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FEBRUARY 2007 PRIMEFACT 417 (REPLACES AGFACT A0.9.66)

Prussic acid poisoning in livestock

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What is prussic acid poisoning?

Prussic acid is not normally present in plants but under certain conditions, several common plants can accumulate large quantities of cyanogenic glycosides which can convert to prussic acid. The risk of prussic acid poisoning in livestock is increased during periods of drought, and even more so after drought breaks, when stressed, stunted plants begin to grow.

Prussic acid is a potent, rapidly acting poison, which enters the bloodstream of affected animals and is transported through the body. It then inhibits oxygen utilisation by the cells so that, in effect, the animal dies from asphyxia.

Prussic acid is also known as hydrocyanic acid (HCN).

Sources of poison

Approximately 200 plants are known to accumulate sufficient quantities of cyanogenic glycosides to cause poisoning. The plant species that commonly cause prussic acid poisoning in livestock in Australia are:

- *Sorghum halepense* (Johnson grass)
- Sudan grass
- Sorghum spp.
- Sorghum–Sudan grass hybrids
- *Cynodon* (blue couch)
- *Brachyachne* (native couches)
- *Eremophila maculata* (native fuschia)
- *Acacia glaucescens* (acacia)
- Linseed meal and cake (especially immature seeds)
- *Heterodendrum oleifolium* (rosewood).

Plant factors

Certain conditions lead to dangerous levels of cyanogenic glycosides in plants. These conditions include:

- periods of rapid regrowth following stunting, for example after a drought breaks, if a crop is eaten back and then allowed to regrow, or if a crop is harvested for hay then allowed to regrow – levels are highest in young plants with green, growing shoots;
- frosted or wilted plants which have a transient increase in glycoside levels;
- herbicide-treated plants which have a transient increase in glycoside levels;
- high nitrogen and low phosphorus levels in the soil;
- plant species such as sorghum, which can contain more prussic acid than Sudan grass – varieties vary in their prussic acid potential;
- plants that are wet with dew or light rain.

Animal factors

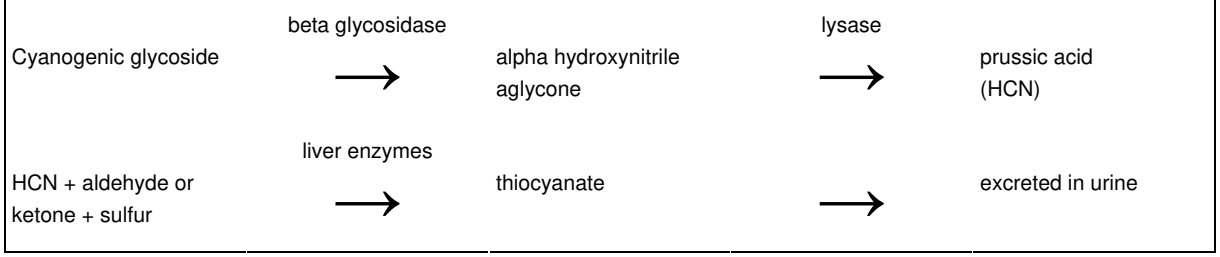
Ruminant animals (cattle and sheep) are more susceptible to prussic acid poisoning than monogastric animals (horses and pigs). The lower pH in the stomach of the monogastric helps to destroy the enzymes that convert cyanogenic glycosides to prussic acid.

For prussic acid poisoning to occur, high levels of cyanogenic glycosides and enzymes necessary to metabolise them need to be present (see Figure 1).

The action of rumen microbes will also metabolise cyanogenic glycosides. Therefore, poisoning is more likely in ruminant animals. Sheep are more resistant to poisoning than cattle due to the different enzyme systems in their fore-stomachs.

Hungry animals are also at greater risk as they will normally consume a larger amount of toxic material in a short time. This ‘overload’ of prussic acid can overwhelm an animal’s ability to metabolise prussic acid to the non-toxic thiocyanate. Large amounts of

Figure 1. Process of prussic acid poisoning



prussic acid can, therefore, be absorbed and lead to poisoning.

Travelling stock and recently introduced stock are at greater risk as they are unaccustomed to local plants. There is also evidence that animals become accustomed to the poison and with experience can tolerate increasing amounts.

