Chapter 17
Poisonous Plants and Feed-Related Poisoning in Horses

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J. Daniel Harkins, D.V.M., Ph.D.
R.A. Smith, Ph.D.
Thomas Tobin, M.V.B., Ph.D., OABT

Quick Reference

Fortunately, most poisonous plants are distasteful to horses, and even the more palatable species are rejected if regular grasses and legumes are available. Some poisonous plants grow in wooded areas, some grow on arid plains, and some can grow anywhere.

Many common varieties of trees are toxic to horses. Horses may be kept on pasture with poisonous trees for many years with no incidence of toxicity. However, if grazing conditions become poor from drought or overgrazing, horses may start to eat plants or plant products they would normally avoid. By providing good-quality hay, owners will reduce the chances of horses eating toxic plant material. Furthermore, to reduce the likelihood of intoxication, it is necessary to prevent access to wilted vegetation by removing downed tree limbs and not feeding prunings to horses. If leaf accumulation in the pasture is large in the fall, leaves should be removed or horses should be moved to a leaf-free pasture.

The primary arrangement of this text is based on the chemical nature of the toxin. There are hundreds of poisonous plants in North America, and many have different names in different parts of the continent. For this reason we have arranged the Latin names of the plants according to the genera to which the plants belong.

PLANTS CONTAINING ALKALOIDS

Miscellaneous Alkaloids

***Taxus* spp.** (Yews). Japanese and English Yews (Figure 1) are dark-colored evergreen shrubs or small trees used as ornamental shrubbery in the United States and Canada. They have received much attention from plant breeders and as a result most ornamental yews are very difficult to speciate. The major toxic compound is taxine, an alkaloid, which depresses the conduction of impulses to the myocardium leading to bradycardia and cardiac arrest (Karns, 1983). All parts of the plant are toxic except for the flesh of the red berries. Darker, older foliage is reported to be more toxic than lighter, younger leaves. The foliage is toxic whether it is fresh-cut or dried. Japanese yew can be found throughout the United States and Canada

The time of greatest risk for all plant poisoning is when normal food supply is at a minimum. This is especially true for *Taxus* since the foliage has significantly higher levels of taxine in winter than during summer. Furthermore, many horses with insufficient foliage will eat vegetation they would otherwise find unpalatable.

Although *Taxus* foliage is usually unpalatable to horses, toxicity is still a problem in areas and climates that permit its growth. If *Taxus* trimmings are mixed with more palatable trimmings from the lawn mower, for example, horses will sometimes eat the mixture in preference to the foliage growing in the pasture. Starvation, boredom, and pica all lead to ingestion of toxic plants. Furthermore, weak gates and fences allow horses access to the shrubs.

Taxus is very toxic to horses, with as little as 0.5 lb being fatal to a 1,000 lb horse. The most common presentation of *Taxus* poisoning is acute death in which the horse collapses and dies within minutes of ingestion. Sometimes the horse is found dead beside the plant with foliage still in its mouth. Rarely, a horse will survive the acute intoxication. These animals often exhibit nervousness, dyspnea, incoordination, and tremors prior to collapse. Death usually occurs within several hours of ingestion.

Diagnosis of *Taxus* poisoning is usually made from a history of ingestion of *Taxus* spp. or the discovery of foliage in the intestinal tract. There are no pathognomonic lesions for *Taxus* toxicity, nor is there any effective treatment (Karns, 1983).

Tropane Alkaloids

Datura stramonium (Jimson Weed). Jimson weed or thorn apple plant (Figure 2) is an annual (recurring every year) herb found in waste land, as well as cultivated areas throughout North America. All parts of the plant are toxic, especially the dark brown seeds which accumulate higher amounts of toxic alkaloids (hyoscyamine and hyoscine) than other plant parts.

Atropine-like alkaloids are found in many different plants included in the nightshade family Solanaceae. The toxic alkaloids include hyoscine (scopolamine) and hyoscyamine, and they act as a competitive antagonist of the actions of acetylcholine and other muscarinic agonists.

Initially, the clinical signs include central nervous system excitation with increased respiratory and heart rates. This is followed by depression and decreased respiratory and heart rates. There is also the characteristic sign of dilated pupils, as well as muscle weakness, colic, and watery diarrhea which may contain blood (Gilman et al., 1990). Polyuria and polydipsia are often present. There is no specific antidote, so therapy is limited to oral administration of activated charcoal and a saline cathartic to limit further absorption of the toxins (Knight, 1995). A horse will return to normal condition within a few days after the source of intoxication is removed.

Exposure of performance horses to Jimson weed has resulted in the identification of scopolamine, an unauthorized substance, in post-race urine samples. Hay and straw used as feed and bedding for performance horses should be checked for Jimson weed, and sources known to be contaminated with the plant should be avoided (Tobin et al., 1995).

Glycoalkaloids

***Solanum* spp.** and ***Lycopersicon* spp.** (Nightshades). The plants containing glycoalkaloids which are commonly eaten by humans include potatoes (*Solanum tuberosum*) and tomatoes (*Lycopersicon* spp.). The tubers of the potato and the fruits of the tomato normally contain very little of the toxins solanine and tomatine respectively. The vines of both, however, are very toxic. Most of the problems occur when the plants are harvested and the vines are tossed over the fence as fodder. Some nightshades also contain the glycoalkaloid solanine.

Pyrrolizidine Alkaloids

Numerous genera of plants contain pyrrolizidine alkaloids. Those found frequently in the United States include ***Senecio*** (Figure 3)**, *Crotalaria, Amsinckia, Cynoglossum, Trichodesma,*** and ***Heliotropium.***

Pyrrolizidine alkaloids are the major plant hepatotoxins for horses and are found in several plants in western North America. The quantities, types, and toxicity of various pyrrolizidine alkaloids differ between plant species. The most common plants containing the toxins are tansy ragwort or stinking willie (*Senecio jacobaea*, Figure 4), fiddleneck or tarweed (*Amsinckia intermedia*), rattlepod or rattlebox (*Crotolaria* spp.), heliotrope or stickseed (*Heliotropium* spp. and *Trichodesma* spp.), hound's tongue

(*Cynoglossum officinale*, Figure 5), and various types of groundsel including common groundsel (*S. vulgaris*), lamb's tongue groundsel (*S. integerrimus*), Riddell's groundsel or Riddell's ragwort (*S. riddellii*), and wooly or threadleaf groundsel (*S. douglasii*).

The concentration of pyrrolizidine alkaloids in plants varies with the stage of growth, and the flowers generally contain the highest toxin concentration of all the plant parts. However, *Crotolaria* spp. contains a pyrrolizidine alkaloid called monocrotaline, which is most concentrated in the seed, with lesser amounts contained in the leaves and stems (Bull et al., 1968). In *Senecio* spp., mature plants are generally more toxic than younger plants. *Cynoglossum* spp. contains the pyrrolizidine alkaloids, heliosupine and echinatine. The greatest concentration of the toxin is found in the preflowering rosette stage (Knight et al., 1984).

Although there are reports of acute death in animals after only a few days of ingesting plants with high levels of pyrrolizidine alkaloids, low-level poisoning over a longer period of time is more common in horses (Knight et al., 1984). Because the liver continues to function at near-normal level until approximately 80% of it is damaged, symptoms of chronic hepatotoxicity are not evident until significant liver damage has occurred. Therefore, the appearance of clinical signs presages a guarded to poor prognosis. Symptoms associated with irreversible liver damage include depression, weight loss, anemia, hepatic encephalopathy, icterus, and photosensitization resulting in dermatitis.

Hepatic encephalopathy may be one of the earliest indications of toxicity. It is characterized by aimless walking, drowsiness, blindness, head pressing, and incessant licking of rocks or other objects (Tennant et al., 1973).

Photosensitization is a result of the liver's inability to remove the normal breakdown products of chlorophyll metabolism, which accumulate in the blood. When exposed to the ultraviolet rays of the sun, the metabolites fluoresce and release radiant energy, causing necrosis of the utaneous cells. Because the presence of pigment will greatly reduce the penetration of the ultraviolet rays, non-pigmented skin is more susceptible to photodamage (Knight, 1995).

Because of the chronic nature of the disease, identification of the plants causing the disease may be difficult. When symptoms appear months after ingestion of the toxins, the offending plants may not be present in the pasture or hay the horses are currently consuming.

Pyridine Alkaloids

Conium maculatum (Poison Hemlock). Poison hemlock (Figure 6) is abundant in much of the United States. Plants growing in the warmer southern states appear to be more toxic than those growing in northern areas. The plant is common in disturbed soils and is especially abundant along roadsides and around buildings. Although the plant has its maximum growth period in the spring with the top dying off by late summer, a secondary growth is seen in the fall. The plant remains green throughout winter when other vegetation may be sparse. The plant contains a number of toxic alkaloids, of which coniceine and coniine are the most abundant. Within an hour of ingestion, horses show marked ataxia and staggering, urination and defecation is frequent, and signs of muscular weakness, trembling,

slobbering, eructation, cyanosis, rapid weak pulse and dilated pupils are noted. Death occurs as a result of respiratory paralysis.

The objective in treating hemlock poisoning is to halt further absorption of the toxin from the GI tract. Activated charcoal (1 lb/1,000 lb horse) should be given in water or saline with a stomach tube. Kaolin and pectin (1 gallon) via stomach is also effective to coat the intestine and prevent absorption of the toxin. After absorbents have had an opportunity to work in the stomach and intestine (4–6 hours), mineral oil (1 gallon/1,000 lbs) can be given as a laxative to facilitate passage of the toxins.

