Common Poisonings in Ruminants

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A **poison** is a substance that has a noxious effect on living organisms. ... A **toxin** is a **poison** produced by a living organism. A venom is a **toxin** injected from a living organism into another. A venom therefore is a **toxin** and a **toxin** is a **poison**, not all **poisons** are **toxins**, not all **toxins** are venoms.

Lecture Plan

Nitrate Poisoning

Oxalate Poisoning

HCN Poisoning

Accidental Lead Poisoning

Ergot Poisoning

Toxic Flurosis

Nitrate Poisoning

- Nitrate in itself is not toxic to animals, but elevated levels
- Nitrates normally found in forages are converted

  $NO_2 \rightarrow NH4 \rightarrow$ Protein

  Nitrite is 10 times as toxic to cattle as nitrate.
- Source – Feed and Water
- Toxicity may be chronic or acute.
- In chronic cases, a sublethal dose may result in abortion, weight loss, reduced milk production and other animal performance issues.
- In acute cases, methaemoglobin formation, respiratory signs and anoxia

Nitrate Poisoning

- Sheep and cattle are more susceptible than goat - because rumen microflora
- Plants fertilized (for seeds) have higher nitrate levels than nonfertilized
- Influencing factors: moisture conditions, soil conditions and type of plant.
- Plant stressors, such as drought, are associated with increased levels of nitrate in plants.
- Soils high in nitrogen readily supply nitrate to plants.
- Additionally, factors such as acidic soils, sulfur or phosphorus deficiencies, low molybdenum and low temperatures are known to increase nitrate uptake by plants
- **Weather – following the first rain after drought**

Crops / Weeds with High Nitrate Content

- cabbages, turnips, sugar beets, lettuce, potatoes, and carrots Tomato
- Millet, Oat, Soybean, Sorghum, Sudan Grass, Sweet Clover, Wheat
- Tomato- Solanine

Growth Stage – Young plant has more nitrate

Leaves, stem and seed have least nitrate

Part near the root – 6 inches – has more nitrate

Presence of moisture helps in nitrate accumulation

Cabbage Poisoning

- Brassica family
- Rich in nitrate / calcium / soluble carbohydrate
- Toxin- glycosinolates, which interfere with the utilisation of iodine in ruminants and cause reduced growth rate and milk yield
- Rumen acidosis as it contains high amount of soluble sugar
- Heinz body anaemia- Due to oxidation of haemoglobin (similar to onion)
- Cabbage is also rich in calcium- reduces absorption of calcium and milk fever, osteoporosis and weakness in hind limbs

Detection of Nitrate in Forages

- Diphenylamine test:
- 0.5 g diphenylamine + 20 ml DW + 88 ml concentrated sulphuric acid
- Put 3 drops of the reagent on the forage- the stalk cut open
- Wait for few seconds
- If blue colour develops positive for nitrate
- Safe level is 0.226%

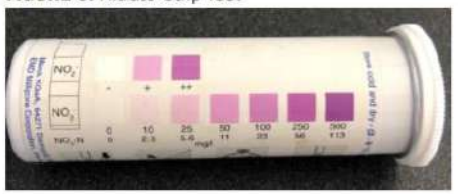
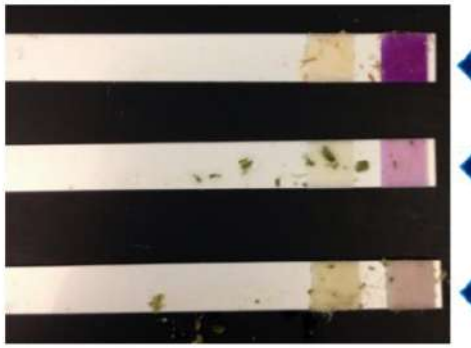


