animal only indicates that the animal was exposed to the pesticide, not that the insecticide produced a toxicosis. On the other hand, failure to find cyanide in body tissues would not guarantee that an animal had not been poisoned by such a chemical.

In summary, it is imperative that a thorough history be obtained, that careful observation be made, and that intelligent questions be asked.

The veterinarian should apply the professional skills that only he possesses in determining signs of illness and in performing a thorough post mortem examination. Properly prepared tissue specimens and other suspected material should be sent without undue delay to a qualified laboratory for chemical and histopathologic examination. All information that can be obtained regarding the case should accompany the specimens to the laboratory. Cooperation and communication between laboratory personnel and the diagnostician will result in the highest usefulness of diagnostic procedures.

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Feed and Water Related Toxicants

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Feed and Water Related Toxicants

Rumensin

Rumensin is a biologically active compound produced by *Streptomyces cinnamonensis*. The compound is soluble in most organic solvents and is only slightly soluble in water. It was first introduced as an aid in the prevention of coccidiosis in poultry. Subsequently, Rumensin was found to increase propionic acid production in the bovine rumen. By so altering rumen fermentation, a greater proportion of dietary energy was retained, resulting in increased feed efficiency of feed lot cattle. This activity led to the introduction of monensin as feed additive for cattle. Inasmuch as horses may be exposed to feed containing monensin, studies have been conducted to determine what effect monensin might have on horses.

Toxicity

Seventy-two cattle were fed Rumensin at levels up to 100 g/ton for 160 days and no changes indicative of toxicity were observed for hematology, serum chemistry, urinalysis, or organ weight parameters. No gross or microscopic tissue changes relating to treatment were observed. The 100 g/ton level did, however, reduce gain.

Oral single doses of Rumensin fed to cattle has caused mortality at 55 mg/kg of body weight which is 80 times the recommended dose of 30 g/ton. However, no mortality ocurred at the 30 g/ton level.

The evidence has accumulated that feed containing 279 ppm monensin is lethal to horses and that 125 ppm may be toxic but not necessarily lethal. Feed with 31 ppm monensin did not cause any clinical changes, aside from partial anorexia in the horse. The single-dose toxicity studies indicate the LD50 of monensin for horses is between 2 and 3 mg/kg of body weight.

Organic Iodides

An organic iodide, ethylene diamine dihydriodide (EDDI) is recommended as a feed additive for non-dairy cattle. (Feed Additive Compendium 1976) at the following rates.

Table 1 - Recommended Oral Levels of EDDI

Animal	Use Level	Indications
Cattle	50 mg/head/day in feed or salt continuously	Prevent footrot, soft tissue lumpy jaw and nutritional source of iodine
	400-500 mg/head/day for 2-3 weeks (not to dairy cattle in production)	Treatment of footrot, soft tissue lumpy jaw, and mild respiratory infections by action as an expectorant

Toxicity

Toxic signs of EDDI in cattle include an expectorant action and nasal discharge with a mild intermittent, non-productive cough. These signs may occur at dosage levels of 500-1000 mg/head/day. After reduction of dosage levels to recommended levels the signs may disappear except for occasional nasal discharge and mild cough.

Cyanide

Sources of Cyanide

1. Cyanogenic plants: Manihot esculate - cassava
   Prunus spp - chokecherry, plum, peach, apricot
   Sorghum spp - Johnson grass, Sudan grass
2. Cyanide is used in meallurgy, electrolplating, metal cleaning, and chemical synthesis.

Causes of Poisoning

1. Ingestion of cyanogenic plants
2. Adverse conditions that increase cyanide content - frost, drought, wilting, herbicides, and nitrate fertilizers.
3. Inhalation of HCN and absorption through the skin.

Mechanism of Action

Cyanide blocks cellular respiration by binding to the terminal cytochrome oxidase in the mitrochondria. Brain tissue is the most sensitive to oxygen deficit. **Note** - Cyanide does not block cytochrome P_{450}. Cytochrome P_{450} and the terminal cytochrome oxidase are both in the Fe^{+3} state.

Signs

1. Depends on the type of exposure
2. Acutely, death can be in seconds from HCN gas
   a. Usually death occurs in 5-20 minutes
   b. Mucous membranes are bright red
   c. Respiratory and cardiac hyperactivity, sinus arrhythmia, excitement, nystagmus
   d. Incoordination with CNS depression in the sequence of: cortex, basal ganglia, hypothalamus midbrain and recovery in the reverse order.
   e. Convulsions
   f. Death is from centrally mediated respiratory arrest
3. Chronically - motor and sensory ataxia, nerve degeneration, posterior myelopathy, and impaired coordination.

