

# Plants Poisonous to Horses

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Plant poisoning of horses continues to be a problem in the western United States because of the diversity of plants and conditions that expose horses to toxic plants in their pastures and hay. In some years the incidence of plant poisoning increases because adverse weather conditions deplete good quality forages which forces horses to eat plants that they would not normally. Snow storms may force horses to browse on any plants that protrude above the snow cover. Drought conditions may similarly cause horses to eat anything they can find when they are hungry. Overgrazing and poor pasture or range management will also put horses at risk as common plants such as leafy spurge (*Euphorbia esula*), Russian knapweed (*Acroptilon repens*), fringed sage (*Artemisia frigida*), field bindweed (*Convolvulus arvensis*) and hounds tongue (*Cynoglossum officinale*) will invade the pasture. Furthermore these same plants and others may be incorporated in hay and can cause poisoning during the winter when plant poisoning would be least expected.

## **Water Hemlock (*Cicuta douglasii*)**

Water hemlock, a native plant, contains the unsaturated alcohol cicutoxin that is found in all parts of the plant, but especially in the roots and the base of the stem. All animals, including man, are highly susceptible to the potent cicutoxin. It is one of the most poisonous plant toxins known, causing severe stimulation and finally paralysis of the nervous system. Poisoning occurs with relatively small quantities of the plant, with as little as 30-60 gm. of the root being sufficient to kill an adult horse.

Signs appear within half an hour of ingesting water hemlock and are characterized by initial salivation followed by muscle tremors that precede violent spasmodic convulsions. Animals have been known to severely lacerate their tongue in episodes of teeth grinding and violent chewing motions. Death from respiratory paralysis and myocardial damage usually occurs within a few hours of the onset of convulsions.

## **Poison Hemlock (*Conium maculatum*)**

Poison or spotted hemlock, a noxious weed, contains several alkaloids, the most toxic of which is coniine. These alkaloids are present in all parts of the plant but especially in the root and the seeds. The alkaloids are toxic to all animals. Since the concentration of alkaloids in poison hemlock varies with the growth stage of the plant, it is difficult to predict with any accuracy the quantity of green plant that must be ingested to cause death. Early studies indicated that somewhere between 2 and 4% of the animal's body weight in green plant have to be consumed before clinical signs develop. A progression of signs starting with muscle tremors, in-coordination, nervousness, dilation of the pupils, rapid heart rate, coma and death due to respiratory paralysis can be expected.

### **Milkweed Poisoning (*Asclepias spp.*)**

Milkweeds are widely distributed throughout the world and much of North America, and have caused poisoning in most domestic animals including horses. Greatest losses occur when other forages are scarce and animals are forced into eating whatever plants are available, or when milkweeds are incorporated in hay. Milkweeds grow in open areas along roadsides, waterways and disturbed areas, preferring sandy soils of the plains and foothills. Over grazing will enhance the spread of milkweeds.

Milkweed poisoning results from the cardiotoxic effects of the cardenolides that act like digitalis glycosides. Milkweeds are most toxic during rapid growth, and retain their toxicity even when dried in hay. Toxicity varies with the species and growing conditions, however all milkweeds should be considered potentially poisonous, especially the narrow-leaved species such as the whorled milkweed (*Asclepias subverticillata*). The highest concentration of glycosides occurs in the latex, with the lowest concentrations in the roots. Fatal poisoning of an adult horse may occur with the ingestion of as little as 2 lbs. of green whorled milkweed plant material.

Signs of poisoning usually begin within 8-10 hours of milkweeds being eaten. The severity of symptoms being dependent on the quantity of plant consumed. In acute milkweed poisoning the animal may be found dead without any prior symptoms. A variety of cardiac arrhythmias may be present prior to death. Poisoned horses, once recumbent, exhibit periods of tetany and chewing movements. There are no specific post mortem signs in animals poisoned by milkweeds but congestion of the lungs, stomach, and intestines, with hemorrhages present on the surfaces of the lungs, kidneys, and heart are commonly found.

### **Death Camas (*Zigadenus spp.*)**

There are approximately 15 species of death camas that are native to North America, and can be found in moist mountain valleys, to drier sandy hills and plains. Death camas appears in early spring, often growing amongst wild onion.

Several poisonous alkaloids are found in all parts of the plant and especially in the bulb. Sheep are most frequently poisoned by death camas, but cattle, horses, and pigs may be affected. Poisoning is most likely to occur in early spring when few other plants are available and the succulent shoots are especially enticing.