Diterpenoid Alkaloids

***Delphinium* spp.** (Larkspurs). Larkspurs (Figure 7) are another type of plant that have been subjected to the attention of plant breeders; therefore, speciation should be left to a botanist. In their wild form, both tall and low larkspurs are known. Several hundred different alkaloids have been characterized from the genus, but only a very small number show evidence of high toxicity. The most dangerous alkaloid in the group appears to be methyllycaconitine, which produces a short-term neuromuscular block when given intravenously (Nation et al., 1982). The tall larkspur, *Delphinium barbeyi,* and the low larkspur, *Delphinium tricorne,* are particularly troublesome.

The alkoloids in *Delphinium* spp. primarily affect the neuromuscular junction, producing curare-like symptoms of muscle weakness and paralysis. Affected horses are hypersensitive and will be stiff or staggering at the walk. Trembling, collapse, and convulsions may follow.

Early diagnosis is critical for successful treatment. The horse should be kept quiet to limit stress and excitability. Intravenous physostigmine (0.04 mg/lb) will counter the curare-like effects of the toxin. This may be repeated as needed over several hours until the clinical signs have subsided (Knight, 1995).

Indole Alkaloids

Claviceps purpurea (Ergot). Hay, along with oats, is a traditional staple of the horse diet. Many members of the grass family are susceptible to infestation with ergot, a condition where individual grains in the head of the grass are replaced with ergot sclerotia. Ergot sclerotia have a purplish black color and tend to be much larger than the grass seed they replace. Before cutting and baling a field of hay, a stroll through the pasture is appropriate. Ergot, when present, is clearly visible to the naked eye. Ergot contains a number of indole alkaloids such as ergotamine. These alkaloids, if consumed in small amounts over a period of time, cause necrosis of the tissues of the extremities. Consumed in greater quantity, ergot can produce neurotropic disorders like convulsions. Grasses especially susceptible to ergot infestation include quackgrass, redtop, bromegrass, reed grass, orchard grass, various types of ryegrass, fescue, bluegrass, dallisgrass and canarygrass (DiTomaso, 1994).

Quinolizidine Alkaloids

***Lupinus* spp.** (Lupines). The lupines (Figure 8) have also been extensively cultivated and some of these cultivars have escaped to the wild. Even among the native lupines, there is evidence of cross-breeding (Majak et al., 1994). Speciation should only be attempted by a botanist with extensive experience in the field. Even though botanists insist that *Lupinus polyphyllus* is the same species in Europe as it is in North America, only those plants grown on the North American continent have been reported to contain the alkaloid anagyrine, a potent teratogen. In general, horses find lupines unpalatable, and because large amounts of the plant must be consumed to cause acute poisoning, horses are not especially threatened by these plants. However, the presence of teratogens does mean that some lupines pose a threat to the fetus. The hay and straw of performance horses are occassionally contaminated with lupines, and lupamine has been identified in post-race urine samples. There is no known treatment for lupine poisoning.

Steroidal Alkaloids

***Zigadenus* spp.** (Death Camas). The death camas (Figure 9) is found mostly on the western ranges where it grows in great clumps. The plant contains a number of alkaloids including zygacine. Occa-

sionally, horses are poisoned by this plant. Initial symptoms include excessive salivation that persists for the duration of the intoxication. Colic, ataxia, muscle weakness, convulsions, and eventually prostration are often seen in horses. Signs of dyspnea are common before the animals become prostrated. In cases where several plants have been consumed, the animals lapse in and out of coma before death.

Treatment for death camas poisoning in horses is usually unrewarding. Effective treatment in sheep has been reported, which consists of subcutaneous injections of 4 mg atropine sulfate and 8 mg picrotoxin in 10 ml of isotonic saline per 220 lbs body weight. Activated charcoal (1 lb/1,000 lb) and kaolin/pectin suspension (1 gallon/1,000 lb) can be given to further halt absorption of the toxin from the gastrointenstinal tract. Intravenous fluid and analgesics are indicated to control colic (Sporke, 1979).

PLANTS CONTAINING TANINS

***Quercus* spp.** (Oaks). Oak blossoms, buds, leaves, stems, and acorns (Figure 10) are toxic to horses and other livestock. Over 60 species of oaks have been identified in the United States and should be considered toxic (Kingsbury, 1964). Young leaves and green acorns are considered more toxic than more mature components, and young leaves are more palatable than mature leaves. The bark of oak trees is also poisonous.

The toxic principle in oak trees is presumed to be tannin, and the tannic acid content of leaves and acorns decreases as they mature (Pigeon et al., 1962). Most episodes of acorn toxicity occur in autumn, soon after the acorns fall. The tannic acid content of acorn shells can be as much as 10% of dry matter.

Tannins are astringents, which means they are potent precipitators of cellular protein, and cause coagulation necrosis in the intestinal tract and in the proximal tubules of the kidneys (Anderson et al., 1983). Clinical signs of oak toxicity vary according to the amount of tannin-containing material ingested by the horse. Initially, horses stop eating, become lethargic, and show signs of abdominal discomfort. The stool will be hard and dark. Choking, nasal mucous, and oral ulcers are sometimes seen. Liver and kidney damage will be evident by elevated serum liver enzymes and blood urea nitrogen. Further renal damage may be indicated by low specific gravity, and the presence of excess protein, sugar, blood, and tubular casts in the urine. Death may occur within a day of ingestion of large amounts of oak leaves and acorns, or the horse may survive up to a week after onset of clinical signs (Knight, 1995). Oak trees are distributed throughout North America.

Treatment includes removal of the horse from the source of poisoning and intravenous fluids to promote diuresis and to maintain hydration and electrolyte and acid-base balance. To help eliminate any tannic acid remaining in the intestinal tract, mineral oil should be given at a dose of 1 gallon per 1,000 lbs. Supportive treatment for signs of colic are also indicated.

PLANTS CONTAINING TOXIC DITERPENES

Kalmia latifolia (Mountain Laurel, Figure 11), ***Rhododendron* spp.** (Azaleas, Figure 12), ***Leucothoe*** spp. (Fetterbushes), ***Pieris*** spp. (Mountain Pieris), ***Lyonia* spp.** (Maleberries), ***Ledum* spp.** (Labrador Teas) and ***Menziesia* spp.** (Mock Azaleas).

Mountain laurel and the other plants noted above are common shrubs or small trees. Mountain laurel produces white or pink flowers and is found in the southeastern part of the United States. The trees produce two toxic principals which are present in all parts of the plants. The toxins are grayanotoxins (andromedotoxin) and a glycoside arbutin. The clinical symptoms caused by these toxic compounds are excessive salivation, colic from intestinal irritation, frequent defecation, depression, and ataxia. Severe intoxication may result in recumbency and death. There is no specific treatment for laurel toxicity; however, mineral oil and intravenous fluids should be administered for supportive therapy (Knight, 1995).

PLANTS CONTAINING GLYCOSIDES

Cyanogenic Glycosides

***Prunus* spp.** (Cherries, Peaches, Plums). The leaves of wild or tame cherry, peach, and plum trees are toxic under certain conditions. Foliage from these trees is not toxic if ingested fresh or browsed directly from the tree. However, wilted leaves from prunings or fallen limbs may produce poisoning by affecting the oxygen-carrying capacity of red blood cells. *Prunus* spp. contains a cyanogenic glycoside which affects the cytochrome oxidase system. When the plants are chewed, the glycosides release cyanide which binds with hemoglobin to prevent release of oxygen to the tissues. The toxin in the plant dissipates with time, and dried leaves and dried bark are non-toxic.

Onset of symptoms occurs within minutes to hours of ingestion. Death due to anoxia will also occur within minutes to hours depending on the amount of toxin ingested. Clinical signs include respiratory distress, bright red to cyanotic mucous membranes, tremors, weakness, and seizures. Mild cases may recover spontaneously; however, horses affected with severe intoxication may die before treatment can be administered (Robbins, 1995).

To successfully treat cyanogenic gylcoside poisoning, the cyanide radical must be detoxified to thiocyanate shortly after ingestion. The traditional treatment consists of intravenous administration of a mixture of 1 ml of 20% sodium nitrate (200 mg/ml=200 mg) and 3 ml of 20% sodium thiosulfate (200 mg/ml=600 mg) per 100 lb body weight. This may be repeated in a few minutes if there is no response (Way, 1984). Other treatment regimes described in the literature include the same medications at a different ratio (100 mg sodium nitrate and 3,000 mg per 100 lb body weight. The addition of a 5% solution of cobaltous chloride (480 mg/100 lb) improves the effect of the sodium nitrite and sodium thiosulfate. Alpha-ketoglutaric acid has been used as a cyanide scavenger in dogs (Knight, 1995).

***Sorghum* spp.** (Sudan Grass and Johnson Grass). Horses grazing Sudan grass (*Sorghum sudanense*), Johnson grass (*S. halepense*, Figure 13), and hybrid plants from each can develop posterior ataxia and urinary incontinence, which results from degeneration of the lower spinal cord. The most prevalent occurrence of the condition is when the plants are young and growing rapidly; however, some incidences have been reported after horses have grazed on mature pastures. Horses fed well-cured *Sorghum* spp. hay are not affected. Not all *Sorghum* spp. are toxic (Adams et al., 1969).

The toxic agent in the poisonous *Sorghum* spp. is cyanogenic glycoside, which has the potential to produce cyanide poisoning and sudden death. However, *Sorghum* spp. poisoning in horses usually results in a chronic neurotoxicosis following prolonged grazing of grasses containing very low concentrations of the toxin.

Clinical signs include rear leg ataxia, urinary incontinence, and weight loss. Posterior ataxia is most evident when the horse is backed or turned sharply. Sequelae of urinary incontinence are urine-stained hair, skin ulcerations in the perineal area and on hind limbs, dribbling of urine, frequent urination, and straining to urinate. Mares may have paralysis of the perineum which will cause the lips of the vulva to remain open, resulting in vaginitis. Males continually drip urine from a relaxed and extended penis. Necropsy findings include focal axonal degeneration and demyelination of the lower spinal cord and inflammation of urinary tract. In horses that die from the disease, death is usually from

ascending pyelonephritis, secondary to the urinary incontinence (Adams et al., 1969; VanKampen, 1970).