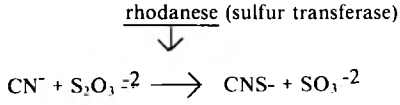
Pathology

1. Neuronal degeneration
2. Multiple foci in the brain
3. Congested and hemorrhagic lungs
4. Congestion of blood vessels, blood unclotted and bright cherry red
5. Reddening and congestion of the rumen or stomach mucosa

Biotransformation

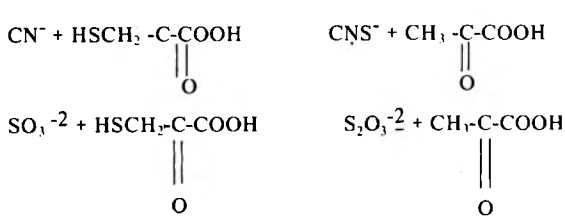
1. Cyanide is detoxified by the liver enzyme rhodanese
2. Rhodanese is present in all tissues
3. Rhodanese can handle large amounts of cyanide but the sulfur stores are quickly depleted.

Cyanide is converted to thiocyanate by rhodanese



Other Reactions

A different transferase



4. Sheep can detoxify 2 mg/kg/hr (HCN)
5. The conversion of cyanide to thiocyanate in low quantities for prolonged periods could reduce thyroid uptake of iodine and produce hypothyroidism.

Toxicity

1. Organic cyanide will induce toxicity only if hydrolyzed to HCN.
2. Ruminants are more susceptible because of the abundance of rumen microflora which produce enzymes that can hydrolyze organic cyanides.
3. The lethal dose is close to the toxic dose of about 2 mg/kg for ruminants.
4. 20 mg HCN/100 g of plant is considered dangerous.

Diagnosis of Cyanide Intoxication

Confirmed by lab exam of: stomach or rumen contents, liver and muscle. The disease must be differentiated from carbon monoxide poisoning.

Treatment

1. 1% solution of sodium nitrite 16 mg/kg
2. 20% solution of sodium thiosulfate 30-40 mg/kg or in combination give .5 ml/kg of 10 g sodium nitrate and 15 g of sodium thiosulfate in 250 ml of solution.
   If treatment is repeated, give only sodium thiosulfate.

$2hb\text{-}Fe^{+2} - O_2 + NO_2^- \longrightarrow 2hb\text{-}Fe^{+3}\text{-}OH + NO_3 + O_2$

$Hb - Fe^{+3} + Cyt\ Fe^{+3} - CN^- \longrightarrow HbFe^{+3} - CN^- + Cyt\ Fe^{+3}$

In mice O^2 or cobaltous chloride can supplement sodium nitrite and sodium thiosulfate treatment. The LD^{50} is increased two-fold over the sodium nitrite and sodium thiosulfate treatment. Cobalt is not as effective in sheep because it must be given in lower doses to avoid toxicity. Oxygen did increase the LD^{50} over sodium nitrite and sodium thiosulfate treatment in sheep.

Prevention

1. Avoid animal exposure to toxic plants containing cyanide
2. Feeding of dietary carbohydrate aids in inhibiting hydrolytic enzymes which act on glycosides.

Nitrite/Nitrate Intoxication

Nitrite/nitrate intoxication is an important cause of livestock toxicosis. It occurs most frequently in sheep and cattle. Ruminant digestion mechanisms increase the susceptibility to high levels of nitrate. The accumulation of nitrate in the plant may be due to a number of factors:

1. Some plants have a tendency to accumulate high concentrations of nitrate naturally.
2. Overfertilization often contributes to the plant storage of excess nitrogen as nitrate.
3. Under the influence of drought or the application of plant hormone herbicides, certain plants will accumulate high levels.

Animals may also be exposed to nitrates through water sources. Nitrates are very soluble in water and may be leached from overfertilized fields or from run off from feed lots.

Nitrite poisoning may occur in any animal. Nitrite is an extremely strong oxidizing agent which is readily absorbed into the bloodstream. The nitrite ion oxidizes the heme iron from the ferrous (Fe++) state to the ferric (Fe+++) state to form methemoglobin. Methemoglobin is incapable of carrying oxygen.