Poisoning may occur in horses after they have eaten about 4 kg of the plants. Salivation, colic, muscular weakness, and staggering gait are reported in horses, with death occurring after several days.

### **Larkspur (*Delphinium spp.*)**

Larkspurs cause more fatal poisoning of cattle in the Western United States than any other naturally occurring plant species. Horses are much less susceptible to larkspur poisoning than are cattle and are unlikely to be fatally poisoned. There are at least 80 species of *Delphinium* in North America, most of which grow in the western states. It is wise to assume that all larkspurs are potentially poisonous, including those that are

cultivated as ornamentals. Larkspurs are often grouped for descriptive convenience into tall and low varieties according to their growth habit.

Tall larkspurs (*D. barbeyi*, and *D. occidentale*, *D. glaucescens*) grow in deep, moist, and highly organic soils at high altitudes, often reaching 6 feet in height. The plants emerge as the snows recede and once established form large and very long-lived, stands. The tall larkspurs generally grow in montane forests, especially where snow drifts form perennially. Low larkspurs (*D. nuttallianum*., *D. andersonii*, *D. virescens*.) grow at lower elevations in drier range land, seldom growing more than 2-3 feet tall, and die-off in early summer as the soil dries out. Foothills larkspur (*D. geyeri*) is intermediate in its growth habit, attaining a height of 3-4 feet when in flower, and is common along the eastern foothills of the Rocky Mountains.

Young rapidly growing larkspur plants are most toxic, the highest concentration of alkaloids being in the leaves. Larkspurs contain many diterpenoid alkaloids that act principally at the neuromuscular junction causing a curare-like blockade with resulting muscle weakness and paralysis. The alkaloids may vary in quantity depending on the species and stage of growth. Even within a small area, certain stands of larkspur appear to be more toxic than others, and are well recognized by ranchers as "hot spots" from which they can frequently anticipate cattle losses. The alkaloid content of tall larkspurs also appears to be consistently higher in plants growing in full sun as opposed to those growing in the shade. Studies with tall larkspur (*D. barbeyi*) have shown it to be the most toxic of the larkspurs.

### **Oak (*Quercus* species)**

Some 60 species of oak grow in North America and all are poisonous. Oaks at any stage of growth are poisonous, but are particularly toxic when the leaf and flower buds are just opening in the spring. As the leaves mature they become less toxic. Ripe acorns are less toxic than when green. Cattle sheep, goats, horses and pigs are susceptible to poisoning. The tannins found in the leaves, bark, and acorns of oaks produce poisoning through their effect on the intestinal tract and kidney. Tannins are potent, precipitators of cellular protein.

Signs of oak poisoning will vary according to the quantity of oak consumed. Initially animals stop eating, become depressed, and develop intestinal stasis. The feces are hard and dark but a black tarry diarrhea often occurs later in the course of poisoning. Abdominal pain is evidenced by teeth grinding and a hunched back. Severe liver and kidney damage is detectable by marked elevations in serum liver enzymes and urea nitrogen. Jaundice, red colored urine and dehydration are further signs encountered in oak poisoning. Animals may live for 5-7 days after the onset of clinical signs.

### **Cyanogenic Plants**

There are at least a 2000 species in 7 plant families that are known to contain cyanogenic glycosides with the potential for producing hydrogen cyanide (HCN, prussic acid) poisoning in animals. Most livestock cyanide poisoning has been associated with **sorghums** including Johnson grass (*Sorghum halapense*) and other forage sorghums.

Arrow grass (*Triglochin spp.*). Chokecherries (*Prunus spp.*), service berry (*Amelanchier alnifolia*), and occasionally crab apple leaves (*Prunus spp.*) have caused cyanide intoxication in ruminants. Nitrate fertilization of plants will increase the cyanogenic glycoside content of plants. Application of herbicides may also increase the cyanogenic glycoside content of plants. Generally all parts of the plant are toxic, the young rapidly growing portion of the plant and the seeds being most toxic. Drying of plants decreases their cyanogenic potential especially over time.

Ruminants are more frequently poisoned by cyanogenic plants than other animals because the rumen bacteria rapidly convert the cyanogenic glycosides to free cyanide (HCN). Horses therefore are unlikely to develop cyanide poisoning from plant sources because their digestive system does not readily convert the glycosides to cyanide.

