in a stoppered bottle or sealed plastic bag.

4. Finely chop or crush a small quantity of plant material and place it in a test tube or bottle that can be sealed with a cork or rubber stopper. Slit one end of the stopper to hold a picrate paper strip.

5. If plant material is dry, moisten with a few drops of water and allow to hydrolyze several minutes in stoppered tube.

6. Moisten the picrate paper with water.

7. Add a few drops of chloroform to the wet plant material in the test tube. Stopper tightly with the picrate paper suspended in the top of the tube by the stopper. Avoid touching the paper to the material in the tube.

8. If the temperature is below 80° F (27° C), warm the solution by holding the container in hand.

9. If the paper changes from yellow to brick red within 30 minutes, prussic acid is present.

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 handfulls per sample should be sealed in a plastic bag, stored in the dark, refrigerated unfrozen, and delivered to the laboratory without delay.

Toxic Levels

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

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 after cutting for hay, grazing or frost 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. Treatment of prussic acid poisoning, with a mixture of sodium thiosulfate or with methylene blue, can be successful if administered by a veterinarian soon after symptoms appear.