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Ruminant Nitrate and Cyanide Toxicosis
Lantana toxicosis

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CSU Veterinary Diagnostic Laboratory

27847 CR 21

Rocky Ford, Colorado

Plants that cause Acute Death due to Asphyxiation

Nitrate → Nitrite-Hbg → Met Hbg

blood can't carry oxygen-muddy brown color

- Species affected – Cattle/buffalo > sheep >>> horses
- Toxic Principle – nitrite (rumen bacteria convert nitrate to nitrite)
- Environmentally stressed plants - water (additive)
- Clinical signs
  - Dyspnea - shortness of breath
  - Muddy, cyanotic mucus membranes
  - Staggering gait
  - Bloat
  - Convulsions -Death
- **Chocolate-brown venous blood and tissues**
- Treatment – Methylene Blue (5-15 mg/kg–1% solution)
  Not approved for use in food or dairy animals

  18 month withholding
- Prevention – adaptation;

  propionibacterium-rumen inoculant-7days preexposure

Hydrogen Cyanide (Prussic Acid)-blood can't release oxygen – cherry red color

- Species Affected - Ruminants – equine?
- Toxic principle – cyanogenic glycosides – (dihurin)
- Regrowth (<24 in) especially important
- Green chop
- Clinical Signs -
  - Dyspnea
  - Cyanosis (blue discoloring of the lining of the mouth)
  - Bloat - rumen contents may smell like almonds
  - Convulsions - Death
- **Blood and tissues are bright cherry red**
- Treatment -

  20% sodium nitrite (10-20 mg/kg)

  methylene blue (3-5 g/230kg)

  20% sodium thiosulfate -500 -660 mg/kg

  can be repeated in 4-6 hrs
- Prevention – none other than prevent exposure

Target Animals

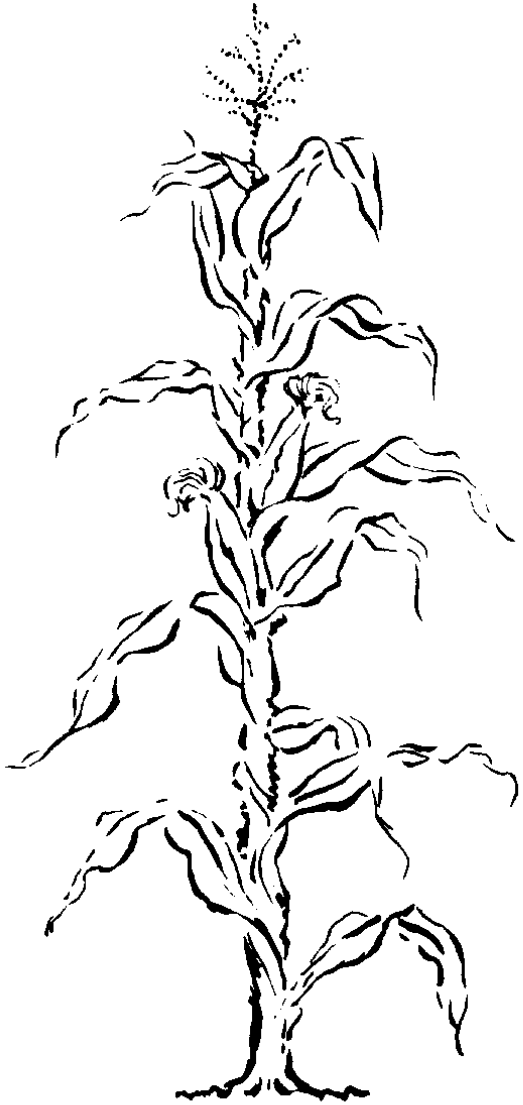
Although nitrate poisoning has occurred in cattle consuming forages with less than 5000 ppm nitrate

- general recommendation is that forages with up to 10,000 ppm, or 1 percent, nitrate are considered safe to feed to non-pregnant cattle that have been acclimated to the high nitrate feed.
- cattle that are stressed or in poor body condition consuming low energy diets are most susceptible to nitrate poisoning.
- sheep are less susceptible than cattle.
- nitrate poisoning has also been reported in goats, alpacas and water buffalo.

A few references consider horses to be as susceptible to nitrates as ruminants, other reports state that no harmful effects occurred in pregnant and open mares consuming forages containing from 25,000 to 35,000 ppm nitrate over a period of several months.

- others have reported no ill effects in horses consuming high nitrate forages that killed cows fed the same hay.
- Kentucky Equine Research News -lists 20,000 ppm as the level of concern for horses.

High nitrate forages do not pose a risk to other non-ruminants.



Top leaves)little accumulation.
Top stalk—small amounts, more than leaves.

Grain—practically zero, nitrogen mostly in protein form.
Middle stalk—moderate accumulation.

Lower leaves—moderate, higher than upper leaves.
Lower stalk—highest range.

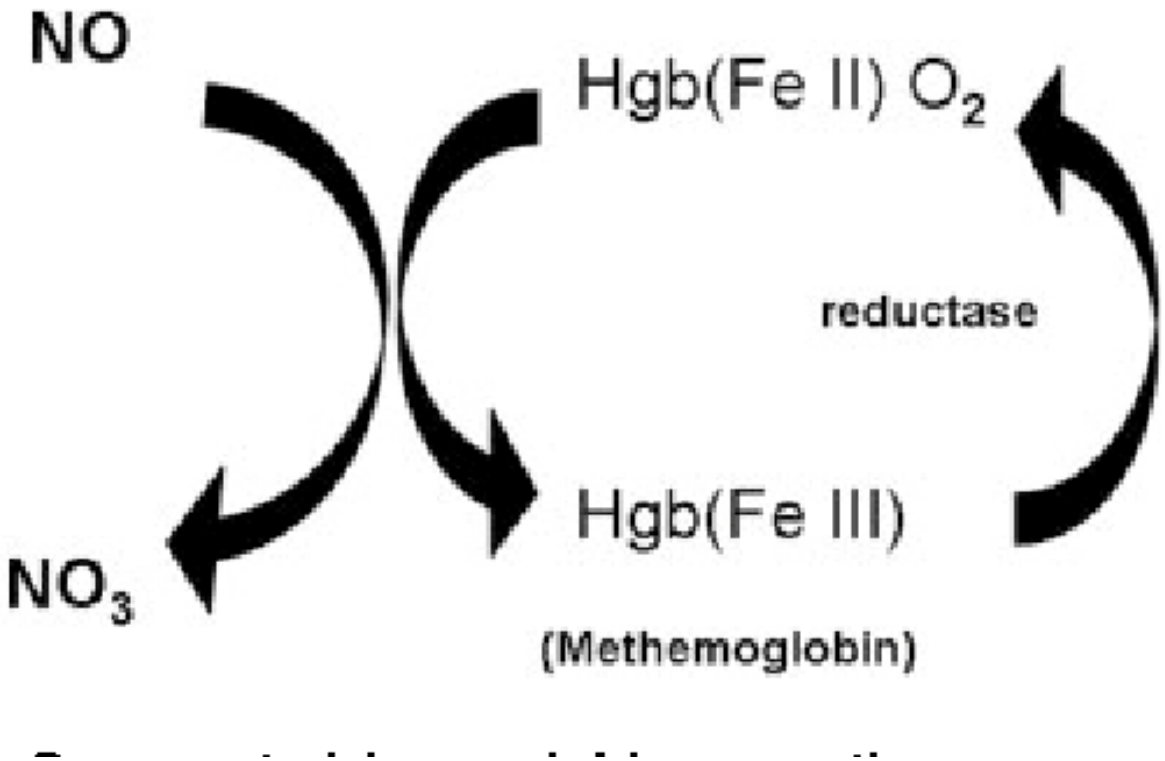
Figure 1.—Normal Accumulation of Nitrates in the Corn Plant

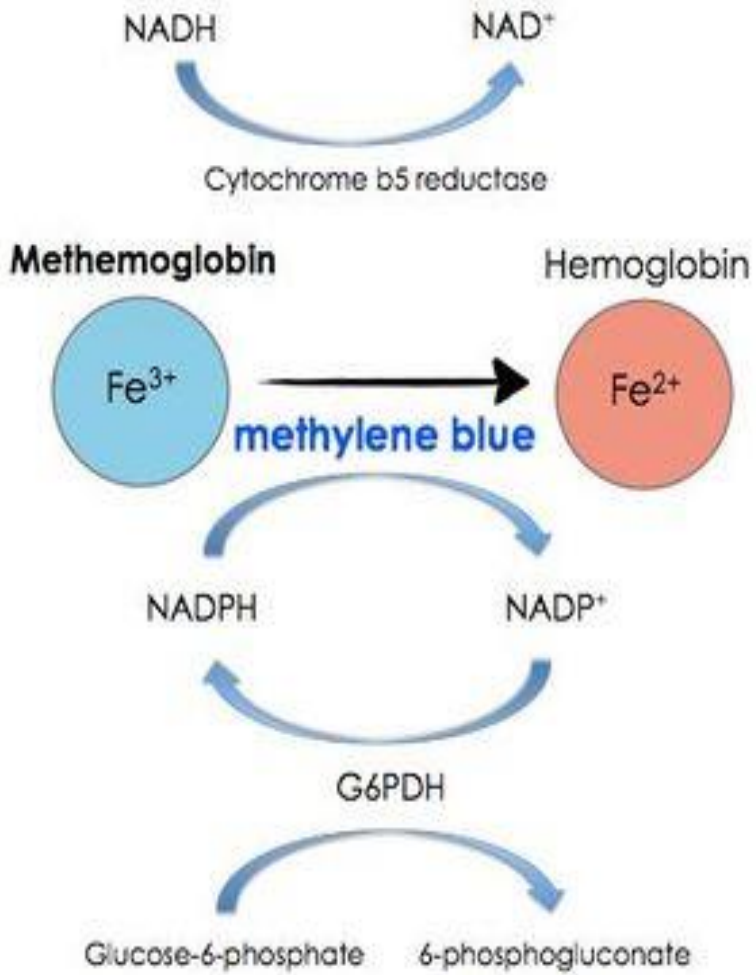
Table 1. Nitrate Nitrogen in 28 Samples of Drought-stressed Corn.

Plant Part	ppm NO_3N[1]
Leaves	63
Ears	17
Upper 1/3 of stalk	153
Middle 1/3 of stalk	803
Lower 1/3 of stalk	5,524
Whole plant	978

