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

helped, but there are no good treatments for animals intoxicated with Buckeyes.

Asclepias spp (Milkweeds) - Milkweeds are worldwide in distribution and a large number of species occur in the United States. Some of the more common species are *Asclepias subverticullata, latifolia,* and *labriformis.* These plants grow in open or semi-open situations, especially in overgrazed pastures. They contain compounds similar in structure to cardiac glycosides. Other toxicants may be present in them. Clinical signs include trembling, staggering, falling down and violent convulsions. Pulse is rapid and weak. Dyspnea progressing to respiratory paralysis and death is a common occurrence. Hypersalivation, bloating, increased rectal temperature and mydriasis may also occur. On post mortem examination the G.I. tract may be inflammed and congested. There is congestion of the lungs and kidneys in some cases. Animals intoxicated with Asclepias, may in addition to general treatment, be treated for cardiac glycoside intoxication.

Astragalus spp and *Oxytropis spp* - (Locoweeds)

Locoweeds are very common in the western United States, and in Northern Mexico. They are common from the rangelands of the middle United States to the higher altitude ranges of the western United States. The toxic principle contained is an alkaloid and there have been many of these compounds identified. Cattle are very susceptible to the disease produced by ingesting this plant. However, it requires the eating of an equivalent of greater than 300 percent of the body weight over three months or longer in some cases. Clinical signs seen are progressive sensory and motor derangements. Cattle show signs of impaired vision and propioreception, progressing to depression. Animals may die in convulsions. Lesions observed include hyperemia and edema throughout the central nervous system. Neuronal degeneration with vascularization has been reported in some cases. The signs may disappear after removal from the infested pastures but may reappear if the animals are stressed. Locoweeds cause many syndromes in cattle. This includes habituation and emaciation, neurological disturbances, posterior paralysis, abortion, teratogenesis, and selenium accumulation. Suppressed estrus and depressed speratogenesis have also been reported with *Astragalus spp.*

Cicuta spp (Water hemlocks)

Water hemlocks occur in the Northern Hemisphere along swampy areas and streams. The toxic principle is cicutoxin. This is a highly unsaturated diol. All animals including man are susceptible to the toxic effects of this agent. Signs may appear within one-half hour of ingestion of a lethal dose. Excessive salivation appears first, then intermittent tremors and violent convulsions. Opisthotonus and grinding of teeth may occur. Mydriasis and elevation of rectal temperature are commonly observed. As death approaches the time intervals between convulsions become shorter. Death follows complete paralysis and respiratory failure. There are no significant lesions on post mortem.

Claviceps spp (Ergot) - Ergots are parasites of many plant species worldwide. The various species of Claviceps grow selectively on seed heads of certain grasses. This includes *Claviceps purpurea* which occurs on ryegrass; *C. cinerea* on Tobossa grass and *C. paspali* which grows on Bahiagrass and Dallisgrass. The toxic principles of the ergot include various derivatives of lysergic acid. Cattle are primarily affected although horses and sheep may be susceptible. There are two syndromes associated with Claviceps intoxication: (1) which is a nervous syndrome, (2) which is a gangrenous syndrome (see Musculoskeletal Section). In cattle the signs are characterized by high excitability, signs of fear of humans and somewhat belligerency. Trembling and incoordination, exaggerated flexing of forelimbs when running and tachycardia often occur. In severe cases the animals may die within 3 days in tetanic convulsions with ostotonus. In cases of average severity animals may die in about one month from thirst or starvation due to incoordination. Post mortem histopathologic examination may reveal degenerative changes in the central nervous system. Abnormally low levels of vitamin A have been detected. The disease can be prevented by mowing the grass which has infested seed heads.

Conium maculatum (Poison Hemlock) - Poison hemlock occurs in the Northern Hemisphere throughout the United States. Its general habitat is disturbed and waste places. The plant contains conine plus four related alkaloids. This hemlock is also toxic to all animals including man. Clinical signs appear quickly and are characterized by nervousness, trembling, ataxia, and dilation of the pupils. Bradycardia, hypothermia, coma and death is quite common. Death appears to be due to respiratory paralysis. There are no remarkable lesions.

Cynodon dactylon (Bermuda grass) - Bermuda grass is a native of Africa which grows or has been introduced to the warmer parts of the United States. It is widely cultivated and is a very adaptable plant where there is sufficient moisture. The toxic agent is unknown, but may be due to a fungus that grows on the grass. Affected cattle show tremors and/or posterior paralysis. These signs may be percipitated by forced exercise. In some cases icterus has been observed; otherwise, there have been no lesions discernible. Treatment is to remove the animals from the affected Bermuda grass.

