

Prussic Acid and Livestock Poisoning

Revised by Christopher D. Allison and John Wenzel¹

aces.nmsu.edu/pubs • Cooperative Extension Service • Guide B-808

The College of
Agricultural,
Consumer and
Environmental
Sciences is an
engine for economic
and community
development in New
Mexico, improving
the lives of New
Mexicans through
academic, research,
and Extension
programs.



New Mexico State University
aces.nmsu.edu



Figure 1. Johnsongrass (*Sorghum halepense*).

CYANOGENIC GLYCOSIDES

Cyanoogenic glycosides are present in over 2,000 species worldwide, and can produce the highly poisonous hydrogen cyanide (HCN), also known as prussic acid. Certain plant enzymes are released when plants are stressed or damaged by chewing, crushing, wilting, or freezing, and these enzymes hydrolyze the glycosides into cyanide.

In New Mexico, cyanide poisoning of livestock is most commonly associated with Johnsongrass (*Sorghum halepense*), Sudangrass (*Sorghum vulgare*), and forage sorghums. Chokecherry (*Prunus virginiana*), serviceberry (*Amelanchier alnifolia*), and arrowgrass (*Triglochin maritima*) have long been recognized as potential threats, but have a much lower incidence of HCN poisoning.

Below is a partial list of plants known to have caused HCN poisoning in livestock (adapted from Knight and Walter, 2001).

- Bermudagrass, *Cynodon dactylon*
- Bracken fern, *Pteridium aquilinum*
- Johnsongrass (Figure 1), *Sorghum halepense*
- Chokecherry (Figure 2), *Prunus virginiana*
- Sudangrass, *Sorghum vulgare*
- Poison suckleya, *Suckleya suckleyana*
- White sweet clover, *Trifolium repens*
- Arrowgrass, *Triglochin maritima*

Cyanoogenic glycosides in the plant are not toxic unless acted on by rumen microorganism enzymes to form HCN. This enzymatic conversion is enhanced when the plant cells are damaged or stressed, such as by drought or freezing. In

¹Respectively, Rangeland Scientist, Lineberry Policy Center for Natural Resource Management; and Extension Veterinarian, Department of Extension Animal Sciences and Natural Resources, New Mexico State University.



Figure 2. Chokecherry (*Prunus virginiana*).

the process, the glycosides (located in the vacuoles) come in contact with the cell enzymes, forming HCN.

At least 55 cyanogenic glycosides are known to occur in plants. Some of the more common include amygdalin (laetrile), prunasin, linamarin, dhurrin, and triglochin. Ruminants are more susceptible to cyanide poisoning than monogastrics. Water drunk after livestock have consumed cyanogenic plants enhances the hydrolysis of the glycosides. Ruminants on high-energy grain rations have a more acidic rumen contents and thus a slower release of HCN than those on a grass or hay diet.

ACUTE CYANIDE POISONING

Hydrogen cyanide is extremely toxic because it rapidly inactivates cellular respiration, causing death quickly. The cherry-red venous blood associated with acute cyanide poisoning results from the failure of oxygen-enriched hemoglobin to release its oxygen to the tissues because the enzyme cytochrome oxidase is inhibited by the cyanide. When exposed to small doses of cyanide, the body can detoxify the cyanide by cellular enzymes and thiosulfates, and then the harmless thiocyanate is excreted in the urine. However, when large amounts of cyanide are absorbed, the body's detoxification mechanism is overwhelmed and cyanide poisoning occurs. The lethal dose of cyanide is in the range of 2–2.5 mg/kg body weight.

Sudden death is often the only sign of poisoning since animals die within 1–2 hours after consuming lethal amounts of a cyanogenic plant. Early signs of acute cyanide poisoning include rapid and labored breathing, frothing at the mouth, ataxia, dilated pupils, muscle tremors, and convulsions. The heart rate is usually elevated, with arrhythmias present. Mucous membranes are bright

red at first, followed by cyanosis (blue or purple coloration) when the animal's tissues are depleted of oxygen.

In addition to the bright red venous blood, hemorrhages are seen in the heart and lungs. The smell of bitter almonds, reputedly characteristic of cyanide poisoning, may be present in the rumen gases. Plants containing 200 ppm or 200 mg/kg of cyanide are toxic to all animals.

TREATMENT

The recommended treatment for cyanide poisoning is the intravenous administration of a mixture of 1 mL of 20% sodium nitrite and 3 mL of 20% sodium thiosulfate per 100 lb of body weight. The dose can be repeated in a few minutes if no response is seen. Administering 1 gallon of vinegar in 3–5 gallons of water via stomach tube will help acidify the rumen contents and reduce the production of hydrogen cyanide.

CHRONIC CYANIDE POISONING

Low levels of cyanide consumed over time cause a variety of chronic effects in livestock. The problem is caused by the loss of the myelin sheath surrounding the peripheral nerves, resulting in loss of nerve function. This demyelination of the nerves is thought to result from the conversion of the cyanogenic glycoside to T-glutamyl B-cyanoalanine, a known lathyrogen that interferes with neurotransmitter activity.

Affected animals develop posterior ataxia, urinary incontinence, and cystitis resulting from lower spinal cord degeneration. Kidney infection is a complicating factor in this cystitis.

REFERENCE

Knight, A., and R. Walter (Eds.). 2001. *A guide to plant poisoning of animals in North America*. Jackson, WY: Teton NewMedia.

Original authors: Christopher D. Allison and R.D. Baker.



Christopher D. Allison is a range scientist for the Lineberry Policy Center for Natural Resource Management. He earned his Ph.D. in range science from Texas A&M University. His professional interests and expertise include range animal nutrition, grazing management, and range plant toxicology.

Contents of publications may be freely reproduced, with an appropriate citation, for educational purposes. All other rights reserved. For permission to use publications for other purposes, contact pubs@nmsu.edu or the authors listed on the publication. New Mexico State University is an equal opportunity/affirmative action employer and educator. NMSU and the U.S. Department of Agriculture cooperating.