The nitrite ion also produces vasodilation causing lowering of blood pressure which can eventually lead to cardiovascular collapse. Its oxidizing effect may operate on vitamin absorption of carotene, A, E, D, B^{12} and K, converting them to forms unavailable to the animals. The metabolism of vitamin A in the liver may be affected by interfering with the conversion of carotene to vitamin A.

Nitrate poisoning occurs primarily in ruminants due to the conversion of the nitrate ion to the more toxic nitrite form. The reducing environment created by the microflora of the rumen is responsible for the conversion.

The mechanism of action then procedes as previously discussed. The conversion within ruminants does occur but the toxic ion is usually too far down the gastrointestinal tract to be absorbed in any quantity.

The nitrate ion in its original form may also cause toxicity. Irritation of the G.I. tract can result in salivation, vomiting, diarrhea and abdominal pain. Thyroid activity can be depressed by the displacement of the I^- from the gland by nitrate. It can also cause alkalosis from displacement of the Cl^- and dehydration from its diuretic effect. These problems appear after long term, low level intoxication. Exposure to low levels of nitrate over a period of time has shown to produce a decrease in weight gain and feed efficiency, decrease in milk production, poor reproductive capacity and impaired health of the epithelial tissues, however, many of these problems may be due to low planes of nutrition.

Acute poisoning usually occurs within 30 minutes to 4 hours after exposure. Clinical signs usually occur when 30-40% of the hemoglobin has been converted. Death usually results from anoxia or from cardiovascular collapse coupled with anoxia. A sudden death syndrome called "nitrate syncope" results from extreme exertion followed by sudden vasodilation and cardiovascular collapse.

Toxicity

Animal	Lethal Dose
Cattle	.55 gm/kg
Sheep	3 gm/kg
Horses	100 gm/kg

Treatment

Methylene blue is usually administered intravenously to counteract the oxidizing effect of the nitrite. The methylene blue is a reducing agent which converts the methemoglobin to hemoglobin. Within the red blood cell the methylene blue is reduced to leucomethylene blue which, in concert with methemoglobin reductase, reduces methemoglobin to hemoglobin. The recommended dosage for methylene blue administration is 1% methylene blue at 8.8 mg/kg. A sympathomimetic can also be used to combat the effects of the vasodilation.

Water Deprivation - Sodium Toxicosis (Salt Poisoning)

Salt poisoning is a term which is going out of style and being replaced by water deprivation - sodium toxicosis; regretably, the physiopathological entity which it represents is still quite fashionable. The mechanism of intoxication by Na+, an ubiquitous and essential substance in the body, is yet poorly understood and not too much more is known about the syndrome than in 1918 when it was established that NaCl, even in large amounts, is not normally toxic as long as the body's salt regulatory mechanisms are intact and fresh water *ad lib* available.

Sodium salts normally make up more than 90% of the solutes determining the effective osmolarity of extracellular fluid. Sodium is the primary cation which with HCo^{3-} maintains the pH balance of the body and is essential for the preservation of normal cell membrane permeability and irritability. Normal plasma levels may vary slightly by species between 135-145 mEg/l; cerebro-spinal fluid values are normally 130-140 mEq/l. In the dynamic relationship between these two fluids, Na+ diffuses passively from the plasma to the CSF, but can cross back into the plasma only by an energy-requiring process. Dehydration, whether absolute or relative, as when a large dose of a Na+ compound enters the body, causes the plasma Na+ to go up above 150 mEq/l, at times even to 190 mEq/l, resulting in a CSF Na+ value of 145 mEq/l or higher. At levels of Na+ greater than 145 mEq/l there is a marked inhibition of anaerobic glycolysis and thus a decrease in energy production for the extrusion of Na+ from the CSF. It is thought that when the plasma osmolality is returned to normal by the addition of water or the removal of Na+, mainly by the kidneys, the Na+ remains trapped in the CSF, forming an osmotic gradient favoring the influx of water from the plasma to the CSF. The subsequent edema is believed to cause the major manifestations of water deprivation - Na+ toxicosis.