Datura spp (Jimsonweed, Thornapple) - Jimsonweed has worldwide distribution and occurs in many areas of the U.S. It grows on fertile, disturbed sights such as stockpens and old farmsteads. These plants are frequently abundant as weeds in cultivation and their highly toxic seeds are thus commonly contained in some cereal grains. The toxic principle of Jimsonweed is the belladonna alkaloids such as atropine and scopolamine. All animals including man are affected, generally herbivors are more resistant than carnivores. Signs are those expected in classic parasympathic blockade. These include tachycardia, hypernea, mydriasis and visual aberations. Hyperirritability and excitement. Retention of urine, constipation, dry mouth and thirst are also characteristic. The disease syndrome progresses to dysphagia, ataxia, muscular trembling, convulsions, and respiratory failure. There are no gross lesions seen with Jimsonweed intoxication. However, the urine of an affected animal will contain sufficient belladonna alkaloids so that one drop of urine in the eye of a cat will produce mydriasis. The treatment of choice is physostigmine. Neostigmine or pilocarpin is not a good therapeutic agent, since it is a charged particle and cannot cross cell membranes.

Delphinium spp (Larkspur) and *Aconitum spp* (Monkshood) - Delphinium species and their close relatives Monkshood are distributed worldwide in the Northern Hemisphere including the northern United States. They are grown as ornamentals and in addition they grow native in many areas of the western United States, including the Plains States. The tall larkspur inhabit high mountain ranges with adequate moisture with their presence from spring to fall. The low larkspurs occur in drier situations at lower altitudes. They appear in early spring and die back in early summer. Monkshood grows along wet meadows and streams. Affected animals include horses, cattle and sheep. However, cattle are the most commonly affected animals. The toxic principle is alkaloid in nature. The clinical signs include colic, depression, vomiting, depression of heart rate and respiration, muscular weakness, paralysis, and terminal respiratory paralysis. Hypersalivation with frequent swallowing, twitching of muscles, bloating, and irregular tachycardia. On post mortem examination widespread congestion and gastroenteritis of variable extent have been reported. Animals affected should be placed in sternal recumbency with the head uphill to prevent bloating. Antiarrhythmics such as procainamide may be indicated.

Eupatorium rugosum (White snakeroot) and *Isocoma wrightii* (Rayless goldenrod) - White snakeroot grows primarily in eastern North America, including Texas.

Iscoma Wrightii (Rayless Goldenrod) occurs in the southwestern United States and Mexico. It is quite common in Texas, New Mexico and Arizona. The plants occur in general proximity to water. The toxic agent in these plants is tremetol an unsaturated alcohol. Most domestic species can be involved. However, the toxic agent is excreted in milk and as a result can become a public health problem. High producing dairy cows are not as apt to be involved because sufficient tremetol would be excreted in the milk to keep the blood concentrations low. Clinical signs may result either by ingestion of plants or milk produced by an intoxicated animal. Signs may become most noticeable after forced exercise. The trembling occurs

especially in muscles around the nose, in the legs and shoulders. Animals may stand in a humped-up appearance and move with stiffness. Debilitation which leads to ultimate recumbency often occurs. Other signs include constipation, vomiting, respiratory and cardiac difficulties and urinary incontinence. On post mortem, congestion of abomasum and intestines has been seen along with pale liver. The gallbladder is generally distended because these animals cease to eat. The brain and spinal cord may be congested. Treatment would include purgative stimulants and laxative feed.

Kallstroemia spp (Caltrops) - Caltrops occur from Kansas to Colorado, south to Texas and Northern Mexico, including southern New Mexico and Arizona. The plants generally occur in heavily grazed pastures and other disturbed soils. The toxic principle is unknown, however, cattle are very susceptible to intoxication by ingestion of this plant. The cattle develop weakness in the rear limbs with knuckling of fetlocks. This condition progresses to posterior paralysis and convulsions often precede death. Congestion and hemorrhage of the lungs, heart, kidneys, stomach, and intestines is often reported. Affected animals should be placed in the shade with feed and water. Handling should be minimized during the time of greatest signs.

Karwinskia humboldtiana (Coyotillo) - Coyotilla is the plant inherent to southern Texas and Northern Mexico. It grows in arroyos and canyons, gravely hills and ridges. The toxic principle is unknown but death can result when animal ingests as little as 2/10 percent of their body weight in fruit. All domestic species including animals may be affected. The signs would include depression, debilitation, trembling, incoordination and posterior paralysis, dyspnea and death. Affected animals show pulmonary edema, histological lesions include degeneration of skeletal and cardiac muscles with mild toxic nephritis and hepatitis. A demyelinating neuropathy often occurs. When animals have progressed to this length there is no recovery. Less severely affected animals may be treated with adequate feed and water and placed in a pen.