[1] ppm = parts per million
Source: University of Wisconsin

Nitrate Mechanism of Action treatment – methylene blue

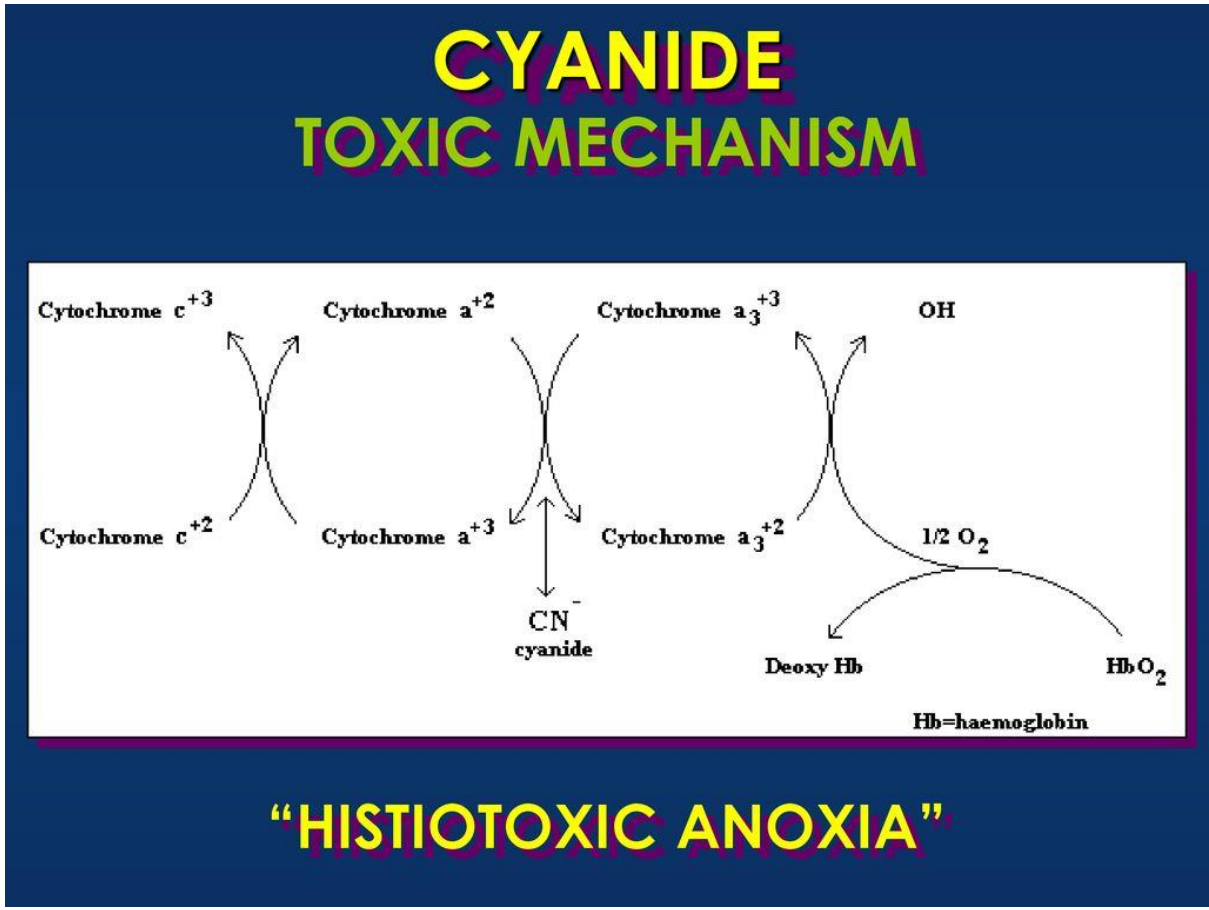




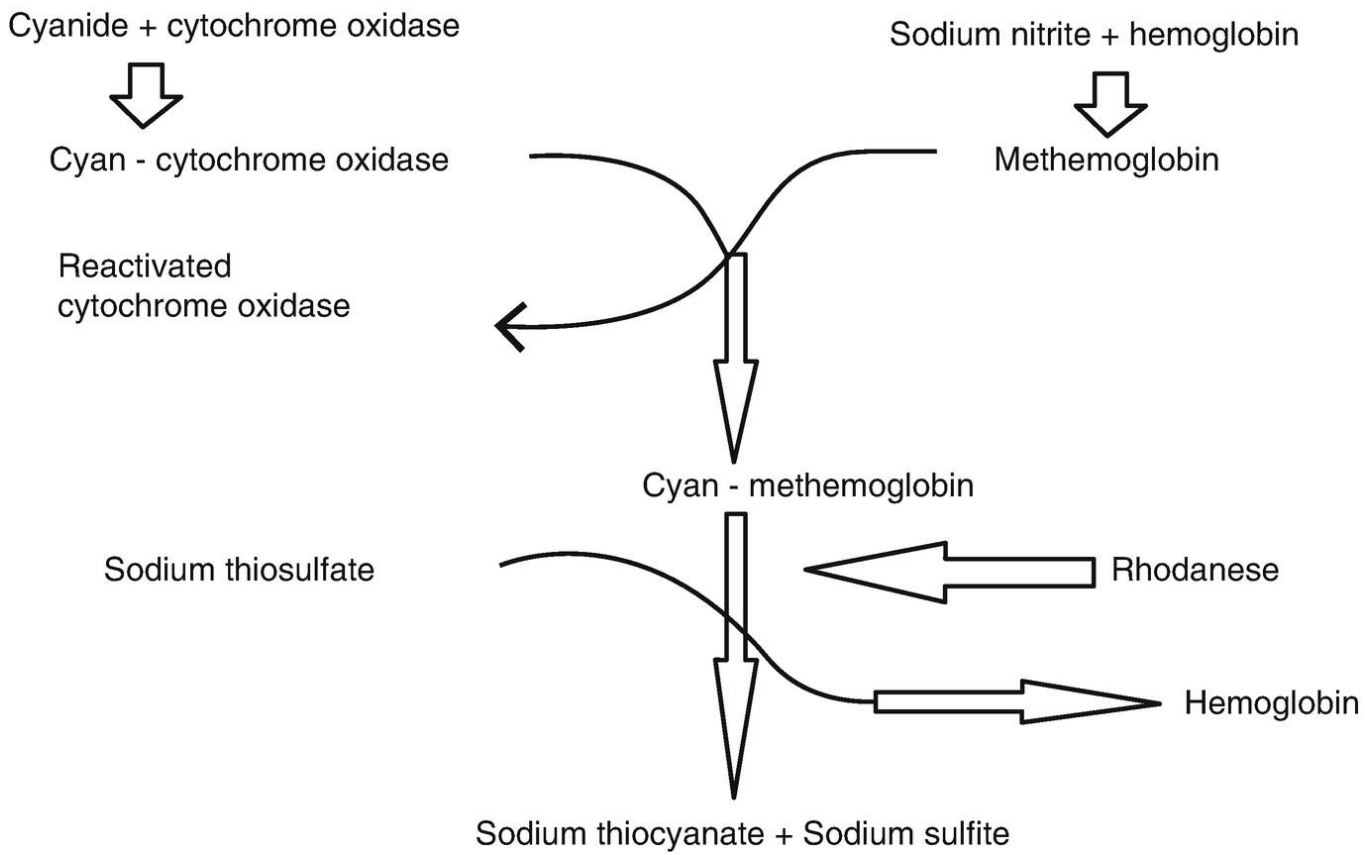
Cyanide Mechanism of Action

Mechanism of Action

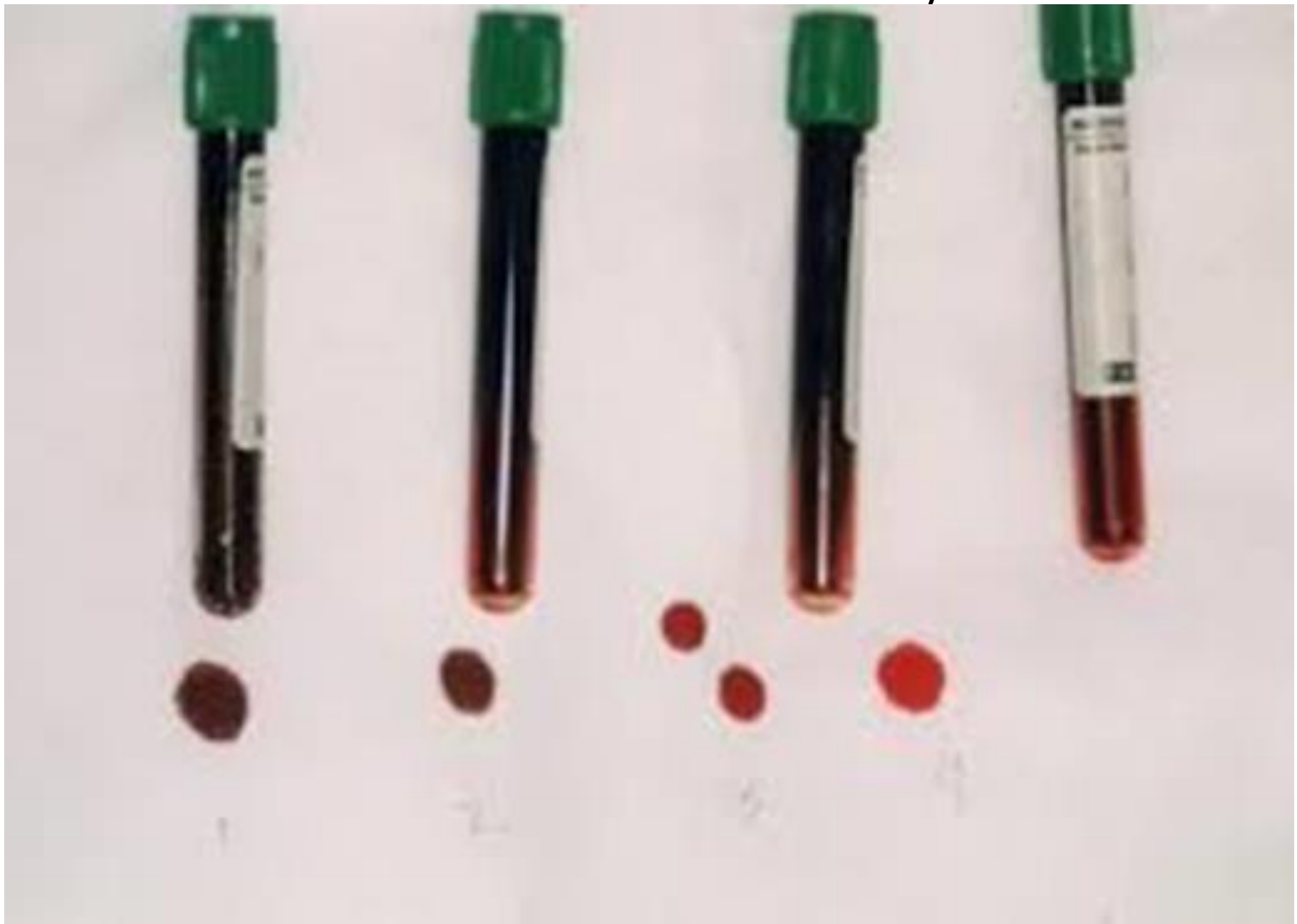
- CN is a chemical asphyxiant……inhibits **CYTOCHROME OXIDASE SYSTEM**
- …….Blocks the **aerobic utilization of oxygen**
- Nitrate reductase, myoglobin, catalase….
- Cellular anoxia….CNS, cardiac arrest, and respiratory arrest
- Cyanide readily bind to many enzymes having a metallic **component…binds avidly to heme Fe^{+3} causing cellular hypoxia……good!**



Cyanide Treatment
Sodium Nitrate and Sodium Thiosulfate



Blood Color Nitrate vs Cyanide



Diagnosis

Nitrate concentration in body fluids – freeze - ship cold

Eye fluid: postmortem - adult >20ppm; aborted fetus >30-50ppm

Serum/plasma: antemortem >20ppm

Post mortem findings

dark brown blood – place on white surface

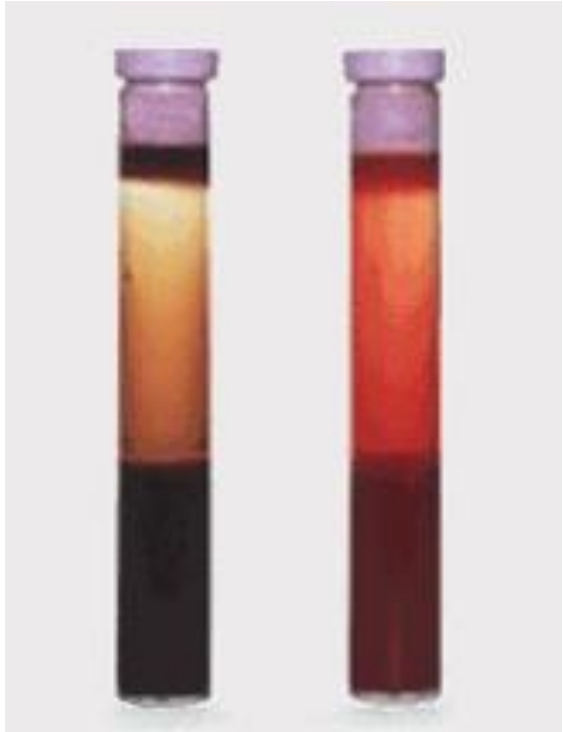
muddy brown membranes

Cyanide – cyanide detected in fresh rumen content

Postmortem – bitter almond smell

cherry red blood & mucus membranes

color fades quickly after death



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Evaluation of Factors Contributing to Excessive Nitrate Accumulation in Fodder Crops Leading to Ill-Health in Dairy Animals

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Abstract

A study was conducted to estimate nitrate content in commonly used fodder crops, viz., berseem (*Trifolium alexandrinum*), bajra (*Pennisetum glaucum*), maize (*Zea mays*), oats (*Avena sativa*), sorghum (*Sorghum vulgare*) and toriya (*Brassica napus*), collected from the fields of different villages of Punjab and farms of the university, and to evaluate the factors associated with nitrate accumulation in these crops. The nitrate level was highest in sorghum on dry matter basis, followed by oats and toriya, berseem, maize and bajra. The nitrate content was also determined in fodder samples harvested from young and mature stages and in different parts of plants. The stem part of forages had higher content than leaves; however, concentrations were low in mature crops as compared to immature ones. The environmental and soil factors associated with it are discussed and correlated with the experimental findings.