Differential diagnoses of the posterior ataxia include equine protozoal myeloencephalitis, equine herpes virus-1 myeloencephalitis, and equine viral encephalitis. A history of grazing sorghum grasses and urinary incontinence suggest sorghum toxicity.

Complete recovery is possible if horses are removed from toxic sorghum grasses. Once severe ataxia or inflammation of the urinary tract occurs, complete recovery is rare. In addition to removing horses from sorghum pastures, any urinary tract infection should be treated with the appropriate antibiotic (Knight, 1995).

***Linum* spp.** (Flaxes). Another cyanogenic glycoside-containing plant is wild blue flax which grows throughout North America. It is a perennial (present at all seasons of the year) with bright blue flowers. The flowers contain five petals that drop off each afternoon and are replaced the following day. Young plants are particularly toxic (Knight, 1995). Symptoms and treatment of linum toxicity are similar to *Prunus* spp. toxicity.

Sambucus spp. (Elderberries). Elderberry bushes produce juicy, edible fruit in the late summer and early fall. However, the leaves and stems are thought by some to contain cyanogenic glycosides that are toxic to horses. The woody shrubs grow 6–10 feet in height and generally prefer rich moist soils along ponds and streams (Knight, 1995). Symptoms and treatment of sambucus toxicity are similar to *Prunus* spp. toxicity.

Cardiac Glycosides

For centuries, cardiac glycosides have been derived from various plants for medicinal purposes. Digitalis and other cardiac glycosides exert a powerful action on the myocardium that is unchallenged in value for treatment of heart failure. However, only very small amounts of the plant are required for a lethal dose when horses graze pastures containing these plants. Less than one ounce of green oleander leaves is lethal to a 1,100 lb horse (Cheeke and Shull, 1985). Fortunately, plants containing cardiac glycosides are very distasteful, and horses avoid them if good-quality pasture is available. Unfortunately, dried leaves of the plants are more palatable, and the cardiac glycosides are retained in dried plants. The dried leaves may pose a greater threat to horses when baled in the hay.

Toxic amounts of the cardiac glycosides inhibit the sodium-potassium pump (Tobin and Brody, 1972). This results in a disruption of the normal cardiac electrical activity and causes a variety of arrhythmias and eventually cardiac arrest. Cardiac glycosides also cause hemorrhagic enteritis, colic, and diarrhea. The extremities of the horse may be cold and the pulse may be weak and irregular due to the decreased cardiac output.

There are no specific antidotes for cardiac glycosides. Affected animals should be removed from the source of the toxin and given fresh water and good-quality hay. All stress should be eliminated to reduce the load on the heart. Activated charcoal (2 g/kg–5 g/kg) is an absorbent that may prevent additional absorption of the toxin into the body. The cardiac arrhythmias may be treated with potassium chloride, procainamide, lidocaine, dipotassium EDTA or atropine sulfate (Szabuniewicz et al., 1972). Animals that

have not ingested a lethal dose of the plants usually recover in a few days. Immunologically-based assays are used clinically in human medicine to quantify cardiac glycosides in therapeutic drug monitoring. These analyses may be useful to detect the wide range of cardiac glycosides found in veterinary toxicology.

***Asclepias* spp.** (Milkweeds). Milkweed (Figure 14) is a perennial (a plant living more than two growing seasons) herb that contains a milky sap from which the plant gets its name. The plant produces flowers that are greenish white to red. The narrow-leafed species are more toxic than the broader-leafed plants. Overgrazing of pastures promotes the establishment of milkweed. The major cardiac glycosides contained in milkweed are the cardenolides. In addition to the toxic effects listed for all cardiac glycosides, the cardenolides in milkweed also cause dyspnea, muscle tremors, and head pressing (Knight, 1995).

Digitalis purpurea (Purple Foxglove). Foxglove is a perennial herb, growing 3–5 feet in height. It has characteristic white or purple flowers with obvious spots. The major cardiac glycosides contained in foxglove are digoxin and digitoxin. Foxglove is the source for medical preparations of these agents (Gilman et al., 1990). Symptoms and treatment of foxglove toxicity are similar to those for milkweed toxicity.

Nerium oleander (Oleander). Oleander (Figure 15) is an evergreen flowering shrub, growing in southern states from Florida to California. It is a perennial shrub or small tree that may reach 25 feet in height. It has a sticky, white sap and produces white, pink, or red flowers. The major cardiac glycosides contained in oleander are oleandroside and nerioside (Knight, 1995). Symptoms and treatment of oleander toxicity are similar to those for milkweed toxicity.

***Thevetia* spp.** (Yellow Oleander). Yellow oleander is a perennial flowering shrub or tree that grows in the southern part of the United States, and Hawaii. It produces clusters of yellow and orange flowers at the ends of its branches. The major cardiac glycoside contained in yellow oleander is thevetin (Knight, 1995). Symptoms and treatment of yellow oleander toxicity are similar to those for milkweed toxicity.

Coumarin Glycosides

***Aesculus* spp.** (Buckeye and Horse Chestnut). Other shrubs or small trees toxic to horses are various buckeye trees and the horse chestnut (*A. hippocastanum*, Figure 16), which is not related to the edible chestnut (*Castanea* spp.). Not all buckeyes have been reported to be toxic. Those that are poisonous to animals are the Ohio buckeye (*A. glabra*), California buckeye (*A.californica*), red buckeye (*A. pavia*), and yellow buckeye (*A. octandra*). They have large palmate leaves with red or white flower spikes at the end of their branches. Their fruit is the characteristic shiny, brown nut. Although *Aesculus* spp. trees are found throughout most of the United States, they are mainly distributed in the eastern and southern states.

The toxic agent in *Aesculus* spp. is the glycoside, aesculin. New-growth leaves and nuts are especially toxic. The agents primarily affect the nervous and gastrointestinal systems. Ataxia and muscle tremors have been reported, with paralysis occurring in more severe cases. However, the main complication reported in horses is colic. There is no specific antidote for aesculin, but mineral oil, fluid therapy, and analgesics as needed for colic are indicated (Tobin, 1981).

MISCELLANEOUS TOXINS

Plants Containing Tremetol

Eupatorium rugosum (White Snakeroot). White snakeroot (Figure 17) or richweed is a perennial plant found on rich soils in wooded and shaded areas of the eastern and southern United States.

Eupatorium adenophorum (Crofton Weed). Crofton weed is an erect perennial plant with soft slender stems, growing up to 6 feet tall. The broad leaves taper toward the tip, and the white flower head forms tight clusters (Harper, 1979).

In a study of intentional intoxication with Crofton weed in horses (O'Sullivan 1985), severe pulmonary fibrosis was the main pathological finding. Ten horses were fed 10–15 g/kg. of the plant daily. It was determined the flowering stage was more toxic than the non-flowering plant. The non-flowering plants produced no visible lesions after 164 days of ingestion. The severity of the pulmonary lesions were directly related to the amount of plant ingested and the duration of exposure. There was no resolution of pulmonary lesions following removal of the animals from the plant.

Tremetol is also found in Jimmy weed or rayless goldenrod (*Haplopappus heterophyllus*), and burrow weed (*H. tenuisectus*), and these plants are also toxic to horses (Knight, 1995). The first sign of intoxication is a dry cough, which is more pronounced after exercise. With continued exposure, animals develop labored breathing accompanied by heaving of the abdomen. Animals become depressed, lose conditioning, and die. Working horses will sometimes collapse during exercise and die of respiratory failure (Harper, 1979). Differential diagnoses for tremetol toxicity include botulism, rabies, lead poisoning, and esophageal obstruction (Knight, 1995).

Treatment is unlikely to be successful in advanced cases. Affected animals should be removed from the Crofton weed source, and stress and exercise should be minimized. Veterinary treatment consists of activated charcoal (1 lb/150 lb body weight) and supportive therapy.

Plants Containing Cannabinoids

Cannabis sativa (Hemp, Marijuana). Hemp is native to Central Asia, but it is now found globally, especially in discreet, remote and well-concealed locations. When grown under these conditions, it often occurs as very dense clumps. A hungry horse left to forage after a day's riding can consume large quantities in a very short time. The symptoms of cannabis intoxication are excitement, difficulty in breathing, and muscular trembling. Despite having subnormal temperatures after exposure to cannabinoids, the animals sweat and slobber profusely prior to death.

Therapy is directed at reducing external stimuli and providing symptomatic treatment using intravenous fluids. If the animal can be maintained with supportive therapy for an adequate period of time and if the temptation to perform euthanasia on the animal can be resisted, the horse will usually recover.

Plants Containing the Thiaminase Enzyme

Pteridium aquilinum (Bracken Fern). Bracken fern (Figure 18) is a perennial fern found in wooded areas. In North America, it is most prevalent in the northwest. The plant contains the enzyme thiaminase, which degrades thiamine (vitamin B_1) in the diet. Because thiamine is an essential amino acid, extensive and repeated ingestion of this plant is required to produce symptoms. *Onoclea sensiblis* (sensitive fern) also caused occasional poisonings in horses.

Symptoms of thiamine deficiency in the horse include: weight loss due to poor appetite, bradycardia, and neurologic symptoms including weakness, depression, and ataxia, especially in the hindquarters. Horses have to ingest a diet containing 30%–50% bracken fern for 30 days before clinical signs appear. If horses continue to ingest the toxin, progressive depression and weakness may result in coma and death (Kingsbury, 1964). Treatment can be administered with oral, intramuscular, or intravenous thiamine (5 mg/kg–10 mg/kg). Intraveous thiamine would be given slowly and diluted in a liter of saline to reduce the chance of adverse reactions. For the next 5–7 days, intramuscular thiamine (5 mg/kg–10 mg/kg) should be given once a day. Recovery usually takes 2–3 weeks.