Nicotiana spp (Tobacco) - Various species of tobacco occur worldwide. *Nicotiana tobaccum* is cultivated. The wild species occur quite frequently in Texas, California and south into South America. They grow primarily along streams, sandy washes, roadsides and other waste places. Nicotiana plants have been used as ornamentals. The toxic principle in nicotine alkaloids and all domestic species are susceptible. Hypersalivation, bloating, weakness, shivering, staggering, and low-class twitching of muscles may be evident. There may be a violent irregular heart beat with pulse becoming rapid and weak. Dyspnea, vomiting and diarrhea are common. Animals become prostrate and death may occur within minutes, or after several days. The signs listed in animals in response to the nicotine alkaloids are similar to initial stimulation of both the sympathetic and parasympathetic nervous system. Later in the syndrome it is more characteristic of a complete blockade of all autonomic ganglia. There are no specific lesions. Cattle may show cyanosis and engorgement of subcutaneous blood vessels and apparent coagulation of blood. There may be congestion of the abomasum.

Solanum spp - The solanum are known as potato weeds, nightshades; are common worldwide, with many species prevalent throughout the United States. Most species occur in waste places or overgrazed pastures. There are several toxic agents which have been identified in *Solanum spp* including glycoalkaloids. Some plants in this genus contain belladonna-like alkaloids. Other alkaloids are nicotine-like in nature. There are also present saponins which produce G.I. irritation and also there are some toxic material which might produce a cutaneous irritation. Signs attributed to the solanum group include dyspnea, hypersalivation, possibly normal to slightly elevated temperature, weakness, ataxia, trembling of muscles of the hind limbs, tachycardia and anemia. Bloating and other gastrointestinal signs are often present. On post mortem examination the animals have exhibited congested lungs and interstitial emphysema. Livers and kidneys have been reported as congested. Animals showing nervous signs with certain solanum species have exhibited necrosis of Purkinje cells in the cerebellum. There is no treatment.

Sophora spp (Mountain laurel or Mescalbean) - There are two species of Sophora. Both species grow in the western United States. Sophora secundiflora grows in the Edwards Plateau and Trans-Texas regions of Texas. *Sophora sericae* (silky sophora) grows in western Texas and north to Wyoming and west to Arizona. Plants contain alkaloid-like substance. Their habitat is near limestone hills, rocks, ledges, and canyons. Their habitat is near limestone hills, rocks, ledges, and canyons. Animals affected include cattle. Forced excercise precipitates a stiffening of the hind limbs and muscular tremors, especially over the shoulders and rump. The animal falls becoming somnelent or comatose. Cattle often die during this convulsive seizure. The seeds are very toxic if crushed, but the hard seed coat may allow the seed to pass through the gastrointestinal tract intact.

Peganum harmala (African Rue) - This plant was introduced to North America. It is a native of North Africa and Asia. It was introduced into New Mexico where it spread to Arizona and western Texas and is continuing to spread southward and eastward. Its primary habitat is desert rangeland, especially disturbed soils such as roadsides and overgrazed pastures. There are toxic alkaloids present in this plant. There is another species, *Peganum mexicanum* or Mexican rue, which is a native of the Big Bend area and down into Mexico. In some instances this plant is also toxic. Cattle are affected quite severely. Signs include both acute and chronic syndromes. In the acute syndrome cattle exhibit stiffness, trembling, incoordination, frequent urination, subnormal temperature and hypersalivation. The chronic is characterized by anorexia, listlessness, weakness of the hind limbs, and knuckling of the fetlock joint. Post mortem examinations may reveal severe gastric enteritis, pulmonary nd renal congestion, and subcapsular hemorrhages in the liver. There is no treatment except to eliminate access to this plant and to supply good feed and water.

Phalaris tuberosa (Canarygrass or Winter harding grass) - Phalaris was introduced to the United States from New Zealand and Australia where it has been used as a cultivated forage. The toxic principle is apparently sypathometic amine. There are three syndromes which are recognized. A peracute syndrome with death following and cardiac failure or rapid recovery. Acute signs range from mild spasms with stiffness to tetanic convulsions in severe cases. Recovery is frequent. The chronic signs include head nodding, ataxia and weakness often with arrythmic tachycardia. Recovery is infrequent. On post mortem examination in the peracute and acute diseases signs are primarily congestion of the abdominal vicera and epicardial hemorrhages. In chronic *Phalaris* intoxication there is a green pigmentation of neural tissue and degenerative changes in the mitochnondria of neurons with demylination of the spinal cord in some cases. There is no treatment. However, the addition of cobalt chloride to the diet of cattle grazing *Phalaris* might prevent intoxication.

Zygadinus spp (Deathcamas) - Deathcamas are native to North America, in many areas in eastern and western United States. Their habitat is grasslands and open woodlands. A steriodol alkaloid similar to those of *Veratrum* species is present in the Deathcamas. Signs in cattle include excessive salivation, followed by signs of nausea vomition. Subsequent signs include muscular weakness, ataxia, trembling and eventual prostration. Decreased blood pressure results in weak rapid pulse. Dyspnea may occur with cyanosis and spasmodic struggling for breath. Coma of varying duration often precedes death. There are no gross lesions associated with ingestion of this plant. Atropine sulfate plus picrotoxin has been reported as a therapeutic measure for *Zygadinus* intoxication in sheep. However, the usefulness of this therapeutic regimen is still to be decided.

