IANR NebGuide
University of Nebraska–Lincoln Extension, Institute of Agriculture and Natural Resources
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G2184

Cyanide Poisoning

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Cyanide, also called prussic acid, poisoning and its treatment are discussed in this NebGuide, along with methods to reduce its occurrence.

Under certain conditions, livestock consuming cyanogenic plants, which are plants capable of producing cyanide, may be poisoned by cyanide. Cyanogenic plants that pose a risk of cyanide poisoning to livestock include forage sorghum, Johnson grass, sudangrass, chokecherry, and arrowgrass.

Exposure to excessive cyanide — also called prussic acid, hydrocyanic acid, or hydrogen cyanide — can be fatal. However, producers can reduce the risk of cyanide poisoning using good management practices.

How do Plants Produce Cyanide?

Cyanide does not occur freely in cyanogenic plants. Instead, they contain cyanogenic glycosides whose molecules contain a cyano group (--CN). It is the cyano group that is the source of cyanide. An example of the cyanoglycoside called dhurrin is illustrated in *Figure 1*. Dhurrin is found in *Sorghum* species.

Cyanoglycosides are packaged in vacuoles within the plant cells. Vacuoles are cavities inside cells surrounded by a membrane.

Cyanogenic plants also contain enzymes within the cells that can remove the cyano group from the cyanoglycoside molecules. When plant tissues are damaged, such as by freezing, chopping, or chewing, the enzymes and cyanoglycosides can come in contact with each other and produce cyanide. An example of how cyanide can be produced from dhurrin is illustrated in *Figure 2*.

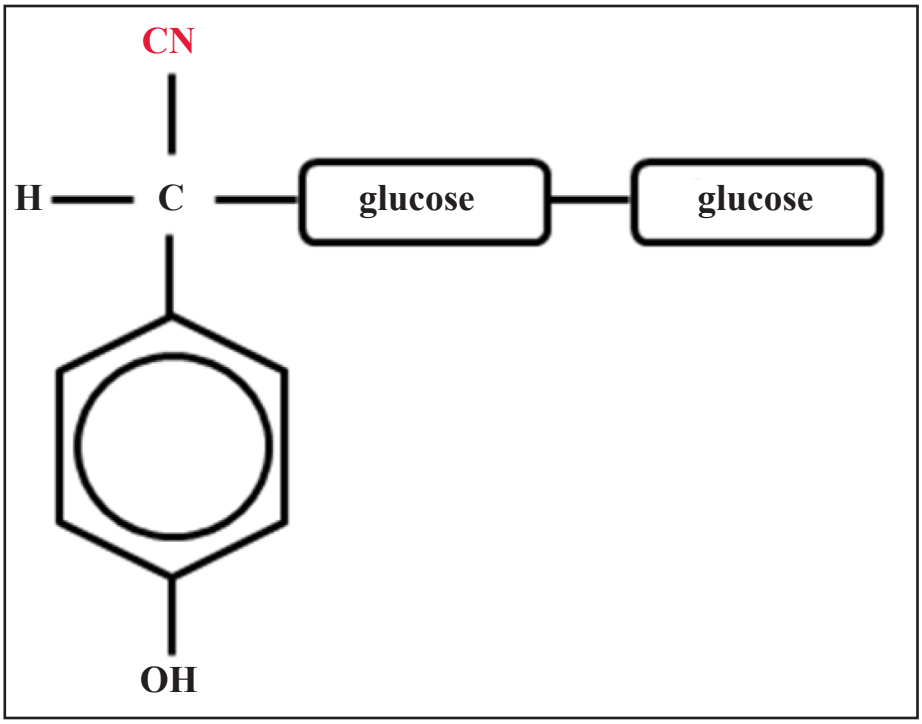

Figure 1. Example of a cyanoglycoside — dhurrin. The cyano group is in the red font. The molecule includes two glucose molecules bonded together.

How Does Cyanide Poison?

Ruminants like cattle and sheep are more likely to be poisoned by cyanogenic plants than are monogastrics like horses and swine because ruminal microorganisms can rapidly break cells apart and release cyanide. Release of cyanide in monogastrics is delayed and slower, so poisoning in such animals is rare, but not impossible. Additionally, the cyanide-releasing enzymes in the plant are more readily inactivated in the acidic gastric fluid of the monogastric's stomach. The enzymes retain their activity in the rumen.

Once cyanide is released from the cyanoglycoside, it can be absorbed quickly into the bloodstream, then readily transported throughout the body.