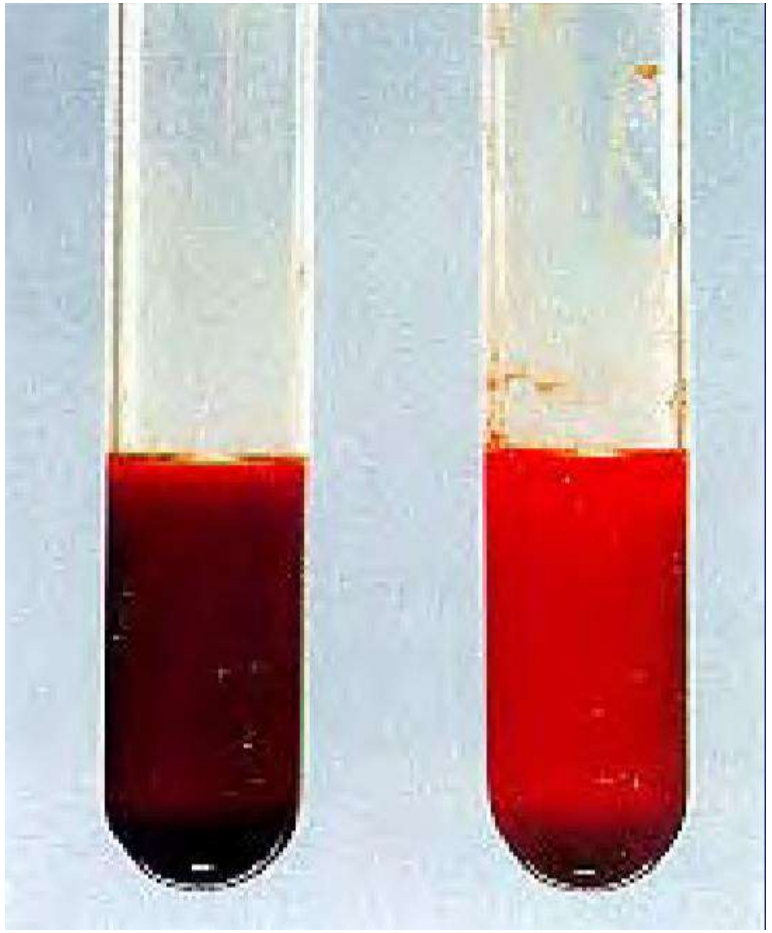
FIGURE 4. Strips that have been used to test plants for nitrates



Species	Nitrate (mg/L NO_3)	Feeding Recommendation
Barley	50,000	DO NOT feed to any livestock
Corn	10,000	DO NOT feed to pregnant livestock
Alfalfa	1,000	Safe to feed to livestock

Clinical Presentation

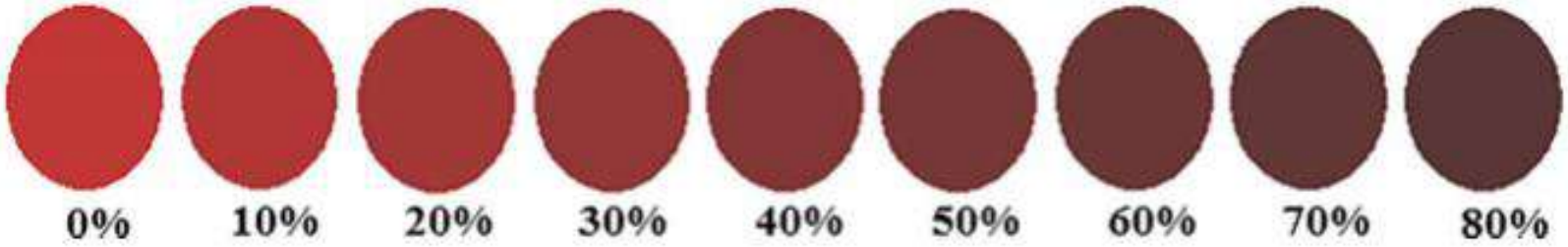
- • bluish/chocolate brown mucous membranes
- • rapid/difficult breathing
- • noisy breathing
- • rapid pulse (150+/minute)
- • salivation, bloat, tremors, staggering
- • weakness, coma, death
- • dark “chocolate-colored” blood
- Pregnant females that survive nitrate poisoning may abort due to a lack of oxygen to the fetus.
- Abortions generally occur approximately 10 to 14 days following exposure to nitrates.



Sub-clinical Nitrate Poisoning

- Reduced reproductive efficiency in adult cattle
- Lower weight gains with or without decreased feed intake in young stock.
- Research has shown that serum progesterone concentrations may be decreased greatly in open animals (50%) more moderately in early pregnancy (25%), and very little, if any, in mid- or later pregnancy.
- Services per conception and first service conception rates may be most noticeably affected, resulting in more repeat breeding
- Check fodder / forages for nitrate content / blood for methaemoglobin

