Prussic Acid Poisoning in Livestock

WASHINGTON STATE UNIVERSITY EXTENSION FACT SHEET • FS129E

Introduction

Prussic acid poisoning (also known as cyanide poisoning) is a metabolic condition in livestock that producers may not see very often, but entire herds can be affected by it, and the resulting death rate can be economically devastating.

To protect livestock from prussic acid poisoning, livestock producers need to understand what causes this toxin, what its sources are, and how to recognize the symptoms of this type of poisoning. Producers should also know how to obtain an accurate diagnosis and apply effective treatment. Finally, producers should understand the risk factors and employ effective methods to prevent prussic acid poisoning.

Causes and Sources

Prussic acid poisoning, sometimes referred to as hydrocyanic acid poisoning or cyanide poisoning, occurs when livestock consume plant parts from specific forage plants, trees, and weed species that contain cyanogenic glycosides (Table 1). Cyanogenic glycosides are plant-specific and at least 55 cyanogenic glycosides have been identified so far (Knight and Walter 2001).

Table 1. Examples of forages and plants that can accumulate prussic acid.

<table>
<thead>
<tr>
<th>Plant type</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grains, forage crops, and grasses</td>
<td>Corn, flax, sorghum, sudangrass, arrow grass, velvet grass, white clover, Indian grass, birdsfoot trefoil, Johnson grass</td>
</tr>
<tr>
<td>Trees</td>
<td>Apricot, peach, cherry, chokecherry, elderberry, apple, wild black cherry</td>
</tr>
<tr>
<td>Landscape and house plants</td>
<td>Eucalyptus, hydrangea</td>
</tr>
</tbody>
</table>

Adapted from *The Merck Veterinary Manual*.

When cyanogenic plants are ingested and chewed, their cell walls and membranes are ruptured, releasing $\beta$-glycosidase enzymes that mix with the cyanogenic glycosides. Freezing weather and mechanically chopping forage can also produce this mixing. The enzymatic reaction results in the removal of the sugar component, which in turn releases free hydrogen cyanide (HCN)—that is, prussic acid or hydrocyanic acid, which is extremely toxic to livestock.

HCN is absorbed into the bloodstream very quickly and inhibits an animal’s ability to deliver oxygen to tissue for cellular respiration. Some HCN is naturally detoxified by the body, converting it to thiocyanate, which is then excreted in the urine (Knight and Walter 2001). However, toxicity will occur when the rate of HCN production exceeds the rate of detoxification. According to *The Merck Veterinary Manual*, the minimum lethal blood concentration level for HCN is approximately 3.0 µg/mL or less.

Ruminants, such as cattle and sheep, are especially susceptible because release of HCN results from ruminal microbial fermentation (Vough and Cassel 2006). While HCN is also toxic to non-ruminants, such as horses and swine, hydrochloric acid in the non-ruminant’s stomach reacts with HCN resulting in some production of formic acid and ammonium chloride, both of which are less toxic than HCN.

Symptoms

Livestock can show symptoms within minutes of ingesting feed containing prussic acid. Excitement, increased respiration rate, labored breathing, salivation, staggering, and collapse are all signs of prussic acid poisoning. Ultimately, death results from asphyxiation, which usually occurs within 30 to 45 minutes following onset of symptoms. The relationship between the level of prussic acid in feed and the associated risk of poisoning is presented in Table 2.

Diagnosis

For treatment to be successful, livestock owners should obtain a swift and accurate diagnosis in consultation with a veterinarian. It is generally recommended that cyanide analysis be performed on the questionable feeds or the animal’s rumen or stomach contents.

An accurate diagnosis is essential because many of the clinical signs of prussic acid poisoning are similar to those of
Table 2. Livestock tolerance to levels of prussic acid in feed (dry matter basis).

<table>
<thead>
<tr>
<th>Prussic acid (HCN) ppm</th>
<th>Effect on livestock</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 500</td>
<td>Generally considered safe</td>
</tr>
<tr>
<td>From 500 to 750</td>
<td>Potential for toxicity exists, thus should not be the sole source of feed</td>
</tr>
<tr>
<td>Greater than 750</td>
<td>Dangerous and will cause death</td>
</tr>
</tbody>
</table>

Adapted from *The Merck Veterinary Manual*.

nitrate poisoning. There are some distinguishing characteristics to help differentiate between prussic acid and nitrate poisoning. Nitrate poisoning causes the animal’s tongue and eyes to turn blue and its blood to turn dark chocolate brown. In contrast, prussic acid causes the animal’s blood to turn a bright cherry red (Krantz 2011). Providing sodium nitrite to animals suffering from nitrate poisoning rather than prussic acid poisoning could make these animals worse.