In animal cells, cyanide prevents production of energy for the cells (adenosine triphosphate or ATP) by inactivating an enzyme called cytochrome oxidase. Even

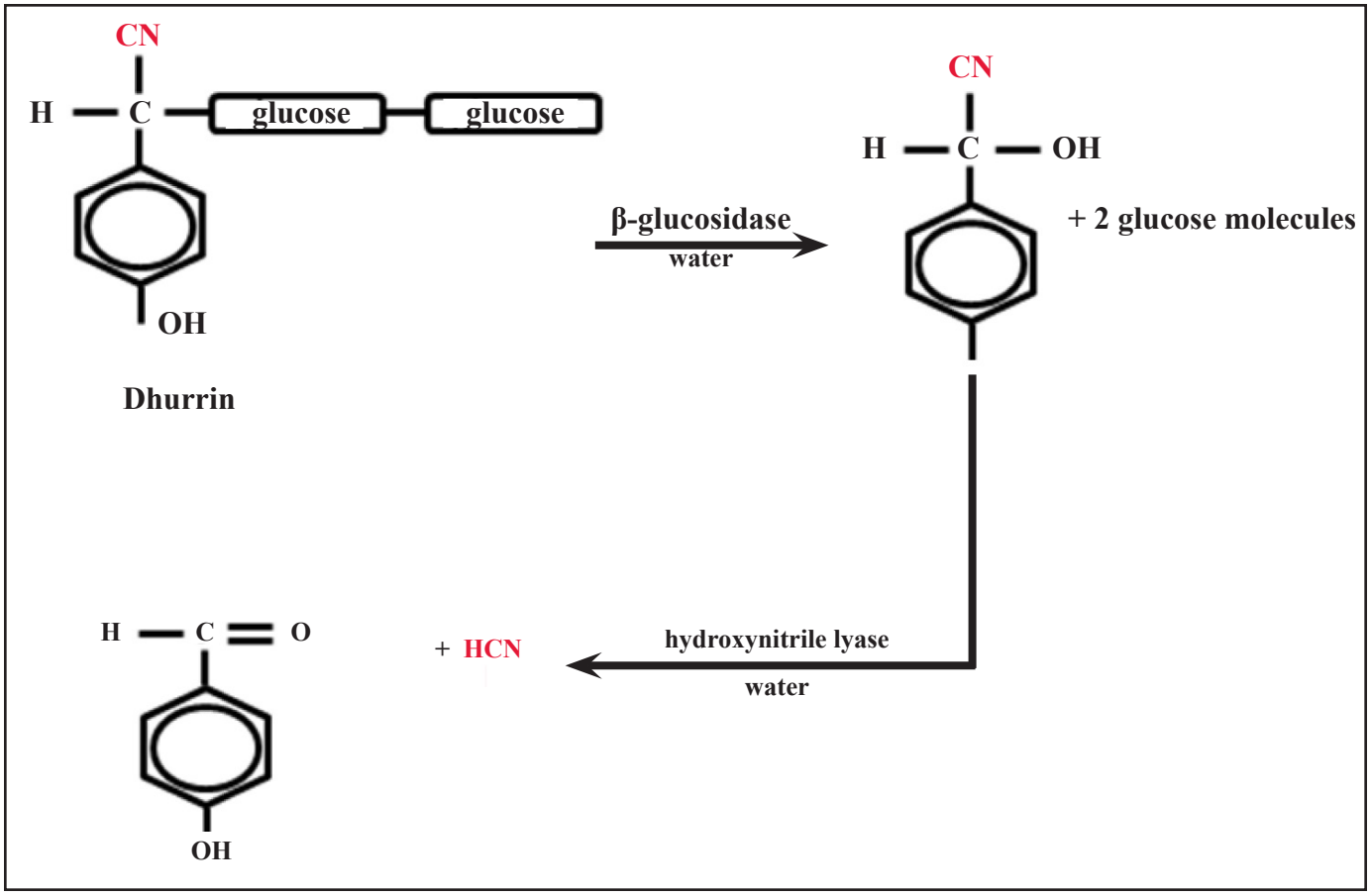

Figure 2. The enzymatic production of hydrogen cyanide (HCN) from dhurrin. It is a two step process involving water and two enzymes, β-glucosidase and hydroxynitirle lyase.

though the blood is saturated with oxygen and can make it available to the cells, the inactivation of cytochrome oxidase prevents cells from using the oxygen so cells die rapidly.

Clinical Signs of Cyanide Poisoning

Cyanide is very toxic to mammals and its effects may be noticed within 15 to 20 minutes to as long as a few hours after the victim consumes the cyanogenic plant.