Sudden death is often the presenting sign of acute cyanide poisoning. Affected animals rarely survive more than 1-2 hours after consuming toxic quantities of cyanogenic plants, and usually die within a few minutes of developing clinical signs of poisoning. If observed early, poisoned animals show rapid labored breathing, frothing at the mouth, dilated pupils, ataxia, muscle tremors, and convulsions. The mucous membranes are bright red in color due to the hemoglobin being saturated with oxygen

## **Plants Affecting the Digestive System**

### **Buttercups**

Native plants including buttercups (*Ranunculus spp*) and related species such as marsh marigold (*Caltha palustris*), clematis (*Clematis spp.*), anemone, and baneberry (*Actaea arguta*) contain irritating substances that cause mouth irritation, excessive salivation and diarrhea if eaten in quantity. The buttercup family contains an oily glycoside, ranunculin, that when converted by plant enzymes to protoanemonin becomes a strong irritant. It primarily causes irritation to the mucous membranes of the mouth and digestive system, with resulting excessive salivation, colic and diarrhea.

### **Nightshades**

Members of the nightshade family (*Solanaceae*) are common weeds and have the potential for poisoning animals that eat them. The more common members of this group include *Solanum spp.* (Nightshades), *Datura stramonium* (Jimson weed), *Hyoscyamus niger* (Black henbane), and *Physalis* (Ground cherries or Chinese lanterns)

A variety of steroidal (tropane) glycoalkaloids are found in these plants, especially in the green parts of the plant and the unripe fruits. The alkaloids have similar effects on the autonomic nervous system blocking normal nerve conduction. This results in decreased salivation and intestinal motility, dilated pupils, and tachycardia. The alkaloids also have a direct irritant effect on the digestive system causing colic, constipation or hemorrhagic diarrhea.

### **Leafy spurge (*Euphorbia esula*)**

Introduced from Eurasia, leafy spurge has become a very troublesome weed of North America where it has infested millions of acres. It is listed as a noxious weed in most states because of its invasive nature and its ability to displace normal forages.

The specific toxin in leafy spurge has not been defined. Most animals, except sheep and goats, will not eat leafy spurge unless forced to do so. When eaten, spurges cause excessive salivation in some animals due to the irritant effects of the plant sap. Diarrhea frequently develops. Recovery is rapid once animals are provided more nutritious food.

Other plants that may induce diarrhea include horse tail (*Equisetum* spp.), wild iris, yarrow (*Achillea* spp.), and sneezeweeds (*Helenium* and *Dugaldia* spp.).

### **Locoweeds (*Astragalus* and *Oxytropis* spp.)**

Locoweeds or vetches, are probably the most important indigenous toxic plants in the Rocky Mountain area. Several hundred species exist in North America, many of them being concentrated in the western States. Not all species of *Astragalus* and *Oxytropis* are poisonous. Horses, cattle, sheep, goats, elk, cats and possibly other animals that eat locoweeds are susceptible to their toxic effects. Signs of poisoning do not become evident until animals have consumed significant quantities of locoweeds over many weeks and the toxic threshold is reached. Animals must consume locoweed for at least 2 weeks and in large quantities before clinical signs develop. Locoweeds are palatable and once animals have learned to eat them they frequently will continue to do so even when normal forages are present. Although horses, cattle, and sheep were thought to develop an addiction to locoweeds, it is more likely habituation as there is no dependence on the plants and they do not actively seek them out, as would be the case if they were addicted. It is entirely possible that the sole reason animals eat locoweeds is that they find them palatable at certain stages!

The alkaloid swainsonine is the principle toxin in locoweed. It is present in all parts of the plant and persists in the dried plant. An endophytic fungus growing mutualistically in the locoweed plants produces the swainsonine, and not the plant itself. The amount of swainsonine in locoweeds varies depending on the species, stage of growth and the growing conditions. The succulent preseed-stage plants appear to be the most palatable with animals preferring to eat the immature seedpods. The palatability of locoweed does not have any relationship to the quantity of swainsonine in the plant. Young animals are most severely affected, as maturing cells are more vulnerable to the effects of the toxin. Compounding this is the fact that swainsonine is also secreted in the milk; so that suckling animals will acquire additional alkaloid to that acquired from locoweed they may eat.

Horses appear to manifest the nervous signs of locoweed poisoning more commonly than do cattle or sheep. Depression, in-coordination, staggering gait, and unpredictable behavior especially if the animal is stressed or excited are common signs of locoism. Some animals become totally unpredictable in their response to being handled. Poor vision, in-coordination, sudden changes in behavior such as rearing and falling over backwards, make horses dangerous and unsafe to ride. If removed from the source of the locoweeds and fed a nutritious diet, animals will show improvement and appear relatively normal after several

months. However, horses with neurological signs from chronic locoism usually only partially recover making them a liability to human safety. There is no proven effective treatment for locoweed poisoning.