Equisetum arvense (Horsetail). Horsetail (Figure 19) is common on sandy, moist soil, in meadows, along roadside ditches, stream banks and railroad embankments. The plant is reputed to contain many toxins, including thiaminase. Horses are very susceptible to horsetail, but fortunately many weeks of exposure are required for symptoms to appear.

bition of these enzymes results in a generalized lysosomal storage disease, which eventually causes irreversible neuronal damage (Dorling et al., 1978).

Horses affected with locoism show abnormal locomotion such as a high stepping gate, ataxia, head bobbing, or dragging of the feet. Alone, the horse usually appears depressed; however, stimuli may cause it to overreact, resulting in excitement or rage. Some horses become completely unpredictable when handled (James et al., 1981). Eventually, horses have trouble eating and become thin with rough coats, followed by death.

If the horse is removed from the source of intoxication and fed a nutritious diet, it will show improvement and may be relatively normal in several months. However, a horse which has eaten locoweed over a long period of time may recover only partially, retaining an irregular gait, making it dangerous to ride.

Locoweed is a cumulative poison, and the more severe signs may not appear until after ingestion of the weed has ceased. It appears horses are more susceptible to intoxication than are cattle or sheep. Horses eat the plant more readily and are affected by smaller amounts of toxin. Young animals are more severely affected, because the maturing neurons are more vulnerable to the toxic effect.

Diagnosis of locoweed poisoning should be suspected in any horse that has been exposed to locoweed and is exhibiting abnormal behavior. Horses that have ingested locoweed within the previous two days may have blood lymphocytes with cytoplasmic vacuoles of diagnostic significance (Harper, 1979).

There are no pathognomonic lesions on postmortem exam. In acutely poisoned horses showing neurologic signs, severe cytoplasmic vacuolation of the brain neurons is common. Similar lesions are also seen in the cells of the pituitary gland, thyroid, pancreas, kidney, and liver. In chronically poisoned horses that have not eaten locoweed for over a month, vacuolations are found only in the liver cells and neurons of the brain.

Other than removal from contaminated pasture and the provision of nutritious feed, there is no treatment for locoism. Horses do appear to crave locoweed if exposed on subsequent occasions; therefore, further access to the plant should be prevented.

Milkvetch Intoxication. Nitroglycosides have been isolated from 263 species of milkvetch (*Astragalus* spp.) which grow on the western plains of the United States, Canada, and Mexico. The plants contain at least two toxic compounds: miserotoxin and 3-nitro propionic acid (Williams and Barneby, 1977). The toxins act primarily on the respiratory and central nervous systems. Depression, incoordination, and hind-leg weakness are the initial symptoms, followed by respiratory difficulty, weight loss, and paralysis of hind legs, if the animal continues to eat milkvetch. If animals are removed from contaminated pastures, they will usually recover if central nervous system symptoms are not severe.

Selenium Toxicosis. Selenium is a necessary trace mineral. A diet containing less than 0.1 ppm (mg/kg) may result in myopathies or steatitis, especially in foals. Diets containing more than 5 ppm may result in chronic selenium toxicity. Diets containing 25–50 ppm, may cause acute selenium toxicosis and sudden death from pulmonary congestion and edema (Knight, 1995).

Twenty-four species of *Astragalus* are obligate selenium accumulators. The high selenium content gives the plants an unpleasant garlic-sulfur taste/odor and horses will avoid eating these plants. It is safe to keep horses on pastures containing these plants, as long as the pasture is not overgrazed.

Acute selenium toxicity from eating selenium-accumulator plants is unusual in horses due to its unpleasant taste. If a horse does eat plants containing very high levels of selenium, death is usually rapid, and occurs before clinical symptoms appear. The usual postmortem findings include: congestion, edema, and necrosis of the lungs, liver, and kidneys.

Chronic selenium toxicosis is more commonly seen and has been divided into two syndromes: blind staggers and alkali disease. Historically, blind staggers is a disease known only to sheep and cattle, occurring after ingestion of selenium-accumulator plants. Symptoms include: aimless wandering, circling, bumping into objects, decreased appetite causing weight loss, and apparent blindness. Eventually, animals develop front-leg weakness and the inability to stand. Death is caused by respira-

tory failure if the plants are not removed from the animals's diet (Rosenfeld and Beath, 1964).

However, a recent study suggests the blind staggers syndrome may be due to locoweed neurotoxicosis, rather than selenium poisoning. Sheep fed two-grooved milkvetch (*Astragalus bisulcatus*), a plant that is an obligate selenium accumulator (containing the toxin causing Locoweed Neurotoxicosis), showed microscopic vacuolation lesions in the brain, typical of locoweed poisoning (Rosenfeld and Beath, 1964).

Alkali disease obtained its name after early western settlers noticed the condition in animals that had grazed for several months in semiarid regions. These regions contain high-alkali soil, with large amounts of readily available selenium for plant uptake. The pathophysiology of the disease demonstrates excess selenium displacing sulfur on the keratin molecule (a main structural component of hooves and hair). Therefore, the mane and tail hairs of affected horses break at the point where sulfur has been displaced, giving the horse a roached mane and bobtailed appearance. The disease is also referred to as "bob-tail disease." Lameness also develops due to hoof wall abnormalities. All feet are affected, and horses will develop severe lameness. Some horses may slough the entire hoof. Other symptoms associated with chronic selenium toxicity include: anemia, liver cirrhosis, emaciation, myocardial degeneration, and bone and joint degeneration (VanKampen and James, 1978).

As with other syndromes produced by *Oxytropis* and *Astragalus* spp, affected horses should be removed from the source of excess selenium, and further access to the plant should be prevented in subsequent years. Providing a diet that is rich in sulfur-containing amino acids (cysteine and methionine) and low in selenium will help reduce the toxic effect of the mineral. The presence of copper in the diet (10–25 ppm) will also minimize the adverse effects of selenium (Stowe, 1980).

***Lathyrus* spp.** (Vetch, Wild Pea, Pea, Flatpea). The vetches contain a number of compounds, many of which are aminonitriles, such as beta-(gamma-L-glutamyl)-aminopropionitrile. Other toxic compounds are found within the genus, such as beta-N-oxalyl-L-alpha-beta-diaminopropionic acid, the toxin in *Lathyrus sativus* (Chickpea). The horse is particularly vulnerable to the chickpea. Numerous horses died in England when fed an exclusive diet of chickpea. Symptoms were evident after only 10 days of feeding (Kingsbury, 1964). A low level of these toxic legumes in the diet causes symptoms to develop only after several months.

Paralysis of the hind legs is one consistent symptom in most species, particularly in the horse. Structural abnormalities such as skeletal deformities, aortal aneurysms, and damage to connective tissue are related to the toxin's adverse affect on collagen. The addition of casein to the diet protected against the paralytic effect and was partially effective against aneurysms (Bachhuber and Lalich, 1954).

***Artemisia* spp.** (Sagebrush). Another plant causing neurologic disease in horses is sagebrush. The toxins in sagebrush are volatile monoterpenoid oils that are most concentrated during the fall and winter months.

Horses develop neurologic signs similar to locoweed poisoning including: ataxia, excitability, circling, and abnormal reactions to common stimuli.

Appetite usually remains normal. The characteristic smell of sage is present on the breath and in the feces of horses that have ingested amounts of sagebrush. Unlike locoweed toxicosis, symptoms of sagebrush toxicity usually disappear if horses are removed from the source and fed a nutritious ration (Knight, 1995).

Plants Susceptible to Fungi Which Produce Mycotoxins Harmful to Horses

Zea mays (Corn) and ***Festuca arundinacea*** (Tall Fescue). Numerous mycotoxins are known to cause diseases in horses. Corn and fescue are two plants in particular that are commonly grown to produce animal feed, and are of special concern to horse owners (Figure 22). Corn is particularly susceptible to infestation by *Aspergillus flavus* which produces alflatoxins, a toxin implicated in the deaths of horses (Vesonder et al., 1991). Corn is also susceptible to *Fusarium moniliforme,* which produces fumonisins. Horses alone develop leukoencephalomalacia if given a diet containing as little as 8 ppm fumonisin (Ross et al., 1992).

Tall fescue is the most widely adapted, cool-season pasture grass in the United States. Many tall fescue pastures, particularly the Ky-31 variety in the southeast, contain an endophyte fungus named *Acremonium coenophialum*. Endophyte-free tall fescue is a nutritious and vigorous forage crop; however, it is estimated that 90% of southern tall fescue pastures are infected with *Acremonium* endophytes. Tall fescue contaminated by this fungus causes reproductive problems in horses including: abortions, prolonged gestations, difficult births, thickened placentas, foal deaths, retained placentas, decreased milk production, and increased mare mortality (Putnam et al., 1991). Only endophyte-free fescue should be offered to horses. However, if pregnant mares are grazed on infested pasture, veterinarians recommend withdrawing mares from the fescue 12 weeks before expected parturition.

Plants with Unknown Toxins

Juglans nigra (Black Walnut). The distribution of black walnut tree growth is contained within the northeastern and central United States. Intoxication from black walnut is usually seen following exposure to black walnut shavings used as stall bedding. However, horses have also been poisoned in pastures containing black walnut trees, especially during pollen shedding in the spring and in the fall when leaflets are shed from the walnut leaves (Knight, 1995).

The clinical symptoms associated with black walnut ingestion begin 8–24 hours after exposure. The horse becomes lethargic and anoretic. The most common symptom is laminitis of varying degrees. The hooves will be tender and warm with an increased digital pulse pressure. If the horse is removed from the source of intoxication early in the disease, the disastrous sequelae of laminitis (rotation of third phalanx) may be avoided. Supportive therapies for laminitis may be needed and include: non-steroidal anti-inflammatory agents, cold water to the hooves, and soft bedding.

