

Prussic Acid Poisoning

Fact Sheet No. 1.612

Livestock Series | Health

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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 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

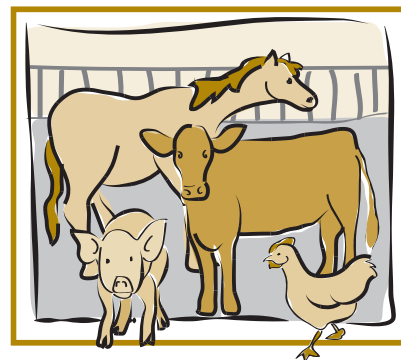
dilated and mucous membranes are bright pink. A characteristic sign of HCN toxicity is a bright cherry-red color to the blood, a symptom that persists for several hours after death. Although blood is oxygenated, HCN interferes with the release of oxygen from oxyhemoglobin to other tissues. This situation contrasts with nitrate toxicity, where oxygenation of blood is restricted. The rumen may be distended with gas, and 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. 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.

Preventing Prussic Acid Poisoning

As with nitrate, most problems with prussic acid can be avoided with proper management of forage and animals. Test any forage crop thought to contain HCN before animals are grazed or fed. Sorghums fertilized heavily with nitrogen and stunted by drought or cool, cloudy weather should be suspected. Reduce risk of poisoning from sorghums by using a maximum of about 50 pounds of nitrogen 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 following frost, delay grazing until a hard freeze kills the entire plant. Do not pasture sorghums following a killing frost until plants thaw and wilt for a few days. Spraying of cyanogenetic



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 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.
- There is a qualitative test for prussic acid potential in forages.

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Test for Prussic Acid

This 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 stoppered bottle or sealed plastic bag.
3. 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.
4. If plant material is dry, moisten with a few drops of water and allow to hydrolyze several minutes in stoppered tube.
5. Moisten the picrate paper with water.
6. If the temperature is below 80 degrees 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.

plants with a 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 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, view as dangerous any forage analyzing more than 200 ppm HCN on an as-fed basis.

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 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.