To perform an accurate chemical analysis that leads to a correct diagnosis, quality test samples are important. Prussic acid levels increase in plants during the morning hours and are highest in leaves (Whittier 2011). Consequently, to obtain a good sample, it is best to sample leaves and regrowth at midday. Collect one or two pounds of forage that are representative of the plant material consumed. The sample must remain fresh. If it is allowed to dry, loss of prussic acid will result. Use a container that can be tightly sealed, such as a plastic bag, freeze the sample, and ship it to the testing lab in a cooler with an ice pack via overnight express (Sulc 2012). Contacting the testing lab to obtain complete shipping instructions will ensure a quality sample reaches the lab in a timely manner. To have feed tested for prussic acid content, contact the Washington Animal Disease Diagnostic Laboratory (WADDL) located in Bus tad Hall, Room 155N, Pullman, WA 99164-7034 or email waddl@vetmed.wsu.edu.

**Treatment**

Treatment can be effective if initiated at the onset of symptoms. However, severely affected animals usually die within 30 to 45 minutes following symptom onset. In cases where poisoning is less severe, your veterinarian may choose to employ intravenous therapy that includes sodium nitrite and sodium thiosulfate. Many times treatment comes too late and the animal cannot be saved. When this is the case, the focus should be on protecting the rest of the herd by keeping them away from affected feed and carefully monitoring them for any symptoms of poisoning.

**Risk Factors**

Ruminants are at the greatest risk of poisoning because they have the ability to consume large quantities of forage and other fibrous materials. However, other species, such as pigs, horses, and house pets, can be at risk as well. It is important to note that the following conditions can increase the risk of prussic acid poisoning.

- The potential for prussic acid poisoning is greatly increased after a frost. Thus, it is critical to delay grazing until reductions in prussic acid are confirmed through forage testing (after regrowth has been frozen and wilted).
- A variety of grasses and trees (Table 1) common to the Pacific Northwest can place animals at risk.
- Excessive nitrogen fertilization can increase the hazard. Consider split applications of nitrogen fertilizer, and apply no more than 60–80 lb of N per acre at one time.
- Using herbicides to control weeds in the pasture can increase the risk of prussic acid accumulation after application.
- Plants under stress from drought or other conditions that inhibit regrowth can concentrate prussic acid in leaves that have been unable to mature.
- Grazing the regrowth of plant species susceptible to accumulation of prussic acid after the end of a drought can be hazardous. The new growth usually contains more prussic acid than old growth.

**Prevention**

Due to the severity of prussic acid poisoning and the potential for economic loss, prevention is certainly the best approach (Knight and Walter 2001; Collins and Hannaway 2003; *The Merck Veterinary Manual*). The following list provides some preventive measures.

- Sorghum and sudangrasses should not be grazed when they are in an immature state. Allow these forages to attain a height of 15 to 18 inches before grazing.
- New varieties of sudangrass and sorghum x sudangrass with lower prussic acid content should be considered when selecting seed.
- Make sure that animals have been provided sufficient feed, such as hay, so they are not hungry when they enter fresh pastures. This will reduce the amount of prussic acid consumed and allow more time for the animal to detoxify low levels of HCN.
- Do not provide animals with yard waste that may include plant material containing prussic acid. In addition, take care to ensure that there is no access to affected fruit and shade tree leaves when animals are allowed to inhabit areas adjacent to orchards and landscape plants. The risk increases when only limited feed is available, resulting in animals being more attracted to nearby leaves.
- Depending on the initial level of prussic acid, processing, such as chopping, haying, or ensiling, allows the prussic acid to volatilize, thus reducing it to acceptable levels in the feed. However, only laboratory testing can confirm these levels.
Further Reading

Illustrations to help identify potentially toxic plants can be found at http://www.animalag.wsu.edu/forages/Kerr-PoisonousPlants1007-2005.pdf.

For more information on a variety of issues associated with pasture management and toxicities for grazing livestock, see http://www.animalag.wsu.edu/forages/HendrixFS1004_2003.pdf.

References