Keywords: Dairy animals, fertilizers, fodder crops, nitrate

INTRODUCTION

Dairy cows fed on high-quality forage produce more milk with less supplemental concentrate than the cows fed lower-quality forages. Forages with high concentrations of crude protein (CP) are considered high quality because feeding high-protein forage cuts down the need of supplemental protein. Secondly, CP content is positively correlated to energy content of forages. High-protein forages generally are more digestible and provide more energy than low-protein forages. Application of nitrogen (N) fertilizer improves both quality and yield by increasing CP content of forage markedly and the available energy. Due to this reason, the use of N-fertilizers on agricultural land has been immensely increased around the world.[1]

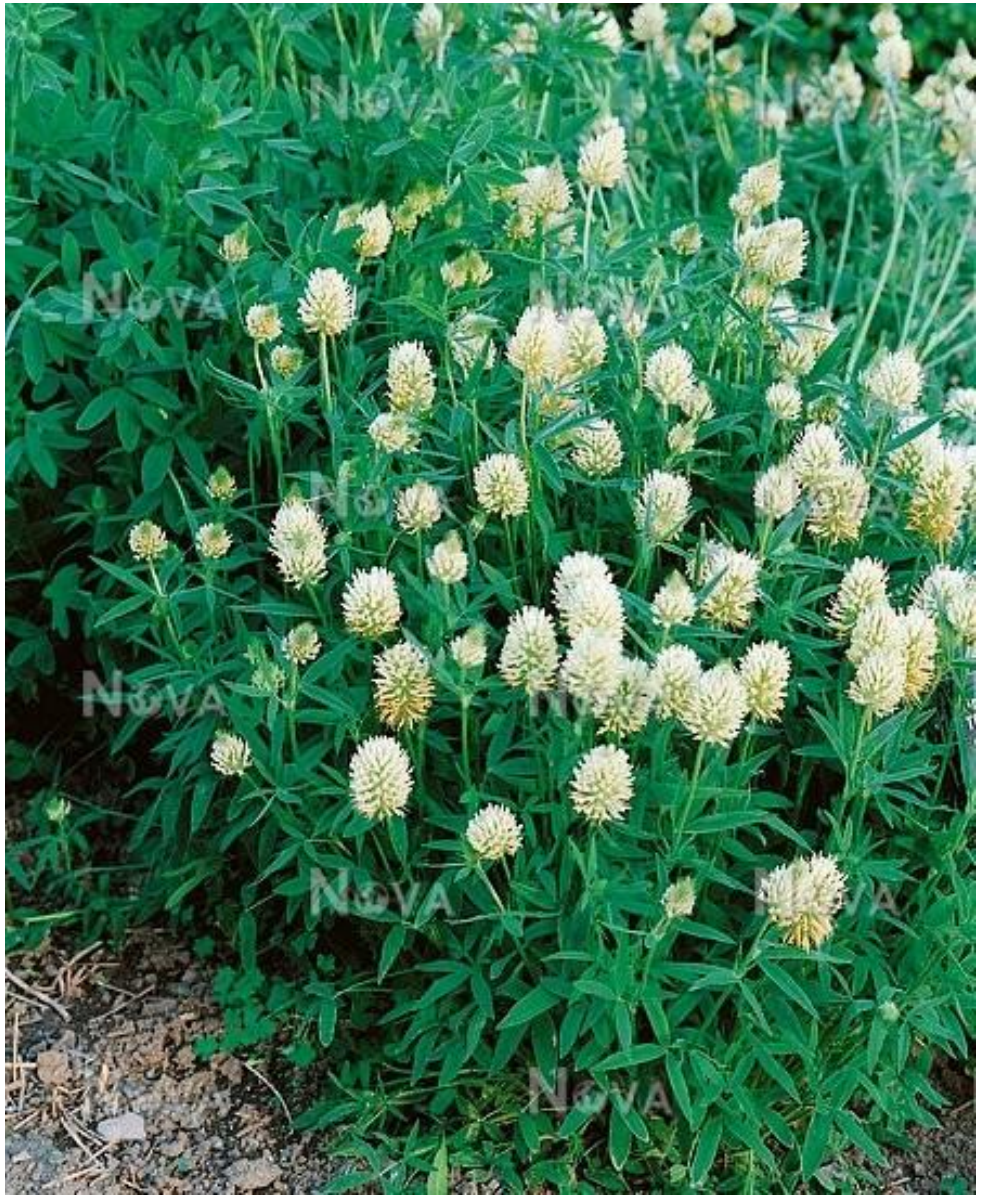
Forages take up and assimilate nitrogen as NH_4^+, NO_3^- and soluble organic compounds such as urea ($CO(NH_2)_2$) and amino-acids.[2] Nitrate is the primary nutrient form of the nitrogen in soils and is a normal constituent of plants. Occasionally, excessive amounts of nitrate accumulate in plants and result

Sorghum
Sorghum vulgare

Toriya (rapseed)
Brassica napus

Bajra (pearl millet)
Pennisetum glaucum

Berseem (Egyptian clover)
Trifolium alexandrinum



Sorghum sp - Sorghum sudan Sudangrass

Shattercane - Sorghum biclor

Nitrates

oats

corn (maize) forages & stalks

Nitrates are Additive – Total Intake

Table 2. Interpretation of laboratory results

Form of Nitrate Measured						
Potassium Nitrate (KNO_3)		Nitrate Nitrogen (NO_3-N)		Nitrate (NO_3)		Recommendations for use in livestock
ppm	%	ppm	%	ppm	%	
Forage (DM basis)						
0-7,220	0-0.72	0-1,000	0-0.10	0-4,430	0-0.44	Generally considered safe for livestock
7,220-10,830	0.72-1.08	1,000-1,500	0.10-0.15	4,430-6,645	0.44-0.66	Safe for nonpregnant animals; limit to 50% of ration dry matter for pregnant animals
10,830-14,440	1.08-1.44	1,500-2,000	0.15-0.20	6,645-8,860	0.66-0.88	Limit to 50% of ration dry matter for all animals
14,440-25,270	1.44-2.52	2,000-3,500	0.20-0.35	8,860-15,505	0.88-1.55	Limit to 30% to 35% of ration dry matter; do not feed to pregnant animals
25,270-28,880	2.52-2.88	3,500-4,000	0.35-0.40	15,505-17,720	1.55-1.77	Limit to 25% of ration dry matter; do not feed to pregnant animals
>28,880	>2.88	>4,000	>0.40	>17,720	>1.77	Danger: do not feed
Water (as received basis)						
0-720	0-0.072	0-100	0-0.01	0-443	0-0.04	Generally considered safe for livestock
720-2,166	0.072-0.21	100-300	0.01-0.03	443-1,329	0.04-0.13	Caution: Possible problems; consider additive effect with nitrate in feed
>2,166	>0.21	>300	>0.03	>1,30	>0.13	Danger: Could cause typical signs of nitrate poisoning

Nitrate Conversion Factors

Percent (%) times 10,000 equals parts per million (ppm) nitrate (NO_3);
ppm divided by 10,000 equals percentage(%).
ppm nitrate-nitrogen (NO_3-N) times 4.4 equals ppm nitrate (NO_3).
ppm nitrate (NO_3) times 0.23 equals ppm nitrate-nitrogen (NO_3-N).
ppm nitrate (NO_3) times 1.63 equals ppm potassium nitrate (KNO_3).

Cattle can adapt to increased nitrate levels in their diets in as little as seven days. Once acclimated, cattle should remain on a constant level of nitrate, as acclimation can be lost as quickly as it is gained.

10 Factors Related to Nitrate Toxicity

1.All plants contain nitrate, but under stress certain forages — corn, sorghum, oats, soybeans, millet, sudan and sorghum/sudan hybrids — can accumulate toxic levels. Pigweed, Canadian thistle, kochia, ragweed and other weeds also tend to accumulate nitrate.

2.Nitrate content varies widely throughout the plant but is greatest in the lower third of the stalk.

3.Concentration is usually high in young plants and decreases as the plant matures. However, at high levels of soil nitrate or under conditions of stress, content may be high at maturity. Highest levels occur just before flowering and decline rapidly after pollination and seed formation.

4.Abrupt setbacks to growth, like drought or freezing, may result in high nitrate. Lack of sunlight, temperature extremes or hail damage can also increase levels.

5.Levels increase immediately after a drought-breaking rain; therefore, harvest should be delayed for a week.

6.When silage is made from high nitrate forages, anaerobic fermentation converts nitrate to ammonia, significantly reducing the nitrate content. Levels in properly ensiled forages can decrease by 30 percent to 60 percent over a month or two. Forages with significantly elevated nitrate levels at harvest should be retested before fed.

7.Green chop should be fed as soon as possible. Storage heating can convert nitrate to nitrite — the toxic agent in nitrate poisoning — increasing potential toxicity up to ten times.

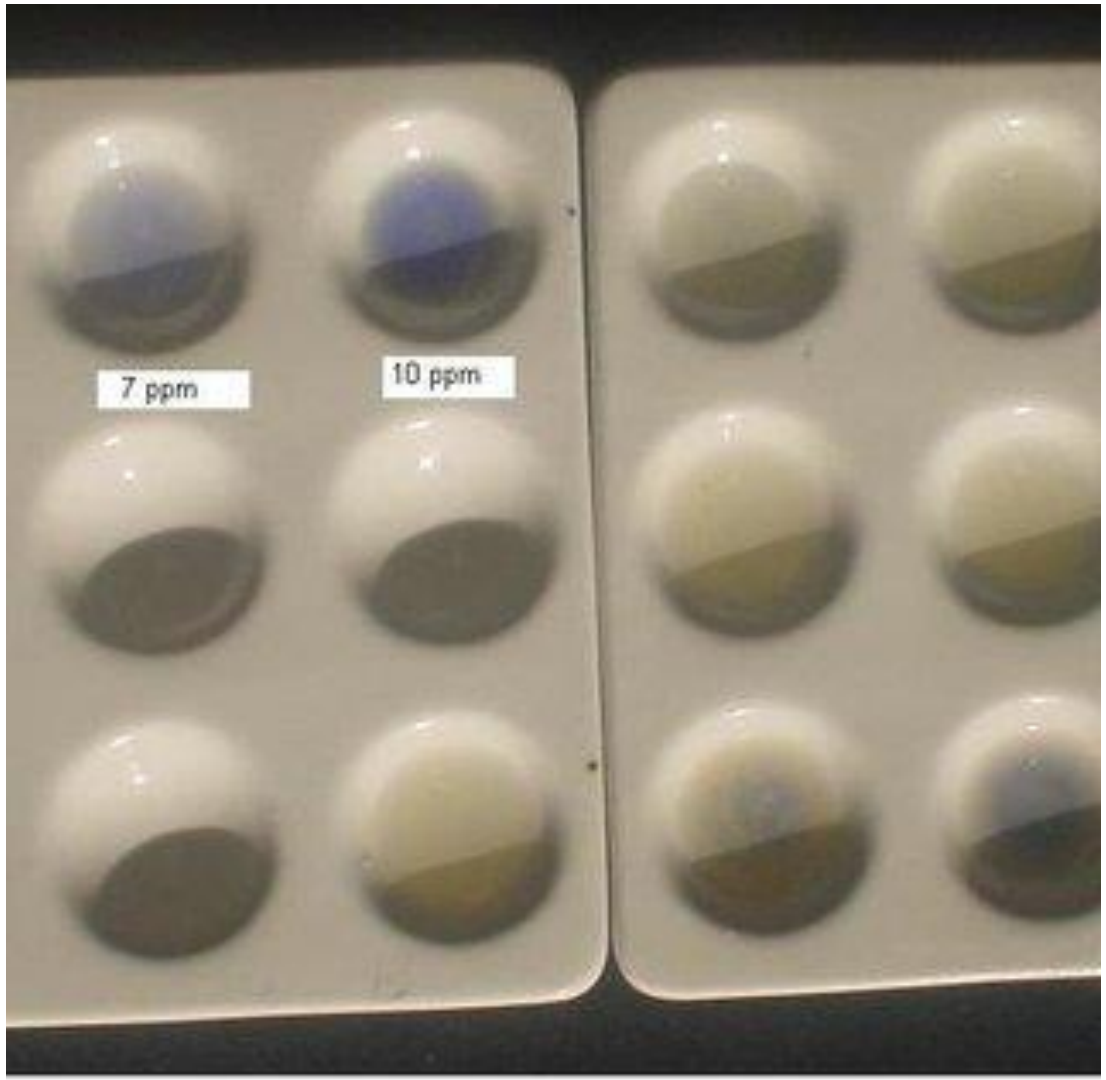
8.To analyze harvested hay or silage, take a composite sample from six to eight different bales or locations in the silo. Cored samples are preferred on bales.

9.The effects of nitrate levels in forage, feed and water are additive. Livestock water containing 1,000 ppm nitrate can contribute to nitrate poisoning even when feed contains only moderate levels.

10.High energy feeds and gradual introduction to high nitrate feeds will increase tolerance. Healthy animals have a higher resistance than ill or poorly nourished animals.

Diphenylamine test

- Diphenylamine may be used as a chemical test for the presence of the nitrate ion. In this test, a solution of diphenylamine and ammonium chloride in sulfuric acid is used. In the presence of nitrates, diphenylamine is oxidized, giving a blue coloration.



Diphenylamine Test

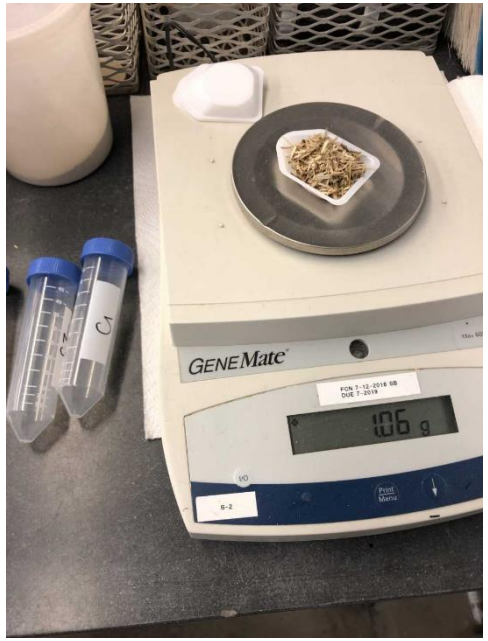
http://www.asi.ksu.edu/doc6194.ashx

- Intense **BLUE** color = forage contains potentially dangerous nitrate levels
- If **POSITIVE** send the forage for analysis prior to feeding.