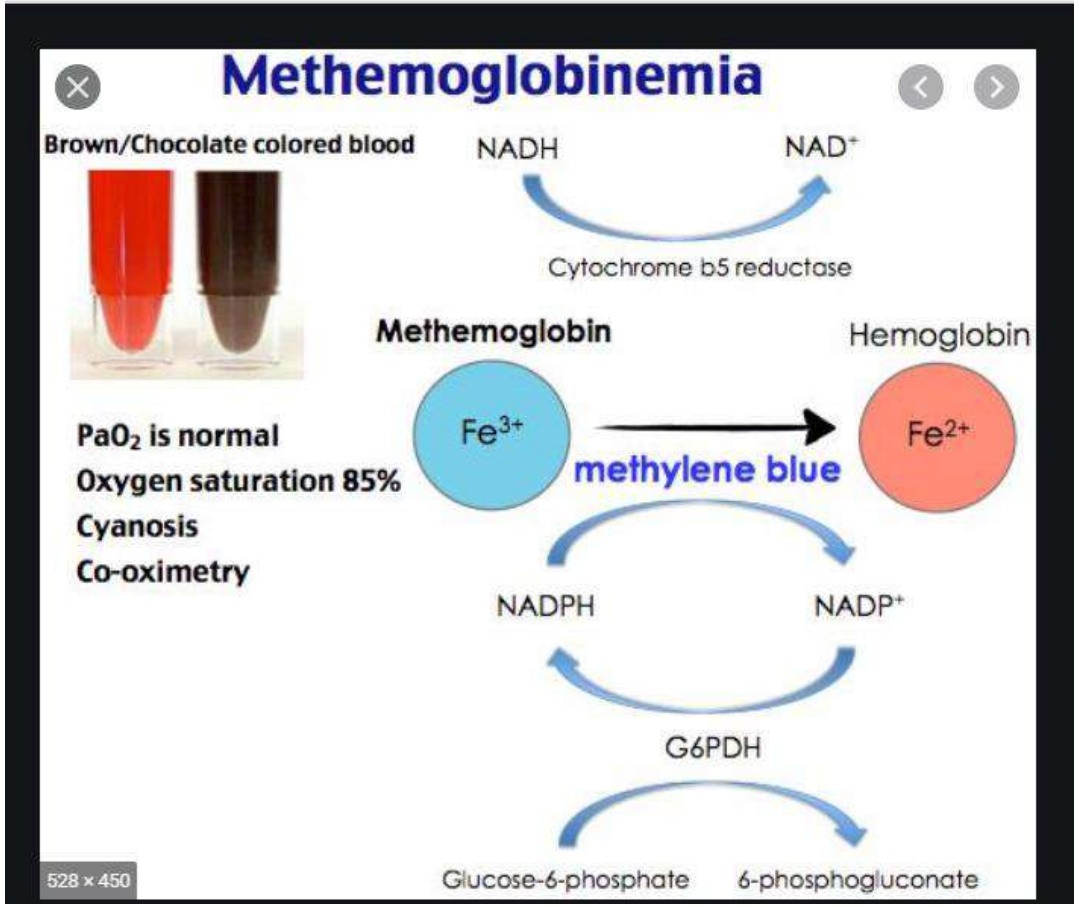
Field Test for measurement of MeHb in Blood (www.indiancattle.com)



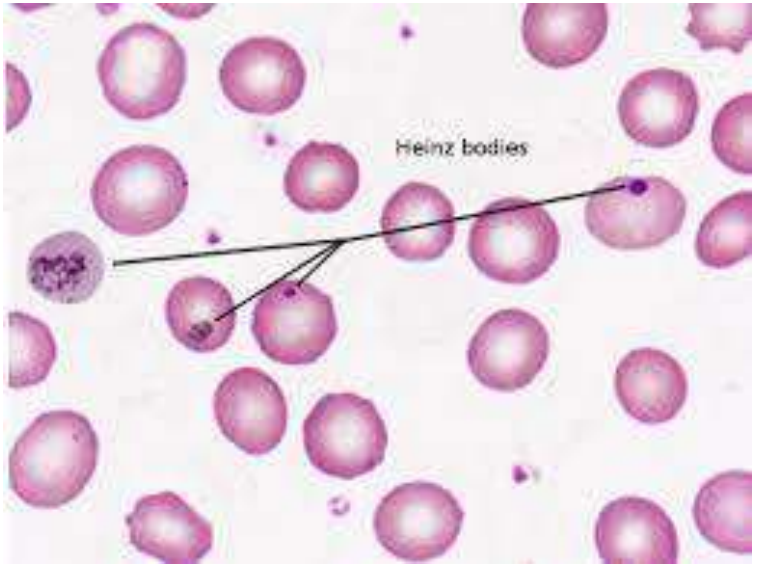
- Measuring MethHb in blood is the direct diagnostic method to decide on onset and cessation of the treatment
- It must be done immediately after collection else results are not accurate.
- Samples to be sent to the laboratory require strict conditions of packing and transport- difficult to follow
- There are spot tests available for field-level diagnosis
- Put a drop of blood on a white filter paper and scan the same using a colour scanner App
- The App gives tentative range of methaemoglobin in blood