### **Sage Poisoning (*Artemisia spp.*)**

Numerous species of sagebrush are native to the western United States and are useful forage plants for wild and domestic ruminants. Horses also eat sage without problem provided they are not forced to eat it exclusively when other forages are scarce. Under adverse conditions horses will readily eat sagebrush and will become intoxicated. A syndrome called "sage sickness" has been reported in horses that eat sand sage (*Artemisia filifolia*). Fringed sage (*Artemisia frigida*) and possibly other species of sage also causes similar signs of poisoning in horses.

Sage poisoned horses exhibit abnormal behavior characterized by ataxia especially of the front legs, and a tendency to fall down or act abnormally to stimuli that would not normally elicit such a response. Normally docile horses will become excitable and unpredictable. The smell of sage is often very noticeable on the breath.

The clinical signs closely resemble those of horses that have been poisoned by locoweeds (*Oxytropis* and *Astragalus spp.*) However, unlike "locoed" horses that do not recover once clinical signs are evident, sage poisoned horses generally recover once they are removed from the sage and are fed a normal balanced diet.

### **Russian Knapweed (*Acroptilon repens*) and Yellow Star Thistle (*Centaurea solstitialis*)**

Horses that eat Russian knapweed and yellow star thistle, two noxious weeds, can develop an irreversible and usually fatal brain disease. Localized necrosis of the brain (nigropallidalencephalomalacia) results in the inability of the horse to prehend and chew its food due to the hypertonicity of the facial muscles. Swallowing is however unaffected. The disease appears to be unique to the horse, and is reported to occur in those areas of the United States, Australia and Argentina where the plants grow abundantly.

Russian knapweed was originally introduced from Russia and has become widely disseminated in the Rocky Mountain States where it is now listed as a noxious weed. It is a persistent, invasive perennial with woody stems up to 3 feet in height, and branching underground stems. The name knapweed comes from the dense covering of white hairs or knap that give the leaves and stems a gray-green appearance.

Yellow star thistle is abundant in northern California and has spread to Colorado. It is an annual herbaceous weed, branching from the base up to 3 feet in height tall. The branches are winged and ascending. Leaves are covered with cottony hair, the basal leaves being deeply lobed; the stem leaves are linear and entire. The disc flowers are yellow and fertile. The bracts are tipped with characteristic stiff yellow spines up to an inch in length.

Horses must consume either the green or dried plant for many weeks before a threshold is reached and clinical signs appear. Experimentally horses were fed 59 - 71% of their body weight in green Russian knapweed before poisoning developed. Slightly greater quantities of yellow star thistle are required to induce poisoning. The plants however are quite palatable to horses and they may develop a preference for the plants.

Affected horses develop hypertonicity of the facial muscles that results in the mouth being held open so that the tongue hangs out. Continuous tongue movements and attempts to eat result in frothing of the saliva. Some horses may wander about with their lips brushing through the grass that can be mistaken for normal grazing. Prehension and mastication is severely impaired, but swallowing is not affected. Weight loss is severe, and eventually the horse will starve to death if does not die from inhalation pneumonia. There is no specific treatment, and the irreversible localized necrosis in the brain warrants a very poor prognosis.

### **Bracken Fern (*Pteridium aquilinum*)**

Bracken fern, an indigenous plant of North America and many parts of the world, occasionally cause poisoning in horses. The plant contains thiaminase, an enzyme that depletes thiamin, a vitamin essential for normal energy metabolism. Affected horses refuse to eat and consequently lose weight. Depression, muscle tremors, uncoordinated gait, especially of the hind legs and paralysis are typical of bracken fern poisoning.

Diagnosis of bracken fern poisoning should be based on evidence that horses have eaten the fern, the clinical signs, and the animal's response to thiamin therapy.

### **Hounds Tongue (*Cynoglossum officinale*)**

Hounds tongue is rapidly spreading noxious weed in the inter mountain area. It is an introduced, biennial weed of cultivated and waste areas. It grows to 3ft. in height, with hairy, alternate tongued-shaped leaves, the basal leaves being up to 20 inches long. The flowers are small, reddish purple in color. The seeds 4 brown nutlets covered with "velcro-like" hooks that readily attach to clothing and animal's hair. The plant is generally not palatable to horses when green but when dried in hay it is readily eaten. Pyrrolizidine alkaloids present in the plant cause irreversible liver disease.

