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Popular Article

Cyanide Toxicity in Animals: Understanding Causes, Effects and Mitigation Strategies

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Introduction

Cyanide is one of the most potent and rapidly acting inorganic poison and it can cause toxicity in animals, principally in ruminants. “Prussic acid poisoning” is older terminology for cyanide poisoning. Hydrogen cyanide was first isolated from a blue dye (Prussian blue) and because of its acidic nature, it became known by the common name “prussic acid”.

Cyanogenic glycosides present in living plant cells and it can be converted to cyanide by enzymes present in the plant when plant cells are crushed, chewed, wilted and ruptured. Ruminants are very susceptible to cyanide poisoning because the rumen environment is mildly acidic, usually has ample water content, and the microflora can rapidly convert cyanogenic glyocosides in plants to free cyanide gas.

Cause

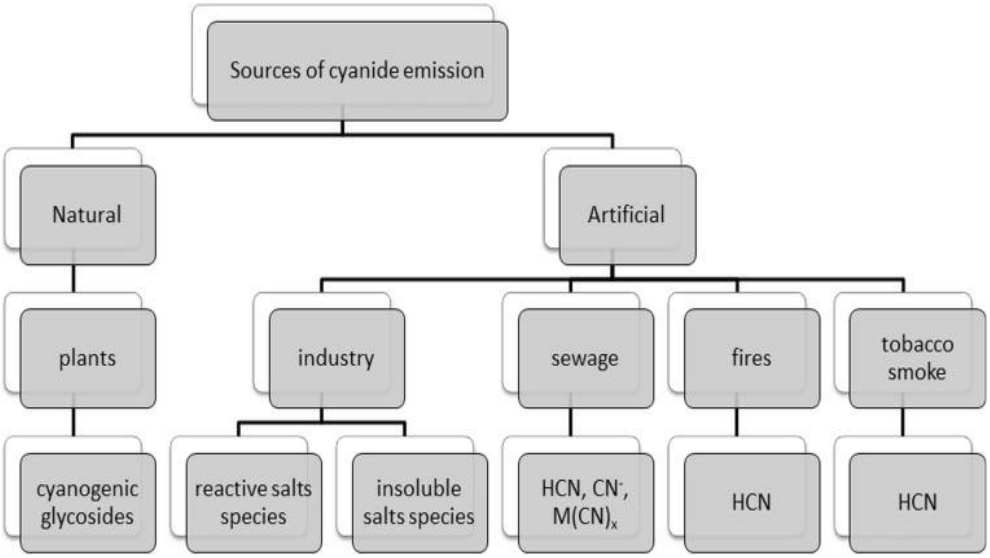
Animals are mostly exposed to cyanide through ingestion of plants containing cyanogenic glycosides. The primary cause of cyanide poisoning in ruminants is the ingestion of plants containing cyanide-producing compounds called cyanogenic glycosides. At least 55 cyanogenic glycosides are known to occur in plants, many being synthesized from amino acids as part of normal plant metabolism.

Source of poisoning and factors

Cyanide poisoning of livestock is commonly associated with johnsongrass, sorghum-

sudangrass, and other forage sorghums. Choke-cherry or wild cherry, elderberry, and arrow grass are less frequent causes.

Young plants, new shoots, and regrowth of plants after cutting often contain the highest levels of cyanogenic glycosides. Some of the sudangrasses, such as Piper, are low in cyanide. Drying plants decreases the cyanogenic potential over time. The cyanogenic potential of plants is affected by species and variety, weather, soil fertility and stage of plant growth. The content of cyanogenetic glycosides in these plants varies between seasons and different parts of the plant, with young, growing leaf usually having the greatest concentration.



Lethal dose

It is shown that, the lethal dosage of HCN in most animal species is in range of 2 mg/kg to 2.5 mg/kg (Schneider, 2012). Within the group of ruminants, goats appear to be the most susceptible to cyanide (Patel et al., 2014).

Table 2: Level of HCN in forages (dry matter basis) and potential effect on animals.

Sr. No	HCN/kg feed(ppm)	Effects of HCN on animals
1	0-500	Generally safe
2	600-1000	Potentially toxic
3	>1000	Dangerous to cattle and usually cause death

Source :(Kraig *et al*., 2012).