Treatment of Nitrate Poisoning

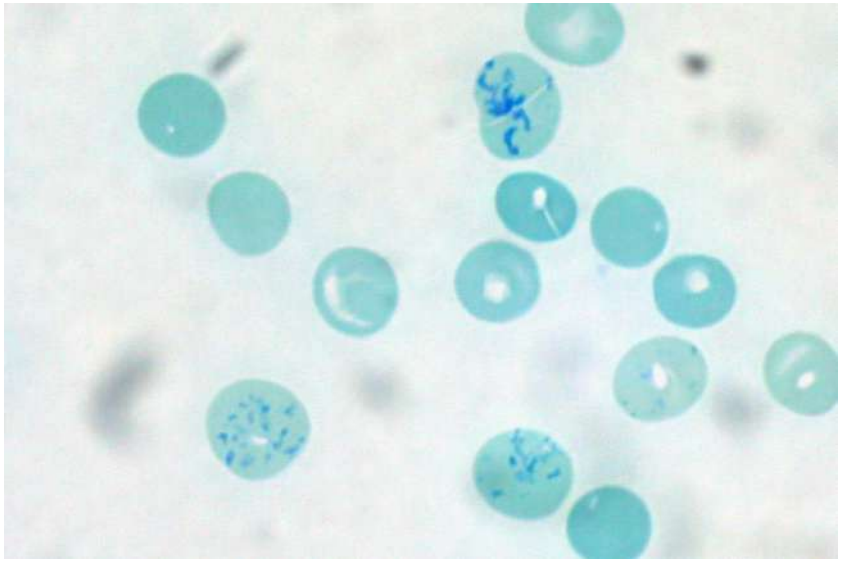
- **Methylene blue Injection**
- (1%) 1 to 2 ml per kg body weight to be given i.v. slowly (Pravon Blue- Avalon Pharma, Mumbai and Flagship Biotech International Pvt. Ltd. Thane)
- Supportive therapy – Diuretic and fluids to clear nephrons
- Removal of the source of nitrate
- Replacement of rumen flora and oral vitamin B. complex to support rumen flora growth (Yeast- media concentrate)
- Novizac (Intas)-Pyruvate and nucleotide with buffered phosphate



Onion Poisoning



- Alkaloid N-propyl disulphide, S-methyl and S-prop(en)yl cysteine sulphoxides (SMCO)
- Rumen microflora can degrade these alkaloids
- Overfeeding usually > 25% of the feed
- Sheep and goats are resistant – even pregnant animals can be fed up to 75%
- **Symptoms:** Constant vocalization, Weakness and haemolytic anaemia , Heinz bodies in blood, abortion, staggering, difficult breathing, hemoglobinuria
- **Treatment** – Novizac (Intas) it contains sodium pyruvate along with buffered phosphorus



General Approach to Treatment of Poisoning

- **Intravenous Dextrose** – 50% 1-4 ml per kg body weight
- I.V. fluid to maintain hydration and induce diuresis
- **Ringers Lactate** – advisable when metabolic acidosis is suspected- do not give with ceftriaxone, citrated blood transfusion
- **Nor epinephrine** 0.5 – 1.0 mg / minute with continuous infusion to combat hypotension (check pulse specially of extremities, cold extremities)
- **Sodium bicarbonate injection-** Rapid and shallow breathing- (Acidosis) Bicarbonate infusion (8.4%) to combat systemic acidosis, 2.5 – 3.5 ml per kg body weight – do not mix in calcium injection
- **Activated charcoal (Carbon)** – Ineffective in small ion toxicity such as cyanide, iron, metals, etc. – This only adsorbs –
- **AC- EDA:** Activated carbon modified with ethylenediamine
- **AC- TETA**: Activated carbon modified with triethylenetetramine

General Antidotes

- **Atropine sulphate-** Is indicated only in organophosphate and carbamate poisoning (0.01 – 0.02 mg / kg)
  - 30 mg / 45 kg body weight to be give slow intravenous 1/3rd i.v. and rest i.m. (t1/2 is 2.5 hours)
  - Tachycardia, arrhythmias, syncope, mydriasis, abdominal pain, tachypnoea, slow respiration, fever
- **Glycopyrroline bromide Pyrolin (Celon Pharma), Vagolate (AHPL)**
  - better option (0.2 mg / ml) dose 0.002 to 0.004 mg per kg
- **Dimercaprol** (British Anti lewisite-BAL)- (Samarth Lifesciences) – 4 mg per kg repeat after 4 hours, but check diuresis. Lead, arsenic, mercury poisoning, daily for 5 days
- **Ca-EDTA (chelating agent)** Livodate-Ca (Liv Bio Pharma)- 200 mg/ ml, 50 mg / kg per day
- **Calcium borogluconate** should be given only when hypocalcaemia is suspected- Fluorine toxicity - Ca-EDTA is an antidote

