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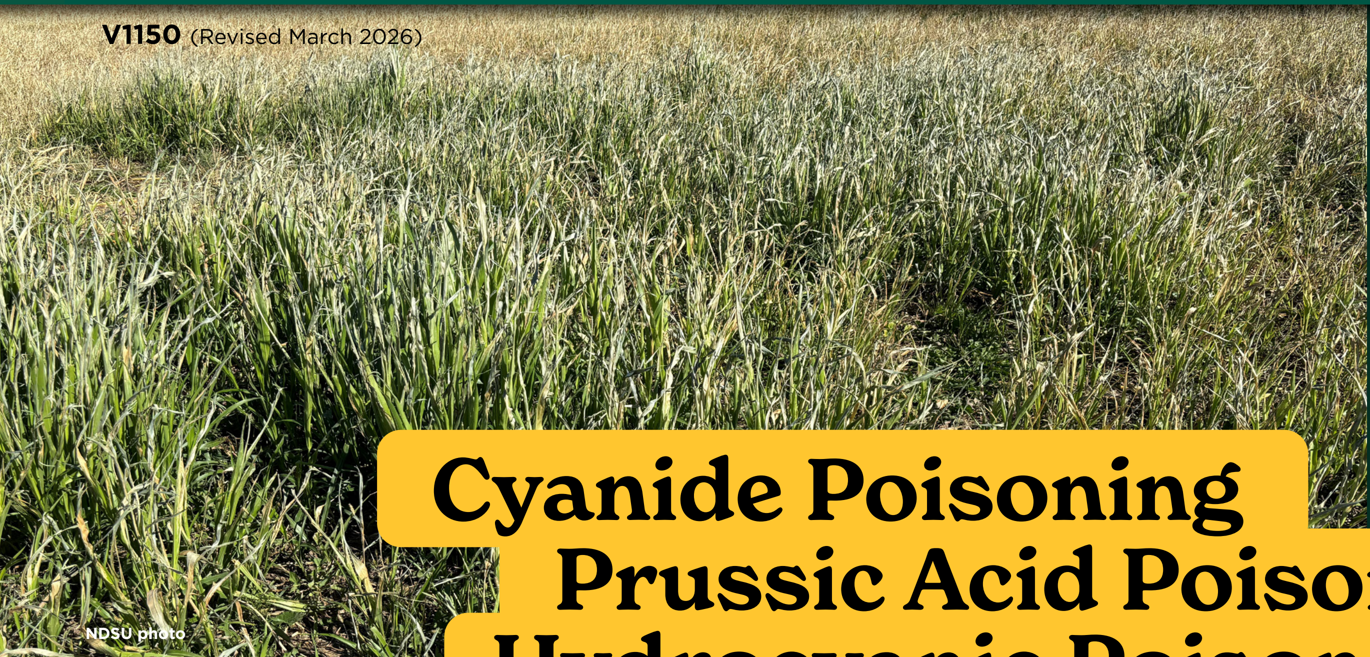

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Cyanide Poisoning
Prussic Acid Poisoning
Hydrocyanic Poisoning

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Cyanide poisoning, prussic acid poisoning and hydrocyanic acid poisoning are all terms describing the same poisoning. Poisoning occurs when livestock consume plants that contain high levels of cyanogenic glycosides, which undergo an enzymatic reaction resulting in the release of hydrogen cyanide (HCN).

Understanding Prussic Acid Poisoning

- There are over 2,500 plants that contain cyanogenic glycosides that can release hydrocyanic acid (prussic acid) under certain conditions. Cyanogenic glycosides are present in the plant to serve as a defense mechanism against pests and pathogens. In general, under normal growing conditions, healthy plants containing cyanogenic glycosides do not produce prussic acid.
- Cyanogenic glycoside levels can increase when the plant comes under environmental stress. Plant stresses that can cause an increased level include drought, frost, freezing, herbicide applications or other factors that disrupt plant growth or health.
- Cyanogenic glucoside levels vary across plant parts. Immature plants will contain higher levels than mature plants. Leaves will contain higher levels than stems. Younger leaves will be higher than older leaves. Resprouts or suckers produced after a harvest or frost can be especially high.
- Nitrogen fertility can increase the accumulation of cyanogenic glycosides. This is especially true if soil phosphorus and potassium levels are low.
- Prussic acid develops when plant cells become ruptured as a result of plant stress. When the plant cells rupture, enzymes that are normally separated from the cyanogenic glycoside molecules come together, causing a reaction in which one of the products formed is prussic acid.

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Common Plants Implicated

While any plant species that contains cyanogenic glycosides can develop prussic acid, the most common plants implicated in prussic acid poisoning are the sorghums, sudangrass and sorghum-sudangrass hybrids. The cyanogenic glycoside found in the sorghums is called dhurrin. Variety differences within the sorghums and sudangrass exist in the concentrations of dhurrin, and therefore prussic acid potential (Table 1).

- When sorghums with elevated levels of dhurrin are consumed by livestock, the chewing and grinding during consumption cause plant cells to rupture. This process mixes the dhurrin and enzymes and results in the release of prussic acid.
- In ruminants, prussic acid release can also be the result of rumen microbial activity. The prussic acid, once released, is absorbed through the rumen wall and into the bloodstream, where it can quickly result in asphyxiation at the cellular level.
- Ruminants (cattle, sheep, goats, etc.) are more susceptible to prussic acid poisoning than nonruminants (horses).
- Cyanide prevents the release of oxygen from hemoglobin to body tissues, resulting in hypoxia (low oxygen supply to tissue).

