K-STATE Research and Extension
FORAGE FACTS MF3040

Prussic Acid Poisoning

Prussic acid is also known as hydrocyanic acid or hydrogen cyanide (HCN). Ingesting plants that have produced excess cyanide causes prussic acid poisoning. Sorghums, sudangrass, sorghum–sudangrass crosses, and closely related species are most commonly associated with prussic acid poisoning. Most sorghums and sudangrasses contain a prussic acid precursor (dhurrin) in their epidermal cells. Dhurrin in itself is not toxic. The mesophyll cells located below the epidermis contain an enzyme that under certain conditions converts dhurrin to prussic acid (HCN). It is the prussic acid that is toxic to livestock.

Grain sorghum generally has higher concentrations of dhurrin than forage sorghums or sudangrass. Under normal growing conditions, the dhurrin concentration is low enough that animals can detoxify it before it causes toxicity. Dhurrin concentrations are highest in young plants, new regrowth, and following rapid regrowth after a period of stunted growth, such as rapid growth of drought-stressed plants following a rain, or regrowth following a frost or freeze. Under these conditions dhurrin concentrations can be high enough to poison livestock.

Appropriate management of these forages combined with sample analysis can minimize poisoning risks and allow safe use of these forages. Delaying grazing until minimum plant heights are achieved or until injured or stressed plants have had adequate time to recover or by proper ensiling or conditioning and drying hay can reduce prussic acid concentrations.

Why Prussic Acid is Toxic

Once the prussic acid precursors are eaten, the rumen is an excellent medium for formation of cyanide, which 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.

Prussic acid poisoning is related to the amount of forage consumed, the rate of consumption, and the animal's physiological condition, but HCN levels in forages exceeding 200 parts per million 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 (Table 1).

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

ppm HCN	Effect on animals
0–500	Generally safe; should not cause toxicity.
500–1,000	Potentially toxic; should not be the only source of feed.
1,000 and above	Dangerous to cattle and usually will cause death.

Prussic acid acts rapidly, often killing animals within minutes. Symptoms of poisoning include increased pulse rate and respiration, excessive salivation and foaming at the mouth, blue coloration of the lining of the mouth, 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 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, shattercane, sudangrass, and sorghum-sudangrass crosses. Potential cyanide production varies widely among varieties and hybrids of most summer annual forages. Grain sorghum and forage sorghum tend to be more toxic than sudangrass or sorghum-sudangrass. Hybrid pearl millet and foxtail millet are generally considered to not have high prussic acid concentrations. Indiangrass, flax, chokecherry, black cherry, elderberry, and some varieties of white clover and birdsfoot trefoil also can cause prussic acid poisoning.

Plant Age and Condition. Cyanide normally is more concentrated in the growing point and young leaves than in older leaves or stems. Young, rapidly growing plants are likely to contain higher levels of prussic acid than older, mature plants. New sorghum growth, especially "suckers" or tillers, following drought or frost are dangerously high in cyanide. Pure stands of Indiangrass 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 can increase prussic acid content. Hydrogen cyanide is released when plant leaves are physically damaged by trampling, cutting, crushing, freezing, 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 into the leaf tissue. After a killing frost, wait at least five days or until the frozen leaf tissue has completely dried out 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.

Herbicides. Herbicide-induced crop injury or the application of a growth regulator herbicide (e.g. 2,4-D or dicamba) could increase cyanide concentration.

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 releasing animals to pasture slows intake and dilutes the amount of cyanide consumed. Animals do not adapt or become immune to cyanide, but they can detoxify low concentrations of HCN. Under normal growth conditions they can consume forage sorghums or sudangrass containing low levels of prussic acid with no ill effects, while elevated levels that cannot be detoxified fast enough cause poisoning.

Harvest Technique. Prussic acid concentrations are greater 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 extremely high cyanide content before cutting, or if the hay was not properly conditioned and 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.

Avoiding Prussic Acid Poisoning

- Avoid grazing or green chopping young sorghum or sudangrass plants or new regrowth.
- Do not allow hungry cattle to graze where prussic acid may be a problem.
- Feed hay or grain before releasing cattle to pasture to slow intake and reduce the poisoning risk.
- 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.

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MF3040 March 2012

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