Mechanism of Toxicity

As ruminants consume plant materials containing cyanogenic glycosides, hydrogen cyanide is liberated in the rumen, rapidly absorbed into the bloodstream and prevents

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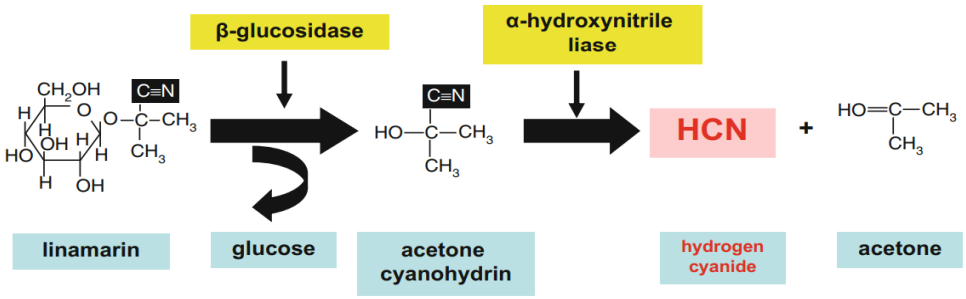
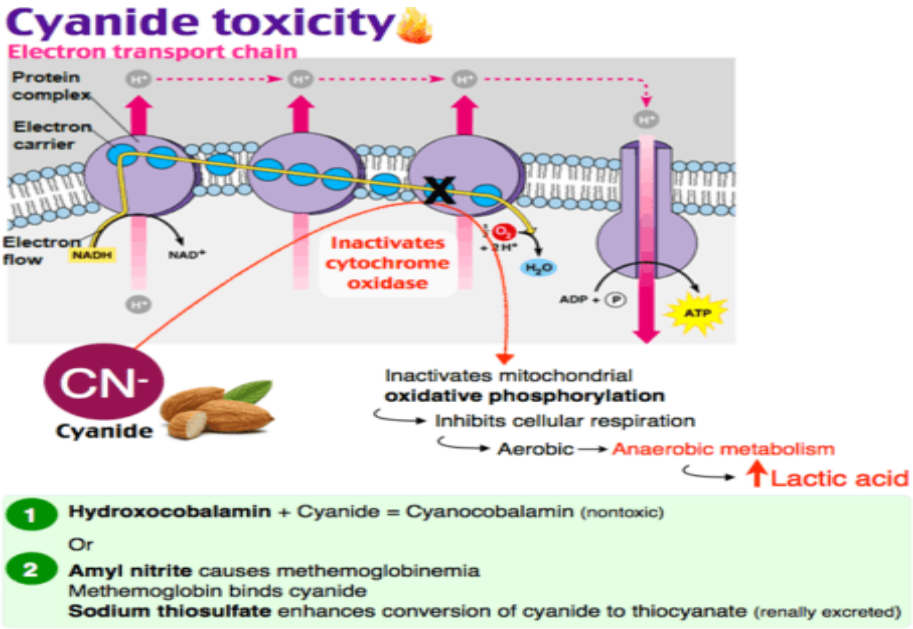
hemoglobin from releasing its oxygen to the tissues.



The mechanism of cyanide intoxication has been attributed to the inhibition of cytochrome oxidase by reversibly binding with ferric ions within the mitochondria, thereby effectively halts cellular respiration by decreasing the tissue utilization of oxygen.



If large quantities of cyanide are absorbed rapidly enough, the body's detoxification mechanisms get disturbed and the animal soon dies. Affected animals rarely survive more than 1-2 hours after consuming lethal quantities of cyanogenic plants and usually die within 5-15 minutes of developing clinical signs of poisoning.

Clinical Findings of Cyanide Poisoning in Animals

Acute cyanide poisoning

- Dyspnea followed by tachycardia.
- The classic "bitter almond" breath smell may be present.
- Salivation, excess lacrimation, and voiding of urine and faeces may occur.
- Muscle fasciculation is common and progresses to generalized spasms and coma before death. Animals may stagger and struggle before collapse.
- Mucous membranes are bright red but may become cyanotic terminally.

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- Venous blood is classically described as "cherry red".

Chronic cyanide poisoning

- Chronic cyanogenic glycoside hypothyroidism will present as hypothyroidism with or without goiter.
- Cystitis ataxia toxidromes are typically associated with posterior ataxia or incoordination that may progress to irreversible flaccid paralysis, cystitis secondary to urinary incontinence, and hindlimb urine scalding and alopecia.
- Signs may include rapid labored breathing, irregular pulse, frothing at the mouth, dilated pupils, muscle tremors, and staggering.

Diagnosis of Cyanide Poisoning in Animals

Appropriate history, clinical signs, postmortem findings, and demonstration of HCN in rumen (stomach) contents or other diagnostic specimens support a diagnosis of cyanide poisoning. Veterinarians should use appropriate personal protective equipment. A rapid qualitative and presumptive diagnosis can be made by testing representative plant samples or stomach contents using the picric acid paper test. Suitable specimens for testing include the suspected food source, rumen/stomach contents, samples of the rumen gas cap, heparinized whole blood, liver, and muscle. Specimens should be sealed in an airtight container, refrigerated or frozen, and submitted to the laboratory without delay.