Toxic Plants which Affect the Gastrointestinal Track of Cattle

Centaurium spp (Mountainpink) - This plant is distributed from Missouri to California, southward into Mexico. The toxic principle is unknown, however, cattle have been reported as becoming intoxicated with ingestion of this plant. Clinical signs in affeted cattle include anorexia, lethargy, diarrhea, signs of abdominal pain, gradual weight loss and death. On post mortem examination congestion of liver and kidneys with a severe gastroenteritis along with hemorrhage and ulceration of the rumen abomasum have been reported. Histopathology-histological examination shows mild toxic nephritis and hepatitis in affected animals.

Fluorensia cernua (Blackbrush) - This plant is distributed primarily from West Texas to Arizona, south into Mexico. It occures primarily on dry hills,

plains and mesas, especially in limestone area. The toxic principle is unknown. Clinical signs include anorexia, lethargy, musclular twitching, groaning, grinding of teeth and poor equilibrium. Post mortem examination reveal affected animals exhibit severe gastroenteritis with some hemorrhage in the abomasum and upper small intestines. Marked congestion of the liver and kidneys with an albumous degeneration of renal tubules is common in some cases.

Lantana - Lantana has a worldwide distribution. Its habitat is primarily where it has escaped as an ornamental. All animals are affected, including man. G.I. signs shown by affected animals include bloody diarrhea, weakness and death in 3 to 4 days. Chronically affected animals may show sings of secondary photosensitization along with hepatic dysfunction. On post mortem examination there is evidence of gastrointestinal stasis, gastroenteritis, along with degenerative changes in the liver.

Quercus spp (Oaks) - Oaks are distributed worldwide and have 35 species alone in Texas. The exact toxic principle of oaks has not been readily identified; however, it is felt that the gallotinins in the oaks possibly play a role in the pathogenesis of the disease. Although *Quercus* are known primarily for producing renal injury, they also produce severe gastrointestinal signs. Clinical signs include emaciation and edema. Constipation and/or diarrhea have been common in a herd of animals intoxicated with oak. There may be blood in the feces. The animals appear to be dehydrated and have a rough coat. Lesions include gastroenteritis with degeneration of mucosal epithelium. Kidneys show inflammation and also petechial hemorrhages. Subcutaneous edema and perirenal edema may occur. Depending on the locale, the disease syndromes associated with oaks may occur twice a year. It may occur in early spring as the new leaves develop and may occur in the fall of the year in heavily wooded areas where the acorns fall off the trees. Keeping animals out of oak-infested pastures during dangerous intervals in the spring and fall is the best method of prevention. Supplementing the diet with alfalfa meal or cottonseed meal to which has been added 10% calcium hydroxide will prevent or reduce losses.

Sesbania spp. (drummundi and vesicaria) - Sesbania spp grow along the Gulf Coast from Florida to Texas and into Mexico. The plant species that have a disturbed area with adequated moisture. A water-soluble toxin has been isolated which will produce G.I. irritation and death in rats and rabbits. Sheep, cattle and goats are all susceptible to the plant. Affected animals exhibit depression, diarrhea and weakness. Signs appear one to two days after injestion. These include depression, diarrhea, and tachycardia with weakness and dyspnea in fatal cases. On post mortem examination, generalized congestion with widespread hyperemia and hemorrhage is evident. The abomasum in affected animals generally is a very inflammed edamatous abomasul mucosal. There is no treatment.

Solanum spp (Nightshades) - See under *Neurolgical Plants*

Xanthium spp (Cockleburs) - Cockleburs are ubiquitous plants in the world. Although swine are commonly poisoned by Xanthium spp cattle are also quite susceptible. In Texas calves appear to be frequently intoxicated by younger cocklebur plants in the Spring. Clinical signs may occur soon after ingestion of the plant. The signs include weakness, hyperthermia, dyspnea, vomiting, and a weak, rapid pulse. Convulsions may ensue. Inflammed edamatous G.I. tract is common. In addition congested liver, icterus and ascites may also occur.

Plants Causing Myopathies in Cattle

Cassia occidentalis (Coffeesenna)
Cassia obtusifolia (Sicklepod senna)
Cassia roemeriana (Twin-leaf senna)
Cassia lindheimerianna (Lindheimer senna)

Cassia spp are widespread throughout the tropic and subtropic areas of the world. Animals affected include cattle and sheep. Clinical signs may appear after the ingestion of 10 gm/kg of plant material daily for 7 days. Signs include anorexia, weakness and diarrhea. Death usually occurs within 24 hours of the onset of signs. CpK, SGOT and LGH enzyme levels are frequently elevated. The signs as produced in cattle are signs similar to the "downer cow" syndrome in which cattle exhibit posterior paralysis. Grossly the animals exhibit pulmonary edema and pale animals and skeletal muscle. Microscopically skeletal muscle degeneration with toxic nephritis and hepatitis occurring in varying degrees. Cassia intoxications are important in the southern United States and affected animals do not respond to either Vitamin E and selenium therapy. In fact such therapy exacerbates the condition.