Sodium Chloride toxicosis usually results from faulty animal husbandry practices allowing and insufficient amount of fresh water to be available to animals when demand for it is increased by a high Na+, usually as NaCl, intake. Water troughs may simply run dry or be frozen over, or the water may be contaminated with salt or an unpalatable substance such as antibiotics which prevent the animal from drinking enough. Sometimes, especially with young animals and fowl, the animal may not find the water when moved to new quarters, or it may be too far from their feeding station. Range cattle, unaccustomed to feed supplements or grain, have been salt poisoned by a ration formulated with a high percentage (8-11%) of salt meant to curb the consumption of the more expensive feed when the nearest source of fresh water was half a mile away. This is not expected in the usual case where feed and water are together and the animals frequently alternate between eating or drinking. Salt deprived animals when given unlimited access to a salt supplement, especially if it is loose rather than in a block, may eat enough in a short time to be poisoned. This has happened after the first rain following a long dry spell which made a salt lick too hard for the cattle to get to. Most often animals are poisoned when the usual water supply is inadequate for a ration with an unaccustomed increase in salt, as when the diet is supplemented with bakery by-products.

Ruminants, particularly sheep and goats, are quite resistent to water deprivation - sodium toxicosis (healthy sheep can drink 2% saline as their only water for at least 6 weeks during the summer with only a moderate loss of weight in most animals, but they are not immune to it). The animals show thirst, salivation, diarrhea, which may be bloody, ataxia, and muscular weakness, especially of the hind quarters with knuckling under at the fetlocks. Some animals "goose-step" or become belligerent. Muscle spasms are soon followed by prostration, convulsions, and death. Subtoxic levels of salt in the drinking water can affect ruminant nutrition. As the salinity of the water increases, more is drunk, increasing the rate and volume of fluid flow from the rumen, decreasing the total rumen microflora population. Calorimetric measurements show that this effect is significant if the chow is pelleted rather than chaffed and that the microflora population stabilizes after a few weeks of adaptation.

Urea and Ammonium Ion Intoxication

Introduction

Urea is a non-protein source of nitrogen that has come into use extensively after it was introduced in WWII as a protein supplement for ruminant animals. Efficient management is necessary, however, since it is more economical than protein, its use has substantially increased. Major sources of the supplement come in a variety of forms and formulations.

1. Feed supplements for mixing and blending
2. Range blocks or cubes
3. Urea-molasses combinations
4. Feed urea in dry granular form
5. Feed grade biuret from controlled urea pyrolysis
6. Urea-gelatinized starch (Starea, tradename) from finely ground grain and urea

Protein Equivalency

	N%	Protein Equivalent %
Urea - Pure	46.7%	292
Urea - Feed Grade	45.0	281
Biuret - Pure	40.8	255
Bieret - Feed Grade	37.0	230

Urea supplements are recommended not to exceed 3% of the grain ration or about 1% of the total feed ration; however, recent formulations have exceeded this composition.

Ammonium salts, especially ammonium nitrate, and solutions containing ammonia and anions such as nitrate and phosphate, are common commercial fertilizer components. Ammonium chloride is used to reduce the incidence of urolithiasis in cattle and sheep at respective daily doses of 0.75-1.5 and 0.25 ounces per animal, and as an expectorant in swine. Monoammonium phosphate, and to a lesser extent, diammonium

phosphate have been used as sources of non-protein nitrogen (NPN) in ruminant rations, a number of products containing this type of compound an NPN are available as follows:

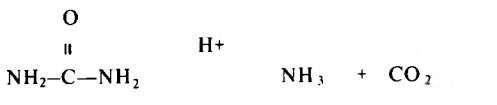
1. Diammonium phosphate - contains at least 17% nitrogen and 20% phosphorous. It may be used in ruminant's feed as a source of this nutrients in amounts that supply not more than 2% of equivalent crude protein in the total animal diet.
2. Ammonium polyphosphate solution - this product is commonly used to add nitrogen and phosphorous to liquid feed supplements. It must contain not less than 9% nitrogen and 13% phosphorous, and must be used to supply not more than 2% of equivalent crude protein in the total animal diet.
3. Ammoniated rice hulls - obtained by the treatment of ground rice hulls with monocalciumphosphate and anhydrous ammonia under conditions of temperature and pressure. This product can be used in beef cattle feeds as a source of NPN and fiber at a level not to exceed 20% of the total ration.
4. Ammoniated cottonseed meal - obtained by the treatment of cottonseed meal with anhydrous ammonia under pressure. It can be used in the feed of ruminants as a source of NPN in an amount not to exceed 20% of the total ration NPN.
5. Ammonium sulphate - results from the neutralization of sulfuric acid with ammonia. It must contain not less than 21% nitrogen and not less than 24% sulfur. It can be used in ruminant feeds as a source of these two nutrients in an amount that supplies not more than 2% of equivalent crude protein in the total daily ration.
6. Monoammonium phosphate - contains not less than 9% nitrogen and 23% phosphorus. It can be used as a source of these two nutrients in ruminant feeds in an amount that supplies not more than 2% of equivalent crude protein in the total daily ration. Table 2 lists the nitrogen, phosphorus, and protein equivalents of this source.