L. Hollis – Vet Quarterly, Fall 2012


PATRIOT


Oster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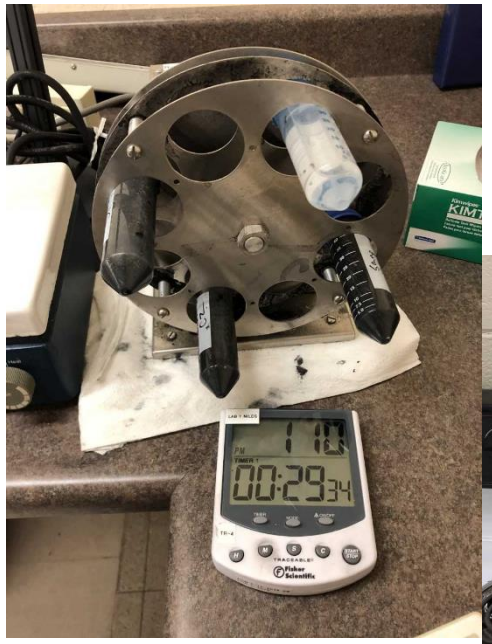
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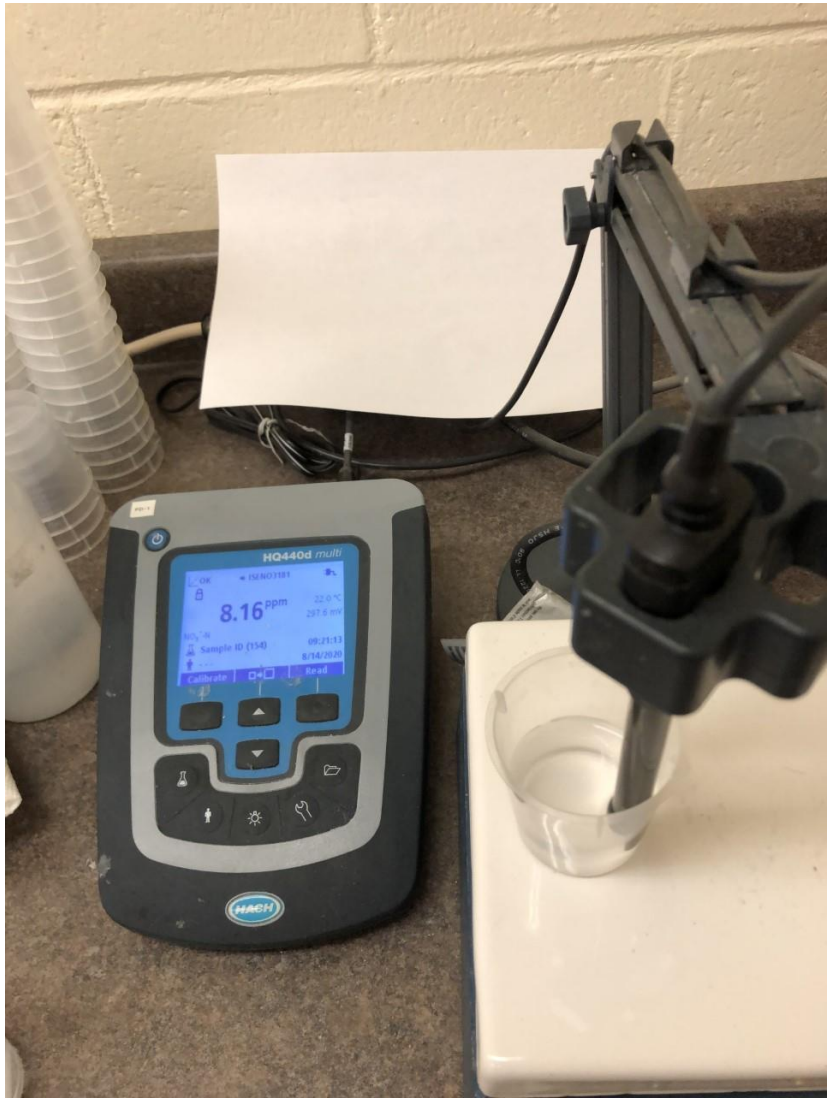
Fisher Scientific

Nitrate Selective Ion Probe

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NO2-
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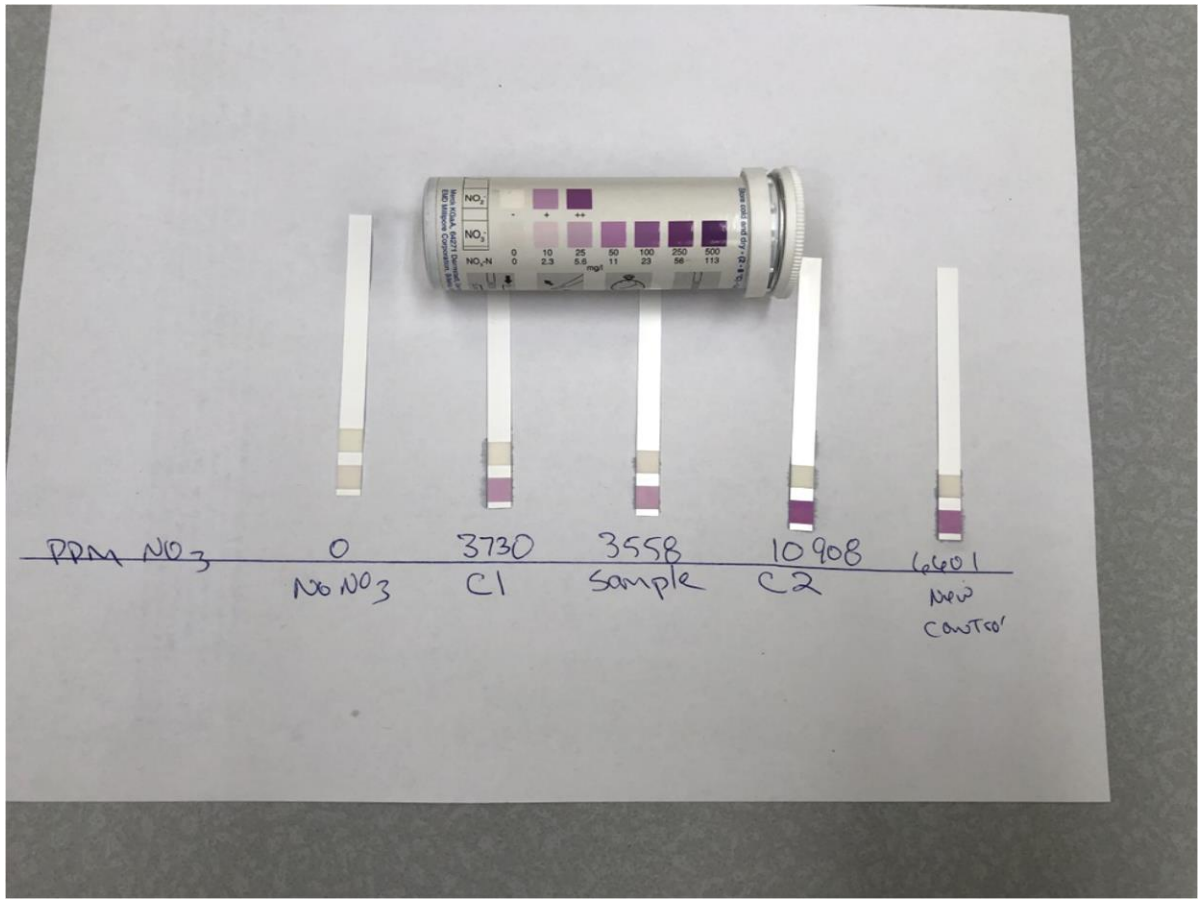
mg/l
Store cold and dry • (2 - 8 °C)

Nitrat-Test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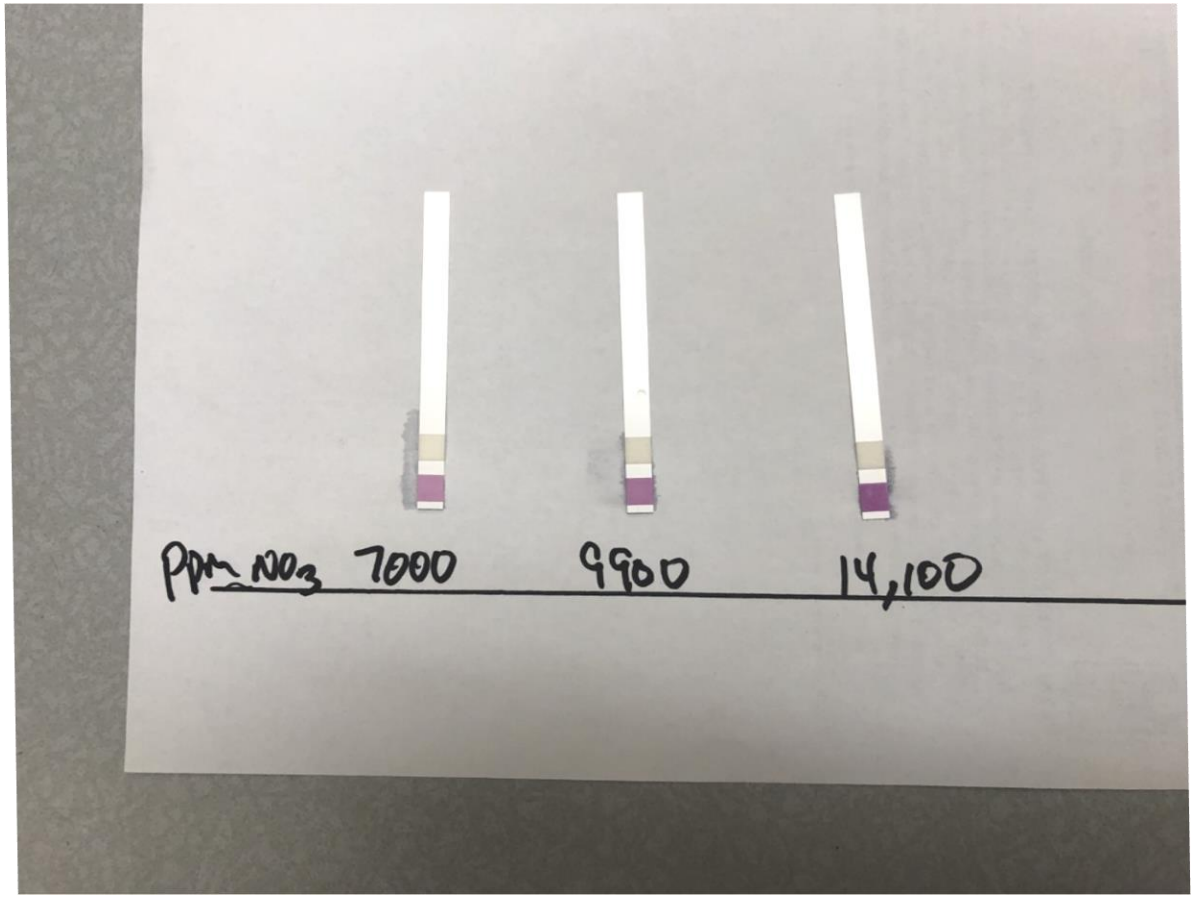
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Management Guidelines

Drought-stressed small-grain forages and other forages suspected of being high in nitrates should be tested before feeding.

Dilute high-nitrate forages with other forages or feedstuffs that are low in nitrates. In some cases, this can reduce nitrate levels in the diet enough to make the forages safe to feed.

Allow for frequent intake of small amounts of high-nitrate feed because that helps adjust livestock to high-nitrate feeds and increases the total amount of nitrate that livestock can consume daily without adverse effects.

Allow cattle time to adapt to increased nitrate in the diet. If nitrate levels are not excessively high (9,000 parts per million nitrate, or NO_3), the animals can adapt to increasing amounts in the feed.

Give livestock access to fresh, nitrate-free water at all times.

Be sure pastures are not overstocked when grazing high-nitrate forages. Overstocking increases the amount of high-nitrate plant parts (stems and stalks) that livestock consume.

Do not strip graze high-nitrate forages. Strip grazing also increases the amount of stem and stalk material that livestock consume.

Do not allow hungry cattle access to high-nitrate forages or pastures. Feed cattle hay or forages low in nitrates before turning them onto high-nitrate pastures.

Supplement cattle grazing high-nitrate forages with other low-nitrate feedstuffs, such as low-nitrate forages, feed grains or byproducts. The addition of high-energy feeds stimulates the conversion of nitrate to nontoxic compounds and helps reduce the potential for toxicity.

Graze cattle on high-nitrate pastures during the day and remove them at night for the first week of grazing if possible. This reduces the amount of high-nitrate forage consumed and helps acclimate cattle to the high nitrate levels.