The toxic principle is unknown; however, juglone, a compound present in the roots, bark, nuts, and pollen of black walnut trees, is the proposed causative toxin. Although direct application of juglone extract to the skin causes skin irritation, it does not produce the classic laminitis associated with black walnut toxicity. Therefore, it appears that ingestion or inhalation of the toxin is required for clinical symptoms to occur. Furthermore, administration of an aqueous extract from black walnut trees is used as a model for the induction of laminitis (Galey et al., 1991).

Bedding with wood shavings is common in areas where shavings can be obtained economically. Light-colored shavings (oak, pine, fir, and other softwoods) are completely safe for horses. In contrast, black walnut shavings are grayish-black to dark brown. Bedding containing 5%–20% black walnut shavings can be toxic to horses (Ralston and Rich, 1983). Although shavings from some trees other than black walnut are dark, to prevent the possibility of toxicity, all wood shavings containing dark wood should be avoided.

Berteroa incana (Hoary Alyssum). Symptoms similar to black walnut toxicity have been seen in horses following ingestion of hay contaminated with hoary alyssum, a member of the mustard family

(Ellison, 1992; Geor et al., 1992). Hoary alyssum is a white-flowered plant with a grayish-green pubescence. Originally, it was a European weed, but is now found in the United States and Canada. It grows in an area bounded by Nova Scotia, Minnesota, New Jersey and West Virginia (Geor et al., 1992). The plant may be an annual, biannual, or perennial and grows to a height of 1–3 feet. The plant appears early in the year and continues to flower and produce seeds until frost. It is difficult to distinguish bare, hoary alyssum stems from alfalfa stems after they are baled in hay. Although the toxin in hoary alyssum is unknown, the agent appears to remain active for up to 9 months after cutting.

In addition to signs of laminitis, horses exhibit depression, leg edema, joint stiffness, fever, and early parturition. In one study, seven horses showed signs of toxicity 17 hours after eating alfalfa hay contaminated with 10%–15% hoary alyssum (Ellison, 1992).

In another study, horses usually recovered 2–4 days after they were removed from the plant source. In the same study, death occurred in 2 of the 98 field cases and rotation of the third phalanx was observed in 3 cases. Not all horses consuming hoary alyssum-contaminated hay developed clinical symptoms (Geor et. al., 1992). In a study where horses ingested hay contaminated with 30%–70% hoary alyssum, circumstantial evidence linked the plant to the death of a few horses (Becker, 1990).

Because the toxic principle of hoary asylum is unknown, treatment for toxicosis in horses after ingestion is empirical based on the severity of the signs. First of all, the source of the toxin should be removed, and veterinary treatment should include phenylbutazone (1 mg/lb, IV, q 12 h), cool water hydrotherapy of the limbs and icing of the feet, and lactated Ringer's solution as needed.

Panicum coloratum (Kleingrass). Similar conditions to hoary alyssum occur sporadically when horses ingest kleingrass. Kleingrass is a tufted perennial widely planted in Texas. Toxicity from kleingrass is highest in Texas during the hot, humid, wet months of June through October, when plant growth is fastest (Cornick et al., 1988). In Australia, the incidence of intoxication is highest in spring and early summer when growth is rapid and rainfall is high. Clinical symptoms include typical signs of liver disease: weight loss, icterus, anorexia, and hepatic encephalopathy. Clinical pathology demonstrates elevations of serum gamma glutamyltransferase, blood ammonia, total and direct bilirubin, and sulfobromophthalein clearance time.

The toxin responsible for kleingrass poisoning has not been identified. However, the sporadic nature of clinical cases and their occurrence associated within wet, humid conditions suggest that mycotoxins or plant metabolites generated during hot, humid conditions may be responsible. Incidence appears to be related to individual susceptibility, duration of ingestion, amount ingested, and the time of year the horse is exposed (Cornick et al., 1988).

Horses usually recover rapidly from the toxicity once they are removed from the source of the toxin. It is possible for horses to graze the areas again when the pasture dries out and under different growing conditions in later years.

Acer rubrum (Red Maple). Red maple trees (Figure 23) are common throughout eastern North America. Their leaves have three or five lobes and

turn a characteristic brilliant red color in the fall. Red, clustered flowers appear before the leaves in the spring. The fruit is red and has two smooth wings.

The fresh green leaves are apparently non-toxic. However, ingestion of wilted red maple leaves by horses can result in severe blood dyscrasias such as hemolytic anemia, methemoglobinemia, and the formation of Heinz bodies within red blood cells (McConnico and Brownie, 1992). Wilted leaves remain toxic for up to 30 days (George et al., 1982). The bark also appears to be toxic (Tennant et al., 1981).

There are two forms of red maple toxicity. A peracute model results from massive methemoglobinemia, causing marked tissue anoxia and sudden death. A second pattern is primarily the result of the Heinz body formation with intravascular and extravascular hemolysis. Methemoglobinemia may also occur with the hemolytic form, but it does not cause hemolysis.

Horses are often found moribund or dead following peracute intoxication. Clinical signs following the hemolytic form include: weakness, depression, icterus, hemoglobinuria, hemoglobinemia, methemoglobinemia, and anemia. Mucous membranes will be dark and discolored, and urine will be brownish-red.

Reports of clinical recovery from hemolytic disease following red maple toxicity are limited. In a recent paper from the School of Veterinary Medicine at the University of North Carolina (McConnico and Brownie, 1992), two horses were successfully treated for red maple toxicity after showing evidence of methemoglobinemia and hemolysis. The exact toxic agent is unknown; however, the toxin is an oxidant. Antioxidant therapy is the current treatment.

The veterinary clinicians questioned the rationale for antioxidant therapy with methylene blue, the classic treatment for methemoglobinemia. Methylene blue can induce Heinz body hemolytic anemia in horses. Rather, large doses (30 mg/kg–50 mg/kg IV, 125 mg/kg orally) of ascorbic acid were administered due to its reported clinical success as a treatment of acetaminophen toxicity in cats. Ascorbic acid has been administered to adult horses in doses of 50 gr/day with no deleterious effects (Wood et al., 1990).

Within 36–48 hours of ascorbic acid treatment, voided urine was clear and negative for blood on urine dipstick. Both horses recovered from the red maple intoxication, although one horse developed laminitis and was eventually euthanized.

Additional treatment included: blood transfusions, IV fluid therapy, mineral oil by nasogastric intubation, nasal oxygen insufflation, and flunixin meglumine (1.1 mg/kg IV BID). Dexamethasone phosphate (0.5 mg/kg IV) was given to reduce red cell destruction. Because of the unpredictable course of the disease (onset of clinical signs may be delayed 5–7 days after ingestion of red maple leaves), all potentially exposed horses should be treated with mineral oil and/or activated charcoal. There are no reports suggesting charcoal is effective against red maple toxicity, but it is suggested that administration be based on charcoal's value as a universal antidote.

Because red maple leaves are toxic when wilted or recently shed, most poisonings occur between late summer and late fall. Cases of toxicity are also seen following tree damage from storms and high winds. Differential diagnoses include: immune-mediated hemolytic anemia, equine infectious anemia, piroplasmosis, congenital methemoglobinemia, wild onion poisoning, and phenothiazine toxicity. Early diagnosis of the disease and treatment, which includes high doses of ascorbic acid, may result in full recovery of the horse.

Centaurea solstitialis (Yellow Starthistle) and ***Centaurea repens*** (Russian Knapweed). Yellow starthistle (Figure 24) is an aggressive weed from the Mediterranean region and is usually found in uncultivated fields, along roadsides, and in open ranges. It is a member of the sunflower family. It is an annual (lives only one growing season) weed, a prolific seed producer, and grows to a height of 1–2.5 feet. The plant begins growth in early spring and continues throughout summer and fall. The leaves and stems are pointed, with the leaf base extending down the stems to form a wing-like structure. Single bright-yellow flowers about one inch in diameter are located at the ends of the branching

stems. The bracts of the head have rigid spines (0.25–1 inch) radiating outward, from which the name yellow starthistle was derived.

Russian knapweed (Figure 25) is an aggressive perennial and inhibits the growth of competing foliage. It is a noxious weed from Eurasia that is now widely established in the western United States. The weeds grow 1.5–3 feet tall. The stems are openly branched. The lower leaves are deeply lobed and 2–4 inches in length. The upper leaves may be entire or serrate and are narrow at the ends and broad at the base. The cone-shaped flower heads are pink to lavender and are 0.25–0.5 inch in diameter. The flowers are solitary at the branch tips and are present June to September. The seeds are covered with white bristles. The plant spreads its growth primarily through the roots which may be over 8 feet deep, thus resistant to dry conditions.

Horses appear to be the only animal known to suffer intoxication by *Centaurea*. Feeding these plants to sheep, guinea pigs, rats, monkeys, dogs, chicks, or mice did not produce visible signs of disease. Cattle have also grazed the plants and plant-infested pastures without evidence of toxicity.

Yellow starthistle and Russian knapweed cause a similar neurologic condition in horses called Equine Nigropallidal Encephalomalacia (ENE) referred to as "chewing disease," which resembles Parkinson's Disease in humans. The toxic principle is unknown; however, the sesquiterpene lactone repin, found in Russian knapweed, is specifically toxic to chick embryo neurons (Stevens et al., 1990). Russian knapweed is the more toxic plant. Onset of the disease is sudden after prolonged ingestion (>30–60 days) of large quantities of *Centaurea* weeds. Before clinical signs develop, it has been calculated that horses must consume 59%–71% and 86%–200% of their body weight of Russian knapweed and yellow starthistle, respectively (Young et al., 1970).