One clinical sign of cyanide poisoning is a bright, cherry-red coloration of the blood due to the abundance of oxygen in the blood and the inability of the cells to use it. Mucous membranes are usually bright and pink because of the cherry-red blood contained in them.

Other clinical signs are the result of the body trying to counteract the effects of the cells' inability to use the oxygen delivered to them by the blood to produce ATP. The body interprets the lower ATP as a lack of oxygen delivery and tries to deliver more oxygen to its tissues.

So the heart rate of the victim initially is increased. Breathing becomes rapid and labored. The victim may be excited.

As its organs start to die, the victim may exhibit muscle tremors, stagger, and collapse. The victim may foam at the mouth and involuntarily urinate and defecate. As the brain dies, the victim may convulse, go into a coma, and die.

Treatment must begin rapidly because death can occur within minutes during severe convulsions.

How is Cyanide Poisoning Treated?

The liver is able to detoxify some cyanide via a reaction catalyzed by the enzyme rhodanese. Cyanide reacts with thiosulfate to produce thiocyanate. That reaction is illustrated in *Figure 3*.

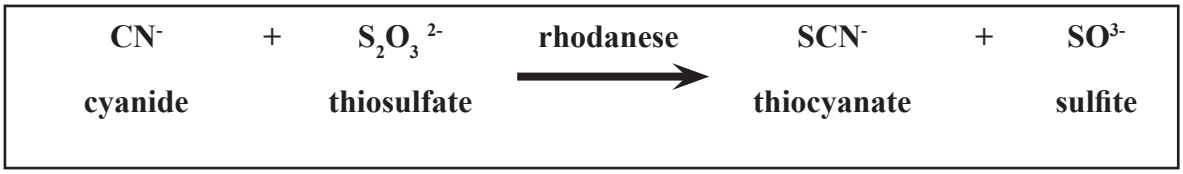

Figure 3. Rhodanese-catalyzed reaction between cyanide and thiosulfate to detoxify cyanide. This reaction occurs in the liver.

When more cyanide is produced than can be detoxified in the liver, additional treatment is necessary to help the victim survive. Treatment must be started rapidly because the victim can die very soon after clinical signs are observed. Cyanide poisoning is a medical emergency.

There are two treatment strategies, both removing cyanide from the blood. One promotes formation of methemoglobin and the other forms thiocyanide.

Treatment Promoting Formation of Methemoglobin

Rapid intravenous administration of a solution of either sodium nitrite or methylene blue (but not both) can be very effective if the victim is treated before significant brain damage has occurred. Both chemicals promote formation of methemoglobin from the hemoglobin in the blood. Methemoglobin then combines with cyanide to form cyanomethemoglobin, which removes cyanide from the blood.

Use of methylene blue is not approved for use in food production animals because it is a suspected carcinogen. Although its withdrawal time for use in the treatment of nitrate poisoning of food production animals is 180 days before market, no withdrawal time has been established for its use in cyanide poisoning.

The clinical signs of nitrate poisoning in cattle can resemble the clinical signs of cyanide poisoning. The diagnosis of cyanide poisoning must include ruling out a possible nitrate poisoning because if sodium nitrite or methylene blue treatment is done on a victim suffering from nitrate poisoning, the treatment will exacerbate and not cure the nitrate toxicosis.

Treatment Promoting Formation of Thiocyanide

Rapid intravenous administration of a solution of thiosulfate helps promote the rhodanese-catalyzed formation of thiocyanide.

Preventing Cyanide Poisoning

What promotes the presence of cyanogenic chemicals in plant tissues?

Plant Species

Leaves and stems of all *Sorghum* species can contain cyanoglycosides. Sudangrass generally has the least amount of cyanide production potential, while forage and grain sorghums, shattercane, and Johnson grass often produce dangerous concentrations. Sorghum-sudangrass hybrids are usually intermediate, but their cyanoglycoside content can vary widely. Use hybrids known to be lower in cyanogenic potential when grazing is planned.

Arrowgrass, velvetgrass, and white clover can also contain cyanoglycosides in their leaves. Chokecherry, pincherry, wild black cherry, apricot, peach, apple, and elderberry trees contain cyanoglycosides in leaves and seeds (pits). However, little or no cyanogenic potential is present in the fleshy part of the fruit. These trees can be hazardous around grazing areas because cattle often graze leaves for variety in their diet.

Plant Parts

Leaves usually produce 2 to 25 times more cyanide than do stems in forage grasses; seeds contain none. Young, upper leaves have more cyanogens than lower leaves. New shoots often contain very high concentrations of cyanogens. New shoots produced after frost can be especially hazardous.