Plants Which Affect The Heart

Datura or Jimsonweed, Thornapples - These plants have a worldwide distribution and generally occur on disturbed fertile sites, barnyards, pens, etc. Toxic principle is the belladonna alkaloids, chiefly atropine and scapalomine. All animals including man are frequently affected. Clinical signs include subnormal temperature, mydriasis, restlessness, muscular twitching, tachycardia, incoordination, paralysis. Death results from respiratory paralysis. No gross lesions are discernible. Physostigmine to effect is the treatment of choice.

Nerium (oleander) - This plant is native to Asia and is cultivated throughout the southern United States as an ornamental. The toxic principle include cardiac glycosides. All animals including man are susceptible. Vomiting, diarrhea, trembling, cardiac stimulation, initially nasal constriction, mydriasis, cold extremities and progressive paralysis is common. The syndrome usually terminates in cardiac depression, coma, and death. Lesions seen in affected cattle include gastroenteritis, widespread. Often oleander leaves may be found in rumen of affected animals. Animals affected with oleander can be treated with a combination of atropine, propranolol and potassium chloride. Treatment of animals with propranolol must be done with electrocardiogram capability since this is a very toxic drug.

Taxus spp (Yew) - Yews are cultivated throughout the United States as ornamentals. Native species occur deep in the woods; however, ornamentals prefer yards and gardens. Taxine, an alkaloid, is the toxic principle. Acute signs occur within a short time after consumption of green foliage at the level of ½% body weight for cattle. The course of the syndrome is short. Trembling, dyspnea, collapse, compression of the heart rate occur. Ultimately the heart stops diastolly. Some acute signs may occur in cattle two days after ingestion of smaller amounts of yew. Signs are the same as for acute poisoning with additional signs of gastroenteritis including diarrhea. Lesions, subacute poisoning, gastroenteritis has been reported. Treatment for *Taxus* intoxication might be the same or is similar as for those plants containing cardiac glycosides.

Veratrum spp (False helebore) - *Veratrum spp* grow in the temperate region of the Northern Hemisphere throughout North America. The habitat is low moist open situation. Complex polycyclic alkaloids are the toxic agents. Cattle are affected if they ingest this particular plant. Clinical signs appear 2-3 hours after ingestion. These include hypersalivation, ataxia, progressing to paresis, rapid, irregular heart beat, dyspnea, emesis, diarrhea and diuresis. Death follows respiratory paralysis. *Veratrum* alkaloids are hypotensive in nature; consequently, the attempted treatment should be made at alleviating the hypotensive syndromes in affected animals.

Plants Affecting the Kidneys

Amaranthus retroflexus (pigweed, carelessweed) is a common occurring plant in most areas of the United States. Although more commonly causing problems in pigs, cattle are known to be involved. The disease syndrome produced is one of perirenal edema with ensuing renal tubular degeneration and necrosis. The blood urea nitrogen is extremely elevated and a tentative diagnosis of urinary tract obstruction or ruptured bladder is frequently suggested.

Quercus spp - The oak species or *Quercus,* also known to be involved with renal dysfunction in the ruminant. The disease syndrome which occurs with the ingestion of oak may be seen at two different times of the year depending on the area of the country. In the southwestern United States the diseas is called oak-bud poisoning because the plant is ingested in early spring or any time after a rainfall which new plant growth occurs. The cattle will ingest the oak buds until the new buds or leaves are about half mature, then they seem to lose their attractiveness to the cow. The syndrome also occurs in late fall when the acorns start to fall to the ground and cattle tend to ingest these

because of the lack of other nutrients or natural feed constituents. Hence, the disease is known as acorn poisoning in the fall. This occurs anywhere there are sufficient quantities of oak or acorns which fall to the ground. Animals may appear drawn and have a rough hair coat and a dry nose, a bloody discharge may come from the nostrils. Lesions include a gstroenteritis, petechial hemorrhages on the kidneys, subcutaneous edema and ascites. Oak poisoning may be managed in the spring. These procedures include removing animals from the infested pastures for up to one month and hand feeding all the herd. Calcium hydroxide can be used as a prophylactic agent if fed for approximately one week before the onset of new leaf development and budding. The formula is as follows: cottonseed meal, 1040 lbs.; alfalfa leaf meal, 600 lbs; calcium hydroxide, 200 lbs, and vegetable oil, 160 lbs. This formula should be made into cubes and fed at the rate of 4 lbs per head per day during the danger period. Calves should be creep fed the same mixture in a meal form. It is not known whether the feeding of the Calcium hydroxide meal or pellets will effectively be prophylactic in nature against *Amaranthus* intoxication.