Supportive therapy

- **Corticosteroids:** limited by the potential side effects, such as prolonged neuromuscular weakness, deregulation of glucose metabolism, superinfection, and sepsis, which could diminish the chances for recovery
- **Diuretic- Furosemide:** 0.5-1 mg per kg body weight max 2g in cows / buffaloes, if no diuresis in six hours stop- preferably give i.v. slow
  - Check for hypokalaemia and hypomagnesemia – former very critical
  - Do not use gentamicin, amikacin and streptomycin when using furosemide.
- **Anticonvulsant** – Daizepam or Midozolam (0.02 to -0.1 mg i.v.)
- **Phenytoin sodium 10-15 mg / kg body weight** to prevent seizures

HCN Poisoning

- Acute poisoning:
- Chronic:
- Hypothyroidism due to disruption of iodide uptake by the follicular thyroid cell sodium-iodide symporter by thiocyanate, a metabolite in the detoxification of cyanide, and
- Chronic cyanide and plant cyanide metabolite (eg, various glutamyl β-cyanoalanines) – associated neuropathy cystitis ataxia syndromes in cattle, sheep, and goats).

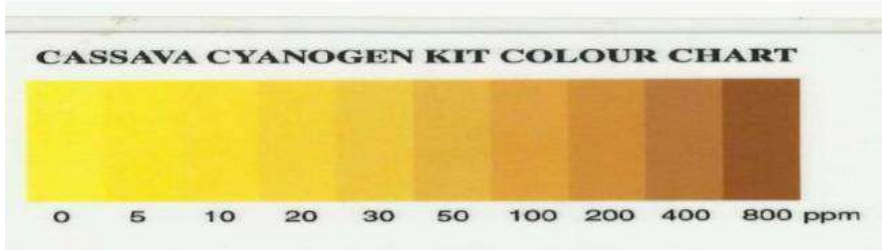
HCN Poisoning

Source:
Sorghum
Casava
Apricot, cherry
Pannacum maximum
Pennicetum clandestinum
Aspergillus niger

The plants are considered as toxic if
they contain over 200ppm of these glycosides

Detecting cyanide in plants

- Prepare aqueous solution of 0.5% picric acid in 5% sodium bicarbonate – Dip the filter paper in the solution and allow it to dry
- Moist the paper before use
- Fiegel-Angel Paper Method:
  - Tetramethyldiaminophenyl methane
  - Methylenebis
  - Copper ethylacetate
  - Each 1 g and dissolve in 100 ml chloroform (Freshly prepared)
- Dip the paper in the above solution and dry in darkness away from light. Before using the material or the paper should be minimally moistened Plae blue turns into Blue-green when positive



Clinical Signs

- Acute: Signs generally occur within 15–20 min to a few hours after animals consume toxic forage, and survival after onset of clinical signs is rarely >2 hr.
- Excitement can be displayed initially, accompanied by rapid respiration rate.
- Dyspnoea follows shortly, with tachycardia.
- Salivation, excess lacrimation, and voiding of urine and faeces may occur
- In other cases, sudden unexpected death may ensue.
- Mucous membranes are bright red but may become cyanotic terminally.
- Venous blood is classically described as "cherry red" because of the presence of high venous blood pO_2; however, this color rapidly changes after death.

Cyanide Poisoning in Ruminants

- Unfortunately, symptoms of prussic acid (cyanide) poisoning are similar to nitrate toxicity.
- When sorghum, sudangrass, or their hybrids, Johnson grass, or pasture containing access to cherry are involved in an acute problem, one must determine whether nitrate or prussic acid is involved.
- Freshly drawn blood from an animal afflicted with nitrate poisoning often is a dark chocolate-brown color, while that from prussic acid toxicity often may turn a **brilliant cherry red upon exposure to air.**
- Forages may be tested for nitrate and prussic acid (cyanide) to help with later confirmation.

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Chronic Cyanide Poisoning

- Chronic cyanogenic glycoside hypothyroidism will present as hypothyroidism with or without goitre
- Cystitis, ataxia are typically associated with posterior ataxia or incoordination that may progress to irreversible flaccid paralysis
- General health and productivity is lowered
- Abortions are common
- Cystitis secondary to urinary incontinence, and hindlimb urine scalding and alopecia.
- Death, although uncommon, is often associated with pyelonephritis. Late-term abortion and musculoskeletal teratogenesis may also occur.