The disease has been reported in the western United States and the subhumid regions of Argentina and Australia. In California, the disease has a biphasic pattern with a small peak of incidence in June–July and a larger incidence in October–November. This biphasic pattern of incidence has also been reported in Argentina, where the first peak is associated with regrowth of yellow starthistle plants acting as biennials (recurring every two years), and the second peak is due to those functioning as conventional annuals (recurring every year). *Centaurea* plants have a minimal moisture requirement and, therefore, may be the only green plants remaining in non-irrigated pastures during dry seasons.

If adequate good-quality foliage is present in a pasture, horses will generally avoid any knapweeds and yellow starthistle in the field. If a pasture is overgrazed, the horses are forced to consume the toxic plants or starve. However, there are a few cases in which horses developed a preference for yellow starthistle and sought the toxic weed growing in good pastures.

Pathologic lesions are typically found in the right and/or left globus pallidus and substantia nigra; the areas of the brain which control chewing functions. No significant differences in clinical signs have been associated with involvement of any one of these regions. The severity of clinical signs correlates with the size of the lesion, rather than the specific site of involvement or the number of sites. Unilateral lesions in both nuclei have resulted in only one side of the horse being affected. Grey matter of the brain is predominantly affected, with white matter being affected to a lesser degree.

Nerve fibers passing through the damaged area undergo necrosis to the same extent as the cell bodies of neurons.

The pathogenesis of the disease appears to involve release of the neurotransmitter, dopamine, from the substantia nigra. The neurotransmitter is subsequently depleted when the nigrostriatal cells are destroyed. The resulting dopamine deficiency produces hypertonicity of the muscles, innervated by the 5th, 7th, and 12th cranial nerves, which explains some of the clinical signs.

Clinical signs of toxicity in the horse appear suddenly. The horse's behavior may be violent and aggressive initially. However, in a few days the violence abates and the horse's condition remains static for the duration of the disease. Initially, horses exhibit hypertonicity of the muscles on the muzzle and lips, causing the mouth to be held open, teeth exposed, and tongue hanging out. Affected horses appear to want to eat and drink; however, their ability to chew food is greatly reduced. Although their ability to swallow does not seem to be impaired, they are unable to put food in position for swallowing. Frothing at the mouth has been observed, and this sign, along with the inability to chew, may cause ENE to be confused with rabies. Other symptoms include aimless walking with the head held close to the ground.

There is no known treatment once clinical signs are apparent. Prognosis is poor, and the horse will usually die from starvation or dehydration. Therefore, euthanasia should be considered for humane reasons.

Trifolium hybridium (Alsike Clover). Most plants that are hepatotoxic to horses contain pyrrolizidine alkaloids as discussed earlier. However, there are a few plants that cause liver damage by non-pyrrolizidine alkaloids. Horses grazing alsike clover have shown three clinical manifestations of toxicity. One manifestation has been described as acute or nervous, in which the symptoms are primarily neurologic. The behaviors of affected horses alternate between depression and excitability. There is also evidence of hepatic neuropathies characterized by head pressing, incoordination, and aimless walking. Yawning and grinding of teeth are also seen. This syndrome rapidly deteriorates toward paralysis, coma, and death.

The second clinical syndrome has been described as chronic or cachectic. Affected horses often have a decreased appetite, progressive weight loss, weakness, sluggishness, and a dry, coarse hair coat. Affected horses often develop acute photosensitization on the non-pigmented and thinly-haired portions of their bodies. Icterus and other signs of hepatic disease are seen.

The toxin responsible for alsike clover poisoning has not been identified. A recent survey of alsike clover poisoning in Canada demonstrated a definite geographical distribution of the disease in the Peace River district of western Alberta and eastern British Columbia (Nation, 1991). Alsike clover is grown in eastern and north-central North America.

Although the two syndromes described can cause death, alsike clover toxicity is usually not serious, causing only photosensitization in unpigmented areas of skin. However, photosensitization has been reported in horses grazing on wet pastures containing other *Trifolium* spp. as well. Both red and white clover have been implicated, and both are heavily infested with *Cymodothea trifolii,* a fastidious fungus causing black blotch on clover (Ames et al, 1994). Red clover can also become infected by another fungus, *Rhizoctonia leguminicola,* which causes the production of slaframine, also known as the slobber factor, because it causes profuse salivation.

It is unknown if the wet clover causes problems by contact or ingestion. The photodermatitis is especially evident on the parts of the body that contact the wet grass (lower legs, mouth).

Treatment consists of removing the animals from the pastures especially in the early morning when the plants are dew-covered. Animals severely affected need to be kept out of the sun until recovered. They may be turned out on pasture at night.

Keep sensitive animals off alsike pastures in the early morning or during wet weather. Provide other feed if animals are consuming large quantities of the clover and if they are showing clinical signs. Keep the pastures mowed to lessen the effects of the toxicity for sensitive animals. Alsike

clover is safe when dry; therefore, prepared feeds containing alsike is safe for consumption.

Gymnocladus dioica (Kentucky Coffee Tree). The Kentucky coffee tree (Figure 26) is a large leguminous tree noted for its large pods (about 5 inches long) which remain on the tree during winter and fall off in the spring. The animals eat the pods at this time if insufficient quality feed is available. When the seed pods fall, they contain several large seeds (about 0.5 in diameter) in a bright green jelly. Early Kentuckians used the dried plant as a coffee substitute, hence its name, and were not poisoned. However, when the jelly is consumed, human fatalities have occurred (Kingsbury, 1964). The symptoms are those of intense gastrointestinal irritation accompanied with certain nervous manifestations which have been described as "narcotic."

Onset of symptoms is rapid. Activated charcoal (1 lb/150 lb body weight) may reduce absorption of the toxin. Other symptomatic treatments such as IV fluids and analgesics for colic may be indicated.

Zephyranthes atamasco (Atamasco Lily, Rain Lily). This lily is confined to the coastal plain from Mississippi to Virginia. The lilies are often found in extensive clumps and are particularly dangerous in the spring when the plant is easy to uproot because the ground is wet. Other forage may be in short supply increasing the likelihood of toxicity. The bulbs seem to be the most toxic part. The plant has been shown to cause "staggers" in horses. Symptoms appear within 48 hours of ingestion and consist of softened feces (often with bloody mucus), staggering, collapse and death. Activated charcoal (1 lb/150 lb body weight) and mineral oil may reduce absorption of the toxin from the GI tract, and analgesics may be indicated for colic.

SUMMARY

Poisonous plants can grow anywhere: in the woods, where horses are ridden and grazed; on the open range, where western horses have access to wild grasses and weeds; in a bluegrass pasture, where limbs and leaves of toxic trees can fall; or in a backyard with ornamental plants (these clippings may be thrown over the fence into a field of horses). Furthermore, toxic plants can be baled with hay and shipped across country to poison horses far from the plant source.

Poisonous plants are common on most ranges of the western United States and are also found, to a lesser degree, in many fields and wooded areas of the eastern Unites States, but they generally cause problems only under certain conditions. For example, on the plains, larkspur consumption may increase during or following summer rains because animals seek shelter in aspen groves which are concentrated in the same area predominated by larkspur. Poisoning can occur from different plants in different seasons in the same pasture. Problems can be greater during drought condition (locoweeds) or during wet years (larkspurs).

Many poisonous plants have both "windows of opportunity," when they are relatively safe to graze, and "windows of toxicity," when their palatability is acceptable, but their toxin level is high enough to be lethal. Larkspur is again a prime example since it is very unpalatable early in the spring when it contains the highest level of toxins, but becomes relatively palatable near the

time of flowering. However, at flowering the toxin levels, while lower, are still well within the dangerous zone. During late summer, larkspur is less toxic.

Symptoms listed in this chapter for each plant toxin are those most likely to be observed. Not all symptoms will be seen in all toxicities, and signs of poisoning may vary a great deal, depending on the amount of toxin ingested and the time over which the toxin was consumed. Also, individual animals respond differently to some poisons.

Furthermore, the symptoms of some poisonings are often based on descriptions from controlled experiments, and "classic" signs may or may not occur in field situations. Also, the signs may be very general and may fit many different plants. Several different species of plants can contain similar toxins, and different toxins may effect the same tissues and organs. A single species can be poisonous in multiple ways; hence, the signs of poisoning vary depending on the toxin most prevalent at the time of poisoning.

Direct losses from poisonous plants include: death, emaciation, reduced growth, decreased reproductive efficiency, abortion and birth defects. There are no known antidotes for most poisonous plants. Even when a treatment is available, affected animals may be in remote places and cannot be reached until it is too late to apply the treatment For this reason, the best weapon against the devastating effects of poisonous plants is prevention of exposure. The following are a few suggestions to help accomplish this:

- Provide adequate forage. Horses are very selective about what they eat and will eat toxic plants only when there is nothing else available. Spring is a particular problem time because toxic plants tend to "green-up" before the nutritious forages appear. Similarly, if a pasture is overgrazed and the proper forage is cropped to the root, horses may consume plants that would not usually interest them. Normally, weeds are not consumed if other green forages are available. Also, provide adequate salt and water to prevent horses from grazing abnormal vegetation
- Check out new pastures. When horses are grazed in new surroundings (e.g., at a rest stop on a trail ride), be aware of what the horse is grazing. Do not allow the animal free access to unfamiliar vegetation.
- Remove known toxic plants from pastures. Some poisonous plants can be eradicated with herbicides. However, some plants actually become more palatable after being sprayed. Some fields may need to be plowed and reseeded with more desirable forage crops.