Toxic Plants Affecting Reproduction in Cattle

The disease syndrome affecting reproduction in cattle must be differentiated into those plants which may cause abortion and other groups of plants which produce teratogenesis or birth defects. Toxic plants which may cause abortion include *Astragalus* species, or locoweeds; Perennial broomweed, or *Gutierriza spp,* also known as *Xanthocephalum* and *Ponderosa pine. Astragalus spp* cause extensive edema of fetal membranes, water belly and/or abortion in cows. The symptoms associated with locoweeds can occur at any time during gestation period. This disease syndrome may occur at the same time as other animals are showing signs of locoism or the more insidious neurological syndromes normally associated with the locoweeds. Ponderosa pine and perennial broomweed are two plants which produce a similar type of disease syndrome, which affect reproductive systems. Ponderosa pine occur from California to British Columbia, east to the Rocky Mountains. Whereas the broomweeds occur from central to west Texas, south into Mexico and west to California and north to Idaho. Generally most abundant on an overgrazed range. The problem with Ponderosa pine occurs when there is a large knock down of pine needles and the animals having nothing to eat, ingest a quantity of these pine needles. Generally there is ensuing abortion. With regard to the broomweed which are also known as turpentine weed, or slink weed, the toxic agent is a triterpeniod saponin which may cause contractions of pregnant uterine muscles. There is some speculation that the same or similar agent also occurs in the pine. Acutely, animals may exhibit anorexia, rough haircoat, diarrhea, constipation, hematuria and death. Abortion may occur in various stages of gestation along with production of weak and underweight calves with retention of placenta when the calves are born. In milder disease stages abortion may be the only sign. In some years 40-50% abortion rates are common on infested premises.

Plants Associated with Birth Defects or Teratogenesis

The common plants are listed in Table 6. The more important plants for cattle include lupines and astragalus. While abortion is more common in cattle than birth defects when associated with **Astragalus** species, there is some speculation that birth defects can occur as a result of ingestion of the species. Especially since this is quite a common occurrence in the Bovine species. The lupines or bluebonnets cause an insidious teratogenic change in the offspring. The calves are known as "crooked calves". They may be born with one or more congenital deformities such as arthrogyrosis, scoliosis, and cleft palate. Abortion may occur at any stage of gestation. Cows which produce crooked calves may show some neurological signs and a rough dry haircoat. The most susceptible period of gestation is day 40 to day 70. The animals which were crooked calves when they were born may be bred and can give rise to normal offspring, unless there is a physical impossibility for normal reproduction. There is no treatment for this disease syndrome; however, if breeding is delayed until one month after cattle have been turned into summer range, generally lupines will not cause a problem.

Plants Which Affect the Liver and Cause Cirrhosis

These plants are listed in Table 7. Many plants which cause a hepatic dysfunction and cirrhosis contain compounds designated as Pyrrolizidine alkaloids. The common plants in the northern United States would be the *Senecio spp. These are very common in the Southwestern, Western and Northern regions including up into Canada. Cattle are primarily affected but other species may also be involved. Some animals have developed hepatic insufficiency up to 6 months after removal from infested pastures. The clinical signs in early stages becomes prominent. Neurological signs may also be seen. On post mortem examination ascites with icterus, defuse hepatic fibrosis, distention of the gallbladder and areas of hepatic regeneration are also evident. The same syndrome may be seen in other pyrrolizidine alkaloid containing plants. Crotalaria* which is an important plant in the southeastern United States, primarily causes problems in chickens and can also affect cattle and other ruminants. The disease syndrome is quite similar to those signs and lesions seen with *Senecio spp.*

Hepatic Fatty Cirrhosis (HFC) - Another disease syndrome which has been classified as hepatic fatty cirrhosis, hard yellow liver also occurs in cattle. It occurs in all ruminant species as far as we know. Hepatic fatty cirrhosis, or also known as hard yellow liver, is a disease which occurs sporadically in cattle, sheep, goats, antelope, and deer in circumscribed areas in West Texas, South Texas, and south into North Mexico. The disease occurs at 4 to 7 year intervals and usually affects practically all ruminants in a pasture. Efforts to transmit this disease have failed and the disease appears to be of toxic origin. The disease appears to be due to an anticytesliptropic agent which allows excessive accumulation of fat in hepatocytes. The excessive fat causes rupture of liver cells followed by gradual destruction of normal liver parenchyma with ensuing fibrosis. When the amount of functioning liver is reduced below that required by the body, the animal starts exhibiting signs of illness. Icterus and photosensitization are not parts of the (HFC) hepatic fatty cirrhosis picture. Approximately 80 different plants where HFC occurs have been fed in varying amounts to sheep and to some extent to cattle. Animals which have early states of hard yellow liver (HYL) may appear to be in excellent condition; however, if these animals are slaughtered at this time, the livers will be condemned on post mortem. Otherwise, the carcass appear to be in good shape. As the disease progresses, the animal becomes emaciated, or slow growers, and gradually waste away and generally die.