Don’t graze high-nitrate pastures until one week after a killing frost if possible.

Observe cattle frequently when you turn them into a suspected field or pasture to detect any signs of toxicity.

Be aware that cattle in poor health and condition, especially cattle suffering from respiratory disease, are more susceptible to nitrate poisoning.

Consider harvesting and feeding high-nitrate forages as silages. The fermentation process that occurs when feeds are ensiled reduces nitrate levels; however, this does not guarantee that silage will contain “safe” levels of nitrate. Testing still is recommended.

Do not allow cattle access to areas where fertilizers are stored.

Do not feed green chop that has heated after cutting or has been held overnight. Heating favors the formation of nitrite, which is more toxic than nitrate.

Nitrate Variation 2.4 ha (6 Acres) Field

2/3 nitrate content in the lower 1/3 of the stock

6 Acres

Chemistry/Toxicology

ethod) - RF

Specimen	Specimen Type	Result Date	Results
1- 6T	Feed	15-Oct-2015	806 ppm(DW)
2- BOT	Feed	15-Oct-2015	7931 ppm(DW)
3- TM	Feed	15-Oct-2015	821 ppm(DW)
4- SB	Feed	15-Oct-2015	2394 ppm(DW)
5- 5B	Feed	15-Oct-2015	3484 ppm(DW)
6- T8	Feed	15-Oct-2015	879 ppm(DW)
7- 3M	Feed	15-Oct-2015	10765 ppm(DW)
8- 1B	Feed	15-Oct-2015	4903 ppm(DW)
9- 4M	Feed	15-Oct-2015	2040 ppm(DW)

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Research Article

TREATMENT OF CYANIDE POISONING IN CROSSBRED COWS: A PROFILE OF DRUG SYNERGISM

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ABSTRACT

Five cross breed cows suffering from cyanide poisoning were treated. Immaturely cut sorghum grass in pre monsoon season fed to animals was identified as a cause of poisoning. All the animals exhibited the severe clinical signs of poisoning like dyspnoea, anxiety, restlessness, staggering gait as well as tremors. All five cows were treated immediately at nearby farms where they were kept. While treating cows, three among five were administered saturated jaggery liquid prior to literature based allopathic SID treatment of 15 gm IV administration of Sodium nitrite and Sodium thiosulphate combination, where as rest two were given only allopathic treatment of Sodium nitrate and Sodium thiosulphate combination (15 gm IV). Cows which were treated with both jaggery and allopathic therapeutics recovered fast as compared to cows treated with allopathic therapeutics alone. Result suggests that jaggery play a synergistic action with allopathic therapeutics and responsible for early recovery from clinical symptoms of poisoning.

Key word: Cyanide, Crossbred Cows, Toxicity

INTRODUCTION

The plants, the most important source of cyanide toxicity in animals, which contain hydrocyanic acid (HCN) either free or in form of cyanogenic glycosides are called as cyanogenetic plants[1]. The glycoside itself is non-toxic, which owe its toxicity to cyanide after hydrolysis. Cyanogenetic plant toxicity is one of the most common plant poisoning among the grazing livestock. Ruminants are more susceptible to poisoning by cyanogenic plants, which release hydrogen cyanide than horses and pigs due to more efficient hydrolysis of the cyanogenic glycosides[2]. Within the group of ruminants, goats appear to be the most susceptible to cyanide. Based on the available data the following intake mg hydrogen cyanide (HCN) equivalents/kg b.w. per day) seems to be tolerated by the following animal species: pigs (2.9 mg/kg per day), poultry (2.8 mg/kg per day), ruminants (on the basis of goat studies) (0.25 mg/kg per day) and horses (0.4 mg/kg per day), respectively[3]. Cattle and buffaloes are also highly susceptible species. This type of toxicity usually occurs in animals when animal ingests large amount of immature sorghum fodder at pre-bloomed stage[4]. Factors that incur likelihood of HCN poisoning from ingestion of cyanogenic plants include: (1) large amount of free HCN and cyanogenic glycoside in plant, (2) rapid ingestion; (3) ingestion of a large amount of plant, and (4) ruminal pH and microflora that continue to hydrolyze glycosides to release hydrogen cyanide. Rapid intake of plant equivalent to about 4 mg HCN/kg of body weight is considered to be lethal amount of plant material[5]. In current trend for earning more profit, farmers are trying to cultivate more crops throughout year, so they may cut the forrage in immature stage, which is the main source of cyanide toxicity. Generally it happens due to lack of forrage for feeding to animals because of improper forrage management and also sometimes it may occur due to accidental ingestion while grazing[6].

About 120 plant species containing cyanogenetic glycosides are found in India. The plant material containing over 20 mg HCN per 100 gm is potentially toxic to livestock[1] (Garg, 2004). The details about toxicity index of HCN are given in Table 1.

The most common source of cyanogenic plant poisoning in livestock is feeding of green Sorghum fodder, particularly immature plant or young shoots. Details of plant forrage prone to cause HCN poisoning in animals is shown in Table 2.

Table 1: Level of HCN in forage (dry matter basis) and potential effect on animals[7]

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

Table 2: Millet and sorghum types and their potential cyanide accumulations[7]

Sr. No.	Type of plants (Millet or Sorghum)	Potential of Cyanide Toxicity
1.	Grain sorghums	High to very high
2.	Johnsongrass	High to very high
3.	Shattercane	High
4.	Forage sorghums	Intermediate to high
5.	Sorghum-sudangrass hybrids	Intermediate to high
6.	Sudangrass hybrids	Intermediate
7.	Sudangrass varieties	Low to intermediate
8.	Pearl and Foxtail millet	Very low

Present case report reflects incidence of cyanide toxicity in crossbred cows.

INTRODUCTION

The plants, the most important source of cyanide toxicity in animals, which contain hydrocyanic acid (HCN) either free or in form of cyanogenic glycosides are called as cyanogenetic plants[1]. The glycoside itself is non-toxic, which owe its toxicity to cyanide after hydrolysis. Cyanogenetic plant toxicity is one of the most common plant poisoning among the grazing livestock. Ruminants are more susceptible to poisoning by cyanogenic plants, which release hydrogen cyanide than horses and pigs due to more efficient hydrolysis of the cyanogenic glycosides[2]. Within the group of ruminants, goats appear to be the most susceptible to cyanide. Based on the available data the following intake mg hydrogen cyanide (HCN) equivalents/kg b.w. per day) seems to be tolerated by the following animal species: pigs (2.9 mg/kg per day), poultry (2.8 mg/kg per day), ruminants (on the basis of goat studies) (0.25 mg/kg per day) and horses (0.4 mg/kg per day), respectively[3]. Cattle and buffaloes are also highly susceptible species. This type of toxicity usually occurs in animals when animal ingests large amount of immature sorghum fodder at pre-bloomed stage[4]. Factors that incur likelihood of HCN poisoning from ingestion of cyanogenic plants include: (1) large amount of free HCN and cyanogenic glycoside in plant, (2) rapid ingestion; (3) ingestion of a large amount of plant, and (4) ruminal pH and microflora that continue to hydrolyze glycosides to release hydrogen cyanide. Rapid intake of plant equivalent to about 4 mg HCN/kg of body weight is considered to be lethal amount of plant material[5]. In current trend for earning more profit, farmers are trying to cultivate more crops throughout year, so they may cut the forrage in immature stage, which is the main source of cyanide toxicity. Generally it happens due to lack of forrage for feeding to animals because of improper forrage management and also sometimes it may occur due to accidental ingestion while grazing[6].

About 120 plant species containing cyanogenetic glycosides are found in India. The plant material containing over 20 mg HCN per 100 gm is potentially toxic to livestock[1] (Garg, 2004). The details about toxicity index of HCN are given in Table 1.

The most common source of cyanogenic plant poisoning in livestock is feeding of green Sorghum fodder, particularly immature plant or young shoots. Details of plant forrage prone to cause HCN poisoning in animals is shown in Table 2.

Table 1: Level of HCN in forage (dry matter basis) and potential effect on animals[7]

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

Table 2: Millet and sorghum types and their potential cyanide accumulations[7]

Sr. No.	Type of plants (Millet or Sorghum)	Potential of Cyanide Toxicity
1.	Grain sorghums	High to very high
2.	Johnsongrass	High to very high
3.	Shattercane	High
4.	Forage sorghums	Intermediate to high
5.	Sorghum-sudangrass hybrids	Intermediate to high
6.	Sudangrass hybrids	Intermediate
7.	Sudangrass varieties	Low to intermediate
8.	Pearl and Foxtail millet	Very low

Present case report reflects incidence of cyanide toxicity in crossbred cows.

Sorghum - Sorghum vulgare
Cyanide toxin in the “green” leaves

Cyanide- Cyanide toxin in the “green” leaves

Foxtail Millet-Setaria italica

Pearl Millet-Pennisetum glaucum

Cyanide - Rubber tree leaves

Hevea Brasiliensis Poisoning in Malnad Gidda Cattle, Karnataka, India

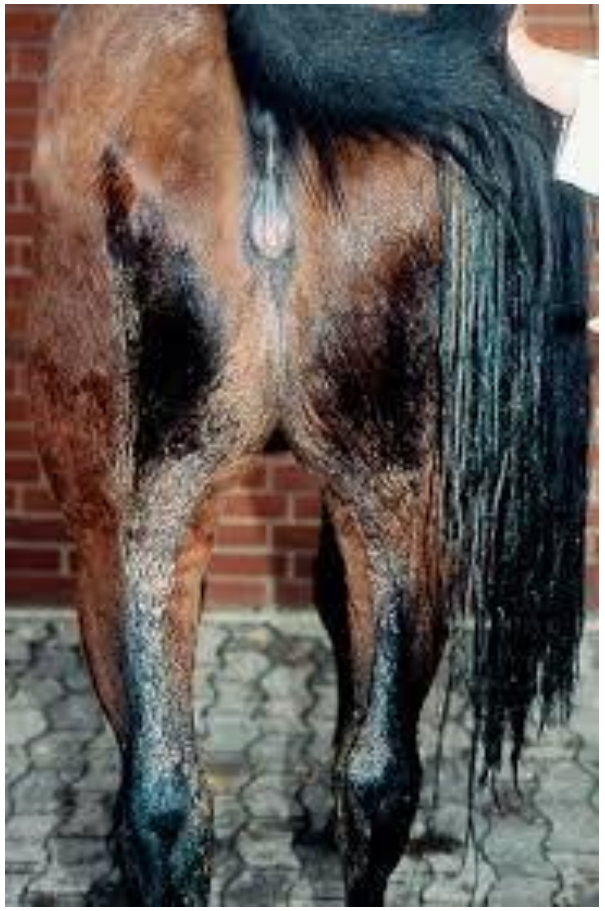
Lohith T. S. et al.

Internal Research Journal of Pharmacy; 2014,5(7)

Cyanide

Sudangrass

ataxia/cystitis & fetal deformities



Johnsongrass - Sorghum halepense

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Experimental poisoning by cassava wastewater in sheep

The main cyanogenic glycoside of cassava is linamarin (the same as in linseed) making up more than 90% of total such compounds in the tuber. Once linamarin is activated by the enzyme beta-glucosidase, it releases prussic acid (hydrogen cyanide, HCN) which is a highly volatile substance characterized by the odor of bitter almonds. It is highly toxic to animals when consumed at high enough quantities, causing asphyxia and death.