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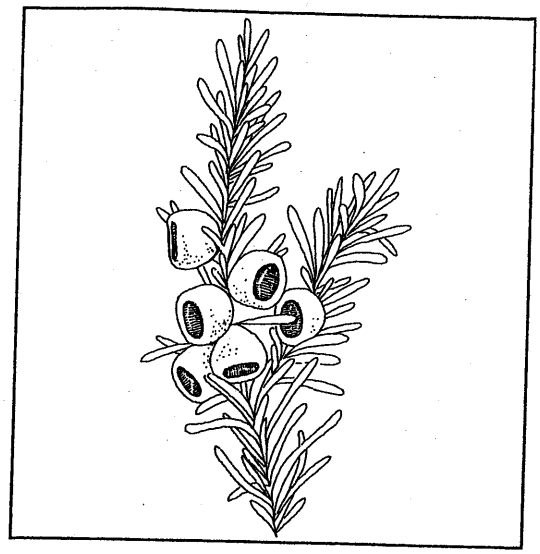

Figure 1. Dark green leaves of yew (*Taxus* spp.) with red fruit that resembles a pitted olive.

Figure 2. The characteristic trumpet-shaped flower of Jimson weed (*Datura stramonium*).

Figure 3. Riddell's groundsel or Riddell's ragwort (*Senecio riddellii*).

Figure 4. Tansy ragwort (*Senecio jacobaea*).

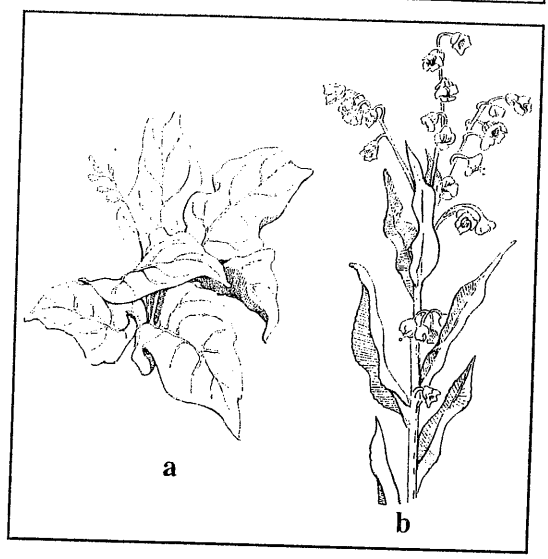

Figure 5. a) Rosette stage and b) flowers of Hound's tongue (*Cynoglossum officinale*).

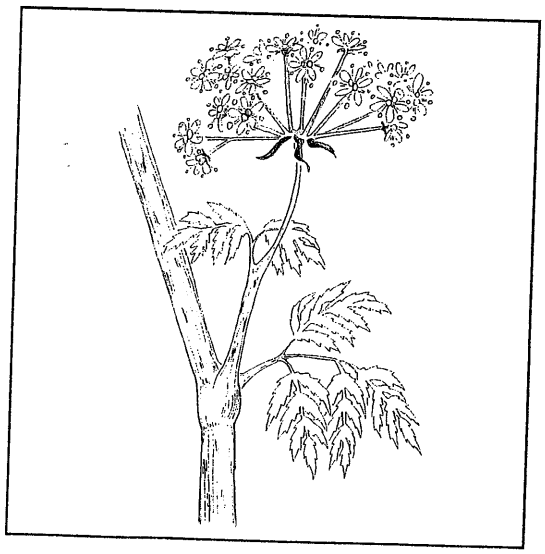

Figure 6. Poison hemlock (*Conium maculatum*).

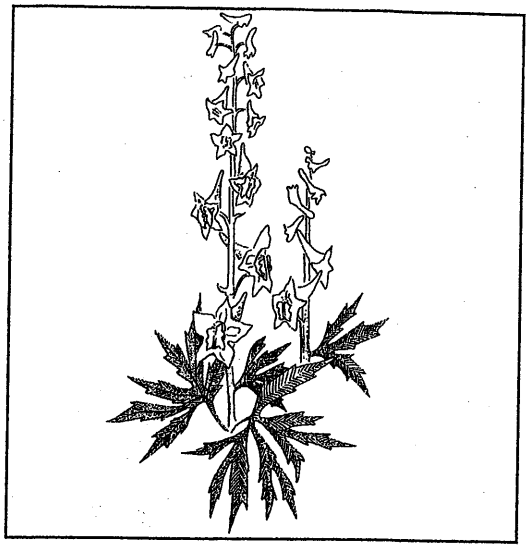

Figure 7. Tall larkspur (*Delphinium barbeyi*).

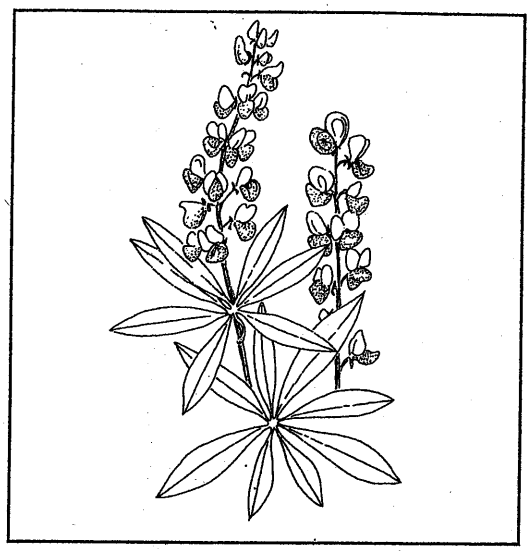

Figure 8. Lupine (*Lupinus* spp.).

Figure 9. Death camas (*Zigadenus* spp.).

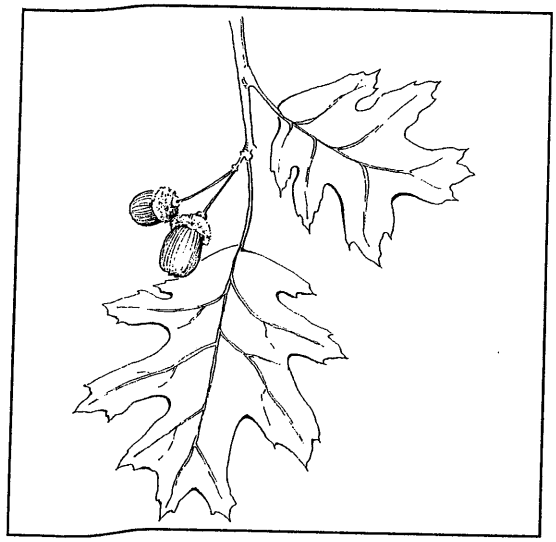

Figure 10. The leaves and acorns of an oak (*Quercus* spp.).

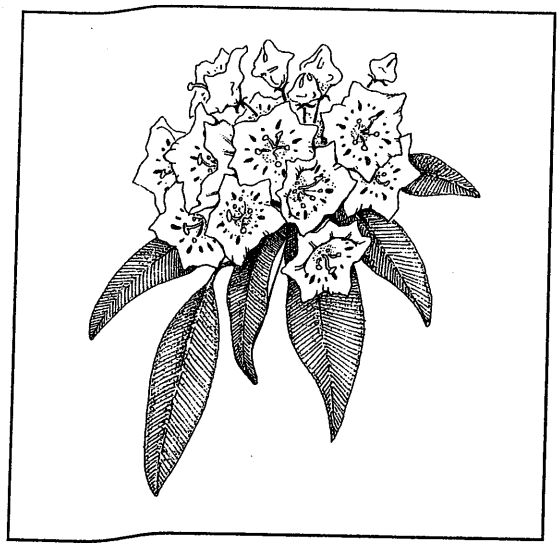

Figure 11. Characteristic white to pink flowers of mountain laurel (*Kalmia latifolia*).

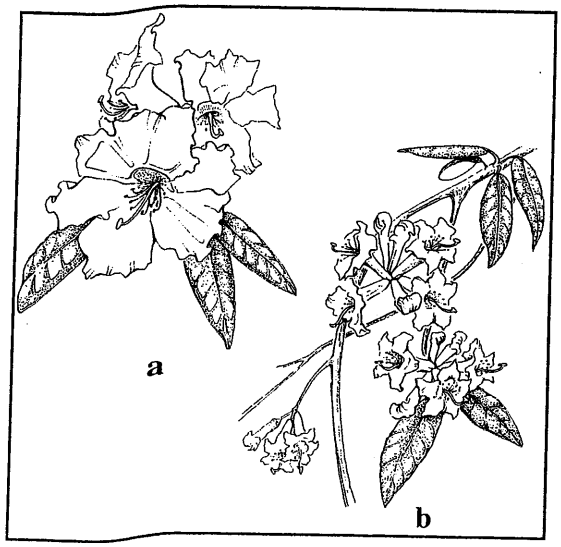

Figure 12. Leaves and flowers of two rhododendrons (*Rhododendron* spp.).

Figure 13. Johnson grass (*Sorghum halepense*).

Figure 14. Flowers of whorled milkweed (*Asclepias* spp.) with horn-like petals.

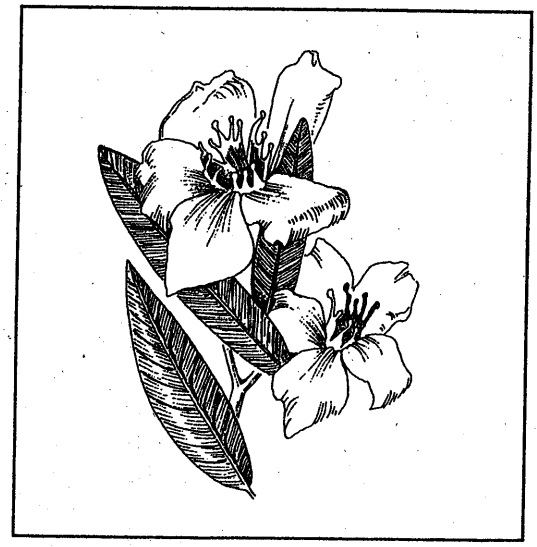

Figure 15. Oleander (*Nearium oleander*).

