PRUSSIC ACID POISONING

INTRODUCTION

Prussic acid is also known as hydrocyanic acid or hydrogen cyanide (HCN). Prussic acid poisoning is caused by cyanide production in several types of plants under certain growing conditions. Sorghums and closely related species are the plants most commonly associated with prussic acid poisoning. Prussic acid precursors are degraded by the animal to release hydrogen cyanide (HCN), which affects the animal. Poisoning occurs when livestock consume young plants, drought stressed plants, or damaged plants that are high in prussic acid.

WHY PRUSSIC ACID IS TOXIC

Once eaten, cyanide is absorbed directly into the bloodstream and binds to enzymes in the cell. This cyanide complex prevents blood hemoglobin from transferring oxygen to individual body cells and the animal dies from asphyxiation.

Cyanide poisoning is related to the amount of forage consumed and the animal’s physiological condition, but HCN levels exceeding 200 ppm on a wet weight (as is) basis are dangerous. On a dry weight basis, forages with more than 500 ppm HCN should be considered potentially toxic.

Prussic acid acts rapidly, often killing the animal within minutes. Symptoms include excessive salivation, difficult breathing, staggering, convulsions and collapse. Death from respiratory paralysis follows shortly. The clinical signs of prussic acid poisoning are similar to nitrate toxicity, but animals with cyanide poisoning have bright red blood that clots slowly, whereas animals poisoned with nitrate have dark, chocolate-colored blood. The smell of bitter almonds is often detected in animals poisoned with cyanide.

Because it occurs quickly, the symptoms are usually observed too late for effective treatment. In the absence of a veterinarian, and if there is little doubt about the diagnosis, the animal can be treated with an injection of sodium nitrate and sodium thiosulfate. Sodium nitrate releases the cyanide from the cell, which then binds with the sodium thiosulfate to form a nontoxic complex that is excreted. Animals still alive one to two hours after the onset of visible signs usually recover.

PRUSSIC ACID CONCENTRATION FACTORS

Plant Species. Crop species most commonly involved with prussic acid poisoning are forage and grain sorghums, Johnsongrass and sudangrass. Potential cyanide production among varieties and hybrids of most summer annual forages varies widely. Grain sorghums are potentially more toxic than forage sorghums or sudangrass, whereas hybrid pearl millet and foxtail millet have very low cyanide levels. Indiangrass, flax, choke cherry, elderberry and some varieties of birdsfoot trefoil can also cause prussic acid poisoning.

Table 1. Level of prussic acid in forage (dry matter basis) and potential effect on animals.

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<thead>
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<th>ppm HCN</th>
<th>Effect on animals</th>
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<td>0–500</td>
<td>Generally safe; should not cause toxicity.</td>
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<tr>
<td>600–1,000</td>
<td>Potentially toxic; should not be the only source of feed.</td>
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<tr>
<td>1,000 and above</td>
<td>Dangerous to cattle and usually will cause death.</td>
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Plant Age and Condition. Young, rapidly growing plants are likely to contain high levels of prussic acid. Cyanide is more concentrated in the growing point and young leaves than in older leaves or stems. New sorghum growth, especially “suckers” or tillers, following drought or frost are dangerously high in cyanide. Pure stands of Indiangrass that are grazed when the plants are less than 8 inches tall can possess lethal concentrations of cyanide.

Generally, any stress condition that retards normal plant growth may increase prussic acid content. Hydrogen cyanide is released when plant leaves are physically damaged by trampling, cutting, crushing, wilting or chewing.

Drought and Frost. Drought-stunted plants accumulate cyanide and can possess toxic levels at maturity. Freezing ruptures the plant cells and releases cyanide. After a killing frost, wait at least four days before grazing to allow the released HCN gas to dissipate.

Prussic acid poisoning is most commonly associated with regrowth following a drought-ending rain or the first autumn frost. New growth from frosted or drought-stressed plants is palatable but can be dangerously high in cyanide.

Soil Fertility. Plants growing in soils that are high in nitrogen and low in phosphorus and potassium tend to have high cyanide concentrations. Split applications of nitrogen decrease the risk of prussic acid toxicity.

Animals. Most losses occur when hungry or stressed animals graze young sorghum growth. Ruminants are particularly susceptible to prussic acid poisoning because cud chewing and rumen bacteria both contribute to releasing cyanide. The plant enzyme responsible for hydrolyzing HCN from dhurrin is destroyed in stomach acid, which allows monogastric animals, such as horses and swine, to be more tolerant of cyanide than ruminants.

Feeding grain or hay before turning animals to pasture reduces rapid intake and dilutes the amount of cyanide consumed. Animals do not adapt or become immune to cyanide, but they can detoxify low HCN levels.

Harvest Technique. Prussic acid concentrations are higher in fresh forage than in silage or hay because HCN is volatile and dissipates as the forage dries or ensiles. However, if the forage had an extremely high cyanide content before cutting, or if the hay was not properly cured, hazardous concentrations of prussic acid could remain. Hay or silage that likely contained high cyanide concentrations at harvest should be analyzed before it is fed.

GUIDELINES TO AVOID PRUSSIC ACID POISONING
• Do not allow hungry cattle to graze where prussic acid may be a problem.
• Do not allow animals to graze potentially troublesome plants after a light frost or after rain has ended a summer drought.
• Hay or ensile plants high in cyanide to reduce toxin levels.
• Have representative samples of any suspect forage analyzed before feeding.

OTHER PUBLICATIONS
Nitrate and Prussic Acid Toxicity in Forage (MF-1018)