Table 2

Compound	Content N%	Content P%	Protein Equivalent %
Monoammonium Phosphate	12.0	27.0	75
Diammonium Phosphate	21.0	23.0	131
Ammonium Polyphosphate	10.0	15.0	62
Ammonium Sulphate	21.0	0.0	131

Absorption

It is within the rumen that a reducing environment is available to split the urea to ammonia due to action by certain microorganisms.



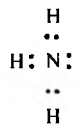
Ammonia is produced from urea in the rumen by the microflora, which incorporate the ammonia into bacterial protein. The protein is then digested in the ruminant's intestinal tract, where it serves as a source of nutrient protein.

Overproduction of ammonia results in the microorganisms inability to utilize all the ammonia. Ammonia poisoning usually results in one to two hours following clinical signs showing within minutes from an excessive amount of ammonia. Urease will also increase the rate of ammonia production from urea, therefore, foods high in urease, urea mixed with soya meal, are extremely dangerous.

pH plays an important part in ammonia poisoning, since ammonia is un-ionized as pH rises above about pH 6.2-7 (alkaline); this form readily passes through the mucosal lining. At low pH levels (acidic) the ammonia is not absorbed since it exists as the ionized species, NH_4^+.

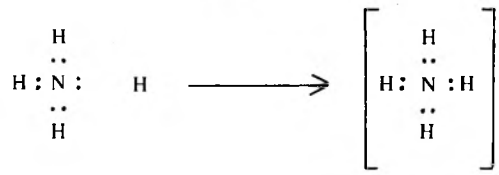
Un-ionized ammonia (NH_3) readily passes cell membranes, ionized (NH_4^+) does not, hence the toxicity increases as the pH value rises. Clearly as the pH increases, ammonia (NH_3) is un-ionized and passes cell membranes quite readily. At the lower pH, ammonia (NH_3) is ionized and does not pass the membrane because $NH_3 + H^+ \longrightarrow NH_4^+$.

Ammonia (NH_3) has four pairs of electrons of which only three are shared, see scheme:



In an acid situation, the H^+ ion is fixed to the NH_3 to form NH_4^+ and so, the positive charge associated with the H^+ ion is retained by the ionic complex (ammonium NH_4) after the union to the ammonia (NH_3):

$NH_3 + H^+ \longrightarrow NH_4^+$ or



Ammonia (Un-ionized) (more absorbed)

ammonium (ionized) (less absorbed)

Excretion

Ammonia is excreted as urea in the urine after it is incorporated into the urea cycle. Small amounts of ammonia are found in the saliva due to the urea in the blood. Unless the animal dies from acute intoxication, ammonia will be rapidly excreted from the body as urea.

Mechanism of Action

Though the animal experiences a rumen alkalosis, it may have a metabolic acidosis which could drop the pH of the blood as much as 0.4 pH units until the time of death. A metabolic alkalosis may also be present in some instances.

Poisoning symptoms appear whenever peripheral blood ammonia nitrogen levels reach 0.84 - 1.3 mg/100 cc (mg%) which usually occurs after rumen ammonia levels reach 80 mg/100 cc (mg%). Severe toxicity has been reported when the blood ammonia level exceeded 0.7-0.8 mg/100 ml.

It is not the systemic acidosis which appears to be the probable cause of death, but rather the inhibition of the citric acid cycle. Compensatory anaerobic glycolysis accompanies this inhibition.

Ammonia is toxic because it leads to the reductive amination of α-ketoglutarate in mitrochondria, catalyzed by glutamate dehydrogenase. $NH_4^+ + \alpha$-ketaglutarate from the tricarboxylic acid cycle and causes both severe inhibition of respiration and excess ketone body formation from acetyl CoA in the liver. One undocumented source (a lab report) suggests that hyperkalemic cardiac blockage and cessation of respiration probably account for termination. The concentration of free ammonia is thus carefully regulated in the liver under normal circumstances.

Toxicity

Toxicity of urea and ammonium compounds may be influenced by several food-related factors. These may include: dehydration or low water intake, lack of natural protein, fasting and high roughage diets with low energy foods. Several body conditions such as high ruminal pH, high ruminal and body temperature, hepatic insufficiency, disease and stress may be other predisposing factors. Lack of adequate time for the animal to become accustomed or adapted to a urea or non-protein diet will contribute significantly to the toxicity.