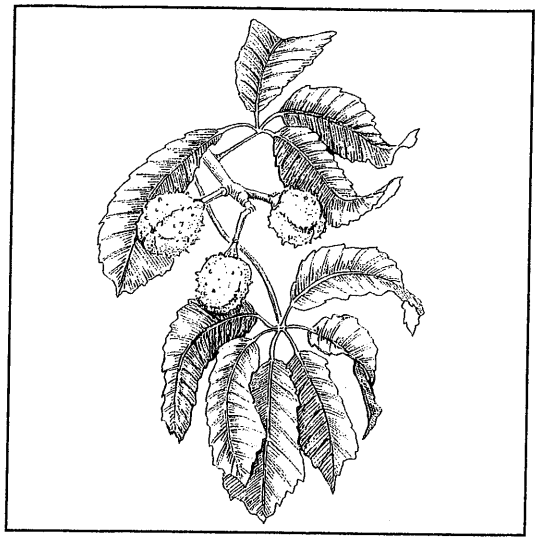

Figure 16. Leaves and nuts of buckeye or horse chestnut (*Aesculus* spp.).

Figure 17. White snakeroot (*Eupatorium rugosum*).

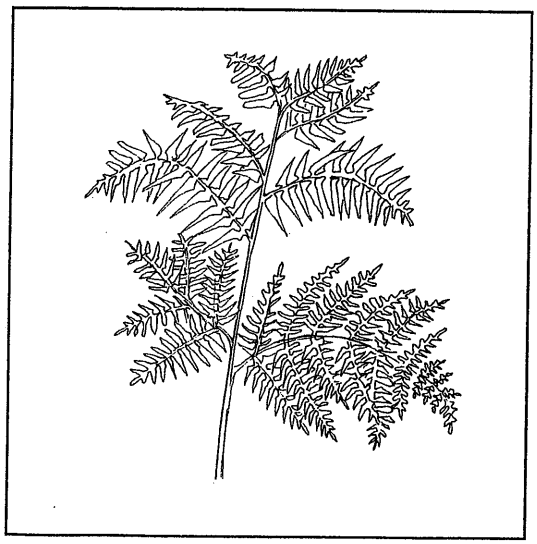

Figure 18. Bracken fern (*Pteridium aquilinum*).

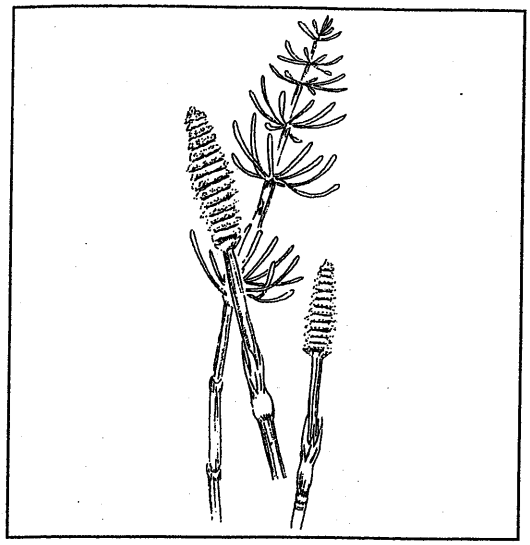

Figure 19. Cone-like head at the end of a field horsetail (*Equisetum arvense*) stem.

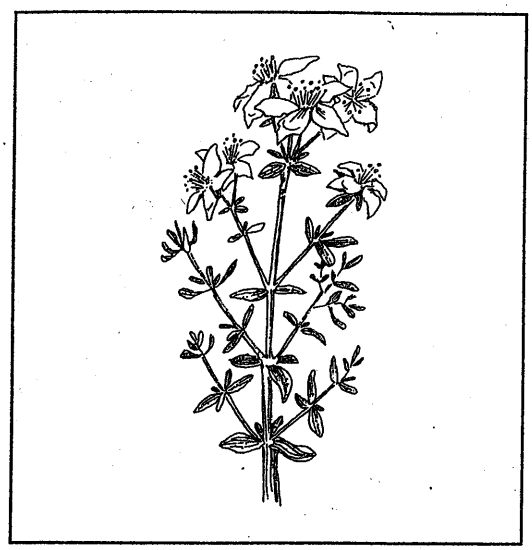

Figure 20. St. Johnswort (*Hypericum perforatum*).

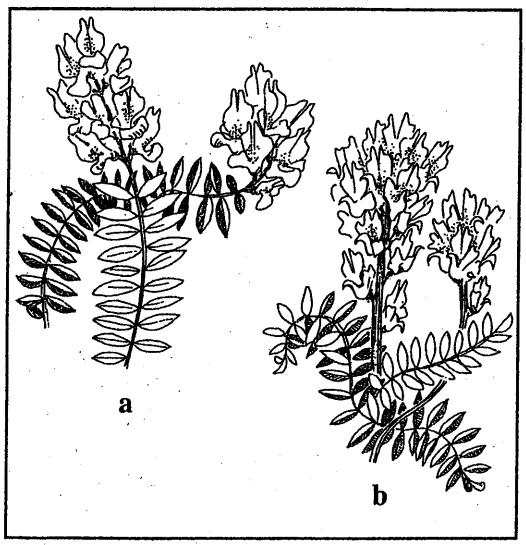

Figure 21. Two examples of locoweed: a) *Astragalus lentiginosus* and b) *Oxytropis sericea.*

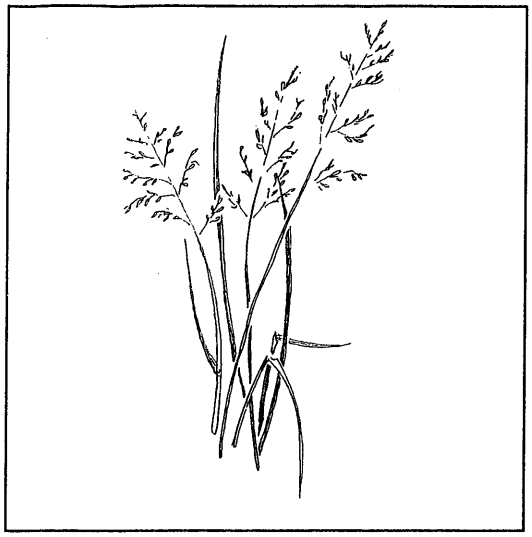

Figure 22. The blades and seed heads of fescue grass (*Festuca arundinacea*).

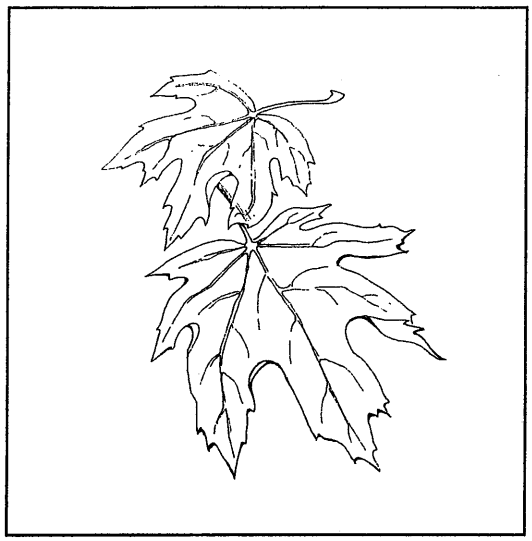

Figure 23. Leaves of red maple (*Acer rubrum*), which is commonly confused with Crimson King Norway maple (*Acer platanoides*), a tree not known to be toxic to animals.

Figure 24. The flowers of yellow starthistle (*Centaurea solstitialis*) surrounded by long, spiny bracts.

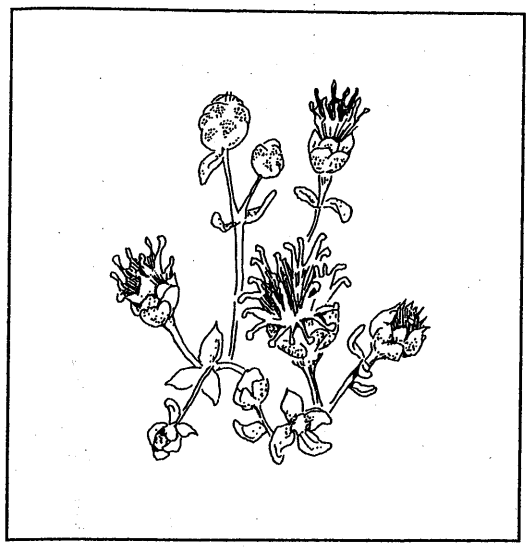

Figure 25. The thistle-like flower of Russian knapweed (*Centaurea repens*).

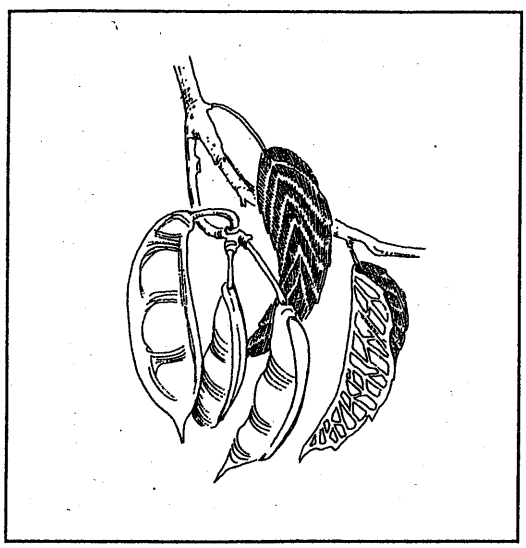

Figure 26. The leaves and pods of the Kentucky coffee tree (*Gymnocladus dioica*).

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