### **Senecio (Groundsel) (*Senecio* species)**

A variety of *Senecio* species are native to North America and can become a problem if animals eat the plants. *Senecio* species are difficult to recognize unless the yellow flowers with their characteristic single layer of green bracts holding the flower are identifiable. All parts of the plant are toxic and care should be taken to ensure *Senecios* are not incorporated in hay, as they remain toxic even when dried. Like hounds tongue, the *Senecios* contain highly toxic pyrrolizidine alkaloids that cause severe and irreversible liver damage. Weight loss, followed by depression, neurological abnormalities secondary to hepatic fibrosis, photosensitization, and eventually death are typical of poisoning from *Senecio* species.

Other plants that may cause photosensitization include Alsike Clover (*Trifolium species*), Alfalfa (*Medicago sativa*), and Tansy mustard (*Descurainia spp.*)

## Selenium Poisoning

Selenium poisoning of livestock has been recognized for many years, occurring when animals consume plants that have accumulated excessive amounts of selenium from seleniferous soils. Geographically, selenium rich soils exist in areas of low rainfall because there is minimal leaching of selenium from the soil. Some plants require selenium for normal growth and as obligatory accumulators, are capable of accumulating ten times the amount of selenium that is present in the soil. Some of these plants may contain up to 10,000 ppm of selenium. Other plants known as secondary accumulators will bind selenium if it is present in the soil but do not require selenium for growth. Plant uptake of selenium is variable and depends on the chemical form of selenium, the species and stage of plant growth, and the climatic conditions. In drought conditions plants take up more selenium.

Plants containing in excess of 5 ppm (5mg/kg) are capable of producing chronic poisoning of livestock. Obligate accumulator plants with 25 ppm or more of selenium may cause acute poisoning. High selenium content of animal water sources may compound a forage selenium toxicity problem since total selenium intake by an animal determines toxicity. Crop plants and pasture grasses in seleniferous soils will accumulate small quantities of selenium that will contribute to the total quantity of selenium consumed by livestock. Recognition of selenium accumulating plants or indicator plants provides a means of identifying seleniferous soils, and the potential for selenium poisoning in livestock kept in these areas.

### Obligate Selenium Accumulator (Indicator) Plants

<i>Astragalus</i> (24 species)	Milk Vetches
<i>Haplopappus spp</i>	Golden weeds
<i>Xylorhiza glabriuscula</i>	Woody aster
<i>Stanleya pinnata</i>	Princes plume

### Secondary Selenium Accumulators

<i>Aster spp</i>	Asters
<i>Atriplex spp</i>	Saltbrush
<i>Castilleja spp</i>	Paintbrush
<i>Penstemon spp</i>	Beard tongue
<i>Grindelia spp</i>	Gum weed

Selenium poisoning occurs most commonly in cattle, sheep, and horses as a result of grazing plants that have accumulated high levels of selenium. Selenium rich plants are seldom eaten because of their distasteful strong odor, but livestock will consume them if other forages are scarce. Selenium poisoning in animals is variable, being dependent on the amount and rate of absorption of selenium from the intestines, the individual animal's susceptibility, the type of selenium present in the plant, and the interaction of selenium with other elements such as sulfur or arsenic that are present in the diet.



## **Toxic Effects of Selenium**

Selenium has numerous complex effects on cellular function, many of which are poorly understood. Selenium inhibits cellular enzyme reactions, especially those involving sulfate and sulfur containing amino acids such as methionine, cystine. Selenium substitutes for sulfur in the amino acids and consequently this causes defective formation of keratin, the essential protein that forms the structure of hooves and hair. The abnormal keratin containing the selenium is structurally weaker than normal keratin containing sulfur. Clinically this is seen initially as circular ridges in the hoof wall and loss of hair from the mane and tail. The hair of the mane and tail breaks off at the point where toxic levels of selenium were incorporated in the formation of keratin. The hoof walls tend to crack along the ridges where keratin formation is defective, and subsequent infection of the sensitive laminae of the hoof results in severe lameness.

### **Chronic Selenium Poisoning**

"Alkali disease" commonly affects horses, cattle, pigs, sheep and poultry that consume forages or cereal crops grown in selenium rich soil. Poisoning is likely to occur after the selenium containing plants have been consumed for several months. Feeds containing more than 5 ppm and less than 50 ppm are most likely to cause disease. Affected animals lose their hair, especially that of the mane and tail. Lameness is due to characteristic abnormal hoof wall growth in all feet, that results in rapid uneven growth and circular ridges and cracks of the hoof wall. Some animals may slough the hoof wall entirely. Cattle will show similar defective horn growth.

