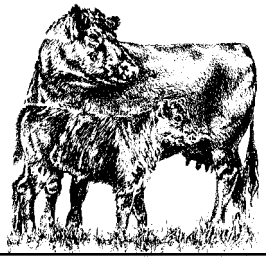

Western Beef Resource Committee *Fourth Edition*

Cattle Producer's Handbook

Animal Health Section 612

Prussic Acid Poisoning

T. L. Stanton, Extension Feedlot Specialist
Colorado State University

Prussic acid, also called hydrocyanic (HCN), normally is not present in plants. However, several common plants can accumulate large quantities of cyanogenic glycoside. When plant cells are damaged by drought, 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 the western U.S., 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, arrowgrass, 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 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 a bright cherry-red color to the blood, a sign that persists for 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

Quick Facts

Prussic acid poisoning can be a lethal problem for cattle grazing sorghum and wilted/stressed plants of the cherry family (*Prunis* sp.) among others.

A characteristic sign of prussic acid toxicity is bright cherry-red blood, a sign 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 intravenously soon after symptoms appear. However, most affected animals die quickly and seldom are presented for treatment.

There is a qualitative test for prussic acid potential in forages (see next box).

Test for Prussic Acid

The following is a qualitative test to evaluate forages (hay, pasture, silage) for prussic acid poisoning potential in ruminants:

1. 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.
2. Dry the paper and cut into strips about 1/4 inch by 1 1/2 inch. Store dried strips in a bottle with stopper or plastic bag.
3. Finely chop or crush 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.
4. If plant material is dry, moisten with a few drops of water and allow to hydrolyze several minutes in a tube with stopper.
5. Moisten the picrate paper with water.
6. If the temperature is below 80°F, warm the solution by holding the container in hand. If the paper changes from yellow to brick red within 30 minutes, prussic acid is present.

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 immediately after symptoms appear. Consult a veterinarian for diagnosis and drug treatment, because HCN toxicity often is 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.

Prevention of 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 be exposed to excessive soil nitrogen and stunted by drought or cool, cloudy weather should be suspected and tested.

Reduce risk of poisoning from sorghums by using a maximum of about 50 pounds of nitrogen per acre per application.

Young plants have a higher HCN potential than more mature ones, so do not graze sorghums until plants are 18 to 24 inches high. This practice also applies to regrowth that occurs after cutting for hay or grazing.

If regrowth occurs after frost, delay grazing until a hard freeze kills the entire plant. Do not pasture sorghums after a killing frost until plants thaw and wilt for a few days. Spraying of cyanogenic plants with an herbicide may increase the toxic hazard.

Graze pastures to a uniform height, then remove animals to prevent selective consumption of lush regrowth. Rotation grazing and heavy stocking rates help in this regard. To acclimate cattle to new pasture, 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 the 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 exists 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.

There is a quick qualitative test for HCN potential in plant tissue. It 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.

Randomly sample fresh forage from several locations. For hay, take cores from several bales. Seal two or three handfuls per sample in a plastic bag, store in the dark, refrigerate unfrozen, and deliver 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, greater than 200 ppm HCN on a dry matter basis suggests that forage may be hazardous.

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, minimize HCN potential through proper fertilization 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.

Issued in furtherance of cooperative extension work in agriculture and home economics, Acts of May 8 and June 30, 1914, by the Cooperative Extension Systems at the University of Arizona, University of California, Colorado State University, University of Hawaii, University of Idaho, Montana State University, University of Nevada/Reno, New Mexico State University, Oregon State University, Utah State University, Washington State University and University of Wyoming, and the U.S. Department of Agriculture cooperating. The Cooperative Extension System provides equal opportunity in education and employment on the basis of race, color, religion, national origin, gender, age, disability, or status as a Vietnam-era veteran, as required by state and federal laws.

Fourth edition; December 2016 Reprint