



Cattle Producer's Handbook

Animal Health Section

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Prussic Acid Poisoning

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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 (*Prunus* sp.) among others.

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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.