### **Diagnosis**

A diagnosis of selenium poisoning is best confirmed by submitting samples of hay or forages for analysis. Water selenium levels should be less than 0.05 ppm (mg/l) Selenium levels greater than 5 ppm should be considered potentially toxic. Blood levels of 1-4 ppm are typical of chronic selenium poisoning, whereas serum levels up to 25 ppm have been reported in acute poisoning. Liver and kidney levels greater than 4 ppm are indicative of selenium toxicity. Generally, the more chronic the selenium toxicity becomes, the lower the levels of selenium in the tissues. Hair and hoof samples however retain high concentrations, and are useful in the diagnosis of chronic selenium poisoning. Hoof wall containing 8-20 ppm is indicative of chronic selenosis. Hair samples containing in excess of 10 ppm selenium are indicative of selenium poisoning.

### **Treatment**

Successful treatment of selenium poisoning is dependent on early recognition of signs and the removal of livestock from the source of the excess selenium. Careful attention should be given to regular trimming of the cracked and over-grown hooves to avoid permanent lameness. Feeding a high protein diet with adequate sulfur and copper levels will counteract the effect of selenium on sulfur containing amino acids. This can be accomplished by feeding a concentrate ration and/or a mineral supplement containing sulfur and copper. Alternatively feeding good alfalfa will provide plenty of sulfur containing amino acids which will counteract the presence of selenium in the animal's diet.

## Cultivated Trees and Plants Potentially Poisonous to Animals

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The following commonly available trees, shrubs and plants are often sold through plant nurseries, and pose a potential hazard to animals if they are planted in or around animal enclosures. If these plants are found to be desirable for landscaping purposes, it is important to position the plants well away from where animals can reach them. Furthermore it is essential to always provide a balanced nutritious diet to animals at all times so that they are not driven through hunger to eating unusual plant material. It is also important to remember that the careless disposal of tree and plant prunnings into an animal enclosure is a frequent cause of poisoning.

### Trees

Black locust	<i>Robinia pseudoacacia</i>
Black walnut	<i>Juglans nigra</i>
Chinese tallow tree	<i>Sapium sebiferum</i> (mildly toxic)
Chokecherry	<i>Prunus spp.</i>
Golden chain tree	<i>Laburnum anagyroides</i>
Horse chestnut, buckeye	<i>Aesculus spp</i>
Kentucky coffee tree	<i>Gymnocladus dioica</i>
Oak	<i>Quercus spp.</i>
Persimmon	<i>Diospyros virginiana</i>
Red Maple	<i>Acer rubrum</i>
Russian Olive	<i>Elaeagnus angustifolia</i>

### Shrubs

Angels Trumpet	<i>Brugmansia spp.</i>
Black laurel	<i>Leucothoe davisiae</i>
Boxwood	<i>Buxus sempervirens</i>
Burning bush	<i>Euonymus atropurpurens</i>
Day or night Jasmine	<i>Cestrum diurnum, C. nocturnum</i>
Hydrangea	<i>Hydrangea spp.</i>
Japanese Pieris	<i>Pieris japonica</i>
Lantana	<i>Lantana camara</i>
Laurel	<i>Kalmia spp.</i>
Mesquite	<i>Prosopis veluntina</i>
Oleander	<i>Nerium oleander</i>
Privet	<i>Ligustrum spp.</i>
Rhododendron (azalea)	<i>Rhododendron spp.</i>
Yellow oleander	<i>Thevetia spp.</i>
Yew	<i>Taxus spp.</i>

**Vines**

Carolina jessamine  
Virginia creeper

*Gelsemium sempervirens*  
*Parthenocissus quinquefolia*

**Perennial Plants**

Castor bean  
Comfrey  
Crown vetch  
Fox glove  
Hairy vetch  
Larkspur  
Lupines  
Monkshood

*Ricinus communis*  
*Symphytum* spp.  
*Coronilla* spp.  
*Digitalis purpurea*  
*Vicia villosa*  
*Delphinium* spp.  
*Lupinus* spp.  
*Aconitum* spp.

**References**

**A Guide to Plant Poisoning of Animals in North America**

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**Web Site: [www.vth.colostate.edu/poisonous\\_plants](http://www.vth.colostate.edu/poisonous_plants)**