Treatment of HCN poisoning

- **Amyl nitrite - Inhalation**
- **Sodium Nitrite (SN) 20%** and **sodium thiosulphate 20%**- (500 mg / kg body weight) Sodium nitrite causes cardiac problems, sodium thiosulphate leads to hypotension – Sodium nitrite slow to act

  (Hope Pharmaceuticals)
- **4 - Dimethylaminophenol**: Aminophenols. 4-dimethylaminophenol (DMAP)-@ dose of 3.25 mg/kg. i.v. of DMAP – 30% methaemoglobin within 10 min and 15% methemoglobinemia was attained within one minute

Treatment Continued

- **Hydroxylamine HCl (HA)**- Another antidote under clinical trials 50 mg / kg – also takes care of convulsions
- **SN+ DMAP or SN+ HA** are found to be superior
- **Thiosulphate, 100-500 g orally-** can be given oral to bind residual cyanide glycosides
- **Chlorpromazine**: (Largactil) significantly potentiate the efficacy of SN and STS combination, ability to sustain cellular calcium homeostasis
- **Diltiazim** 0.25 mg / kg body weight as bolus in 2 minutes, repeat after 15 minutes @ 35 mg / kg
- **Naloxone** (0.4 mg / ml)- in adult cattle up to 2 mg i.v.
- **Hydroxocobalmin** (Vitmain B12a)- 1g / ml- Maya Biotech Pvt. Ltd.
- Dose- 20 mg / kg body weight
- **Cobalt-edetate –** 1.5-3.00 g i.v.
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Oxalate Poisoning (Mycotoxins)

- The poisoning can occur both due to high oxalate content of plants or contamination with fungus- high in oxalate
- Rice straw, Panicum maximum (Guinea grass), Pennisetum clandestinum
- Oxalis
- Setaria, Halogeton, Pigweed, amanthus
- Napier Bajra Hybrid – Oxalate content high 4% after the first cut, sown in early summer accumulates more oxalate
- Contamination with fungi
- Aspergillus niger- high in exalate

Oxalate Toxicity

- Rice straw stored and wet due to early rains carry Aspergillus mould
- Potential toxicity
- Nephrosis, tremors, osteoporosis
- Renal failure
- Urolethiosis

Plant species and oxalate content

Plant species	Oxalate content (%)	Reference
Napier Bajra Hybrid	3.3-4.0	Kaur *et al.*, 2016
	2.58-5.62	Sidhu *et al.*, 2014
Guinea Grass	1.05-2.40	Garcia-Rivera and Morris (1955)
Pearl millet	0.63-1.98	Kaur *et al.*, 2012
Bathu	3.48-5.98	Sidhu *et al.*, 2014
Sorghum	0.92-1.70	Sidhu *et al.*, 2014
Setaria	2.61-4.98	Katoch, 2013
Napiergrass	3.24-4.78	Rahman *et al.*, 2006
Kikuyu grass	0.4-2.4	Marais (1997)
Baffel grass	1.3	Rahman *et al.*, 2013
	1.2-2.2	Playne (1976)

Napier-Bajra Hybrid and Oxalate Toxicity

- Napier-Bajra Hybrid is known to be rich in soluble oxalate and cause toxicity signs
- Aged plants have more oxalate
- Nitrogen fertilizers increase oxalate content
- In hot and humid weather oxalate content is increased
- It should be preferably sown during later winter and avoid in early summer
- Contamination with fungus increases oxalate to > 10%
- No3:NH4 35:65 ideal- less oxalate

Clinical Signs and Treatment

- Ataxia, incoordination, bloat, rumen impaction, hypocalcaemia, no rumen activity, colic, weakness of legs
- Blood clotting Time - > 12 minutes (when serum calcium is less than 4 mg / dl)
- **Treatment**

Calcium borogluconate – 350-450 ml slow i.v. **(half s/c)**

Dictionic calcium – 20 ml i.m.