Table 1. Sorghum Types and Prussic Acid Potential

Sorghum Types	Prussic Acid Potential
Piper Sudangrass	Low
Sudangrass x Sudangrass Hybrids	Fairly Low
Sorgo (sweet sorghum)-Sudangrass Hybrids	Intermediate
Sorghum-Sudangrass Hybrids	High
Grain Types (Varieties and Hybrids)	High to Very High

Forage Analysis

Cyanide analysis estimates the potential of the plant to cause poisoning. During the forage analysis, the plant cells are broken, allowing the cyanogenic compound to mix with the plant enzymes that release the cyanide. The toxin is then measured. Table 2 provides some guidelines for evaluating forages for cyanide toxicity.

Management

- **Prussic acid-free (PF trait) in sorghum hybrids –** A recent development in the management of prussic acid in sorghums is the release of the prussic acid-free trait. The PF trait works by blocking the plant pathway that leads to the development of dhurrin. Without dhurrin, the plant cannot produce prussic acid, eliminating the risk of prussic acid poisoning.
- **Alternative forages –** Millets, corn and other annuals that do not produce prussic acid can be substituted in place of forages with prussic acid potential.
- **Frost or freezing –** Frost may only kill the top portion of the sorghum plant, allowing for young shoots at the base of the plant to survive or resprouting to occur. Young shoots or resprouts are preferentially grazed and can contain high concentrations of prussic acid. Delay grazing for 1-2 weeks after frost to allow prussic acid to dissipate and reduce the chance of poisoning. An alternative would be to wait until a killing frost, then allow the plant to completely dry for 1-2 weeks before grazing.
- **Most prussic acid poisoning in livestock occurs when sorghums are grazed less than 18 inches tall.** Sorghums less than 18 inches tall can have high levels of dhurrin; these levels decrease as the plant grows and matures. Allow sorghums to reach 18-24 inches in height prior

Table 2. Cyanide (HCN) Content of Forages

As-fed Basis (Moisture Content Unknown)	Cyanide Content (Dry-matter Basis)	Comments
< 200 ppm	0-500 ppm	This feed should not cause cyanide poisoning.
200-600 ppm	500 to 1,000 ppm	This feed is potentially toxic; it should be fed at a restricted rate.
> 600 ppm	> 1,000 ppm	This feed is potentially very toxic. Drying, ensiling or allowing it to mature should reduce the cyanide content. Retest before feeding.

to grazing in order to reduce concentrations of cyanogenic glycosides and prussic acid potential. Allow sorghums to regrow to 18-24 inches following grazing, silage or hay harvest prior to grazing.

- **Feeding grain or hay before turning animals into pasture may reduce the rapid intake of forage and the amount of cyanide consumed.** Animals do not develop immunity to cyanide, but they can detoxify low levels of cyanide.

Sorghum silages or hay are generally safe. Plants are normally beyond the minimum grazing height when harvested for hay or silage. Allow sorghums to recover from a period of stress prior to haying. If the hay was not properly cured, cyanide can remain.

Clinical Signs

Signs of cyanide poisoning can occur within 15-20 minutes to a few hours after animals consume the toxic forage. Animals are often found dead. Clinical signs, when noticed, occur in rapid succession. Excitement, rapid pulse and generalized muscle tremors occur initially, followed by rapid and labored breathing, staggering and collapse. Signs may also include salivation (drooling), lacrimation (runny eyes) and voiding of urine and feces. Animals rarely survive more than two hours after the onset of clinical signs. The mucous membranes are usually bright pink, and the blood will be a bright cherry red, but both of these characteristics will fade quickly after death.

Diagnosis

When livestock losses occur and cyanide poisoning is suspected, proceed with the following:

- Contact your veterinarian.
- Send the suspect forage to the diagnostic laboratory for analysis. Do not send grains, stomach contents or blood samples.
- Change forages or remove animals from suspected pastures until results of the analysis are returned.
- Veterinarians should use caution on necropsy, as opening the rumen can release cyanide gas. A respirator and other personal protective equipment (PPE) should be utilized in such cases.
- As with any case of animals showing signs of salivation and/or neurologic impairment (staggering, incoordination or collapse), rabies should be on the list of potential diagnoses. Avoid contact with saliva and consider submitting appropriate brain tissue for testing.

Treatment

Treatment for cyanide poisoning is often not possible due to the rapid progression of clinical signs leading to death. Activated charcoal is not effective in preventing the absorption of cyanide and is not recommended. Since affected animals are likely hypoxic, any stress from handling or treatment could be fatal. Immediate treatment goals are to re-establish oxygen transport from red blood cells to body tissues. This can be accomplished by veterinarian administration of certain compounds. Intravenous sodium nitrite converts hemoglobin to methemoglobin, which binds to cyanide and allows for detoxification to thiocyanate. This detoxification requires sulfur, so concurrent treatment with sodium thiosulfate is recommended.

However, care must be taken to avoid confusing cyanide poisoning with nitrate toxicity, as these can present with similar clinical signs. Blood from animals with nitrate toxicity will be chocolate brown as opposed to bright cherry red for cyanide poisoning. If a case of nitrate toxicity were misdiagnosed, and treatment with sodium nitrite were initiated, this would rapidly worsen the animal's condition.

The medications necessary to attempt treatment for cyanide poisoning may not be regularly stocked by veterinary clinics. If conditions are favorable for cyanide poisoning, this should be discussed with a veterinarian so that all parties can be adequately equipped to respond should the need arise.

500-9-12; drought-7-17; web-5-20; web-3-26