Necropsy Findings

Venous blood is classically described as being "bright cherry red"; The rumen may be distended with gas; in some cases, the odour of “bitter almonds” may be detected after opening. Subendocardial and subepicardial petecchial and ecchymotic hemorrhages typical of an agonal death may be present. Cyanotic mucosa, dark muscles, lung edema and hemorrhages may be seen.

Differential diagnoses include poisonings by

Nitrate or nitrite, urea, organophosphates, carbamates, chlorinated hydrocarbon pesticides, toxic gases (carbon monoxide and hydrogen sulfide) as well as infectious or noninfectious diseases that cause sudden death

Treatment, Control, and Prevention of Cyanide Poisoning in Animals

Immediate treatment with hydroxocobalamin and oxygen and removal from the source of exposure. In cases of acute cyanide poisoning, immediate treatment is necessary. Hydroxocobalamin (vitamin B12a) is the gold-standard antidote for cyanide because of its effectiveness and low toxicity. When available, oxygen should be used to supplement antidotal

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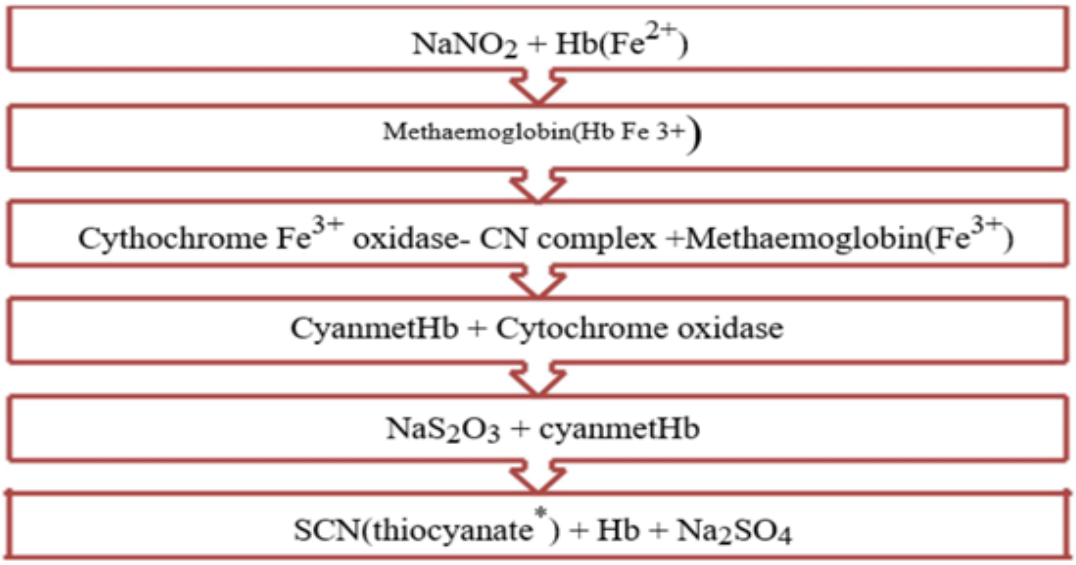
therapy. Classically, various nitrites have been used for this purpose; eg, inhaled amyl nitrite followed by IV injection of a nitrite salt (typically sodium nitrite) has been used to rapidly induce methemoglobinemia. Treatment with nitrites is usually followed up by injection of sodium thiosulfate.



Biochemical mechanisim of reversal of the cyanide poisoning.

The best preventive step is to test suspect feed and/or pastures before allowing consumption.

Mitigation Strategies

- Manage grazing and feed conditions for environmental stress to minimize risk, and analyze feed before allowing consumption.
- Hydroxocobalamin plus 100% oxygen should be administered as soon as possible after suspected cyanide poisoning.
- Treatment should not be delayed for diagnostic confirmation.
- Removal from the source of exposure is the main clinical priority in chronic cyanide-associated toxidromes.

Conclusion and recommendations

Cyanide poisoning is one of the most important poisonings that can affect livestock. It is highly lethal and rapidly acting toxin to which ruminants are more susceptible. The risks of poisoning can be affected by level of cyanide in plant material that varies with environmental stressors to plants as well as stage of plant growth. Immediate diagnosis and treatments with sodium thiosulphate and sodium nitrite can be done in together with supportive therapy.

The recommendations are: The rapid administration of sodium thiosulphate alone or in combination with sodium nitrite intravenously (IV) to early noticed should be done to reduce

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losses of livestock. Lowering of rumen pH by feed supplement and management.

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