Fertilization

High application rates of nitrogen fertilizer are often needed to obtain high yields of *Sorghum* species. However, cyanogenic potential also may increase with high nitrogen fertilization rates. This problem is most severe on soils deficient in phosphorus.

Maintain proper soil phosphorus levels according to soil test recommendations to reduce cyanide potential in forage. Use split applications of nitrogen when the total amount exceeds 60 pounds of N per acre to decrease the risk of cyanide toxicity.

Herbicides

Foliar herbicides such as 2,4-D can increase cyanogenic potential in forages for several weeks after application, so plan grazing operations accordingly.

Drought

Drought-stricken plants containing mostly leaves are slow to decrease their cyanogenic potential, so grazing stunted plants during drought is a common cause of cyanide poisoning of livestock.

Frost

Plants that are frozen may release hydrogen cyanide for several days thereafter. After wilting, hydrogen cyanide may be released and the plant's cyanogenic potential will decrease. Dead plants usually have less cyanogenic potential. Do not graze or green chop forage for several days after a killing frost.

When only plant tops have been frosted, new shoots may regrow at the base of the plants. These can be very dangerous because of high cyanogenic potential and because cattle will selectively graze the new growth. Do not graze frosted summer annuals until regrowth of shoots is 15 to 18 inches tall, or until several days after the entire plant and shoots are killed by subsequent frost.

Control Exposure

Pasture

To reduce danger from poisoning, do not graze sudangrass and sorghum-sudangrass hybrids until they are 15 to 20 inches tall; forage sorghums should be several feet tall. Heavy stocking rates and rotational grazing will reduce the

opportunity for livestock to selectively graze young, succulent, and potentially hazardous shoots.

Hungry animals may consume forage too rapidly to detoxify cyanide. Feed ground cereal grains or a full feed of hay before first turning animals onto pasture. Turn animals out to new pasture later in the day, since cyanide release potential is reported to be highest during early morning hours. Use less valuable, test animals to graze potentially toxic pastures prior to risking the entire herd.

Sulfur is used to detoxify cyanide, and most *Sorghum* species contain marginally adequate amounts in forage. Thus, free choice salt and mineral with added sulfur may help protect against cyanide toxicity. Always have plenty of clean, fresh water available.

Monitor grazing closely during periods of environmental stress, such as drought or frost. Feed good quality grass or alfalfa hay with pasture during these times to reduce potentially high cyanide concentrations in the animals' total intake.

Grain sorghum stubble is usually safe to graze, but small green shoots of sorghum or shattercane may develop in the stubble. Abundant regrowth can be dangerous. Wait for these shoots to be frozen and wilted before grazing.

Green Chop

Green chop forces livestock to eat both stems and leaves, thereby reducing problems caused by selective grazing. The cutting height can be raised to minimize the inclusion of regrowth. If nitrates are a potential problem, green chop can still be dangerous. See NebGuide G1779, *Nitrates in Livestock Feeding,* for more information.

Hay and Silage

Sorghum hay and silage usually lose 50 percent or more of the cyanide they contain during curing and ensiling processes. Free cyanide is released by enzyme activity and escapes as a gas. However, hazardous concentrations of cyanide may still remain in the final product, especially if the forage had an extremely high cyanide content before cutting. Hay has been dried at oven temperatures for up to four days with no significant loss of cyanide potential.

Although hay and silage rarely contain hazardous concentrations of cyanide, analyze these feeds in a laboratory prior to use whenever high concentrations are suspected. Dilute or mix potentially toxic feed to a safe concentration with grain or forage that is low in cyanide.

Measuring Cyanide Concentrations in Plant and Animal Tissues for Safety and Diagnosis

Chemical analysis of cyanide in forages is the only reasonably accurate method of predicting potential toxicity to animals. Hay, green chop, silage, or growing plants containing more than 200 ppm (20 mg%) cyanide as HCN on a wet weight (as is) basis are very dangerous as animal feed. Forage containing less than 100 ppm HCN, wet weight, is usually safe to pasture. Analyses performed on a dry weight basis have the following criteria: more than 75 mg% HCN is hazardous, 50 to 75 mg% HCN is doubtful, and less than 50 mg% HCN is considered safe. Ideally, forage samples should be quick frozen as soon as possible and maintained in a frozen state until submitted for analysis. Dry hay or silage also may be submitted.

Blood or liver from a victim can be analyzed for cyanide, but such analytical services are not always readily available. Additionally, the specimen must be collected as soon after death as possible and preserved because cyanide is produced as the tissue decays, so the presence of elevated cyanide concentrations in tissues alone cannot confirm that a cyanide poisoning has occurred. Contact the laboratory for specimen collection and preservation instructions.

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