Plants Associated with Photosensitization in Cattle

Photosensitizing plants are listed in Table 8. Photosensitization is described as abnormal sensitivity of skin to sunlight. Requirments for disease conditions to occur include the presence of a photosensitizer or photodynamic agent, exposure to sunlight, a susceptible skinned animal and elemental oxygen which would be supplied by hemoglobin. The photosensitizing agent may either be ingested as it is or it may be manufactured by the biliary system, or the liver may be rendered incapable of biotransforming normal constituents such as chlorophill, which would give rise to the development of photosensitizing agents. Normally liver involvement is evidenced in photosensitization. However, a few plants which are classified as primary photosensitizers have a photodynamic chemical present in them. Animals which are affected with photosensitization may initially show signs of erythema puritis, shaking of head and ears. These animals seek out shade. As the disease progresses the reaction of the animal will depend upon the length of exposure, the degree of sensitivity and the intensity of the light. Secondary infection often ensues. In a few days the swelling may subside leaving the skin in a necrotic state in which it may slough. If the liver is involved then the animals may show evidence of a hepatic dysfunction.

Toxic Plants Which Accumulate Selenium

Selenium accumulating plants are listed in Table 9. Selenium is a metaloid which has caused a problem in the United States and other areas of the world for some time. Plants grown on seleneferous soils may contain sufficient amounts of selenium to cause poisoning in domestic animals. The selenium might be present as selenates, elemental selenium or organic

selenium. Plants may use selenium in either form. Animals and humans are susceptible to selenium intoxication. However, intoxication is most common in forage-eating animals, including ruminants.

The syndrome in ruminants may be differentiated into acute poisoing, subacute and chronic poisoning. The acute disease is characterized by changes in movement of posture of the animals. The animals may walk a short distance with an uncertain gait and then show a very depressed edigy. The temperature may be elevated and the pulse may be rapid and weak. Bloating and abdominal pain is usually pronounced. Mucous membranes become pale to bluish in color. The course of the illness is from a few hours to several days, depending upon the amount of toxicity of the selenium ingested. Generally if the selenium content of the plants is of several hundred to a thousand parts per million the acute disease will be produced. Subacute and chronic is divided actually into three groups: The blindstagger syndrome, alkali disease, and chronic selenosis, which has been produced experimentally. The characteristics of these forms of selenium intoxication are the following.

With alkali disease the source of selenium would be seleneferous grains and grasses. Lameness, loss of vitality, elongated hoof, loss of hair from the tail is common. There is a large quantity of selenium accumulated in the hoof and hair of affected cattle. Gross lesions include atrophy and cirrhosis of the liver, chronic nephritis, enlarged gallbladder, soft, flabby heart. Blind staggers generally occurs from ingesting of selenium indicator plants, such as *Astragalus*, and other accumulators. Emaciation, neuromuscular involvement is often common with loss of hair from the tail. Lesions seen in affected animals are similar to the alkali disease. With chronic selenosis cattle are characterized as being emaciated with some neuromuscular involvement. The lesions observed are similar to the other chronic signs. Hooves of chronically affected animals may show malformations. A circular break generally appears in the wall of the hoof below the coronary band. As the new growth of the hoof continues the rings break in the wall of the coronary band of the hoof moves downward. In severe cases the crack is so deep that the upper part of the old wall becomes separated from the new growth. As the new hoof develops the old hoof is sloughed. During this time the animal is in severe pain and may die of thirst or starvation due to inability to move properly. Cattle may be seen grazing on their knees. Plants which accumulate selenium are classified into three categories. These are: obligate selenium accumulators, facultative selenium accumulators, and those plants which might pick up the level present in the soil. Or these plants may be restricted from growth. Selenium accumulators include some *Astragalus* species, goldenweed or *Vonupsis* and *Stanyuleya* or prince's plume. Faculative indicator plants include woody asters and atrplex. Grains produced on seleniferous soils can also accumulate selenium to some extent. The treatment of selenium intoxication has at best limited. Rations containing 50-100 ppm arsenic acid has provided benefit to calves. Mature cattle have been treated successfully by the oral administration of 405 gm of napthlolene daily for 5 days, resting for 5 days and repeating the dose. One must be cautioned about the administering of some of these compounds. Various forms of chronic selenosis may be treated by the feeding of a salt preparation containing about 40 ppm arsenic. Removal of animals from selenium containing forage or grain or supplementing with hay and grain is always indicated. In the case of selenium deficiency problems the addition of selenium bearing grains or rations would be indicated.

Table 1 - Toxic Plants Which Affect the Nervous System.