Signs of poisoning

Signs of poisoning usually occur 15–20 minutes after the toxin is consumed. Death occurs very quickly, approximately 2–3 minutes after the onset of clinical signs in peracute cases, and within 1–2 hours in acute cases. Usually, animals are found dead with no signs observed. The brain and heart are the first to be affected by lack of oxygen, and so the resulting clinical signs prior to death include:

- breathing difficulties
- rapid, weak, irregular pulse
- anxiety and restlessness followed by depression
- stumbling/staggering
- muscle tremors
- moaning
- dilated pupils
- recumbency
- bloat, and sometimes salivation and vomiting
- terminal convulsions
- bright red mucous membranes.

Diagnosis

A diagnosis of prussic acid poisoning is made on clinical and/or post-mortem findings and by the recovery of the poison from the plants/feed and from the animal.

On post-mortem examination, the blood may also be bright red and clot poorly (the blood will return to a dark colour a few hours after death). Muscles may be dark and there may be haemorrhaging in the trachea and lungs. Haemorrhages will be evident on the surface of the heart. There may also be a smell of bitter almonds in the rumen. Samples for laboratory analysis include rumen contents (which should be frozen as soon as possible), muscle, liver and a sample of feed.

Prussic acid poisoning can be confused with:

- nitrite poisoning
- acute pulmonary oedema and emphysema
- blue-green algae poisoning
- anaphylactic reactions.

Treatment

Urgent veterinary attention is necessary. It is essential to obtain the correct diagnosis, as confusion with nitrite poisoning may be disastrous. (See Primefact 415 *Nitrate and nitrite poisoning in livestock)*.

Treatment aims to re-establish oxygen transport at the cellular level. Sodium nitrite is injected intravenously to convert haemoglobin to methaemoglobin, which reacts with cyanide (prussic acid) to form cyanmethaemoglobin. A simultaneous injection of sodium thiosulfate provides sulfur to convert cyanmethaemoglobin to the non-toxic thiocyanate, which is excreted in the urine. (See note below.)

An alternative treatment is to inject a large dose of sodium thiosulfate alone. This is the preferred treatment if there is suspicion that prussic acid poisoning is combined with nitrate poisoning. Treatment will need to be repeated. (See note below.)

Animals should be removed from the source of poison immediately and fed safe feed to help dilute the amount of poison in the rumen/stomach. Animals exposed to the poison source should be treated even if they are not showing any clinical signs. Animals should be handled as quietly as possible.

Note: Sodium nitrite and sodium thiosulfate are not approved for use in food-producing animals. This matter is under review. Contact your veterinarian for advice.

Prevention

- Do not allow stock to graze risky food sources. Have feed analysed if safety is in doubt.

- Do not allow stock to graze drought-stressed, immature, wilted or frost-damaged plants that are known cyanogenic glycoside accumulators (see ‘Sources of poison’).
- Never allow stock to graze sorghum that is less than 50 cm high.
- Feed hungry stock with hay before allowing them to graze forages which may contain high levels of cyanogenic glycosides / prussic acid.
- If buying sorghum hay, make sure that it was cut during low-risk conditions. There is no decrease in prussic acid content in the process of haymaking.
- Feeding material as silage will reduce the risk of poisoning, as correct ensilage for 3 weeks reduces levels of toxin by approximately 50%. On feeding out, some toxin will be released as gas. It is still recommended that this feed be tested prior to use.
- Green chop forage may be safer than the same plant material in a pasture because selective grazing of high-risk leaf material is prevented.
- Linseed gruel should be thoroughly boiled to remove any free prussic acid.
- Supplementation of sulfur (if a deficiency exists) will increase the animal’s efficiency at converting prussic acid to the non-toxic thiocyanate.

Testing feed samples

NSW Department of Primary Industries Regional Veterinary Laboratories (RVLs) can test feed samples for prussic acid. Samples may be sent via your veterinarian to any NSW DPI RVL for testing.

Further information

For further information, contact NSW DPI or your veterinary advisor.

ISSN 1832-6668

Replaces Agfact A0.9.66

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Job number 7296