Cassava

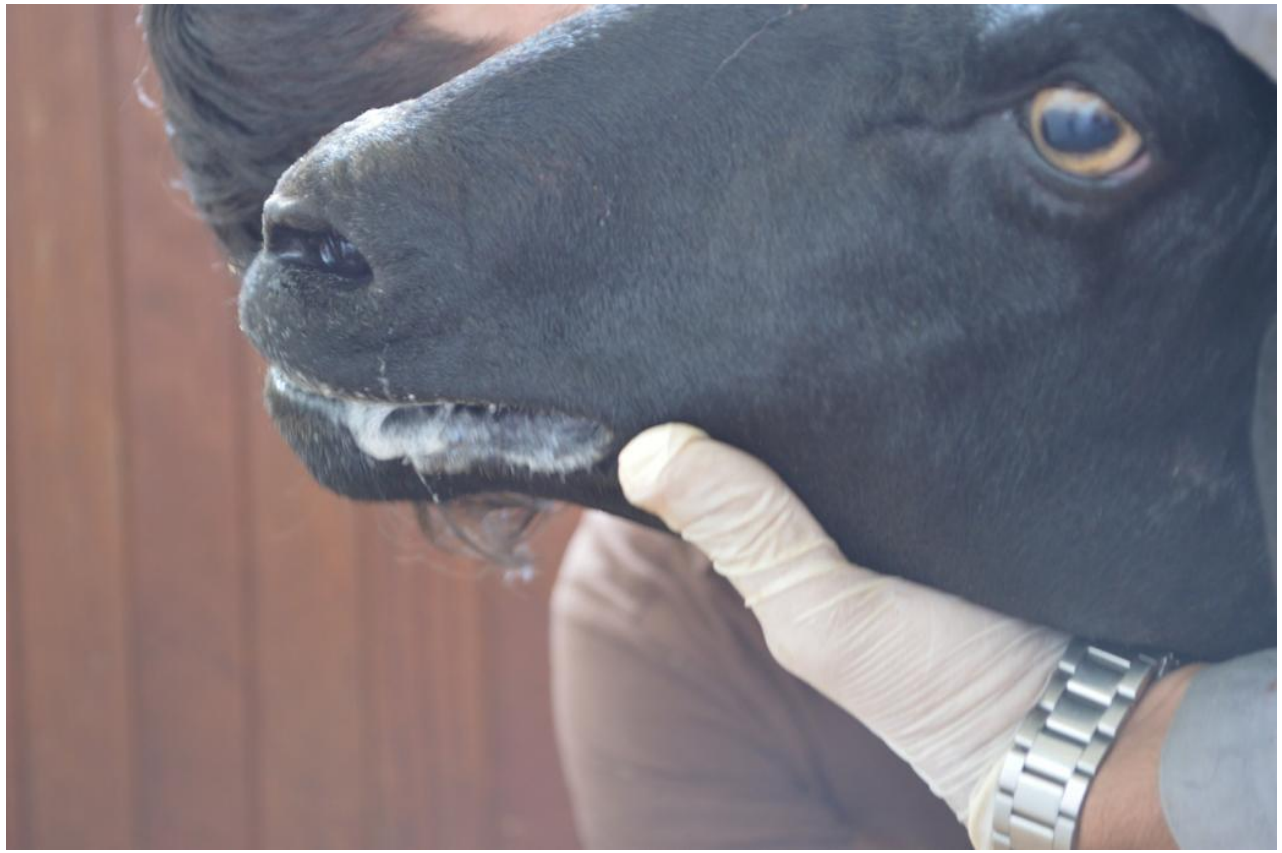
The flesh of sweet cassava varieties contains low levels of hydrogen cyanide (less than 50 mg/kg hydrogen cyanide), but bitter varieties are rather rich (over 200 mg/kg), with certain ones containing more than 1,000 mg/kg. Therefore, knowing the initial variety is important as 90% reduction of 50 mg/kg is substantially different compared to 90% reduction of 200 mg/kg.

Vet and Human Toxicology 2002 Dec;44(6):366-9.

Cyanogenic potential of cassava peels and their detoxification for utilization as livestock feed

Robert Tweyongyere[1], Ignatious Katongole

This study determined the cyanogenic potential of the cassava peels and assess the effectiveness of sun drying, heap fermentation and wet fermentation (soaking) in reducing the cyanide potential of the peels. Fresh cassava peels from major fresh food markets in Kampala and cassava grown in various parts of Uganda from Namolonge Agricultural and Animal Research Institute were used. The fresh peels from the market were subjected to the different detoxification methods for 5 d; the cyanide potential was determined by enzymatic assay. The mean potential of the cassava peels from the food markets Kampala was 856 mg cyanide equivalen/kg of dry matter. The potential of the peels of the 14 cultivars fell between 253 and 1081 mg cyanide eQuivalent/kg of dry matter. High cyanogenic potential cultivars dominate on the market and pose danger of poisoning to livestock fed on fresh cassava peels. Treatment of the peels by sun-drying, heap fermentation on soaking reduced the cyanide potential to below 100 mg cyanide equivalent/kg of dry matter at 48, 72 and 96 h respectively. Sun-dying caused an early sharp fall in the cyanide potential, but heap fermentation or soaking gave the lowest residual cyanide after 120 h. Cassava peels could be safely used as livestock feed if they are treated to reduce the cyanogenic potential.



Cyanide Picric Acid Test


REF
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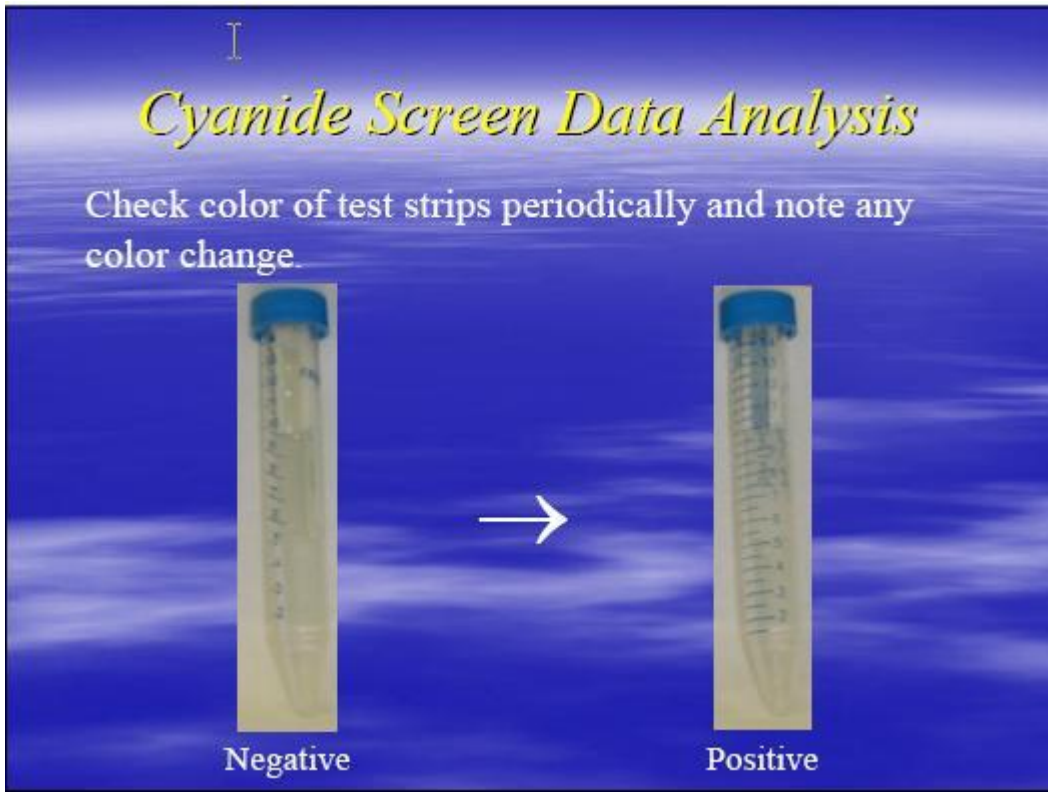
Cyanide Screen Data Analysis
Check color of test strips periodically and note any color change.
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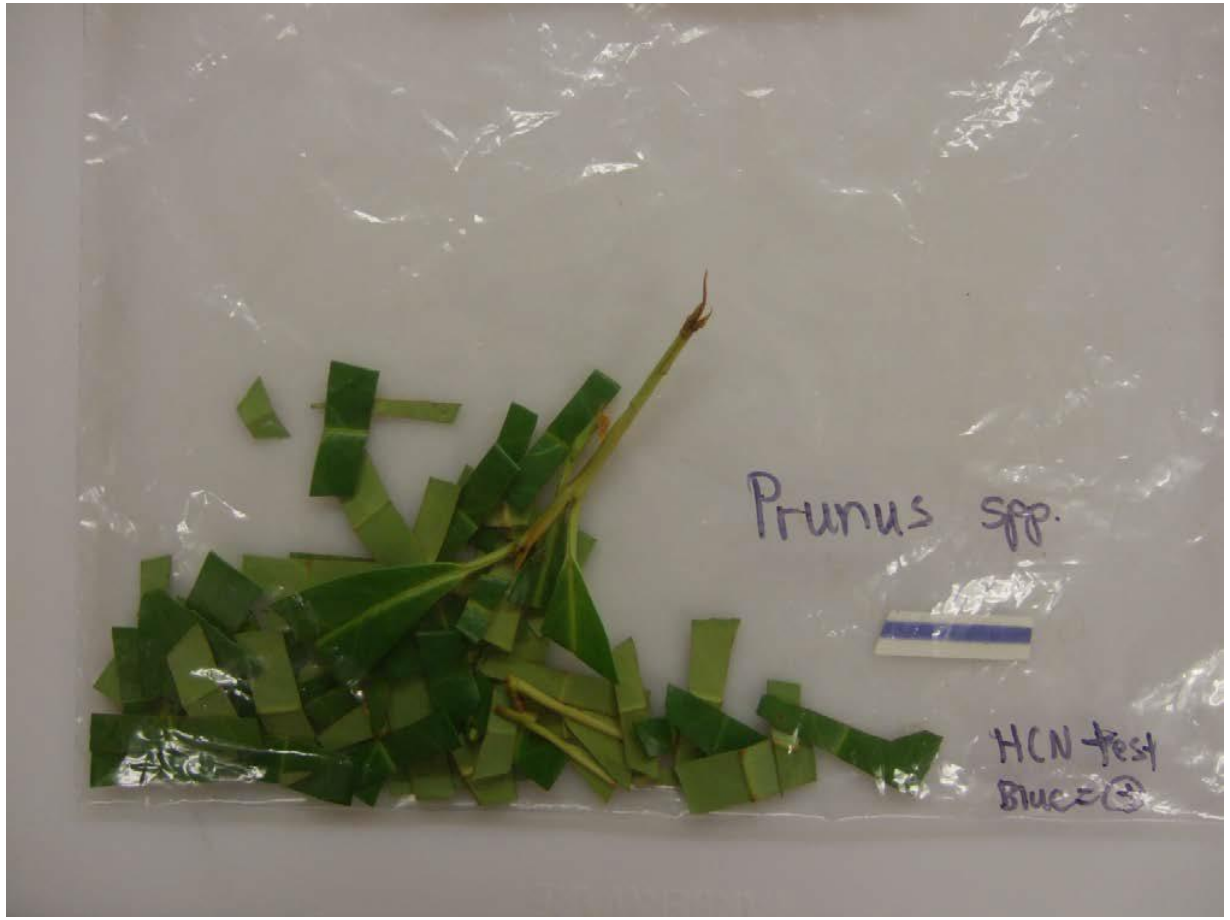
Positive