Scientific Name	Common Name
Aconitum spp	Monkshood
Aesculus spp	Buckeyes
Asclepias spp	Milkweeds
Astragalus spp	Locoweeds, Peavine
Cicuta maculata	Water Hemlock
Claviceps spp	Ergot
Conium maculatum	Poison Hemlock
Cynodon dactylon	Bermuda Grass
Datura spp	Jimson Weed
Delphinium spp	Larkspur
Eupatorium rugosum	White Snakeroot
Isocoma spp	Rayless Goldenrod
Kallstroemia spp	Caltrop
Karwinskia humboldtiana	Coyotillo
Nicotiana spp	Tobacco
Oxytropis spp	Locoweed
Peganum harmala	African Rue
Phalaris spp	Harding Grass
Solanum spp	Nightshade
Sophora spp	Mescalbean, Mountain Laurel
Zygadenus nuttallii	Deathcamas

Table 2 - Toxic Plants Which Affect the Gastrointestinal Tract

Scientific Name	Common Name
Centaurium spp	Mountain Pink
Lantana spp	
Phytolacca americana	Pokeweed
Quercus spp	Oak
Sesbania spp	Sesbane, Rattlebox
Solanum spp	Nighshade
Xanthium spp	Cocklebur

Table 3 - Plants Which Affect the Heart

Scientific Name	Common Name
Datura spp	Jimson Weed
Nerium oleander	Oleander
Taxus cuspidata	Yew
Veratrum spp	False hellebore, Skunk Cabbage

Table 4 - Toxic Plants Which Affect the Kidneys

Scientific Name	Common Name
*Amaranthus spp	Pigweed, Carelessweed
Quercus spp	Oak

*Oxalate-producing plant

Table 5- Toxic Plants Which May Cause Abortion

Scientific Name	Common Name
Astragalus spp	Locoweeds
Gutierrezia spp	Perennial
(Xanthocephalum spp)	Broomweed
Pinus ponderosa	Ponderosa Pine

Table 6 - Toxic Plants Associated with Teratogenesis (Birth Defects)

Scientific Name	Common Name
Astragalus spp	Locoweeds
Conium maculation	Poison Hemlock
Lathyrus odoratus	Sweet Pea
Lupinus sericeus	Bluebonnet
Nicotiana spp	Tobacco
Veratrum spp	False Hellebore
Vicia spp	Vetch

Table 7 - Toxic Plants Which Affect the Liver and Cause Cirrhosis

Scientific Name	Common Name
*Amsinckia intermedia	Fiddleneck
*Crotalaris spp	
*Echium plantagineum	Viper's bugloss
*Heliotropium europeum	
Phyllanthus abnormis	Abnormal leafflower
Sartwellia flaveriae	
*Senecio spp	Groundsel

* Contain Pyrrolizidine Alkaloids

Table 8 - Toxic Plants Associated with Photosensitization

Scientific Name	Common Name
Ammi majus	Bishop's weed
Agave lecheguilla	Lechugilla
Avena sativa	Oats
*Cymopterus watsonii	Desert Parsley
Cynodon dactylon	Bermuda Grass
*Hypericum performatum	St. Johnswort
Lantana spp	
Medicago spp	Alfalfa
Nolina spp	Sacahuista
Panicum coloratum	Kleingrass
*Polygonum fagopyrum	Buckwheat
Tetradymia spp	Horsebrush
Tribulus terrestris	Goat Head, Puncture Vine

* Primary Photosensitizer

Table 9 - Toxic Plants Which Accumulate Selenium

Scientific Name	Common Name
Astragalus spp	Locoweeds
Atriplex nuttallii	Saltbrush
Oonopsis spp	Goldenweed
Stanleya pinnata	Prince's Plum
Xylorrhiza spp	Woody Aster

Mycotoxicoses of the Bovine with Reference to Fungi and Toxins Associated with Disease

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Introduction

The mycotoxicoses are a group of diseases caused by toxins produced by a number of different fungi. In many instances, the toxins represent secondary metabolites produced by fungi following a growth phase of the fungus. There are approximately 50 different genera of fungi in four major toxonomic groups capable of producing over 100 different toxins. We are concerned, however, only with those that may become involved with animal health because they (1) produce toxins orally active or potentially poisonous by other exposure routes for animals and man, (2) can be isolated from food and feedstuffs or (3) (toxins) are found naturally occurring in food and feedstuffs in amounts sufficient to be responsible for disease.

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The Fungi

The major genera of fungi concerned with bovine mycotoxicoses discussed in this paper are *Aspergillus, Penicillium, Fusarium, Rhizoctonia,* and *Claviceps. Pithomyces, Stachybotrys, Phompsis, Phoma Diplodia,* and *Myrothecium* are genera of fungi implicated in disease in other countries. Although it is helpful to have identification of fungal contaminants of foods and feedstuffs, definitive indentification of the toxin is necessary to prove the diagnosis. There are several reasons for this. First presence of the fungus is no assurrance it was producing its toxin. Second, a given toxin may persist in a food or feedstuffs when the fungus producing it is no longer present. Third, a given fungus may be capable of producing more than one toxin, i.e., *Aspergillus flavus* may produce both aflatoxin and a tremorgenic mycotoxin, and *Aspergillus parasiticus* may produce both aflatoxin and rubratoxin. Fourth, a given toxin may be produced by