- **Penicillamine- Dose** 5-7.5 mg / kg 4 times a day max 2 g
- **Thiamine:** 1 g i.v. with or before glucose
- **i.v. fluid with nor-epinephrine- If severe hypotension** 0.5 mg / minute

Oxalate Toxicity – Treatment

- Magnesium sulphate orally
- Oral Calcium salts to bind excess oxalate
- Hydration – enough water to drink
- Replace the rumen flora from healthy cattle / goat
- Dicationic calcium is better as a follow up
- Replace rumen microflora

Lead Poisoning

- Even a small amount of lead can kill cattle.
- Cattle will readily drink crankcase oil, lick grease from machinery and chew on lead plumbing and batteries.
- Other frequent causes of poisoning include flaking high lead paint, ash from fires in which lead materials were burnt, lead shot from shooting.
- The lead in these materials settles in the stomachs of cattle, where stomach acids gradually change the lead into poisonous salts.
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Clinical Signs

- Although clinical signs of poisoning normally precede death, most animals are simply found down or dead on the pasture. Symptoms include:
- Depression
- Loss of appetite
- Occassionally diarhhea
- Apparent blindness
- Odd behaviour including grinding teeth, bobbing head, twitching eyes/ ears
- Frothing at mouth
- Muscle tremors
- Staggers
- Excitable
- Convulsions

Treatment

- **Dimercaprol (BAL):** 4 mg per kg body weight repeat every 4 hours, next day every 8 hours
- **Thiamine hydrochloride** (vitamin B1) can reduce the effects of lead on the central nervous system.
- Drenching with small amounts of **magnesium sulfate** (Epsom salts) may also help to reduce absorption of lead from particles held in the reticulum of cattle, sheep and other ruminants.
- These relatively low-cost treatments may improve the survival rate of clinically affected animals.
- **Edetate calcium disodium** 3%, 25-35 mg / kg dep i.m. or i.v. every day for 5-7 days
- Treating poisoning cases is arduous, tiring and require time investment but professionally rewarding
- Please post your queries on www.indiancattle.com or my email address: drasamad@hotmail.com

- THANK YOU

Degnala Disease in Buffaloes

- As a consequence symptoms like drying and necrosis of tail and ear tip, swelling of legs and then drying, necrosis and gangrenous lesion on foot, reduction in milk production by 70-80% were observed in buffaloes fed to such straw. In Nepal such conditions were reported from districts like Jhapa, saptari, siraha, Rautahat, Chitwan, nawalparasi, Kathmandu, Nuwakot,Banke and Lamjung. The vet doctors and technicians working in such areas were treating such condition diagnosing as FMD, Black Quarter etc but without success

Fescue Toxicosis

- Ergotism is a worldwide disease of farm animals that results from ingestion of sclerotia of the parasitic fungus *Claviceps purpurea*, which replaces the grain or seed of rye and other small grains or forage plants, such as the bromes, bluegrasses, fescues, and ryegrasses. The hard, black, elongated sclerotia may contain varying quantities of ergot alkaloids, of
  which ergotamine and ergonovine (ergometrine) are pharmacologically most important. Cattle, pigs, sheep, and poultry are involved in sporadic outbreaks, and most other species are susceptible. Poisoning can come from grazing seed heads or from infected grains in concentrate rations.