Adaptation is significant in urea poisoning. Whereas 50 g, sometimes 100-200 g, of urea is usually lethal to cattle, 400 g may produce no ill effect if the dosage is increased slowly. Cattle usually develop or lose tolerance in a few (3) days.

Signs

Signs appear after rumen ammonia levels exceed about 80 mg/100 ml or after blood ammonia levels exceed about 1 mg/100 cc. Signs include colic, groaning, shivering, staggering, bellowing, salivation, grinding of teeth, polyuria, forced rapid breathing, marked jugular pulse and violent struggling followed by death. Ataxia has been shown to occur when the blood ammonia level reaches 2 mg/100 cc. Death occurs when this level equals or exceeds 5 mg/100 cc.

Lesions

Vascular injury and pulmonary edema are usually present from acute toxicity studies. Pulmonary edema is thought to be caused by increased pulmonary permability due to neurogenic-adrenergic factors. There are general hemorrhagic patches of the mucuos membranes on the stomach and intestines. There may be congestion in the liver, however, the blood fluid is generally lighter in color, making the liver pale. The liver is usually friable. Some swelling of the kidney has been reported along with nephritis. One quoted source, Fujimoto and Tajima (1953), observed degenerative changes in the central nervous system.

Diagnosis

Diagnosis should include history of exposure, diagnostic tests and differential diagnoses. There are several diagnostic tests and one feed analysis, which may help in the diagnosis of urea poisoning. These include:

1. Blood ammonia level test (1-4 mg/100 cc)
2. Rumen ammonia level test (80 mg/100 ml)
3. Rumen pH level test (pH 7.5 or greater)
4. Urea level in feed test

If tests cannot be performed within one hour after termination, then the specimen should be preserved with a 1-3% saturated mercuric chloride solution or frozen. These actions are necessary to prevent false positive tests from ammonia due to specimen autolysis, proteolysis and warm temperature.

Differential diagnosis should include the following suspect items, which may cause similar signs and lesions:

- Cyanide poisoning
- Encephalitis
- Enterotoxemia
- Grain engorgement
- Insecticide poisoning
  - Chlorinated hydrocarbon
  - Organophosphate
- Meningitis
- Protein engorgement

Various rumen pH values are often associated with rumen disorders. Several common ones are given:

Condition	Rumen pH
Grain	
Acute overload	3.4 - 4.5
Mild indigestion	4.5 - 5.5
High diet	5.5 - 6.2
Fasting	6.8 - 7.3
Protein engorgement	7.5 - 8.5
Urea poisoning	8.0 - 9.2

Treatment

Treatment should include a weak acid, demulcents and stimulants. Cold acetic acid (2-8 l of 5% acetic acid) or vinegar should be given orally. Three to 10 gallons of cold water may be given prior to the acetic acid solution. Reducing the rumen temperature will decrease the production of ammonia. The excess fluid dilutes the present ammonin-ammonium concentration and also acts as a diuretic. Ergotamine may control some ammonia effects via this adrenergic blocking agent. However, the value of this agent and barbiturates, acting as central nervous system depressants, is questionable. Saline solutions with calcium and magnesium salts are indicated. Bloat should also be relieved.

Major Poisonous Plant Problems In Cattle

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Major Poisonous Plant Problems In Cattle

Animal agriculture plays an important role in the economic well being of the United States. Harvesting of the rangelands by ruminants is one of the main stays of this aspect of agriculture. As a result the occurrence of toxic plants in pastures causes a tremendous economic loss to livestock owners in the form of animal deaths, loss of production of meat and milk, and reproduction problems. In some years it has been estimated in Texas alone that the loss due to cattle deaths may be as high as 24 to 30 million dollars, annually. Therefore, the purpose of this paper is to attempt to describe some of the more common plants toxic to cattle in the United States.

Toxic Plants Affecting the Central Nervous System

Aesculus spp (Buckeyes) - Buckeyes grow in Europe and North America along river bottoms, banks of streams and canyons. It is especially common in the eastern United States. The plant contains several glycosides-notably, aesculin and fraxin. Cattle are affected during early spring as the Buckeyes start to leaf out. The signs seen in cattle intoxicated by Buckeyes are an uneasy staggering gait, weakness and trembling, congestion of mucous membranes, and mydriasis. The animals become depressed and progress on to coma and death. There are no lesions descernible. If the animals are given stimulants and purgatives soon after discovery of the illness, it may be