Shelby Filley
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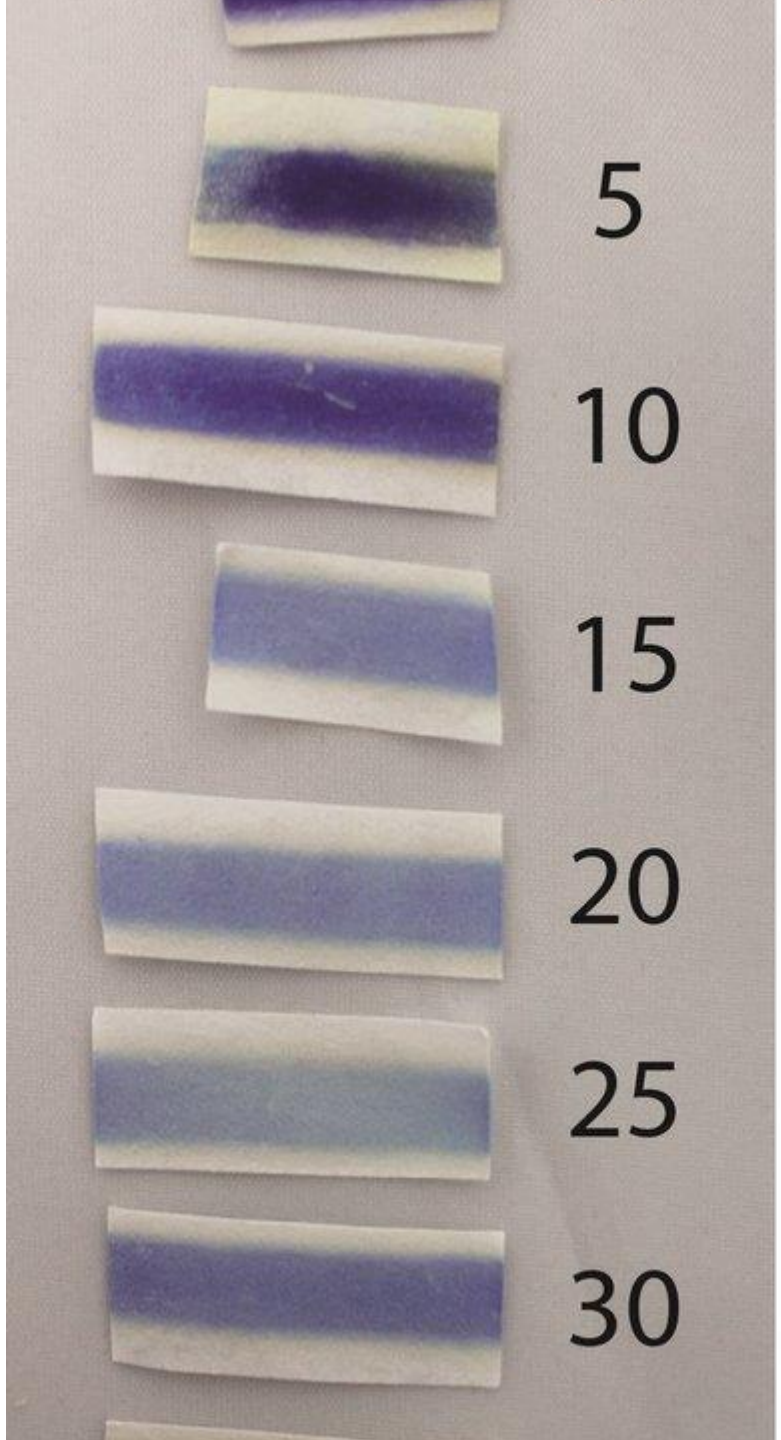
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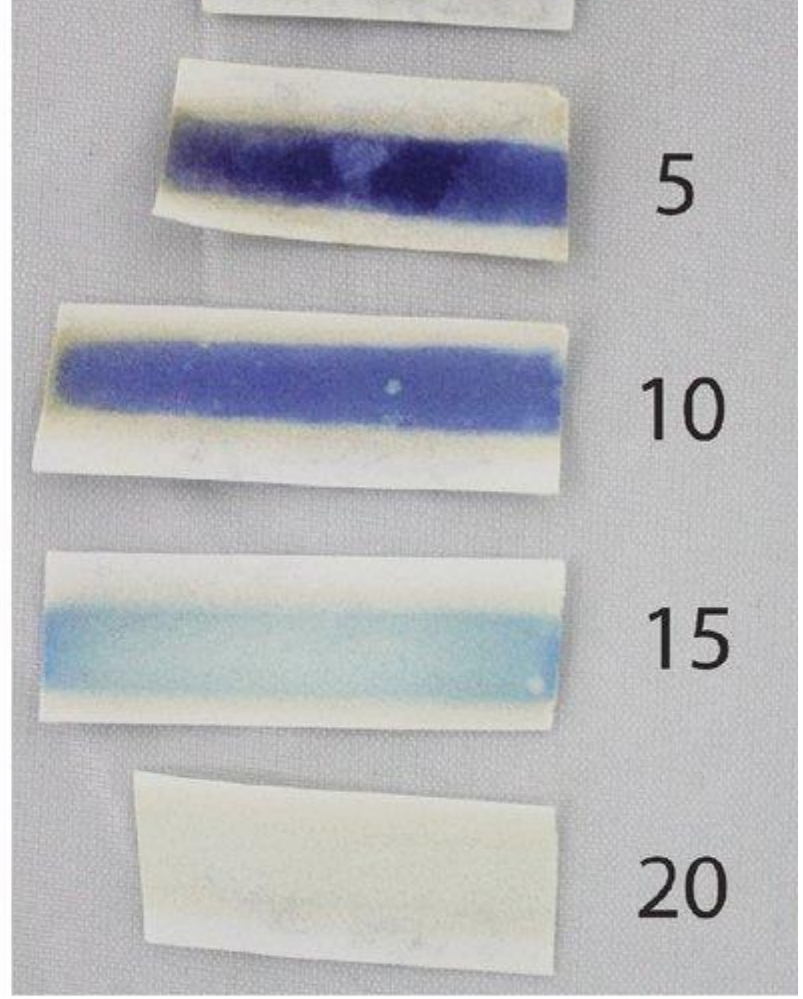
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TREATMENT OF CYANIDE POISONING IN CROSSBRED COWS: A PROFILE OF DRUG SYNERGISM

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PUBUSHING HOUSE
TREATMENT OF CYANIDE POISONING IN CROSSBRED COWS: A PROFILE OF DRUG SYNERGISM
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ABSTRACT
Five cross breed cows suffering from cyanide poisoning were treated. Immaturely cut sorghum grass in pre monsoon season fed to animals was identified as a cause of poisoning. All the animals exhibited the severe clinical signs of poisoning like dyspnoea, anxiety, restlessness, staggering gait as well as tremors. All five cows were treated immediately at nearby farms where they were kept. While treating cows, three among five were administered saturated jaggery liquid prior to literature based allopathic SID treatment of 15 gm IV administration of Sodium nitrite and Sodium thiosulphate combination, where as rest two were given only allopathic treatment of Sodium nitrate and Sodium thiosulphate combination (15 gm IV). Cows which were treated with both jaggery and allopathic therapeutics recovered fast as compared to cows treated with allopathic therapeutics alone. Result suggests that jaggery playa synergistic action with allopathic therapeutics and responsible for early recovery from clinical symptoms of poisoning.
Key word: Cyanide, Crossbred Cows, Toxicity

What is Jaggery? (Acc. to Reg. 5.7.6 of Food Safety and Standard Regulations, 2010)

- **GUR OR JAGGERY** means the product obtained by boiling or processing juice pressed out of sugarcane or extracted from Palmyra-palm, date-palm or coconut-palm. It shall be free from substances deleterious to health and shall confirm to the following analytical standards, on dry weight basis :

Substances	Analytical Standards
Total sugars expressed as invert sugar	Not less than 90 percent and Sucrose not less than 60 percent
Extraneous matter insoluble in water	Not more than 2 percent
Total ash	Not more than 6 percent.
Ash insoluble in hydrochloric acid (HCL)	Not more than 0.5 percent.

???

Lantana camara

Introduced Plants, Negative Effects of

William G Lee,Landcare Research, Hamilton, New Zealand

Lantana camara is an important weed of plantation crops. In India, it has taken over land used for tea and sugar cane production, causing the displacement of whole villages, and in other countries it is a major nuisance in cotton, coffee, coco-nut, oil palm, bananas, pineapple, rubber, and rice crops. In continental countries such as Australia, India, and South Africa, it is natural grasslands that have been most extensively modified by Lantana camara.

Lantana

Hepatogenous Photosenitization

(Secondary Photosensitization)

Major cause of photosensitization in livestock

Photosensitization results from increased circulating phylloerythrin secondary to intrahepatic cholestasis

-Phylloerythrin is formed when chlorophyll is degraded in the intestine

-normally excreted in bile.

Toxic principle - triterpene acids lantadene A and B. induce an intrahepatic cholestasis.

At least 15 of the 29 described taxa of Lantana camara are known to be toxic to livestock.

About 1% body weight of green leaves will induce poisoning.

All parts of the plant are toxic including the ripe black fruits.

Description

The plant is a shrub with square stems and a few scattered spines. The leaves are simple, opposite or whorled and oval-shaped. The margins are serrate.

The flowers born in flat-topped clusters, are small, tubular and white, yellow, orange, red or purple in color. The fruits are produced in clusters and turn black when ripe.

Gastrointestinal

Acute cases develop hemorrhagic diarrhea. Chronic poisoning results in constipation

Cardiovascular system

Rapid heart rate

Respiratory System

Difficulty in breathing.

Integumentary System

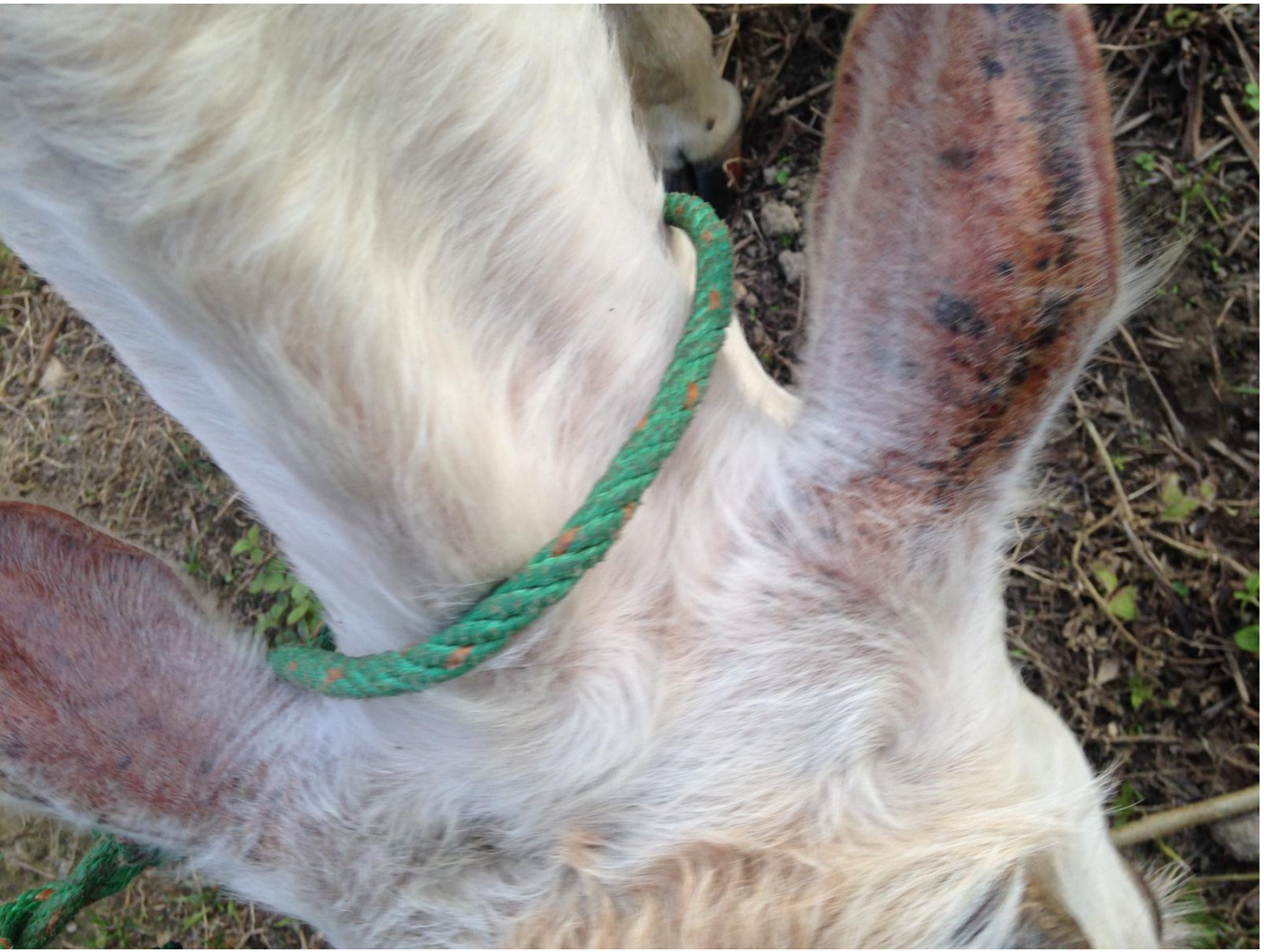
Photosensitization, especially of the white skinned areas.

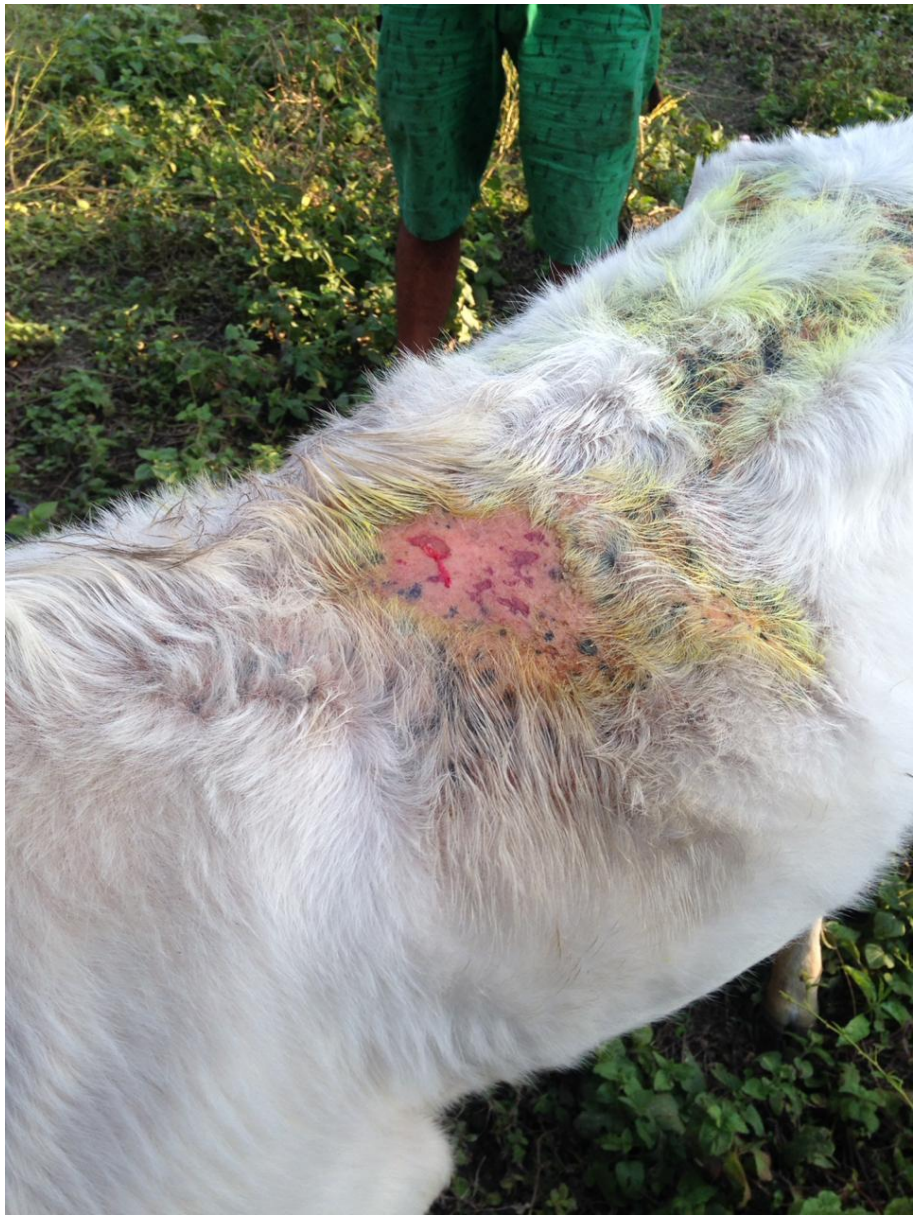
Ocular System
Conjunctivitis.