- Ergot causes vasoconstriction by direct action on the muscles of the arterioles, and repeated doses injure the vascular endothelium. These actions initially reduce blood flow and eventually lead to complete stasis with terminal necrosis of the extremities due to thrombosis. A cold environment predisposes the extremities to gangrene. In addition, ergot also causes stimulation of the CNS, followed by depression. Ergot alkaloids inhibit pituitary release of prolactin in many mammalian species, with failure of both mammary development in late gestation and delayed initiation of milk secretion, resulting in agalactia at parturition. Ergot alkaloids have also been associated with heat intolerance, dyspnea, and reduced milk production in dairy cattle, similar to the “summer syndrome” described for fescue toxicosis.
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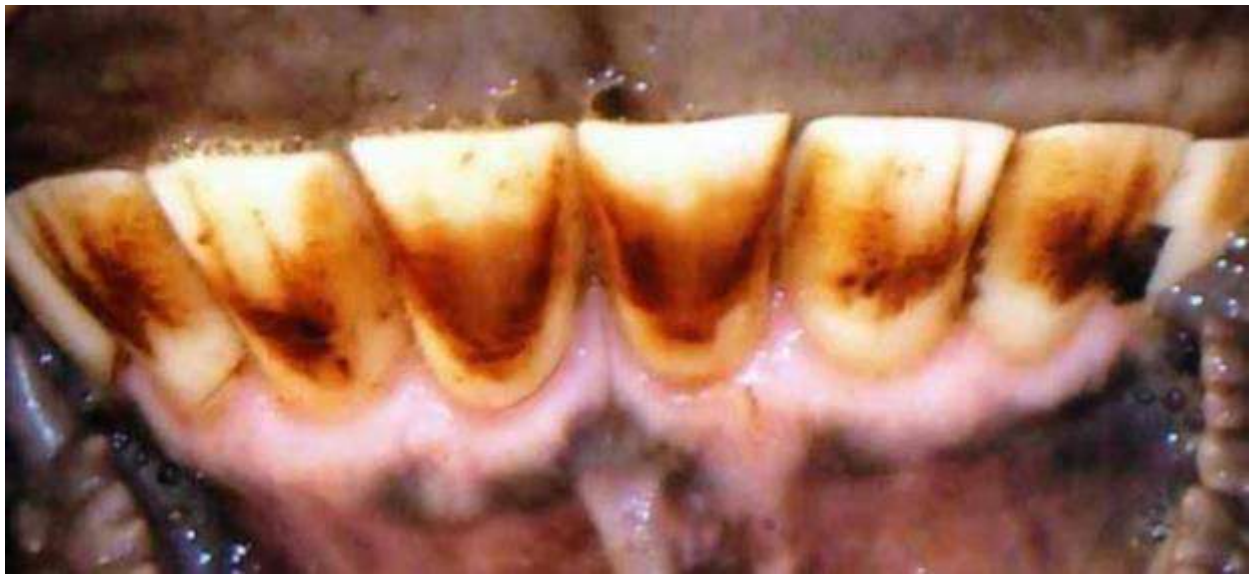
- Cattle may be affected by eating ergotized hay or grain or occasionally by grazing seeded pastures infested with ergot. Lameness, the first sign, may appear 2–6 wk or more after initial ingestion, depending on the concentration of alkaloids in the ergot and the quantity of ergot in the feed. Hindlimbs are affected before forelimbs, but the extent of involvement of a limb and the number of limbs affected depends on the daily intake of ergot. Body temperature and pulse and respiration rates are increased. **Epidemic hyperthermia** and hypersalivation may also occur in cattle poisoned with *C purpurea* (Also see Fescue Poisoning). Ergot alkaloids may interfere with embryonic development in pregnant females.
- Associated with the lameness are swelling and tenderness of the fetlock joint and pastern. Within ~1 wk, sensation is lost in the affected part, an indented line appears at the limit of normal tissue, and dry gangrene affects the distal part. Eventually, one or both claws or any part of the limbs up to the hock or knee may be sloughed. In a similar way, the tip of the tail or ears may become necrotic and slough. Exposed skin areas, such as teats and udder, appear unusually pale or anemic. Abortion is not seen.
- There was a significant decrease in the diameter of arteries and veins servicing the ovary and uterus on day 10 and 17 of the estrous cycle. Reduction in blood flow to the reproductive organs during critical times in the estrous cycle may contribute to the reduced ovarian function and pregnancy rates associated with fescue toxicosis.

e Nalgonda defluoridation

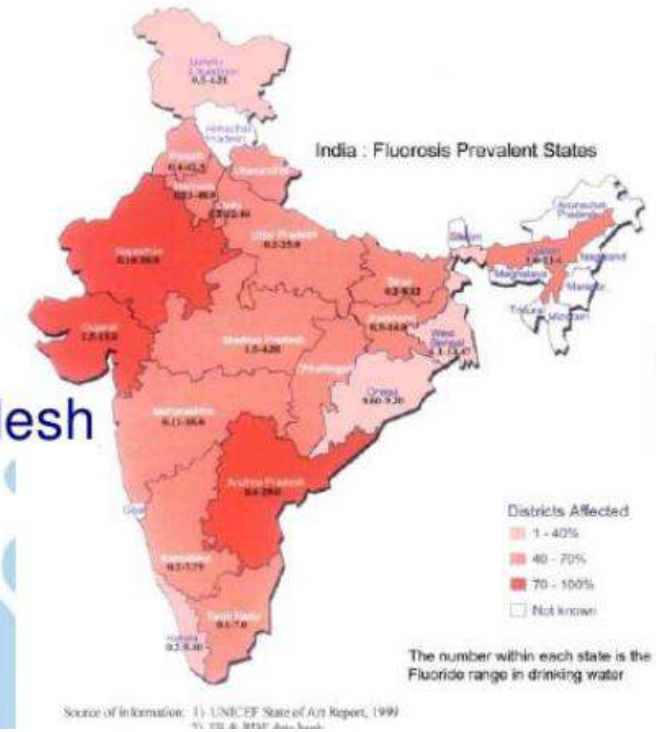

Photo courtesy of Dr. Steve Ensley.



Fluoride Contamination -India

Most Affected:

- Rajasthan
- Gujarat
- Andhra Pradesh



Source of information: 1). UNICEF State of Art Report, 1999