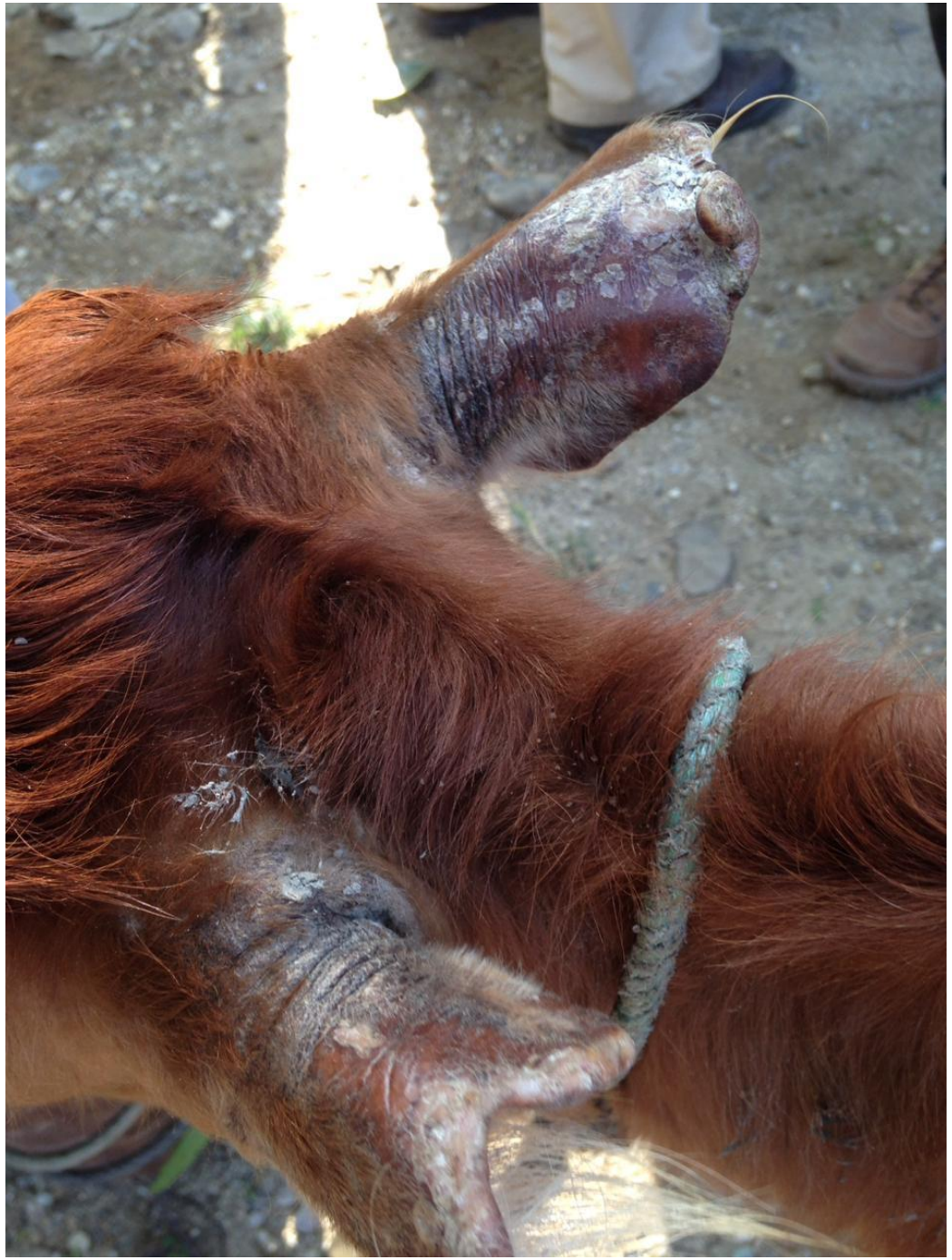
Hepatic System
Bile duct canaliculi are affected causing bile stasis and resulting jaundice. death results after animals become severely emaciated.

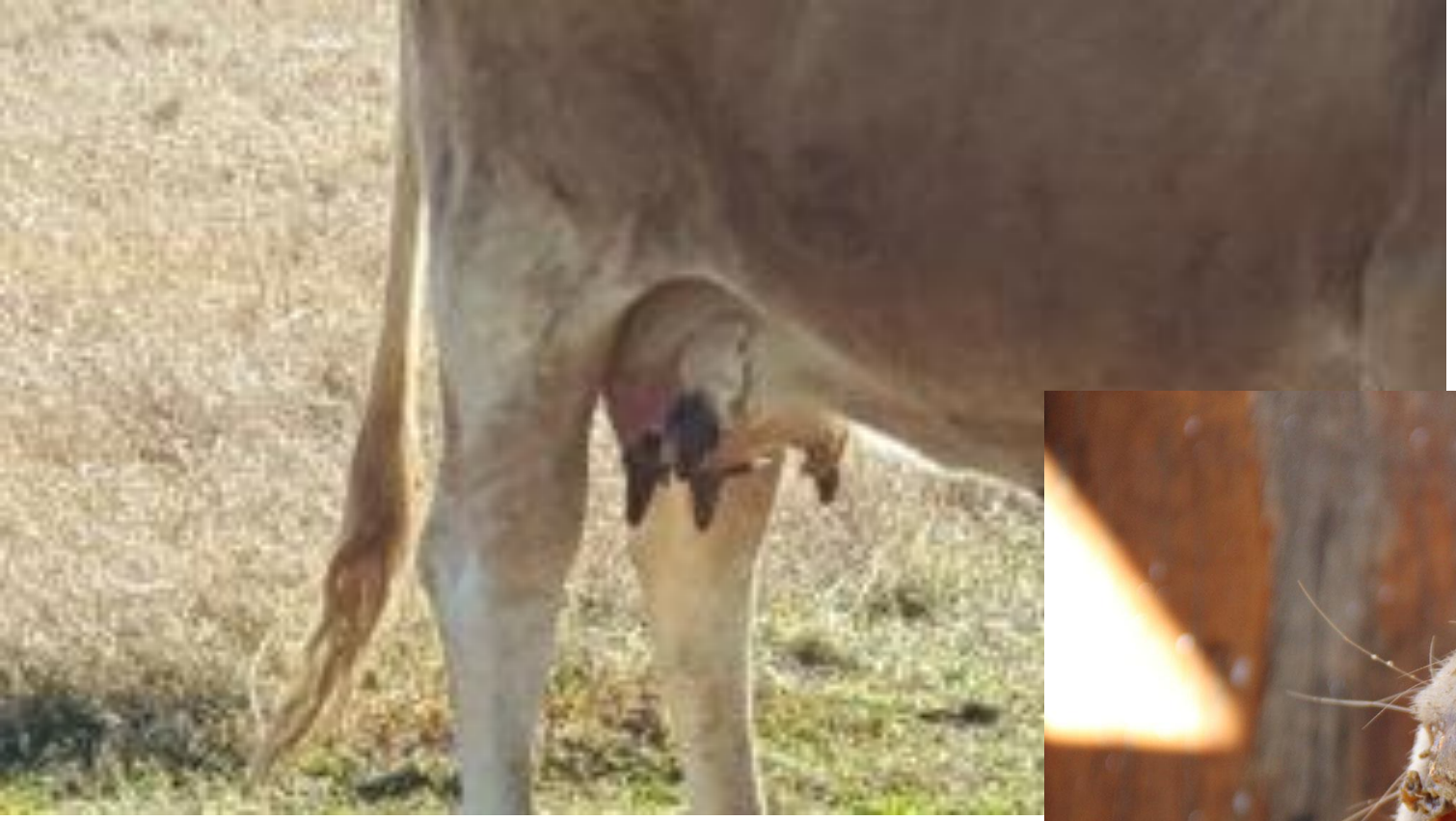
Diagnosis
Elevated liver enzymes and bilirubin

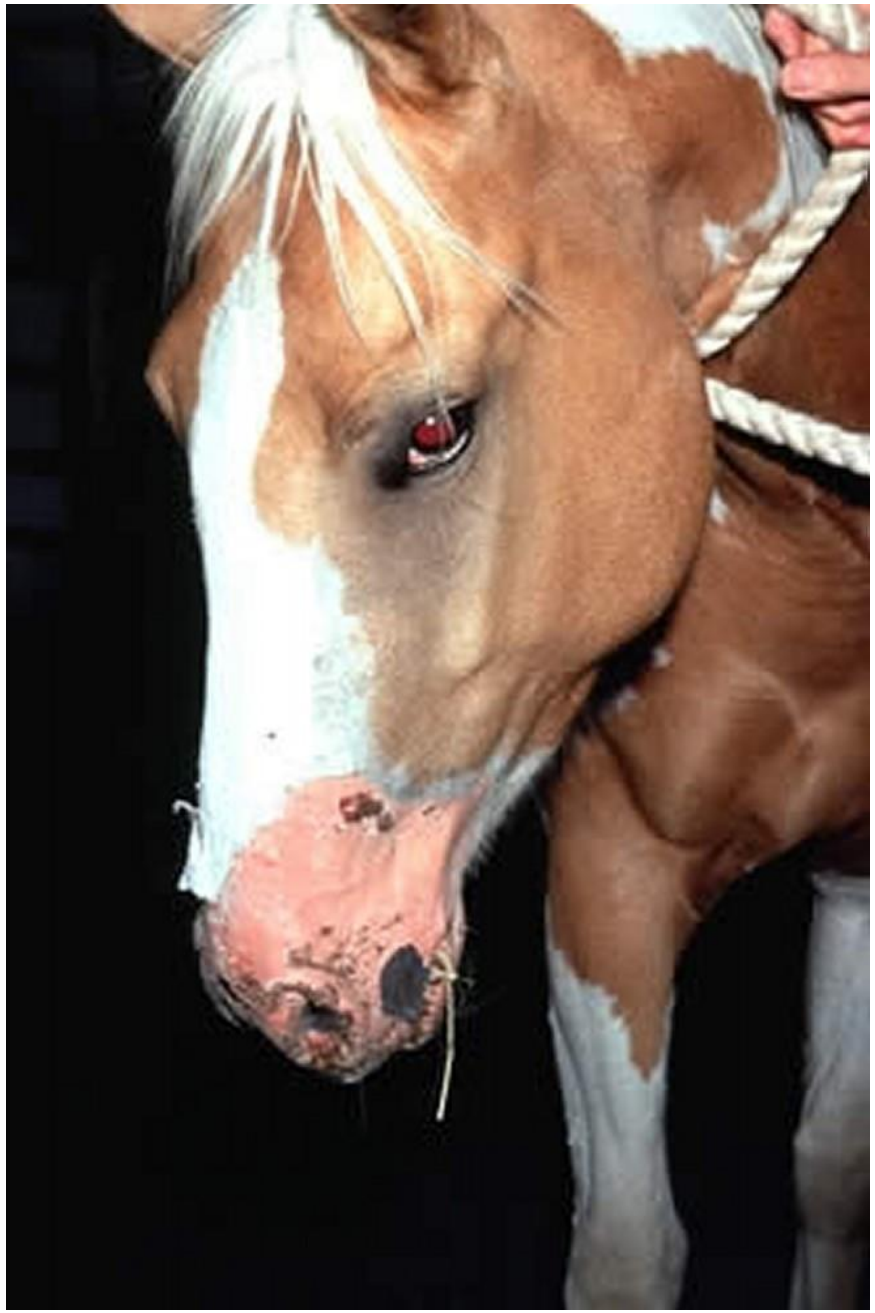
Special Notes
Lantana is an invasive weed that should be vigorously controlled by herbicides or grubbing.











Lantana Treatment

Activated charcoal

cattle - 2.5 kg in 20L water; small ruminants – 500g in 4L water

activated charcoal is effective but expensive

a second dose may be required 24 hours after the first.

Bentonite can be substituted for activated charcoal

it is not as effective, taking up to two days longer

- a more economic treatment of large numbers

use the same dosage as for charcoal in a slurry with water.

the prognosis is good, with rapid treatment

delayed treatment decreased the prognosis

kidney function may be seriously damaged.

Supportive therapy to prevent dehydration

Prognosis good if treated quickly